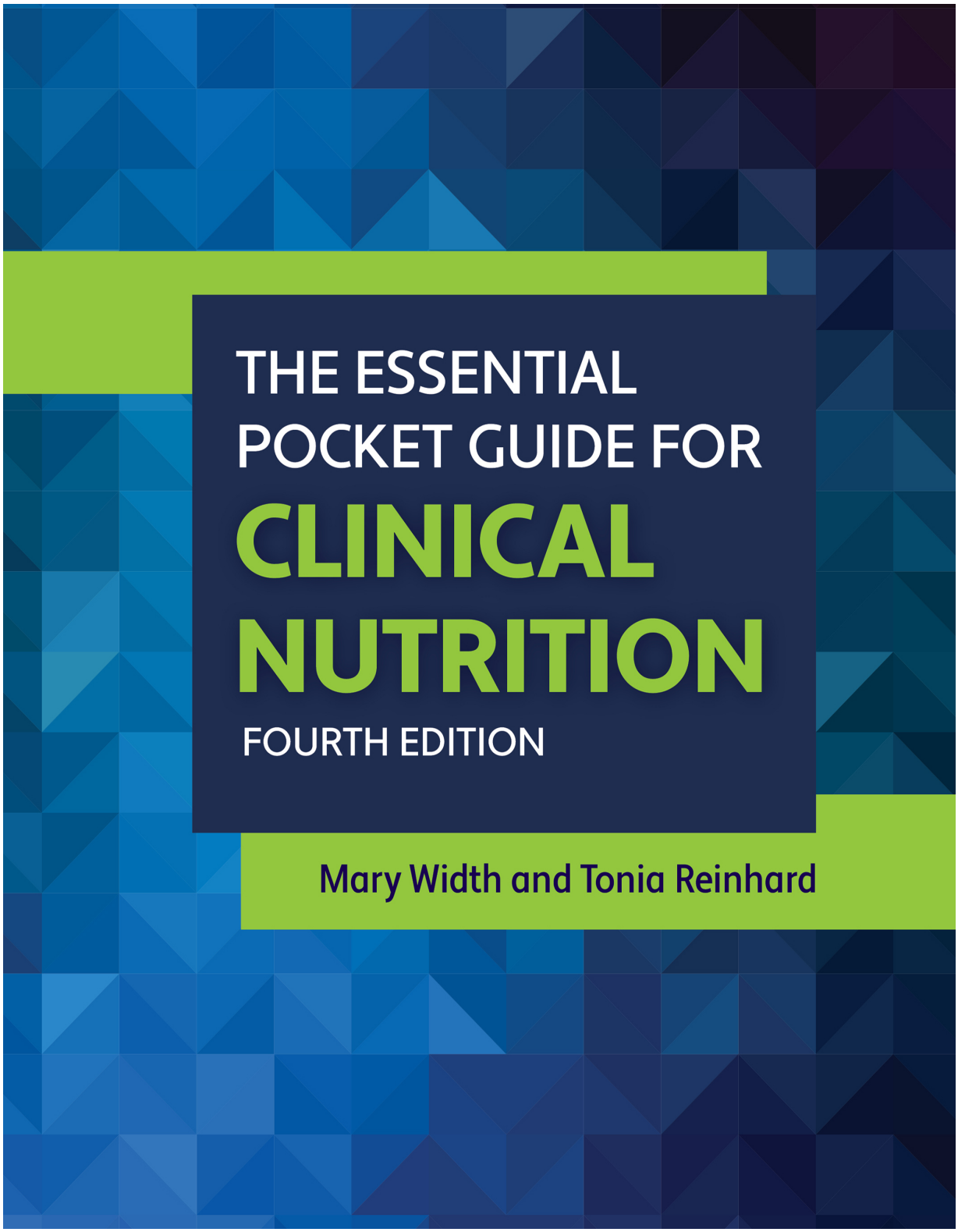
THE ESSENTIAL POCKET GUIDE FOR CLINICAL NUTRITION

FOURTH EDITION

Mary Width and Tonia Reinhard


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01934-6

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Cover Image (Title Page, Part Opener, Chapter Opener): © Attitude/Shutterstock.
Printing and Binding: Lakeside Book Company – Crawfordsville

Library of Congress Cataloging-in-Publication Data
<insert CIP data>

6048

Printed in the United States of America
29 28 27 26 25 10 9 8 7 6 5 4 3 2 1

“This book is dedicated to my bonus daughters, Dr. Alicia Width and Dr. Megan Width. I am so proud of the strong, brilliant, and accomplished women you have become.”

—Mary Width

“This book is dedicated to the Reinhard Crew: John, Faye and Jeff, Brendan and Karen, and the next generation: Claire, Zoe, Gea, Anthony, Mary, Maeve, and Gus.”

—Tonia Reinhard



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Preface

When we first thought about publishing this book, we wanted a resource for students enrolled in our dietetics program to take to their clinical rotations. Like most dietitians, we had created our own portable clinical books full of information we needed at our fingertips and easily jammed into a lab coat pocket. Once the book was published, we were delighted when our students would come back from rotations and tell us that their preceptors had ordered the book for their own use.

After several editions and much input from practicing dietitians, as well as students, we believe that the *Essential Pocket Guide for Clinical Nutrition* is an invaluable resource that healthcare professionals from a variety of disciplines can tailor to their own practice in applying evidence-based nutrition information. In this fourth edition, we worked diligently to adhere to our original goal of producing a concise pocket-sized reference, which has become one of the book's main assets based on feedback. We were also excited to hear from other health professionals using the

book because we have worked to expand the user base beyond dietitians.



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Organization of This Text

Part I contains chapters covering nutritional assessment, life-stage assessment, and nutrition support. Part II includes chapters on the major nutritionally relevant diseases. The book includes appendices on food and drug interactions, laboratory assessment, herbs and dietary supplements, dietary reference intakes, and food sources of vitamins and minerals.



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Features of This Text

This edition includes the following features:

- The **compact size** fits into any lab coat pocket.
- The **colored tabs** enable quick, easy searching of contents.
- Use of the **Nutrition Assessment, Diagnosis, Intervention, and Monitoring and Evaluation** (ADIME) format for quick synopses of all major nutritionally relevant diseases, including sample nutrition diagnosis statements in **Problem-Etiology-Signs and Symptoms** (PES) format.
- Each chapter contains **quick reference tables** that highlight important information.

This book could not have been developed without the help of our expert contributors, and we thank them for their hard work and valuable contributions to the book. In addition, we thank all of the countless colleagues and preceptors who offered constant encouragement and excellent suggestions during the writing process.

—Mary Width, MS, RD, FAND

—Tonia Reinhard, MS, RD, FAND



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PART 1

Nutrition Assessment and Support



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CHAPTER 1

Nutrition Assessment

The Nutrition Care Process

The Nutrition Care Process (NCP), developed by the Academy of Nutrition and Dietetics (the Academy), is a systematic approach to providing high-quality nutrition care, which provides dietetic professionals with a framework for critical thinking, problem-solving, and decision-making to address nutrition-related problems.[1] The NCP was designed to improve the consistency and quality of nutrition care and the predictability of nutrition care outcomes. Dietetic professionals, including registered dietitians (RD) and registered dietitian nutritionists (RDN), use the NCP in diverse venues to provide nutrition services and document the process.

The NCP consists of four steps: Nutrition Assessment and Reassessment, Nutrition Diagnosis, Nutrition Intervention, and Nutrition Monitoring and Evaluation.[1] The NCP culminates in documentation of the process, often referred to by the acronym "ADIME." A feature in this book is a highlight box called "ADIME At-A-Glance," which includes common aspects of each NCP step for a specific condition or disease state.

Nutrition assessment (NA) of the patient, client, or resident is the first step in effective nutrition care to identify and diagnose nutrition risks and plan appropriate interventions. This step also includes reassessment when new data can be compared to previous information that may lead to new or revised nutrition diagnoses.[1] Data collected during NA and reassessment can be organized into nine assessment domains. The following are examples of data collected within each domain but are not all-inclusive:

1. Anthropometric measurements: height, weight, body frame, body mass index (BMI), weight change, growth

pattern indices, and body compartment estimates.

2. Biochemical data, medical tests, and procedures.
3. Physical exam findings: Findings from a physical exam, interview, or the health record.
4. Client history: current and past information related to personal, health, family, and social history.
5. Food/Nutrition-related history: food and nutrient intake and administration, medication and complementary/alternative medicine use, knowledge/beliefs/attitudes, behavior, food availability, physical activity and function.
6. Assessment, monitoring, and evaluation tools: Tools used during assessment, reassessment, and monitoring and evaluation.
7. Etiology category: Categories to communicate the type of nutrition diagnosis etiology.
8. Comparative standards: What indicator data are compared against.
9. Progress evaluation: Evaluation of progress toward nutrition-related goals and resolution of nutrition diagnoses.

Nutrition diagnosis is the second step in the NCP. After assessment, the patient's nutrition-related problems and needs are determined, which form the nutrition diagnosis. The nutrition diagnosis, in turn, is expressed and documented in a specific format: problem, etiology, and signs and symptoms (PES). **Boxes 1.1** and **1.2** illustrate and provide an example for the PES format.

Box 1.1 Nutrition Diagnosis: Problem/Etiology/Signs

The problem (P) describes alterations in patient nutritional status:

- A diagnostic label (qualifier) is an adjective that describes the physiologic response (e.g., altered, impaired, risk of).

The etiology (E) refers to cause(s) or contributors(s) to the problem:

- It is linked to the problem by the term “related to” (RT).

The signs/symptoms (S) are clusters of subjective and objective factors that provide evidence that a problem exists:

- They also quantify the problem and describe severity.
- Linked to (E) by the term “as evidenced by” (AEB).

Box 1.2 Writing the Nutrition Diagnosis (ND) Statement

Example for the ND Statement Format (**P**)roblem/(**E**)tiology/(**S**)igns/symptoms:
Excessive energy intake (**P**) related to frequent consumption of large portions of high-fat meals (**E**), as evidenced by:

1. Daily caloric intake exceeding estimated energy intake by 500 kcal (**S**)
2. 12 lb weight gain during past 18 months (**S**)

The nutrition diagnostic terms are classified according to the following four domains and subclasses:

1. Intake: Actual problems related to intake of energy, nutrients, fluids, and/or bioactive constituents through oral diet or nutrition support.
2. Clinical: Nutrition problems that related to medical or physical conditions.
3. Behavioral-Environmental: Nutrition problems that relate to knowledge, attitudes, beliefs, physical environment, access to food, or food safety.
4. Nutrition Situation: Nutrition related findings providing additional context relevant to a person's nutrition care.

Nutrition intervention is the third step in the NCP. The interventions are planned to positively change nutrition-related behaviors, environmental conditions, or aspects of health status for the patient/client. The interventions should

address the problems identified in the nutrition assessment that have been formulated into the nutrition diagnosis. The intervention typically targets the etiology of the nutrition diagnosis/problem identified in the PES statement. The nutrition intervention is less frequently directed at the signs and symptoms to minimize their impact.[1] The intervention step includes two interrelated components: planning and implementation. Planning involves prioritization of the nutrition diagnoses as to each problem's severity or importance, and the use of evidence-based interventions and practice guidelines to establish patient-focused expected outcomes for each nutrition diagnosis. Implementation of the nutrition intervention is the action phase that includes carrying out and communicating the plan of care to the patient/client, caregivers, and other members of the healthcare team; continuing data collection; and revising the nutrition intervention strategy, based on the patient/client response. The nutrition intervention terminology is organized into eight domains (2 planning and 6 implementation):

1. Goals planning
2. Prescription planning
3. Food and/or nutrient delivery
4. Nutrition education
5. Nutrition counseling
6. Coordination of nutrition care by a nutrition professional
7. Population based nutrition action
8. Nutrition intervention encounter context

ADIME 1.1 ADIME At-A-Glance: Nutrition Diagnoses for Malnutrition

Severe: Chronic Illness or Condition

- Chronic disease/condition-related malnutrition (may have severe protein-calorie malnutrition) RT increased energy expenditure because of catabolic illness AEB estimated energy intake ≤75% over 2 months, severe pectoralis and deltoid muscle loss (shoulder square in appearance), malignancies

Severe: Acute Illness or Injury

- Acute disease/injury-related malnutrition (may have severe protein-calorie malnutrition) RT alteration in gastrointestinal tract structure AEB weight loss of >5% in 1 month, severe quadricep wasting (severe depression of muscle on thigh, prominent femur bone), major gastrointestinal surgery

Severe: Social or Environmental (Starvation-related)

- Starvation-related malnutrition (may have severe protein-calorie malnutrition) RT disordered eating pattern AEB intake of ≤50% of estimated energy needs for ≥1 month, severe deltoid muscle loss (prominent acromion protrusion), anorexia nervosa

The final step in the NCP is monitoring and evaluation. This is a critical component of the NCP because it identifies important measures of patient/client outcomes relevant to the nutrition diagnosis and intervention and describes how best to measure and evaluate these outcomes. During this step, the patient's status is re-examined by gathering new data (monitoring) and assessing to see how it compares with previous status, expected outcomes, and established standards (evaluation). This fourth step helps to determine whether the patient/client is meeting the nutrition intervention goals or desired outcomes. The domains and

terminologies used in monitoring/evaluation are the same as those used in nutrition assessment and reassessment, except for client history (there is no client history domain in this step because there are no nutrition care outcomes associated with client history).

Anthropometric Assessment

There are several anthropometric measurements that are useful in the clinical setting. Measurements of body weight, height, and composition can be used by the clinician when evaluating nutritional status.

Estimating Height

An estimation of height is necessary for patients who are confined to bed or a wheelchair, have curvature of the spine or contractures, or are otherwise unable to stand for an actual height measurement.[2]

Demi-Span

- This measurement is particularly useful in the clinical setting because it requires no special equipment. It is also helpful to use with patients who have lower limb dysfunction.
- Using the left arm, if possible, measure the distance from the tip of the middle finger to the middle of the sternal notch.
- Make sure the patient's arm is horizontal and in line with the shoulders.
- Height (in cm) is calculated using the following formulas:

Females: Height in cm = (1.35 × demi-span in cm) + 60.1

Males: Height in cm = (1.40 × demi-span in cm) + 57.8

Knee Height

- Knee height is measured using a sliding broad-blade caliper (available commercially at

http://www.weighandmeasure.com).

- Patient should be in the supine position, and the left leg is preferable for measurement.
- With both the knee and ankle at 90-degree angles, place one blade of the caliper under the heel of the foot and the other on the anterior surface of the thigh.
- The shaft of the caliper is held parallel to the long axis of the lower leg, and pressure is applied to compress the tissue.
- Height (in cm) is calculated using the following formulas:

Females: Height in cm = 84.88 – (0.24 × age in years) + (1.83 × knee height in cm)

Males: Height in cm = 64.19 – (0.04 × age in years) + (2.02 × knee height in cm)

Evaluating Body Weight

Ideal Body Weight

Body weight relative to height is a frequently used measurement to determine risk of morbidity and mortality.[3] Although there is limited published research on their validity, many ideal body weight (IBW) equations and height/weight tables are used to assess an individual's nutrition status by comparing actual body weight with IBW standards in various settings. One common method frequently used in the United States is the Hamwi method, shown below:

Females: IBW = 100 lb for 5 ft + 5 lb for each inch >60 in

Males: IBW = 106 lb for 5 ft + 6 lb for each inch >60 in

For light- or small-framed individuals, the IBW, using this method, can be reduced by 10%, and for heavy- or large-

framed individuals, the IBW can be increased by 10%. See the following sections for determining frame size.

Frame Size Adjustment

To adjust for differences in body build (muscularity, bone thickness, and body proportions), it is necessary to determine an individual's frame size when calculating IBW.[4] Frame size can be estimated by two methods. Measuring wrist circumference is easy and straightforward, although wrist size can be influenced by body weight. Measuring elbow breadth is more complex but tends to provide a more accurate estimate of frame size. Both methods use the measurements in relation to the patient's height.

Wrist Circumference

- Measure the wrist circumference just distal to the styloid process at the wrist crease of the left hand, in inches, using a tape measure. Compare the measurement with the values in **Table 1.1**.

TABLE 1.1 Estimating Frame Size Using Wrist Circumference

Female Wrist Measurements			
	Height <5′2″	Height 5′2″–5′5″	Height >5′5″
Small	<5.5″	<6.0″	<6.25″
Medium	5.5″–5.75″	6.0″–6.25″	6.26″–6.5″
Large	>5.75″	>6.25″	>6.5″
Male Wrist Measurements			
			Height >5′5″
Small			5.5″–6.6″
Medium			6.5″–7.5″
Large			>7.5″

Data from U.S. National Library of Medicine (NLM). Calculating body frame size. Reviewed July 25, 2022. Accessed July 24, 2023. http://www.nlm.nih.gov/medlineplus/ency/imagepages/17182.htm.

Description

Elbow Breadth

- Subject should stand, if possible, and extend the arm forward so that it is horizontal and parallel to the ground.
- Turn palm so that it is facing up and bend elbow so that the forearm is at a 90-degree angle to the ground.
- Measure the distance between the two prominent bones on either side of the elbow (the epicondyles of the humerus). This measurement can be taken with a ruler or tape measure, but using commercially available calipers is preferable.
- Compare the measurement with the values in **Table 1.2**.

TABLE 1.2 Estimating Frame Size Using Elbow Breadth

Female Elbow Measurements

Medium Frame

If elbow breadth is less than those in the table for a specific height, subject is small framed, and if elbow breadth is greater, subject is large framed.

Height	Elbow Breadth
4′10″–4′11″	2¼″–2½″
5′0″–5′3″	2¼″–2½″
5′4″–5′7″	2⅜″–2⅝″
5′8″–5′11″	2⅜″–2⅝″
6′0″–6′4″	2½″–2¾″

Male Elbow Measurements

Medium Frame

If elbow breadth is less than those in the table for a specific height, subject is small framed, and if elbow breadth is greater, subject is large framed.

Height	Elbow Breadth
5′2″–5′3″	2½″–2⅞″
5′4″–5′7″	2⅝″–2⅞″
5′8″–5′11″	2¾″–3″
6′0″–6′3″	2¾″–3⅛″
6′4″–6′7″	2⅞″–3¼″

Data from Frisancho AR, Flegel PN. Elbow breadth as a measure of frame size for U.S. males and females. *Am J Clin Nutr*. 1983;37(2);311. doi: 10.1093/ajcn/37.2.311.

Amputation Adjustment

For patients with amputations, estimation of IBW should be adjusted with the following equation using the factors in **Table 1.3**.

$$\frac{100 - \%\ \text{amputation}}{100} \times \text{IBW for original height}$$

TABLE 1.3 Amputation Adjustments for Estimating Ideal Body Weight

Percentage Body Weight Contributed by Body Part	
Hand	0.7%
Forearm and hand (below elbow)	2.3%
Entire arm	5.0%
Foot	1.5%
Lower leg and foot (below knee)	5.9%
Above knee	10.1%
Entire leg	16.0%

Data from Osterkamp LK. Current perspective on assessment of human body proportions of relevance to amputees. *J Am Diet Assoc.* 1995;95(2):215-218.

Example: A male patient with a height of 6´0˝ and a right below the knee amputation.

$$\text{IBW} = \frac{100 - 5.9\%}{100} \times 81 \text{ kg (IBW for original height)}$$

$$\text{IBW} = 0.941 \times 81 \text{ kg}$$

$$\text{IBW} = 76.2 \text{ kg or round to } 76 \text{ kg}$$

Spinal Cord Injury Adjustment

For patients with spinal cord injuries, estimation of IBW should be adjusted as follows:

- Paraplegia: subtract 10%–15% from IBW
- Tetraplegia/Quadriplegia: subtract 15%–20% from IBW

Interpretation of Body Weight Data

Percentage of Ideal Body Weight

$$\% \text{ IBW} = \frac{\text{current body weight (CBW)}}{\text{IBW}} \times 100$$

Percentage of Usual Body Weight (UBW)

$$\% \text{ usual body weight (UBW)} = \frac{\text{CBW}}{\text{UBW}} \times 100$$

Table 1.4 shows how to evaluate % IBW data.

TABLE 1.4 Interpreting % IBW

% IBW	Nutritional Risk
>120	Obesity
110–120	Overweight
90–109	Adequate

% IBW	Nutritional Risk
80–89	Underweight
70–79	Moderately underweight
<70	Severely underweight

Percentage of Weight Change

This calculation is useful in assessing variations from the patient's usual weight, especially in the elderly population where unintentional weight loss is associated with increased morbidity and mortality.[5,6] Once the percentage of weight change has been calculated, use **Table 1.5** to assess the significance of any weight changes.

$$\%\ \text{weight change} = \frac{\text{UBW} - \text{CBW}}{\text{UBW}} \times 100$$

TABLE 1.5 Interpreting Unintentional Weight Changes

Time Frame	Significant Weight Loss	Severe Weight Loss
1 week	1%–2%	>2%
1 month	5%	>5%
3 months	7.5%	>7.5%
6 months	10%	>10%

Data from (i) Academy of Nutrition and Dietetics. Nutrition Care Manual: Unintended weight loss. Accessed July 24, 2023. https://www.nutritioncaremanual.org, (ii) Gupta R, Evans AT. Approach to the patient with unintentional weight loss. Updated June 10, 2021. Accessed July

Time Frame	Significant Weight Loss	Severe Weight Loss

24, 2023. https://www.uptodate.com, and (iii) *State Operations Manual: Appendix PP-Guidance to surveyors for long-term care facilities*. U.S. Department of Health and Human Services: Centers for Medicare and Medicaid Services. Published August 25, 2020. Revision 211, February 3, 2023. Accessed July 24, 2023. https://www.hhs.gov/guidance/document/guidance-surveyors-long-term-care-facilities.

Assessment of Overweight and Obesity

Body Mass Index

BMI, or Quetelet's index, is a calculation based on height and weight, regardless of gender, that is considered an indicator of body fatness and can be used to assess the severity of obesity. However, BMI is not a direct measure of body fat and does have limitations. Factors such as age, sex, ethnicity, distribution of body fat, and muscle mass can influence the relationship between BMI and body fat. In addition, the presence of edema should be considered when interpreting BMI results. For these reasons, BMI should not be used as a diagnostic tool but instead can be used as a screening tool for overweight or obesity in conjunction with other valid measures of risk such as measurements of visceral fat, body composition, waist circumference, and genetic or metabolic factors.[7] See **Table 1.6** for weight classifications based on BMI.

$$\text{BMI} = \frac{\text{weight (kg)}}{\text{height (m}^2\text{)}} \quad \text{or} \quad \text{BMI} = \frac{\text{weight (lb)}}{\text{height (in}^2\text{)}} \times 703$$

TABLE 1.6 Classification of Overweight and Obesity by BMI, Waist Circumference,

and Associated Disease Risk

BMI (kg/m²)	Weight Status	Disease Risk[a] Relative to Normal Weight and Waist Circumference	
		Men ≤40″ Women ≤35″	Men >40″ Women >35″
<18.5	Underweight	—	—
18.5 to <25	Healthy range	—	—
25.0 to <30	Overweight	Increased	High
30.0 to <35	Obesity class 1	High	Very high
35.0 to <40	Obesity class 2	Very high	Very high
≥40.0	Obesity class 3	Extremely high	Extremely high

[a]Disease risk for type 2 diabetes, hypertension, and cardiovascular disease.

Data from (i) Assessing Your Weight and Health Risk. National Heart, Lung, and Blood Institute. Accessed July 24, 2023. https://www.nhlbi.nih.gov/health, (ii) *Defining Adult Overweight and Obesity*. Centers for Disease Control and Prevention. Reviewed June 3, 2022. Accessed July 24, 2023. https://www.cdc.gov/obesity/basics/adult-defining.html, and (iii) Ross R, Neeland IJ, Yamashita S, et al. Waist circumference as a vital sign in clinical practice: a consensus statement from the IAS and ICCR working group on visceral obesity. *Nat Rev Endocrinol*. 2020;16(3):177-189. doi: 10.1038/s41574-019-0310-7.

Description

Waist Circumference and Waist-to-Hip Ratio

The presence of excess fat in the abdomen, out of proportion with total body fat, is an independent predictor of risk factors and morbidity.[8] Two methods for measuring abdominal fat are waist circumference (WC) and the waist-to-hip ratio (WHR). Both methods have been used to show increased risk for diabetes, coronary artery disease, and hypertension for individuals with excess abdominal fat. In addition, combining WC and/or WHR with BMI can better identify obesity-related risk versus using each measurement on its own.[9] Regardless of which method the clinician uses, measuring abdominal fat can help identify risk level for several chronic diseases (see **Tables 1.6** and **1.7**).

TABLE 1.7 Waist-to-Hip Ratio

Male	Female	Health Risk
≤0.95	≤0.80	Low risk
0.96–1.0	0.81–0.85	Moderate risk
≥1.0	≥ 0.85	High risk

Data from (i) Waist Circumference and Waist-Hip Ratio. World Health Organization. Geneva, December 2008, and (ii) Yusuf S, Hawken S, Ounpuu S, et al. Obesity and the risk of myocardial infarction in 27,000 participants from 52 Countries: A case-control study. *Lancet.* 2005;366:1640-1650.

Estimating Nutrient Requirements

Energy Requirements—Noncritical Care

Many prediction equations and formulas are available for estimating resting metabolic rate (RMR). The Mifflin–St. Jeor (MSJE) and Harris–Benedict (HBE) equations are two widely used formulas for estimating resting energy expenditure in the noncritically ill population. According to the Academy's Evidence Analysis Library (EAL), the MSJE performed better than the HBE when predicting RMR in both nonobese and obese, noncritically ill populations.[10] Once RMR has been calculated, the total energy expenditure (TEE) would need to be estimated using an activity factor, and for some patients, a stress factor as well. Stress factors are used for hospitalized patients in a hypermetabolic state owing to disease, infection, or trauma. The clinician's judgment should be used to determine the appropriate activity and/or stress factors to estimate TEE for each individual patient, and TEE should be reviewed regularly as the clinical situation changes. See **Table 1.8** for activity and stress factors (note: factors can vary by facility and clinician).

TABLE 1.8 Activity and Stress Factors for Determining Total Energy Expenditure

Condition	Factor
Activity Factors	
Confined to bed	1.2
Ambulatory	1.3
Stress Factors	
Burns	

Condition	Factor
≤20% BSA	1.5
20%–40% BSA	1.8
>40% BSA (use max. of 60% TBSA)	1.8–2.0
Infection	
Mild	1.2
Moderate	1.4
Severe	1.8
Starvation	0.85
Surgery	
Minor	1.1
Major	1.2
Trauma	
Skeletal	1.2
Blunt	1.35
Closed head injury	1.4

TBSA, total body surface area.

Mifflin–St. Jeor Equation (MSJE)

$$\text{Females: REE} = 10W + 6.25H - 5A - 161$$

$$\text{Males: REE} = 10W + 6.25H - 5A + 5$$

where, REE, *resting energy expenditure*; W, *actual weight in kilograms*; H, *height in centimeters*; A, *age in years.*

Harris–Benedict Equation (HBE)

Females: BEE = 655.1 + 9.6W + 1.9H – 4.7A

Males: BEE = 66.5 + 13.8W + 5.0H – 6.8A

where, W, *weight in kilograms (use of actual vs. ideal vs. adjusted body weight is determined by the clinician*);* H, *height in centimeters;* A, *age in years.*

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*The EAL does report that the HBE was most accurate when used with the actual body weight.[11]

Kilocalories per Kilogram

A fast, easy, and gender-inclusive method for estimating energy needs is using kilocalories per kilogram of body weight, with the reference weight as actual or IBW, based on the clinician's judgment (**Table 1.9**).

TABLE 1.9 Energy Requirements Based on Kilocalories per Kilogram of Body Weight in Non-Critical and Critical Care

Condition	Energy Requirement (kcal/kg)
Normal	25–30
Stress	
Mild	30–35
Moderate to severe	35–45
Critical Illness	
Critically ill (BMI <30)	25–30 (using dry body weight or UBW)
Obese, critically ill (BMI 30–50)	11–14 (actual body weight)
Obese, critically ill (BMI >50)	22–25 (ideal body weight)

UBW; usual body weight.
Data from (i) Academy of Nutrition and Dietetics. Nutrition Care Manual. Accessed July 24, 2023. https://www.nutritioncaremanual.org. and (ii) McClave, SA, Taylor BE, Martindale, RG, et al. Guidelines for the provision and assessment of nutrition

Condition	Energy Requirement (kcal/kg)

support therapy in the adult critically ill patient. JPEN *J Parenter Enteral Nutr.* 2016;40(2):159-211. https://doi.org/10.1177/0148607115621863.

Energy Requirements—Critical Care

The Academy and the American Society for Parenteral and Enteral Nutrition (ASPEN) recommend that indirect calorimetry (IC) be used to determine energy requirements in critically ill patients, and in the absence of IC, that published predictive or weight-based equations be used.[12,13] The Academy's EAL recommends that if IC is not available, the Penn State University equations have the best prediction accuracy.[12] For *nonobese*, ventilator-dependent, critically ill patients, and for *obese*, ventilator-dependent, critically ill patients <60 years of age, the EAL recommends using the Penn State equation [PSU(2003b)]. For *obese*, ventilator-dependent, critically ill patients >60 years of age, the Modified Penn State equation [PSU(2010)] has better accuracy. See Table 1.9 for kilocalories per kilogram in critical illness.

Penn State Equation [PSU(2003b)]

(Use with nonobese and with obese <60 years of age)

$$RMR = Mifflin\ (0.96) + V_E\ (31) + T_{max}\ (167) - 6{,}212$$

Modified Penn State Equation [PSU(2010)]

(Use with obese >60 years of age)

$$RMR = Mifflin\ (0.71) + V_E\ (64) + T_{max}\ (85) - 3{,}085$$

where, V_E, *expired minute ventilation*; T_{max}, *maximum body temperature in previous 24 hours in degrees Celsius.*

Determining energy requirements for non–ventilator-dependent, critically ill patients is dependent on protocol at each facility and clinician judgment. One method in use is the MSJE modified by a factor of 1.25, although accuracy rates for this equation are only around 50%.[12]

$$RMR = MSJE \times 1.25$$

Recommendations by ASPEN and the Society for Critical Care Medicine (SCCM) for kcal/kg energy requirements for critically ill patients can be found in **Table 1.9**.

Protein Requirements

Table 1.10 contains protein recommendations for patients with several general conditions. Disease-specific protein requirements can be found in respective chapters.

TABLE 1.10 Daily Protein Requirements for Hospitalized Patients

Condition	Protein Requirement (g/kg)
Normal–maintenance	0.8–1.0
Metabolic stress	
Mild	1.2–1.5
Moderate to severe	1.5–2.0
Obese, critically ill	2.0 (using IBW, for BMI 30–40); up to 2.5 (using IBW, for BMI >40)
Pressure injury	1.2–1.5 (for adults with a pressure injury who are malnourished or at risk of malnutrition).
Protein repletion	1.2–2.0
Severe trauma, burns	1.5–2.0

Data from (i) Academy of Nutrition and Dietetics. Nutrition Care Manual. Accessed July 24, 2023. https://www.nutritioncaremanual.org, (ii) McClave, SA, Taylor BE, Martindale, RG, et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient. JPEN *J Parenter Enteral Nutr.* 2016;40(2):159-211. https://doi.org/10.1177/0148607115621863, (iii) American Society for Parenteral and Enteral Nutrition. *Feeding the Patient with Obesity in the Critical Care Setting: A*

Condition	Protein Requirement (g/kg)

Case-Based Approach. 2021. Accessed July 24, 2023. https://www.nutritioncare.org/uploadedFiles/Documents/Malnutrition/MAW_2021/MAW-Critically-Ill-Obese-Patient-Factsheet.pdf, and (iv) European Pressure Ulcer Advisory Panel, National Pressure Injury Advisory Panel and Pan Pacific Pressure Injury Alliance. Prevention and Treatment of Pressure Ulcers/Injuries: Clinical Practice Guideline. The International Guideline 2019. https://internationalguideline.com/2019.

Fluid Requirements

Methods for estimating fluid requirements are typically based on kilocalorie intake (energy method), body weight, fluid balance, or body surface area (BSA).[14] The energy method uses 1 mL/kcal for adults and 1.5 mL/kg for infants. The fluid balance method, which requires the measurement of urine output, is urine output plus 500 mL/day. The BSA method uses 1,500 mL/m^2/day × BSA (see the Pediatric chapter (Chapter 3) in this book for information on calculating BSA). **Table 1.11** shows additional methods for estimating fluid requirements based on body weight. Fluid requirements will also depend on the medical condition of the patient, with additional or restricted fluids required in certain situations. The individual disease chapters may list fluid requirements for specific conditions.

TABLE 1.11 Estimating Fluid Requirements Based on Body Weight

Method 1 (Adults)	
Body Weight (actual)	**Fluid Requirement**
Average adult: 18–54 years	30–35 mL/kg
Older: 55–65 years	30 mL/kg
Elderly: >65 years	25 mL/kg
Method 2 (Commonly Used in Pediatric Populations)	

Method 1 (Adults)	
Body Weight (actual)	**Fluid Requirement**
Body Weight (actual)	**Fluid Requirement**
1–10 kg	100 mL/kg
11–20 kg	1,000 mL + 50 mL/kg each kg >10 kg
>20 kg	1,500 mL + 20 mL/kg each kg >20 kg

Data from Academy of Nutrition and Dietetics. Nutrition Care Manual: Methods for Estimating Fluid Requirements. Accessed July 24, 2023. https://www.nutritioncaremanual.org.

Biochemical Data

The purpose of collecting laboratory data for nutrition assessment is to determine status inside the body. Blood and urine samples can be used to directly measure a nutrient or metabolite that is affected by the nutrient. Each test is associated with a distinctive sensitivity and specificity. Sensitivity indicates the degree to which the assay for a particular constituent is accurate in determining the amount of that constituent in a sample. Specificity refers to how specific the test is in reflecting a particular function or diagnosis, for example, how specific blood urea nitrogen is for assessing renal function.

Basic concepts in the interpretation of laboratory data include the following: No single test is diagnostic on its own; repeated draws are more valid; there can be diurnal variation for some tests; and some constituents can be affected by other superimposed conditions, diseases, and medications.

Protein Status

Protein status, both visceral protein and somatic protein compartments, were historically an important aspect of nutrition assessment in various settings, including acute care. The three major hepatic proteins used to assess visceral protein are albumin, transferrin, and prealbumin (**Table 1.12**). However, these hepatic proteins also reflect the physiologic response to injury, stress, infection, surgery, and trauma.[15] They are negative acute-phase proteins, in that their levels decrease during the physiologic response to stress when the liver reprioritizes protein synthesis from visceral proteins to acute-phase reactant proteins. Systemic inflammation, a hallmark of the stress response, not only

reduces albumin synthesis but can also increase its degradation and promote transcapillary leakage of albumin.[16] In light of this, markers of protein status, such as albumin and prealbumin, have little value in assessment or monitoring of nutritional status in the acute-care setting. Instead, they can be used in conjunction with positive acute-phase protein lab values, such as C-reactive protein (CRP), to identify patients who might be at nutritional risk because of acute or chronic health conditions that contribute to inflammation. In addition, hypoalbuminemia has been studied extensively among subsets of patients, such as those with burns, pressure ulcers, severe sepsis, cancer, renal disease, and surgical patients, as an important predictor of morbidity and mortality.[17-20] The use of multifactorial prognostic indices may be useful in some situations to predict nutrition risk (see **Table 1.13**).[21-24]

TABLE 1.12 Visceral Protein Parameters

Protein	Normal Range	Implications and Considerations
Albumin	3.5–5.0 g/dL	Low when visceral protein is depleted; 17–20 d; low in liver disease, malabsorption syndromes, protein-losing nephropathies, ascites, burns, overhydration, inflammation; elevated in dehydration
Fibronectin	220–400 mg/dL	Low when protein is depleted; half-life 15 hr; low in inflammation, injury; affected by coagulation factors
Prealbumin	15–36 mg/dL	Low when visceral protein is depleted; half-life 1.9 d; low in liver disease, burns, inflammation; elevated in nephrotic syndrome, chronic kidney disease, pregnancy, Hodgkin lymphoma
Retinol-binding	3–6 mg/dL	Low when visceral protein is depleted; half-life 12 hr; low in chronic pancreatitis or carcinoma,

Protein	Normal Range	Implications and Considerations
protein		cystic fibrosis, intestinal malabsorption, chronic liver diseases, vitamin A deficiency; elevated in renal failure
Transferrin	188–341 mg/dL	Low when visceral protein is depleted (only if iron status is normal); half-life 8–10 d; low in chronic infection, malignancy; elevated in late pregnancy, use of oral contraceptives, viral hepatitis

Data from Pagana KD, Pagana TJ, Pagana TN. *Mosby's Manual of Diagnostic and Laboratory Tests*, 7th ed. Elsevier-Mosby, 2022.

TABLE 1.13 Formulas for Assessment of Nutrition Risk

Risk Assessment	Formula	Interpretation
Nutrition Risk Index	(1.519 × albumin) + (41.7 × % IBW)	>100, no risk 97.5–100, mild risk 83.5–97.5, moderate risk <83.5, severe risk
Prognostic Nutrition Index	158 – 16.6 (albumin) – 0.78 (triceps skin fold in mm) – 0.2 (transferrin) – 5.8 (delayed skin hypersensitivity)	<40, normal ≥40, compromised
Prognostic Inflammatory and Nutritional Index	$\frac{\text{C-reactive protein} \times \text{alpha 1-acid-glycoprotein}}{\text{prealbumin} \times \text{albumin}}$	≤1, no risk 1–10, low risk 11–20, moderate risk 21–30, severe risk

Total lymphocyte count (TLC) reflects visceral protein status; although because of its association with immune system function, it will not be accurate under some circumstances (**Table 1.14**).

TABLE 1.14 Total Lymphocyte Count and Visceral Protein Status

Formula: TLC = % of lymphocytes × No. of WBCs (10^3)

Interpretation (Values in cells/mL^3)	
Normal	>1,500
Mild degree of depletion	1,200–1,500
Moderate degree of depletion	800–1,199
Severe degree of depletion	<800

Affected by
Injury, viral infection, radiation therapy, surgery, chemotherapy, and other immunosuppressive medications.

Note: TLC may not be a reliable indicator of malnutrition in the elderly.
WBCs, white blood cells.
Data from (i) Academy of Nutrition and Dietetics. Nutrition Care Manual: Biochemical data, medical tests, and procedures. Accessed July 24, 2023. https://www.nutritioncaremanual.org, and (ii) Pagana KD, Pagana TJ, Pagana TN. *Mosby's Manual of Diagnostic and Laboratory Tests*, 7th ed. Elsevier-Mosby, 2022.

Another parameter for the assessment of visceral protein status is nitrogen balance, although it more properly reflects total body protein (**Table 1.15**). Adults are normally in nitrogen balance, in that dietary protein (source of nitrogen) is used for protein synthesis. When protein degradation

exceeds synthesis, a person is in negative nitrogen balance, which occurs in malnutrition. Children and pregnant women are in positive nitrogen balance, because protein synthesis exceeds degradation during phases of growth.

TABLE 1.15 Use of Nitrogen Balance in Assessment of Total Body Protein

1. Monitor dietary intake of protein (PRO) for 24 hr.
2. Convert dietary protein intake to nitrogen intake:

$$\text{Nitrogen} = \frac{\text{PRO intake}}{6.25 \text{ g nitrogen}}$$

3. Collect 24-hr urine; obtain urinary urea nitrogen (UUN).
4. Nitrogen balance = Nitrogen intake – (UUN + 3[a]).
5. Interpretation: Adults are normally in nitrogen balance (0); pregnant women and children (growth states) are in positive balance; negative balance may suggest malnutrition.

[a]Insensible losses.

The somatic protein compartment is assessed using both anthropometric and biochemical measurements, the latter through the use of creatinine excretion and nitrogen balance. Creatinine is a catabolic product of creatine phosphate, a compound needed in muscular contraction. It is excreted at a constant daily rate proportional to muscle mass. Kidney disorders and high intake of meat products can raise levels. **Table 1.16** provides the formula for calculating creatinine height index (CHI) and standards for comparison to determine protein depletion severity.

TABLE 1.16 Calculation and Interpretation of

Creatinine Height Index

24-Hour Urine Collection; Meat-Free Diet (Ideally); Compare Creatinine with Standard for Height/Gender

$$\% \text{ CHI} = \frac{\text{24 urinary creatinine} \times 100}{\text{expected 24-hr urinary excretion (mg)}}$$

Interpret: 60%–80%, mild depletion
40%–59%, moderate depletion
<40%, severe depletion
Affected by: Renal dysfunction, advanced age, stress, trauma, sepsis, strenuous exercise, use of corticosteroids

24-Hour Urinary Creatinine Excretion in Adults

Males: 23 mg/kg/IBW		Females: 18 mg/kg/IBW	
Height (cm)	**Creatinine (mg)**	**Height (cm)**	**Creatinine (mg)**
157.5	1,288	147.3	830
160.0	1,325	149.9	851
162.6	1,359	152.4	875
165.1	1,386	154.9	900
167.6	1,426	157.5	925
170.2	1,467	160.0	949
172.7	1,513	162.6	977
175.3	1,555	165.1	1,006
177.8	1,596	167.6	1,044
180.3	1,642	170.2	1,076
182.9	1,691	172.7	1,109
185.4	1,739	175.3	1,141
188.0	1,785	177.8	1,174
190.5	1,831	180.3	1,206
193.0	1,891	182.9	1,240

CHI, creatine height index.

Data from (i) Blackburn GL, et al. Nutritional and metabolic assessment of the hospitalized patient. *J Parenter Enteral Nutr.* 1977;1(1):11-12, and (ii) Saleh MA, Obeidalla AA, Thabet AF, Abdel Aal AM. Creatinine height index as a predictor of nutritional status among patients with liver cirrhosis. *J Public Health Epidemiol.* 2016;8(10):220-228. doi:10.5897/JPHE2016.0850.

Description

Biochemical assessment also includes review of hematologic parameters, as they may be indicators of

nutritionally related anemia (**Table 1.17**). Several of these parameters can also be expected to be affected by compromised visceral protein status.

TABLE 1.17 Hematologic Parameters Related to Anemia

Constituent	Normal Range	Implications and Considerations
Erythrocyte protoporphyrin	<5 µg/dL RBCs	High in later stages of iron-deficiency anemia
Ferritin	Males: 18.0–350 ng/mL Females: 15–49 years: 12.0–156 ng/mL >49 years: 18.0–204 ng/mL	Low in early deficiency state in the presence of depleted iron stores
Folate, RBC content	95 ng/mL	Low in later stages of folate-deficiency anemia
Folate, serum	1.9 ng/mL	Low as folate-deficiency progresses
Hematocrit	Males: 41%–50% Females: 35%–46%	Low in anemia; represents percentage of RBCs in total blood volume
Hemoglobin	Males: 14.0–17.2 g/dL Females: 12.0–15.6 g/dL	Low in anemia; represents total amount of hemoglobin in RBCs
Mean corpuscular hemoglobin concentration	32–36 g/dL	Low in iron-deficiency anemia (hypochromic); normal in B_{12} and folate deficiency (normochromic);

Constituent	Normal Range	Implications and Considerations
		represents hemoglobin (pigmentation) contained in an average RBC
RBC count	Male: 4.4–5.8 $\times 10^6$ µL Female: 3.9–5.2 $\times 10^6$ µL	Low in anemia; the number of RBCs in sample
Transferrin	188–341 mg/dL	High in iron-deficiency anemia as transport of iron increases
Vitamin B_{12}	200–800 pg/mL	Low in B_{12} deficiency

RBC, red blood cell.
Data from Pagana KD, Pagana TJ, Pagana TN. *Mosby's Manual of Diagnostic and Laboratory Tests*, 7th ed. Elsevier-Mosby, 2022.

Nutrition-Focused Physical Examination: Identifying Malnutrition

Physical assessment is a vital part of the nutrition assessment of a patient. The goal is to identify signs and symptoms that may be associated with specific nutrient deficiencies and compromised nutritional status or malnutrition.

Adult malnutrition in the clinical setting is diagnosed using an etiology-based approach that was developed in a collaboration between the Academy of Nutrition and Dietetics and the Society for Parenteral and Enteral Nutrition (AND/ASPEN).[25] The three etiology-based nutrition diagnoses are acute illness or injury-related malnutrition (inflammation is acute and severe), chronic disease or condition-related malnutrition (inflammation is chronic and mild to moderate), and starvation-related malnutrition (chronic starvation without inflammation). Six characteristics have been identified as vital in the detection and diagnosis of malnutrition: insufficient energy intake, weight loss, loss of subcutaneous body fat, loss of muscle mass, fluid, accumulation that may mask weight loss, and diminished functional status. As no single parameter is definitive for adult malnutrition, two or more of the six characteristics are recommended for diagnosis (see **Table 1.18**).

TABLE 1.18 Determining Adult Malnutrition

Context of Malnutrition	Chronic Illness		Acute Illness or Injury		Social/Environmental Circumstances	
Degree of Malnutrition	**Malnutrition of Moderate Degree**	**Severe Protein-Calorie Malnutrition**	**Malnutrition of Moderate Degree**	**Severe Protein-Calorie Malnutrition**	**Malnutrition of Moderate Degree**	**Severe Protein-Calorie Malnutrition**
Energy intake: Compare recent intake with estimated energy needs and report inadequate intake as a percentage of estimated needs over time.	<75% of estimated energy requirement for ≥1 mo	≤75% of estimated energy requirement for ≥1 mo	<75% of estimated energy requirement for >7 d	≤50% of estimated energy requirement for ≥5 d	<75% of estimated energy requirement for ≥3 mo	≤50% of estimated energy requirement for ≥1 mo
Weight loss: Evaluate weight in light of other clinical findings including hydration. Report weight change over time as a percentage of weight lost from baseline.	5% in 1 mo 7.5% in 3 mo 10% in 6 mo 20% in 12 mo	>5% in 1 mo >7.5% in 3 mo >10% in 6 mo >20% in 12 mo	1–2% in 1 wk 5% in 1 mo 7.5% in 3 mo	>2% in 1 wk >5% in 1 mo >7.5% in 3 mo	5% in 1 mo 7.5% in 3 mo 10% in 6 mo 20% in 12 mo	>5% in 1 mo >7.5% in 3 mo >10% in 6 mo >20% in 12 mo
Body fat: Loss of subcutaneous fat (orbital, triceps, fat overlying ribs).	Mild depletion	Severe depletion	Mild depletion	Moderate depletion	Mild depletion	Severe depletion
Muscle mass: Loss of muscle (temples, clavicles, shoulders, scapula, thigh, calf).	Mild depletion	Severe depletion	Mild depletion	Moderate depletion	Mild depletion	Severe depletion
Fluid accumulation: General or local fluid accumulation (extremities, ascites, or vulva/scrotal edema).	Mild	Severe	Mild	Moderate to severe	Mild	Severe
Functional status: Based on standards supplied by manufacturer of dynamometer.	Not applicable	Measurably reduced	Not applicable	Not recommended in intensive care setting	Not applicable	Measurably reduced

Reproduced from White JV, Guenter P, Jensen G, Malone A, Schofield M. Consensus statement of the Academy of Nutrition and Dietetics/American Society for Parenteral and Enteral Nutrition: Characteristics recommended for the identification and documentation of adult malnutrition (undernutrition). *J Acad Nutr Diet*. 2012;112(5):275-283, 730-738.

Description

The nutrition-focused physical exam (NFPE) is conducted by the dietitian to help identify malnutrition by determining fat loss, muscle loss, and presence of edema (**Table 1.19**), as well as to identify possible nutrient deficiencies (**Table 1.20**).[26,27] The Academy offers training workshops on conducting an NFPE and more information can be found on their website (https://www.eatrightpro.org/nfpe). When analyzing NFPE results, it is crucial to recognize that identifying a standalone nutrient deficiency solely from this data is uncommon. The indications and manifestations of a deficiency usually arise following prolonged inadequate dietary intake. Findings should be analyzed in conjunction

with other assessment data including diet history, lab tests, and the medical condition of the patient.

TABLE 1.19 Nutrition-Focused Physical Examination: Malnutrition Assessment

Fat Area	Normal	Mild to Moderate	Severe
Orbital region—surrounding the eye (fluid retention may mask loss)	Slightly bulged fat pads	Slightly darker circles; somewhat hallow look	Dark circles; hallow look; depression; loose skin
Upper arm region—triceps/biceps	Large space between fingers when pinching	Some depth pinch but not ample; fingers almost touch	Very little space between folds, fingers touch
Thoracic and lumbar region—ribs, lower back, midaxillary line	Chest is full; ribs do not show; slight to no protrusion of the iliac crest	Ribs apparent; depressions between them less pronounced; iliac crest somewhat prominent	Depression between the ribs very apparent; iliac crest very prominent
Muscle Area	**Normal**	**Mild to Moderate**	**Severe**
Temple region—temporalis muscle	Can see/feel well-defined muscle	Slight depression	Hollow; scooping depression
Clavicle and acromion bone region—deltoid muscle	Rounded curves at arm/shoulder/neck	Acromion process may slightly protrude	Shoulder to arm joint looks square; bones prominent; acromion protrusion very prominent
Clavicle bone region—pectoralis major, deltoid, trapezius muscles	Not visible in male; visible but not prominent in female	Visible in male; some protrusion in female	Protruding, prominent bone
Scapular bone region—trapezius, supraspinatus, infraspinatus muscles	Bones not prominent; no significant depressions	Mild depression or bone may show slightly	Prominent, visible bones; depressions between ribs/scapula or shoulder/spine
Dorsal hand—interosseous muscle	Muscle bulges could be flat in some well-nourished people	Slightly depressed	Depressed area between thumb–forefinger
Patellar region—quadriceps muscle	Muscles protrude; bones not prominent	Knee cap less prominent; more rounded	Bones prominent; little sign of muscle around knee
Anterior thigh region—quadriceps muscles	Well rounded; no depressions	Mild depression on inner thigh	Depression/line on thigh; obviously thin
Posterior calf region—gastrocnemius muscle	Well-developed bulb of muscle	Not well developed	Thin; minimal to no muscle definition
Edema	Normal	Mild to Moderate	Severe
Edema—rule out other causes of edema; patient at dry weight	No sign of fluid accumulation	Mild to moderate pitting; slight swelling of the extremity; indentation subsides quickly (0–30s)	Deep to very deep pitting; depression lasts a short to moderate time (31–60s); extremity looks swollen (3–4+)

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Description

TABLE 1.20 Nutrition-Focused Physical Examination: Nutrient Deficiency Assessment

	Observed Conditions		
Body System	**Healthy**	**Abnormal**	**Implications**
Hair	Normal distribution, shiny	Thin, dull, dry, brittle, corkscrew hairs	Chemotherapy, protein or biotin deficit, vitamin C deficiency
Eyes	Bright, clear, shiny, pink conjunctiva	Sunken, dull, pale, dry conjunctiva, photophobia, xerosis	Deficiency of vitamin A, zinc, riboflavin
Lips	Moist, good color	Swollen, dry, red, cracked	Deficiency of riboflavin, pyridoxine, niacin
Gums	Pink, firm	Sore, spongy, red, swollen, bleed easily	Vitamin C deficiency
Tongue	Pink, presence of papillae	Purple, white or gray coating, smooth, slick	Deficiency of riboflavin, pyridoxine, folic acid, niacin, vitamin B_{12}, iron
Teeth	Clean, intact, all present	Dentures, missing teeth, loss of tooth enamel	Calcium deficiency, poor diet
Neck	No swelling	Presence of nodule(s), goiter	Excess or deficiency of iodine
Skin	Smooth, slightly moist, good color	Pale, dry, scaly, bruises easily, pressure ulcers, dermatitis	Deficiency of iron, vitamins A or C, zinc, essential fatty acid, protein; excess of niacin
Legs	Well-developed, firm musculature, no joint or bone pain	Calf tenderness, flaccid muscles, pain, edema, rickets, bone or joint pain	Deficiency of protein; vitamins A, C, or D; calcium
Abdomen	No swelling or pain	Mildly edematous, diarrhea, ascites	Deficiency of protein, niacin, zinc
Hands/Nails	Smooth	Brittle nails, atrophied fine muscles, spoon-shaped nails	Deficiency of protein, iron
Musculoskeletal, adipose	Normal bone, muscle, and fat development	Calf tenderness, loss of subcutaneous fat, emaciated appearance, pain, decreased grip strength, hollow cheeks, fractures, osteoporosis	Deficiency of protein, thiamin, vitamin C; energy or fluid deficit
Neurologic	Normal reflexes	CVA, limited reflexes, disorientation, paralysis, convulsions, dementia	Deficiency of thiamin, niacin, vitamins B_6 or B_{12}, folic acid, iodine, phosphorus, calcium, magnesium

CVA, cerebrovascular accident.

Data from (i) Academy of Nutrition and Dietetics. Nutrition Care Manual. Nutrition-Focused Physical Findings. Accessed July 24, 2023. https://www.nutritioncaremanual.org, and (ii) Escott-Stump S. *Nutrition and Diagnosis-Related Care*, 9th ed. Academy of Nutrition and Dietetics, 2022.

Description

The Subjective Global Assessment (SGA) is another nutrition assessment tool that has been in use since the 1980s. It has been found to be highly predictive of nutrition associated complications and a recent study found that the Academy/ASPEN criteria for diagnosing malnutrition correlated with SGA in identifying nutritional status in hospitalized adult patients.[28] The SGA uses patient assessment data such as weight, intake, functional status, disease state, symptoms, metabolic stress, and physical examination to assess and monitor risk for malnutrition and to evaluate effects of interventions. An online, scored patient-generated SGA is available for health professionals to utilize at http://pt-global.org.

Patient History

A comprehensive nutrition assessment includes many aspects of the patient's history. The main types of history include health or medical, medications, personal, and food and nutrition. Sources for historical information include the medical record, the patient, and significant others.

The **health or medical history** identifies factors that affect nutrient needs or nutrition education needs or that place the client at risk for poor nutrition status. One important component is the nutritional relevance of the current diagnosis (**Box 1.3**). The diagnosis may present a specific level of risk based on the potential to adversely affect nutritional status. In addition to the disease's potential to alter nutritional status, the degree of risk posed to an individual patient depends on several factors, including its duration and severity, the presence of other physiologic stressors, and the individual (genetics, age, and nutritional status). Some diseases are nutritionally relevant because of the likelihood of the need for nutrition intervention.

Box 1.3 Nutritionally Relevant Conditions

AIDS-HIV	Dehydration	Multiple sclerosis
Alcohol and/or drug abuse	Diabetes	Nephrotic syndrome
Cachexia	Dysphagia	Neutropenia
Cancer	Eating disorders	Obesity
Celiac disease	Gastrointestinal	Pancreatitis

	bleeding	
Cerebrovascular accident	Hepatic encephalopathy	Parkinson disease
Chronic obstructive pulmonary disease	Hepatitis	Peritonitis
Cirrhosis	Hypertension	Pressure ulcers
Coronary heart disease (MI)	Inflammatory bowel disease	Renal failure
Crohn's disease	Malabsorption	Sepsis
	Malnutrition	Tuberculosis

MI, myocardial infarction.
Data from Academy of Nutrition and Dietetics, Evidence Analysis Library, "Energy Expenditure: Evidence Analysis: Estimating RMR with Prediction Equations," last modified 2006, http://www.eatrightpro.org/resources/research/evidence-based-resources/evidence-analysis-library.

The **medication history** identifies substances that can affect nutrient needs or alter nutritional status. This includes prescribed medications, over-the-counter medications, and dietary supplements (see Appendix B for a listing of food/supplement and drug interactions). **The food and nutrition history** identifies food consumption patterns, specific nutrient intake and imbalances, reasons for potential nutrition problems, and dietary factors important for shaping the nutrition care plan. **Personal history** includes several aspects of psychosocial and lifestyle patterns that may affect nutrient needs, influence food choices, or limit therapy options. **Table 1.21** lists general information for all categories of history in addition to specific information to collect from the patient or the medical record.

TABLE 1.21 Patient History Categories

History Category	Specific Information
Health/Medical	
Current health status and diagnoses	Date of first diagnosis, previous education on diagnosis
Previous medical history and health status	Previous diagnoses and conditions, particularly nutritionally relevant
Family history	Specific family members affected by nutritionally relevant diseases and onset age
Surgical history	Recent diagnostic procedures requiring NPO status Difficulty chewing (state of dentition) or swallowing Chronic gastrointestinal problems (diarrhea, constipation, nausea, vomiting)
Medications	
Prescription medications	Use of multiple medications Duration of medication use Frequency of use (chronic or as needed) Changes in sense of smell or taste related to medications Previous education on potential interactions
Over-the-counter medications	
Dietary supplements (nutrient, herbal)	
Illegal drugs	
Food and Nutrition/Diet	
Food intake	Food intolerances or allergies
Eating habits and patterns	Appetite (current and prior to admission) Weight history (recent weight loss in particular) Physical disabilities affecting food preparation or intake

History Category	Specific Information
Health/Medical	
	Typical daily intake (types and amounts of foods and beverages consumed) Meal pattern Religious dietary restrictions
Lifestyle patterns	Ethnic dietary habits Alcohol consumption Frequency of dining out; types Exercise, physical activity (type and frequency) Attitude regarding diet and health Previous diet instruction (location, year, topic) Interest in diet instruction or outpatient counseling Stage of change/readiness to learn
Personal	
Age	Income
Gender	Use of or eligibility for government programs
Cultural/Ethnic identity	Communication barriers
Occupation/Economic status	Cognitive function
Role in family	Smoking
Educational level	Ability to perform daily functions
Motivational level	Person responsible for grocery shopping, meal preparation
Access to transportation	Recent loss of spouse or partner

Food and Nutrition History

A comprehensive food and nutrition history (diet history) is generally not possible with most patients in the acute care setting, although components of the history are vital in nutrition assessment. In other practice settings, it is both possible and desirable to collect detailed information of this type. The goal of a food and nutrition history is to identify nutrient intake and imbalances, the reasons for potential food and nutrition problems, and all dietary factors important in generating the nutrition diagnosis and subsequent intervention. The following types of data are needed: Food intake, eating habits and patterns, and lifestyle patterns related to nutrition and health. If it is likely the patient will require bedside instruction, and depending on the diagnosis, more detailed information about the patient's food intake may need to be collected.

Of the various methods for obtaining a patient's food intake, the most practical in the acute or extended care settings are the typical daily intake (TDI) (**Table 1.22**) and simplified food frequency (FF) (**Table 1.23**). In the TDI, the patient is asked what he or she typically eats on a daily basis to identify patterns of deficiency or excess in particular food groups. In contrast to a 24-hour food recall, in which the patient recounts his or her intake beginning with the last meal eaten, the TDI begins with the first meal of the day. To avoid a judgmental approach, it is best not to label meals as breakfast, lunch, and dinner, but rather based on time of day. For example, you could ask, "What is the first thing you typically eat or drink when you get up for the day?" The TDI should also indicate the amount of food eaten and where eating typically takes place.

TABLE 1.22 Typical Daily Intake Form

Meal Timing	Food Item	Amount	Where Eaten
8:00 a.m.	Oatmeal	1 cup	Kitchen table at home

Description

TABLE 1.23 Food Frequency Form

Food Item/Group	Amount/Day	Amount/Week
Milk or other dairy products (yogurt, cheese)		
Meat, poultry, and eggs		
Fish		
Nuts, legumes, and legume products		
Fruits		
Vegetables		
Starches (breads, cereals, grains)		
Fats, added (oils, margarine, salad dressing)		
Snack foods (chips, pretzels, crackers)		
Desserts/Sweets		
Dining out		
Fast food		
Restaurant dining		
Beverages		
Alcohol		
Coffee, tea		

Food Item/Group	Amount/Day	Amount/Week
Carbonated beverages		
Fruit juices		

A simplified FF is not as detailed as a comprehensive FF questionnaire but is advantageous for two reasons. The first advantage is that the simplified FF is a quick method for determining whether the patient avoids any major category of food, and the second is that it will provide a crosscheck of the TDI. After collecting the food intake information, it is compared with an appropriate standard, such as the Dietary Guidelines for Americans or the Dietary Reference Intakes. A simplified evaluation form of general aspects of diet can also be used (**Table 1.24**).

TABLE 1.24 Evaluation of Dietary Intake

Group	Servings/Day	Recommended	Adequate/Excess
Dairy			
Protein			
Fruit			
Vegetable			
Starch			
Fat/sweets			
Overall Diet Adequacy: _____ yes _____ no			
Specific Nutrients:			
Deficit of: _____ kcals _____ PRO _____ fiber _____ Vit A _____ Vit C _____ Fe _____ Ca _____			
Other			
Excess of: _____ kcals _____ Fat _____ SFA _____ Chol _____ Sugar _____ Alcohol _____ Na			
Other: _____			
Summary:			

PRO, protein; Fe, iron; Ca, calcium; SFA, saturated fatty acid; Chol, cholesterol; NA, sodium.

Description

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CHAPTER 2

Pregnancy

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Pregnancy represents a vulnerable life stage relative to a person's nutritional status. Both the dietary intake and the nutritional status of a person before and during pregnancy greatly influence fetal development and, in turn, pregnancy outcome. Research has also shown an association between suboptimal nutritional status and abnormal fetal growth patterns including low birth weight (LBW; <2,500 g), small for gestational age (SGA; <10% birthweight for gestational age), fetal growth restriction (FGR), macrosomia (>4.4–4.5 kg) and large for gestational age (LGA; >90% birthweight for gestational age). These are associated with the risk of developing chronic diseases such as obesity, insulin resistance, and diabetes in both childhood and adulthood.[1] Several of the complications of pregnancy can also adversely affect nutritional status. For these reasons, nutritional assessment is imperative to help ensure optimal pregnancy outcome.

It is for this reason that the Centers for Disease Control and Prevention (CDC) and the US Department of Health and Human Services Office on Women's Health recommend that women and men should prepare for pregnancy at least 3 months prior to getting pregnant.[2] Some preparations, such as achieving a healthy weight, managing chronic health conditions, discontinuing smoking and alcohol use, and getting regular dental care should begin earlier[3] (**Box 2.1**).

Box 2.1 Considerations for Preconception Health

1. Have a reproductive plan.
2. See your doctor. Discuss and manage any known medical conditions and sexually transmitted infections (STIs). Discuss all prescription and over-the-counter medications as well as dietary/herbal supplements and vitamins. Review and update vaccinations, including T-dap and flu.
3. Consume at least 400 µg of folic acid daily (from fortified foods and/or a dietary supplement, and foods rich in folate).
4. Stop drinking alcohol. There is no known safe level of consumption at any time during pregnancy or while trying to get pregnant.
5. Stop smoking and avoid exposure to tobacco smoke.
6. Stop using drugs and seek assistance for substance use disorder.
7. Avoid toxic substances and environmental contaminants such as chemicals or cat/rodent feces.
8. Reach and maintain a healthy weight via lifestyle habits and regular physical activity.
9. Learn your and your partner's family health history and discuss findings with your provider.
10. Address mental health issues.
11. Seek assistance for concerns such as domestic violence or lack of support.
12. Get regular dental check-ups.
13. Discuss with your provider the possible risks and consequences of travel to areas with Zika virus outbreak or other areas with known risk.

Data from (i) Preconception Health. US Department of Health and Human Services Office on Women's Health. Last updated February 22, 2021. Accessed April 25, 2023. https://www.womenshealth.gov/pregnancy/you-get-pregnant/preconception-health, and (ii) Planning for Pregnancy. Centers for Disease Control and Prevention. Last reviewed February 15, 2023. Accessed April 25, 2023. https://www.cdc.gov/preconception/planning.html.

One of the most important aspects of pregnancy is body weight. Both weight gain during gestation and, more importantly, prepregnancy body mass index (BMI) significantly influence pregnancy outcomes.[4] However, many people fail to meet weight and weight gain recommendations prior to and during pregnancy. As of 2019, 29% of people met the criteria for obesity prior to

pregnancy, which is an 11% increase from 2016. Additionally, only 32% of the US people gain weight within the recommended range during gestation.[1]

A person with either excess or low body weight before pregnancy has a higher risk for poor outcome. In addition, weight gain during pregnancy, specifically total amount and rate, is correlated with infant birth weight, which, in turn, is associated with infant mortality.[5] Therefore, achieving and maintaining a healthy weight prior to pregnancy should be encouraged.

Guidelines for Pregnancy Weight Gain

In 2009, The Institute of Medicine (IOM) issued pregnancy weight gain recommendations based on current body weight for both total weight and rate of weight gain. These guidelines are based on the World Health Organization (WHO) BMI categories for all pregnant people regardless of age or racial/ethnic background (**Table 2.1**).[5]

TABLE 2.1 Guidelines for Pregnancy Weight Gain

Category	BMI (WHO)	Weight Gain (kg)	Rate of Weight Gain for 2nd and 3rd Trimesters*
Underweight	<18.5	12.5–18	0.44–0.58 kg/wk
Normal weight	18.5–24.9	11.5–16	0.35–0.50 kg/wk
Overweight	25.0–29.9	7–11.5	0.23–0.33 kg/wk
Obese (all classes)	≥30.0	5–9	0.17–0.27 kg/wk

*Calculations assume a 0.5 to 2 kg weight gain in the first trimester.

Data from Institute of Medicine and National Research Council. 2009. *Weight Gain During Pregnancy: Reexamining the Guidelines.* Washington, DC: The National Academies Press. https://doi.org/10.17226/12584.

Description

Provisional recommendations are also provided for twin pregnancies, encouraging pregnant people with a normal prepregnancy (BMI 18.5–24.9) to gain 17–25 kg, overweight (BMI 25.0–29.9) to gain 14–23 kg, and obese (BMI ≥30) to gain 11–19 kg. Optimal maternal weight gain for underweight (BMI <18.5) is 23–28 kg.[6] There is insufficient data to provide weight gain recommendations for triplet or other multifetal pregnancies (**Table 2.2**). As these pregnancies typically result in a higher rate of low birth weight, it is necessary to ensure adequate weight gain.

TABLE 2.2 Weight Gain Recommendations for Multifetal Pregnancy

Twin Gestation	
Underweight BMI (<18.5)	23–28 kg*
Normal BMI (18.5–24.9)	17–25 kg
Overweight BMI (25.0–29.9)	14–23 kg
Obese BMI (≥30.0)	11–19 kg
Triplet Gestation	
(Any BMI)	Insufficient data to determine

*Data from Luke B, Hediger ML, Nugent C, et al. Body mass index—specific weight gains associated with optimal birth weights in twin pregnancies. *J Reprod Med*. 2003;48(4):217-224.
Data from Institute of Medicine and National Research Council. 2009. *Weight Gain During Pregnancy: Reexamining the Guidelines*. Washington, DC: The National Academies Press. https://doi.org/10.17226/12584.

Nutrient and Energy Recommendations for Pregnancy

Requirements for most essential nutrients and calories increase during pregnancy over nonpregnant status (**Table 2.3**). Meeting energy needs during pregnancy is crucial for appropriate maternal weight gain. The most recent Dietary Reference Intakes for Energy suggests that a pregnant person's estimated energy requirement (EER) is equal to their nonpregnant EER during the first trimester for any given physical activity level (PAL). However, during the second and third trimesters, an energy deposition increment is added to account for fetal growth and plasma volume expansion. This increment is based on prepregnant BMI (underweight, +300 kcal/day; normal weight, +200 kcal/day; overweight, +150 kcal/day; obese, –50 kcal/day). Continuous monitoring of weight gain and subsequent adjustment of energy intake is required to ensure optimal pregnancy outcome (**Table 2.4**).[7]

TABLE 2.3 Percentage of Increase Over Nonpregnant Female for Daily Recommended Nutrient Intakes

	Female, 14–18 yr	Pregnant, 14–18 yr	% Increase	Female, 19–30 yr	Pregnant, 19–30 yr	% Increase	Female, 31–50 yr	Pregnant, 31–50 yr	% Increase
Macronutrient									
Carbohydrates	130 g	175 g	35%	130 g	175 g	35%	130	175	35%
Protein	46 g	71 g	54%	46g	71g	54%	46g	71g	54%
Fiber[a]	25 g	31 g	24%	28 g	34 g	21%	25 g	31 g	24%
Vitamin									
Vitamin A	700 µg	750 µg	7%	700 µg	770 µg	10%	700 µg	770 µg	10%
Vitamin D	600 IU	600 IU	0%	600 IU	600 IU	0%	600 IU	600 IU	0%
Vitamin C	65 mg	80 mg	24%	75 mg	85 mg	13%	75 mg	85 mg	13%
Vitamin E	15 mg	15 mg	0%	15 mg	15 mg	0%	15 mg	15 mg	0%
Vitamin K	75 µg	75 µg	0%	90 µg	90 µg	0%	90 µg	90 µg	0%
Thiamin B_1	1.0 mg	1.4 mg	40%	1.1 mg	1.4 mg	28%	1.1 mg	1.4 mg	28%
Riboflavin	1.0 mg	1.4 mg	40%	1.1 mg	1.4 mg	28%	1.1 mg	1.4 mg	28%
Niacin	14 mg	18 mg	29%	14 mg	18 mg	29%	14 mg	18 mg	29%
Vitamin B_6	1.2 mg	1.9 mg	59%	1.3 mg	1.9 mg	46%	1.3 mg	1.9 mg	46%
Vitamin B_{12}	2.4 µg	2.6 µg	8%	2.4 µg	2.6 µg	8%	2.4 µg	2.6 µg	8%
Folate	400 µg	600 µ/day	50%	400 µg	600 µg	50%	400 µg	600 µg	50%
Choline	400 mg	450 mg	12.5%	425 mg	450 mg	6%	425 mg	450 mg	6%
Mineral									
Calcium	1,300 mg	1,300 mg	0%	1,000 mg	1,000 mg	0%	1,000 mg	1,000 mg	0%
Iodine	150 µg	220 µg	47%	150 µg	220 µg	47%	150 µg	220 µg	47%
Iron	15 mg	27 mg	80%	18 mg	27 mg	50%	18 mg	27 mg	50%
Magnesium	360 mg	400 mg	11%	310 mg	350 mg	13%	320 mg	360 mg	13%
Phosphorus	1,250 mg	1,250 mg	0%	700 mg	700 mg	0%	700 mg	700 mg	0%
Potassium	2,300 mg	2,600 mg	13%	2,600 mg	2,900 mg	12%	2,600 mg	2,900 mg	12%
Selenium	55 µg	60 µg	9%	55 µg	60 µg	9%	55 µg	60 µg	9%
Sodium	2,300 mg	2,300 mg	0%	2,300 mg	2,300 mg	0%	2,300 mg	2,300 mg	0%
Zinc	9 mg	12 mg	34%	8 mg	11 mg	38%	8 mg	11 mg	38%

[a]For the 2nd trimester only.

Data from (i) *Dietary Guidelines for Americans, 2020-2025*. U.S. Department of Agriculture and U.S. Department of Health and Human Services. 9th Edition. December 2020. Accessed April 25, 2023. https://www.dietaryguidelines.gov, and (ii) *Dietary Reference Intakes: The Essential Guide to Nutrient Requirements. Institute of Medicine*. 2006. Washington, DC: The National Academies Press. https://doi.org/10.17226/11537.

Description

TABLE 2.4 Energy Needs in Pregnancy

Trimester	PAL (Physical Activity Level) Category	Estimated Energy Requirement Equation (kcal/day)
First trimester of pregnancy (conception to 13 completed weeks) for women 19 years and above	Inactive Low active Active Very active	584.90 − (7.01 × age) + (5.72 × height) + (11.71 × weight) 575.77 − (7.01 × age) + (6.60 × height) + (12.14 × weight) 710.25 − (7.01 × age) + (6.54 × height) + (12.34 × weight) 511.83 − (7.01 × age) + (9.07 × height) + (12.56 × weight)
2nd and 3rd trimesters of pregnancy For women 19 years and above.	Inactive Low active Active Very active	1,131.20 − (2.04 × age) + (0.34 × height) + 12.15 × weight) + (9.16 × gestation) + energy deposition 693.35 − (2.04 × age) + (5.73 × height) + (10.20 × weight) + (9.16 × gestation) + energy deposition −223.84 − (2.04 × age) + (13.23 × height) + (8.15 × weight) + (9.16 × gestation) + energy deposition −779.72 − (2.04 × age) + (18.45 × height) + (8.73 × weight) + (9.16 × gestation) + energy deposition

Prepregnant BMI (WHO)	Energy Deposition (kcal/day)
Underweight	+ 300 kcal
Normal weight	+ 200 kcal

Trimester	PAL (Physical Activity Level) Category	Estimated Energy Requirement Equation (kcal/day)
Overweight		+ 150 kcal
Obese		− 50 kcal

Age is in years, height is in centimeters, weight is in kilograms, and gestation in weeks.
Data from *Dietary Reference Intakes for Energy*. National Academies of Sciences, Engineering, and Medicine. 2023. Washington, DC: The National Academies Press. https://doi.org/10.17226/26818.

Although the dietary reference intakes (DRIs) consider only single-fetus pregnancy, data from research have generated energy recommendations for a twin pregnancy, based on prepregnancy BMI. For people beginning pregnancy with a BMI of 18.5–24.9, an energy intake of 40–45 kcal/kg/day is recommended. Suggested intakes for underweight and overweight people are 42–50 kcal/kg/day and 30–35 kcal/kg/day, respectively.[8] However, calorie intake may need to be adjusted to ensure appropriate weight gain throughout the second and third trimesters. Due to increased maternal metabolic requirements, carbohydrate intake recommendations also increase to 175 g/day for a prepregnancy normal-weight person and 200 g/day and 163 g/day for an underweight and overweight person, respectively.[7]

Specific nutrients become “nutrients of public health concern,” as described by the U.S. Department of Agriculture, because their underconsumption is associated with health concerns. During pregnancy, these nutrients also include iron, folate, iodine, and choline (**Table 2.5**).[9]

Pregnant people are routinely prescribed a vitamin and mineral supplement, but a healthful daily eating pattern is important. The MyPlate Daily Food Plan can serve as the basis for such a pattern. The following example illustrates a food plan for a pregnant person with a normal prepregnancy BMI (**Table 2.6**).

TABLE 2.5 Recommendations for Nutrients of Concern in Pregnancy

Nutrient	Age (yr)	Amount
Calcium	14–18 19 and older	1,300 mg 1,000 mg
Choline	All ages	450 mg
Folate	All ages	400–800 µg
Iodine	All ages	220 mcg
Iron	All ages	27 mg
Protein	All ages	71 g

Data from: (i) *Dietary Guidelines for Americans*, *2020-2025*. U.S. Department of Agriculture and U.S. Department of Health and Human Services. 9th Edition. December 2020. Accessed April 25, 2023. https://www.dietaryguidelines.gov, and (ii) US Preventive Services Task Force. Folic Acid Supplementation for the Prevention of Neural Tube Defects: US Preventive Services Task Force Recommendation Statement. *JAMA*. 2017;317(2):183-189. doi:10.1001/jama.2016.19438

TABLE 2.6 The USDA MyPlate Plan

Food Group	Servings		Serving Types
	1st Trimester[a]	2nd and 3rd Trimesters[b]	
Whole grains	6 oz	8 oz	Whole wheat bread and chapati Whole grain cereals and crackers Brown and wild rice
Fruits	2 cups	2 cups	Fresh, frozen, canned, or dried whole fruits
Vegetables	2.5 cups	3 cups	Fresh, frozen, or canned dark green, red, and orange varieties
Protein foods	5.5 oz	6.5 oz	Seafood Beans, peas and lentils Unsalted nuts and seeds Soy products Eggs Lean meats and poultry
Dairy	3 cups	3 cups	Low-fat or fat-free dairy milk, yogurt and kefir Lactose free dairy Fortified soy beverages Cheese
Added sugars	<50 g/day	<60 g/day	
Saturated fat	<22g/day	<27 g/day	
Sodium	<2,300 mg/day	<2,300 mg/day	

[a] Based on a 2,000 kcal diet (1st trimester).
[b] Based on a 2,400 kcal diet (2nd and 3rd trimesters).

Data from (i) MyPlate Plan. Food and Nutrition Service U.S. Department of Agriculture; July, 2021. Available at https://www.myplate.gov/myplate-plan/results/2000-calories-ages-14-plus and https://www.myplate.gov/myplate-plan/results/2400-calories-ages-14-plus. (Accessed April 30, 2023), and (ii) *Dietary Guidelines for Americans, 2020-2025*. U.S. Department of Agriculture and U.S. Department of Health and Human Services. 9th Edition. December 2020. Accessed April 25, 2023. https://www.dietaryguidelines.gov.

Description

Nutritional adequacy in pregnancy may be further compromised by special dietary conditions and practices. These may include celiac disease and vegetarian or vegan diets. Pregnant people adhering to a gluten-free diet may consume less than adequate iron, folic acid, calcium, and zinc. Therefore, diet recommendations include consumption of fish and seafood (low in mercury), dark green vegetables, fortified dairy products, and legumes. The use of a gluten-free prenatal vitamin/mineral supplement may also be

necessary.[10] For pregnant people adhering to a vegetarian or vegan diet, nutrients of concern include iron, vitamin D, vitamin B_{12}, choline, and n-3 fatty acids (eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA)).[11] The 2020–2025 Dietary Guidelines for Americans provides a healthy vegetarian eating pattern and includes recommended foods in nutrient dense forms (**Table 2.7**).

TABLE 2.7 Healthy Lacto-Ovo Vegetarian Eating Patterns

Food Group	Daily Servings (Based on 2,000 Calories)	Weekly Servings (Based on 2,000 Calories)	Serving Types
Vegetables	2.5 (cup-eq)		
Dark-green vegetables		1.5 cup-eq/wk	Cooked kale, broccoli, bok choy, collard greens Raw spinach or romaine lettuce
Red and orange vegetables		5.5 cup-eq/wk	Carrots, red pepper, and winter squash
Beans, peas, and lentils		1.5 cup-eq/wk	Black beans, lentils, chickpeas, pinto beans, or edamame
Starchy vegetables		5 cup-eq/wk	Potatoes, sweet potatoes, corn, green peas, or green lima beans
Other vegetables		4 cup-eq/wk	Mushrooms, cabbage, zucchini, or green peppers
Fruits	2 cup-eq		Orange, berries, melon, mangos, banana
Grains	6.5 oz-eq		
Whole grains	3.5 oz-eq		Whole wheat bread, whole grain cereal, brown rice, millet, bulgur, and oatmeal
Refined grains	3 oz-eq		Enriched pasta, white rice, or cereal
Dairy	3 cup-eq		Low-fat milk, cheese, yogurt, or fortified soy beverage/yogurt
Protein Foods	3.5 oz-eq		
Eggs		3 oz-eq/wk	Egg
Beans, peas, and lentils		6 oz-eq/wk	Black beans, lentils, chickpeas
Soy products		8 oz-eq/wk	Cooked tofu
Nuts and seeds		7 oz-eq/wk	Peanut butter, almonds, sunflower seeds
Oils	27 g		Olive oil, avocado oil, canola oil

Data from *Dietary Guidelines for Americans, 2020-2025*. U.S. Department of Agriculture and U.S. Department of Health and Human Services. 9th Edition. December 2020. Accessed April 25, 2023. https://www.dietaryguidelines.gov/sites/default/files/2021-03/Dietary_Guidelines_for_Americans-2020-2025.pdf.

Description

Nutrition Assessment

An early nutrition assessment is crucial in pregnancy, given the relationship between a person's nutritional status and pregnancy outcome. In addition to the standard nutrition assessment categories, it is important to consider key risk factors, which may be present prior to pregnancy or arise during prenatal care (**Box 2.2**). Providers of individualized nutrition counseling should consider a pregnant person's access to food and prenatal care, socioeconomic status, race/ethnicity, and cultural food practices.[12]

Box 2.2 Nutrition Assessment Risk Factors in Pregnancy

1. Pregnancy at a young age (conception at ≤17 years of age)
2. High parity and young age (under the age of 20 at conception with three or more previous pregnancies)
3. Prepregnancy weight: BMI <18.5 or ≥ 25.
4. Low maternal weight gain: < 1 lb/wk (underweight), <0.8 lb/wk (normal weight), <0.5 lb/wk (overweight) or <0.4 lb/wk (obese)
5. High maternal weight gain: >1.3 lb/wk (underweight), > 1 lb/wk (normal weight), >0.7 lb/wk (overweight), >0.6 lb/wk (obese) in the 2nd and 3rd trimesters for singleton pregnancies
6. Low hemoglobin/hematocrit or elevated blood lead levels
7. Hyperemesis gravidarum
8. Nutrient deficiency or disease
9. Lactose intolerance
10. Food allergies
11. Current or history of gestational diabetes
12. History of preeclampsia
13. Chronic disease (e.g., diabetes/prediabetes, hypoglycemia, cardiovascular disease, gastrointestinal diseases, thyroid disorders, hypertension/prehypertension, renal disease, cancer, inborn errors of metabolism, celiac disease, central nervous system disorders, genetic/congenital disorders, infectious diseases)

14. Recent major surgery, trauma or burns
15. Multi-fetal gestation
16. Alcohol and substance use
17. Smoking or exposure to environmental tobacco smoke
18. Drug nutrient interactions
19. Short interpregnancy interval
20. History of low-birth-weight or large for gestational age infant
21. Fetal growth restriction
22. Pregnant woman currently breastfeeding
23. Pica (compulsively ingesting non-food items)
24. Eating disorders
25. Depression
26. Development, sensory, or motor disabilities interfering with ability to eat
27. Oral health condition
28. Consuming a diet very low in calories/nutrients; impaired intake/absorption
29. Inadequate vitamin/mineral supplementation
30. Consuming dietary supplements with potentially harmful consequences
31. Ingesting foods that could be contaminated with pathogenic organisms (*E. coli*, *Listeria monocytogenes*)
32. History of bariatric surgery
33. Lack of/inadequate prenatal care (care beginning after the 1st trimester)
34. Homelessness, migrancy, recipient of abuse, inability to make feeding decisions or prepare food
35. History of fetal or neonatal loss
36. History of birth with nutrition-related congenital or birth defect

Data from Nutritional Risks Criteria and Descriptions. California WIC Policy and Procedure Manual; Revised October 1, 2021: 1-33. Available at: https://www.cdph.ca.gov/Programs/CFH/DWICSN/CDPH%20Document%20Library/LocalAgencies/WPPM/960-01NutritionRiskCriteriaUSDA.pdf. Accessed May 1, 2023.

Anthropometric Measurement

As an assessment category, the anthropometric measure is perhaps the most important because of the relationship between prenatal weight gain and pregnancy outcome. Using the IOM weight gain guidelines, based on prepregnancy weight and multifetal pregnancy, allows the

establishment of weight gain goals. Recording prenatal weight gain on a growth grid to monitor weight gain is important in assuring both adequate total weight gain and appropriate rate of gain.[5]

Variations in weight gain goals (amount or rate) require further investigation to determine whether the cause is pathophysiologic or related to inadequate/excess dietary intake.

Biochemical Data: Laboratory Data

Blood volumes during pregnancy may increase by approximately 6% in the first trimester, 29% by the end of the second trimester, and 48% by term.[13] This expansion may affect blood-based biomarker concentrations. Since this relationship is complex, laboratory values should be interpreted carefully (**Table 2.8**).[13]

TABLE 2.8 Laboratory Values in Pregnancy

Constituent (Serum[a])	Normal Level/Range (Common Units)	
Albumin	1st trimester: 3.1–5.1 g/dL 2nd trimester: 2.6–4.5 g/dL 3rd trimester: 2.3–4.2 g/dL	
Calcium (total)	1st trimester: 8.8–10.6 mg/dL 2nd trimester: 8.2–9.0 mg/dL 3rd trimester: 8.2–9.7 mg/dL	
Creatinine (s/p)	1st trimester: 0.4–0.7 mg/dL 2nd trimester: 0.4–0.8 mg/dL 3rd trimester: 0.4–0.9 mg/dL	
Glucose (OGTT, 100-g test load to diagnose GDM) (s/p)	**Time (hr)**	**mg/dL**

Constituent (Serum[a])	Normal Level/Range (Common Units)	
–GDM diagnosed:	0	>105
2 abnormal values	1	190
	2	165
	3	145
Hematocrit (whole blood)	1st trimester: 31%–41% 2nd trimester: 30%–39% 3rd trimester: 28%–40%	
Hemoglobin (whole blood)	1st trimester: 11.6–13.9 g/dL 2nd trimester: 9.7–14.8 g/dL 3rd trimester: 9.5–15 g/dL	
White blood cell count	1st trimester: 5.7–13.6 × $10^3/mm^3$ 2nd trimester: 5.6–14.8 × $10^3/mm^3$ 3rd trimester: 5.9–16.9 × $10^3/mm^3$	
Lipids: cholesterol	1st trimester: 141–210 mg/dL 2nd trimester: 176–299 mg/dL 3rd trimester: 219–349 mg/dL	
Lipids		
Triglyceride	1st trimester: 40–159 mg/dL 2nd trimester: 75–382 mg/dL 3rd trimester: 131–453 mg/dL	
Osmolality	1st trimester: 275–280 mmol/kg 2nd trimester: 276–289 mmol/kg 3rd trimester: 278–280 mmol/kg	

Constituent (Serum[a])	Normal Level/Range (Common Units)
Thyroid Hormones	
Thyroxine total, T_4	1st trimester: 6.5–10.1 mcg/dL 2nd trimester: 7.5–10.3 mcg/dL 3rd trimester: 6.3–9.7 mcg/dL
TSH	1st trimester: 0.6–3.4 mIU/L 2nd trimester: 0.37–3.6 mIU/L 3rd trimester: 0.38–4.04 mIU/L
Thyroxin-binding globulin (TBG)	1st trimester: 1.8–3.2 mg/dL 2nd trimester: 2.8–4.0 mg/dL 3rd trimester: 2.6–4.2 mg/dL
Urea nitrogen	1st trimester: 7–12 mg/dL 2nd trimester: 3–13 mg/dL 3rd trimester: 3–11 mg/dL
Uric acid	1st trimester: 2.0–4.2 mg/dL 2nd trimester: 2.4–4.9 mg/dL 3rd trimester: 3.1–6.3 mg/dL

[a]Serum values, unless denoted as (p), plasma, or s/p, serum or plasma, or as noted. TSH, thyroid-stimulating hormone.
Data from F. Gary Cunningham, MD. Normal reference ranges for laboratory values in pregnancy. In: UpToDate, Post, TW (ed), UpToDate, Waltham, MA, 2023.

Patient History

The history component of nutrition assessment in pregnancy is a key in identifying underlying risk factors related to dietary intake, which may influence pregnancy outcome. The following relevant historical components provide descriptions of such factors.

Clinical History

A thorough clinical history aims to identify underlying pathologies as well as risks/indications for malnutrition. This includes asking about symptoms such as loss of appetite, unanticipated weight changes, poor wound healing, and poor dentition. It also includes history of chronic illnesses or infections, trauma, or eating disorders, as well as clinical/surgical illnesses, including any mental illness.[14]

Food and Nutrition History

The importance of nutritional intake during pregnancy, especially for specific nutrients, necessitates a thorough food and nutrition history as part of nutrition assessment. Potential nutrients of concern, because of their importance or increased need in pregnancy, include calcium, vitamin D, iron, zinc, magnesium, folate, vitamin B_6, and energy and protein. **Box 2.3** provides dietary intake details and lifestyle factors, which are a critical component of a complete food and nutrition history.[14] Pregnant women, unable to meet DRI recommendations, are routinely prescribed a prenatal vitamin and mineral supplement containing key nutrients (**Table 2.9**).

Box 2.3 Food and Nutrition History

I. Socioeconomic, Lifestyle, and Health Beliefs/Attitudes

1. Perceived nutrition/dietary conditions
2. Diet restrictions (e.g., vegan, food allergy/intolerance)
3. Economics: adequate resources/availability of food. Adequate housing and transportation.
4. Ethnic/cultural dietary practices
5. Home life/meal patterns
6. Appropriate physical activity

II. Physiologic Considerations

1. Nausea, vomiting, diarrhea

2. Constipation
3. Gastroesophageal reflux
4. Recent weight change

III. Nutritional Adequacy: Food Groups/Nutrients

1. Appetite
2. Multiple gestation
3. Dairy products (calcium, vitamins D and B_{12})
4. Fruits and vegetables (folate, vitamin C, fiber, magnesium)
5. Whole grains (B_6, folic acid, fiber)
6. Protein foods (iron, zinc, B_{12} B_6, iodine, choline)

IV. Medical Considerations

1. Chronic conditions
2. Medications
3. Oral/dental health

V. Contaminants/Other Substances

1. Fish (mercury/contaminants: check local health department for advisories)
2. Pica
3. Excessive caffeine (>2 cups coffee/day)
4. Alcohol
5. Smoking, including electronic nicotine delivery systems
6. Substance use disorder
7. Foodborne pathogens (*Listeria monocytogenes, Toxoplasma gondii*)

TABLE 2.9 Key Nutrients in a Prenatal Supplement

Vitamins	Amount	% DV[a]
A	4,000 IU	156
B_{1} (Thiamine)	1.84 mg	129
B_{2} (Riboflavin)	3 mg	215

Vitamins	Amount	% DV[a]
B_6	10 mg	527
B_{12}	12 µg	460
Folic acid	1 mg	170
Niacin	20 mg	111
C	120 mg	141
D	400 IU	150
E	22 mg	150
Calcium	200 mg	20
Iron	27 mg	100
Zinc	25 mg	227

[a]Daily values for pregnant and lactating women.
Data from (i) *Dietary Guidelines for Americans, 2020-2025*. U.S. Department of Agriculture and U.S. Department of Health and Human Services. 9th Edition. December 2020. Accessed April 25, 2023. https://www.dietaryguidelines.gov/sites/default/files/2021-03/Dietary_Guidelines_for_Americans-2020-2025.pdf, and (ii) Drugs.com, [Internet]. PNV Prenatal Plus Multivitamin Prescribing Information. Updated May 23, 2022. Accessed May 13, 2023. Available from https://www.drugs.com/pro/pnv-prenatal-plus-multivitamin.htm.

Medication and Supplement History

Approximately 90% of pregnant people take medication during pregnancy and about 70% take at least one prescription medicine.[15]

Many common medications, both prescription and over the counter, may have adverse effects during pregnancy, including birth defects, preterm birth, low birthweight, nausea, loss of appetite and constipation. Medication use

may also lead to nutrient deficiencies or drug-nutrient interactions.[14] **Box 2.4** provides a listing, although not comprehensive, of common medications that may increase the risk of birth defects.

Box 2.4 Medications Causing an Increased Risk of Birth Defects

Prescription Drugs	
ACE inhibitors	Captopril (Capoten®), Enalapril (Vasotec®)
Acne medications	Isotretinoin (Amnesteem®, Claravis®, Myorisan®, and Zenatane®)
Anticonvulsants	Carbamazepine (Carbatrol®, Epitol®, Equetro®, Tegretol®), valproic acid (Depacon®, Depakene®, Depakote®, Stavzor®, Valproic®)
Anticoagulants	Warfarin (Coumadin®, Jantoven®)
Opioids	Morphine (Avinza®, Kadian®, Duramorph®, MSContin®), codeine, hydrocodone (Vicodin®, Norco®), oxycodone (OxyContin®, Percocet®, Percodan®)
Antidepressants/mental health medications	Fluoxetine (Prozac®), Paroxetine (Paxil®), lithium
Others	Thalidomide (Thalomid®), Molnupiravir (Lagevrio™),

Data from Prescription medicine during pregnancy. March of Dimes. Reviewed July, 2022. Accessed May 5, 2023. https://www.marchofdimes.org/find-support/topics/pregnancy/prescription-medicine-during-pregnancy.

A detailed medication history should be obtained, and pregnant people should consult their healthcare provider to discuss the medicines they take, the dosage, when it will be taken during pregnancy and all current and preexisting health conditions.[15]

While medication use is common in pregnancy, it is also estimated that up to 20% of those using medications may also use herbal medicinal products adjunctively. This combination may increase the risk of herb–drug interactions and may potentially be harmful to the pregnant person or fetus.[16] A recent systematic review confirmed that these herb–drug interactions are under-studied and were not reported or investigated in most of the studies reviewed.[16] Additionally, the U.S. Food and Drug Administration does not require the same quality standards for herbal medicinal products that it does for medicines. Therefore, the authors concluded that herbal medicinal products should not be recommended during pregnancy until additional evidence of their safety is obtained.[16]

Another emerging concern in the United States is the increasing use of marijuana for medical and recreational use during pregnancy. Self-reported use ranges from 2% to 5% in many studies and may be as high as 15% to 28% when limited to populations that are socioeconomically disadvantaged or living in urban environments.[17] As the legalization of recreational marijuana increases, its use in pregnancy may likely increase, given that 34% to 60% of those using it continue to do so while pregnant.[17]

While current studies are limited by self-reporting of use, evidence suggests that marijuana use during pregnancy is associated with increased risk of low birth weight (< 2500 g), small for gestational age diagnosis, preterm delivery (<37 weeks), NICU admission and decreased mean birth weight (in grams), low Apgar score at 1 minute, and low infant head circumference (in centimeters).[17] For this reason, the American College of Obstetricians and

Gynecologists (ACOG) has issued recommendations stating that “Women who are pregnant or contemplating pregnancy should be encouraged to discontinue marijuana use.”[18] The American Academy of Pediatrics (AAP), the ACOG, and the American Society of Addiction Medicine recommends that those considering pregnancy, and pregnant people throughout their pregnancy, be screened routinely for marijuana and other drug use by using a validated screening tool. Abstinence counseling intervention is recommended for those using substances, and referral to treatment for those meeting the criteria for substance use disorder. Providers are encouraged to be aware of state-specific laws and educate their patients accordingly.[19]

Complications During Pregnancy

The most common nutrition-related complications during pregnancy include preexisting diabetes, gestational diabetes, hyperemesis gravidarum (HG), morning sickness, and pregnancy-induced hypertension. Such problems, either preexisting or arising during pregnancy, pose varying degrees of risk to maternal and fetal health.

Other factors such as a pregnant person's preconception health and nutritional status also influence the risk of miscarriage, prematurity, birth defects and fetal growth restriction.[20]

This emphasizes the importance of nutritional assessment regarding specific conditions to help ensure a positive pregnancy outcome.

Nausea and Vomiting of Pregnancy

Nausea and vomiting of pregnancy (NVP), also known as morning sickness, occurs in approximately 50% to 80% of all pregnancies and is not confined to only morning hours.[21] It generally begins prior to 9 weeks of pregnancy, and resolves by approximately 14 weeks gestation. Some pregnant people experience continued symptoms throughout pregnancy.[22] A severe form of NVP, hyperemesis gravidarum (HG), occurs in up to 3% of pregnancies and sometimes requires hospitalization.[22] Diagnosis of this condition occurs when there is a 5% loss of prepregnancy weight along with dehydration or loss of body fluids.[22] As HG can have significant impact on energy and nutrient intake, parenteral nutrition may become necessary in severe cases.

Approaches to nutrition therapy for NVP begin with dietary changes such as eating six small meals daily and choosing lower-fat foods. The use of ginger has been found to be an

effective non-pharmacological treatment for NVP and is readily available without a prescription.[21] **Box 2.5** provides Morning Sickness Nutrition Therapy recommendations.

Box 2.5 Morning Sickness Nutrition Therapy Recommendations

- Eat small, more frequent meals and snacks with added protein.
- Avoid an empty stomach.
- Drink fluids between meals instead of with meals.
- Consume adequate liquids throughout the day (8–12 cups).
- Avoid stimuli such as strong odors from food cooking. Try cold foods such as smoothies or frozen fruit.
- Avoid high-fat foods.
- Eat dry foods such as crackers or dry cereal in the morning before getting out of bed.
- Try sour foods such as vegetable juice or citrus fruit/juice.
- Avoid highly spiced foods if this exacerbates nausea.
- Take a prenatal vitamin.
- Try eating foods with ginger which has been shown to be beneficial in reducing nausea.

Data from (i) Academy of Nutrition and Dietetics. Nutrition Care Manual®. Morning Sickness Nutrition Therapy. https://www.nutritioncaremanual.org/client_ed.cfm?ncm_client_ed_id=49. Accessed May 18, 2023; and (ii) Morning Sickness: Nausea and Vomiting of Pregnancy FAQ126. The American College of Obstetricians and Gynecologists. Published: May 2020. Last reviewed: December 2021. Accessed May 18, 2023.

When dietary interventions are unsuccessful, medical treatment may be needed including certain medications such as pyridoxine (vitamin B_6), a combination of pyridoxine and doxylamine (diclegis and bonjesta), or diphenhydramine (benadryl).[23]

In severe cases of NVP, such as hyperemesis gravidarum, other antiemetics/medications, such as metoclopramide (reglan), promethazine (phenergan), prochlorperazine (compro), or ondansetron (zofran), may be prescribed.[23] The

decision to use these medications during pregnancy is based on whether the benefits outweigh the potential risk.[22]

Diabetes Mellitus

About 1% to 2% of pregnant people in the United States have type 1 or type 2 diabetes, and recent studies indicate that this rate is increasing. Between 2000 and 2010 the percentage of people with type 1 and type 2 diabetes before pregnancy increased 37%.[24] In that same time period, the percentage of pregnant people with gestational diabetes increased by 56%.[24] This form of diabetes currently affects 6% to 9% of pregnant people.[25] All forms of diabetes during pregnancy increase maternal and infant risk for adverse outcomes. These risks include preeclampsia, fetal macrosomia, shoulder dystocia, neonatalhypoglycemia, and cesarean section.[25] However, management of blood glucose levels via daily self-monitoring of blood glucose (SMBG), physical activity, nutrition therapy, and/or medications significantly reduces these risks and provides improved birth outcomes.[24]

ADIME 2.1 ADIME At-A-Glance: Nausea and Vomiting of Pregnancy

Assessment

- Prepregnancy BMI and weight, weight loss
- Frequency/Severity of nausea and vomiting
- Use of herbal supplements for nausea, adherence to prescribed prenatal vitamin
- Dietary intake, meal pattern (eating one large meal per day in the evening when foods are better tolerated)

Nutrition Diagnosis

- Inadequate oral food and beverage intake related to nausea and vomiting of pregnancy, AEB weight loss of 2 lb in 9 weeks
- Inadequate vitamin/mineral intake related to nausea and vomiting of pregnancy, AEB inability to consume prescribed nutrient supplement
- Increased nutrient needs related to pregnancy, AEB diet history of low intake and weight loss
- Harmful nutrition practices related to taking over-the-counter (OTC) herbal supplement, AEB diet history of unsafe dietary supplements

Intervention

- Nutrition counseling for small, frequent meals with adequate protein and avoidance of high-fat foods
- Nutrition counseling for safe alternatives to OTC herbal supplement (i.e., ginger, B_6, or prescribed medication)
- Nutrition counseling for adequate fluid intake between meals vs. with meals
- Suggest temporary alternatives to prenatal vitamins such as a chewable/liquid children’s vitamin

Monitoring and Evaluation

- Body weight
- PO intake (food, fluid)
- Vitamin/mineral supplement tolerance/usage
- Status of nausea and vomiting
- Usage of non-FDA-approved herbal supplements

Gestational Diabetes

Gestational diabetes mellitus (GDM) is carbohydrate intolerance developing during pregnancy. If the condition is managed with nutrition therapy alone and without medication, it is termed as class A1GDM. Gestational diabetes managed with medication to achieve glycemic control is termed as class A2GDM.[26] The U.S. Preventive Services Task Force (USPSTF) has recommended screening for gestational diabetes in asymptomatic pregnant persons at 24 weeks gestation or after. Those with late entry into prenatal care (after 28 weeks of gestation) should be screened as soon as possible.[25] There are two options for GDM diagnostic criteria: A "one-step" strategy using a 2-hour, 75-g oral glucose tolerance test (OGTT), which is the consensus of the International Association of Diabetes and Pregnancy Study Groups (IADPSG); or a "two-step" approach consisting of a 1-hour 50-g (nonfasting) screening with a subsequent 3-hour 100-g OGTT for those with a positive screen, which is the consensus of the National Institutes of Health (NIH). This method is widely accepted and is used by 95% of obstetricians in the United States (**Table 2.10**).[26] Screening for GDM may be performed prior to 24 weeks if a pregnant person presents with risk factors for type 2 diabetes, including obesity, family history of type 2 diabetes, or experiencing fetal macrosomia during a prior pregnancy.[25] Additional risk factors for gestational diabetes may be found in **Box 2.6**.

TABLE 2.10 Detection and Diagnosis of Gestational Diabetes

One-Step Strategy (IADPSG Consensus)
• Perform a 75-g OGTT, with plasma glucose measurement at fasting and at 1 and 2 hr, at 24–28 wk of gestation in women not previously diagnosed

One-Step Strategy (IADPSG Consensus)

with diabetes.

- The OGTT should be performed in the morning after an overnight fast of at least 8 hr.
- The diagnosis of GDM is made when any of the following plasma glucose values are met or exceeded:
  - Fasting: 92 mg/dL (5.1 mmol/L)
  - 1 hr: 180 mg/dL (10.0 mmol/L)
  - 2 hr: 153 mg/dL (8.5 mmol/L)

Two-Step Strategy (NIH Consensus)

- Step 1: Perform a 50-g glucose load test (GLT) (nonfasting), with plasma glucose measurement at 1 hr, at 24–28 wk of gestation in women not previously diagnosed with diabetes. If the plasma glucose level measured 1 hr after the load is >140 mg/dL (7.8 mmol/L), proceed to 100-g OGTT.
- Step 2: The 100-g OGTT should be performed when the patient is fasting.
- The diagnosis of GDM is made when at least two of the following four plasma glucose levels (measured fasting, 1, 2, 3 hr after the OGTT) are met or exceeded:

	Carpenter/Coustan	NDDG
Fasting	95 mg/L (5.3 mmol/L)	105 mg/dL (5.8 mmol/L)
1 hr	180 mg/dL (10.0 mmol/L)	190 mg/dL (10.6 mmol/L)
2 hr	155 mg/dL (8.6 mmol/L)	165 mg/dL (9.2 mmol/L)
3 hr	140 mg/dL (7.8 mmol/L)	145 mg/dL (8.0 mmol/L)

Data from (i) Jin J. Screening for Gestational Diabetes. *JAMA*. 2021;326(6):577. doi:10.1001/jama.2021.11187 and (ii) ACOG Practice Bulletin No. 190: Gestational Diabetes Mellitus. *Obstet Gynecol*. 2018;131(2):e49–e64. doi:10.1097/AOG.0000000000002501.

Box 2.6 Risk Factors for Gestational Diabetes

1. Obese/overweight and not physically active.
2. Family history of type 2 diabetes.
3. Older than 25 years of age.
4. Previous pregnancy history of:
    - Gestational diabetes
    - Giving birth to a baby weighing more than 9 lb
5. Presence of polycystic ovary syndrome (PCOS) or another health condition linked to problems with insulin?
6. Diagnosed with prediabetes.
7. Have high blood pressure or heart disease.
8. Being a member of a racial or ethnic group that has a higher prevalence of diabetes that isn't entirely explained by race or ethnicity, such as Black, American Indian or Alaska Native, Asian, Hispanic/Latino or Pacific Islander. However, being a person of color is not a cause for having gestational diabetes.

Data from Gestational Diabetes. March of Dimes. Last reviewed March, 2022. Accessed May 21, 2023. https://www.marchofdimes.org/find-support/topics/pregnancy/gestational-diabetes.

Pregnant persons diagnosed with GDM should receive medical nutrition therapy (MNT) with a registered dietitian nutritionist (RDN) at diagnosis. The RDN should complete a nutrition assessment then monitor blood glucose and food records, weight changes, physical activity, and medication therapies at each follow-up visit. Since GDM is a risk factor for development of type 2 diabetes, a post-delivery encounter should focus on lifestyle changes, weight management and increased physical activity if appropriate.[27] In an effort to standardize nutrition therapy for GDM, the Academy of Nutrition and Dietetics developed evidence-based nutrition practice guidelines.[27] Three key components include SMBG with medical nutrition therapy (MNT), diet modifications including adjustment of carbohydrate intake, and physical activity. In addition, they

developed guidelines for nutrition monitoring and evaluation (**Box 2.7**).

Box 2.7 Recommended MNT Guidelines for GDM

Screening/Referral

1. Screen for GDM risk.
2. Most women with low risk screened using OGTT between 24 and 28 wk gestation.
3. For women with average to high risk, screen for undiagnosed type 2 diabetes at first prenatal visit. Monitor nutritional intake, weight gain, and physical activity. Risk increases with obesity and excessive weight gain prior to pregnancy.
4. Provide first MNT encounter within 1 week of initial referral and provide a minimum of three visits. Remote/virtual encounters may be an option. Schedule a visit after delivery to discuss postpartum lifestyle modifications.

Nutrition Assessment

1. Food/beverage/nutrient intake (i.e., total carbohydrate and energy intake, dietary patterns, food preferences, appetite, allergies/intolerances, access to food, cultural/religious considerations, food safety).
2. Physical activity (consistency, frequency).
3. Pharmacologic therapy (insulin or oral glucose-lowering agent).
4. Substance use and use of dietary supplements.
5. Knowledge, beliefs, and attitudes (willingness and ability to make lifestyle changes).
6. Assessment of biochemical data, medical tests and procedures (glycemic tests, SMBG, ultrasounds, nutrient levels, thyroid/kidney function, urine ketones if necessary).
7. Patient/Family/Client medical health history.
8. Psychosocial/economic issues (social supports, procurement of safe food).

Nutrition Intervention

1. Encourage adequate caloric intake to promote fetal and maternal health, achieve glycemic goals and promote appropriate weight gain.
2. Individualize amount and type of carbohydrates based on nutritional assessment, treatment goals, blood glucose response, and patient needs.
3. Consume adequate amounts of protein and fat based on DRIs.

4. Prevent nutritional deficiencies by recommending a multivitamin/mineral supplement for women whose intake does not meet the DRIs.
5. Perform physical activity daily for at least 30 minutes unless contraindicated.
6. Perform daily self blood glucose monitoring (SBGM). Initially test 4 times/day: fasting and 1–2 hr after each meal. Thereafter, adjust frequency to meet target goals: ≤95 mg/dL fasting, ≤140 mg/dL 1 hr postprandial and ≤120 mg/dL 2 hr post prandial.
7. Use only FDA-approved nonnutritive sweeteners in moderation.
8. Avoid consuming alcohol, even in cooking.

Nutrition Monitoring and Evaluation

1. Monitor blood glucose levels, change in weight, food/nutrient intake, and physical activity.
2. Review need for/use of pharmacologic therapy (insulin) if MNT has been unsuccessful.
3. Review ketone testing results (if previously recommended or women with weight loss or inadequate carbohydrate/calorie intake).

MNT, medical nutrition therapy.

Data from Academy of Nutrition and Dietetics. Nutrition Care Manual®. Gestational Diabetes (2018). https://www.nutritioncaremanual.org/topic.cfm?ncm_category_id=1&lv1=5517&lv2=18188&lv3=273823&ncm_toc_id=273823&ncm_heading=Nutrition%20Care. Accessed May 20, 2023.

Specific dietary measures are outlined in **Box 2.8**, and if this fails to control blood glucose levels, insulin is the preferred treatment for diabetes in pregnancy. Oral antidiabetic medications (e.g., glyburide and metformin) have not been approved by the FDA for the treatment of GDM and may not be safe during pregnancy.[26]

Box 2.8 Specific Dietary Measures for GDM

- Recommend a minimum of 175 g carbohydrate, 71 g protein (or 1.1 g/kg/day) and 28 g of fiber daily.
- Space carbohydrate evenly throughout the day.
- Eat several small meals and snacks versus large meals without snacks.

Data from Academy of Nutrition and Dietetics. Nutrition Care Manual®. Gestational Diabetes (2018). https://www.nutritioncaremanual.org/topic.cfm?

ncm_category_id=1&lv1=5517&lv2=18188&lv3=273823&ncm_toc_id=273823&ncm_heading=Nutrition%20Care. Accessed May 20, 2023.

Preexisting Diabetes

Pregnancy requires heightened vigilance with regard to glycemic control, so those who enter pregnancy with diabetes may need to increase the frequency of SMBG and adjust insulin dose. Research suggests that insulin requirements differ between those with type 1 and type 2 diabetes throughout pregnancy. In type 1 diabetes, insulin requirements (IRs) generally decrease in the first trimester (3.7%) and late third trimester (4.1%) with an approximate 20% increase in both the second and third trimesters. However, pregnant people with type 2 diabetes require a higher dose of insulin from the beginning to the end of each trimester with this change progressively increasing as gestation advances. Also, the decrease in insulin requirements noted in the beginning and end of pregnancy for those with type 1 diabetes was not seen in those with type 2 diabetes.[28] A person with preexisting type 2 diabetes who previously managed glycemic control without insulin may require insulin. Those using oral hypoglycemic agents to control diabetes before pregnancy will likely require insulin because of a lack of consensus on their use and potential complications. The American Diabetes Association (ADA) recommends target ranges for pregnant people with type 1 and type 2 diabetes as follows: fasting glucose 70–95 mg/dL, 1-hour postprandial glucose 110–140 mg/dL and 2-hour postprandial glucose 100–120 mg/dL. The lower limits are based on the mean normal blood glucose in pregnancy and do not apply to those with type 2 diabetes treated solely with diet.[29]

ADIME 2.2 ADIME At-A-Glance: Gestational Diabetes

Assessment

- Prepregnancy BMI and weight, current weight
- OGTT results
- Previous history of GDM or large-for-gestational age deliveries
- Access to transportation, social support
- Dietary intake, physical activity

Nutrition Diagnosis

- Excessive oral food/beverage intake related to nutrition knowledge deficit and inactivity, AEB prepregnancy BMI of 30, and weight gain of 20 lb at 28 weeks gestation
- Excessive carbohydrate intake related to knowledge deficit and lack of social support, AEB 1-hr nonfasting OGTT result of 150 mg/dL
- Limited access to food related to lack of transportation and social support, AEB consumption of high-calorie convenience/fast foods
- Physical inactivity related to lack of safe environment, AEB reported sedentary lifestyle

Intervention

- Provide nutrition counseling for IOM weight gain recommendations.
- Provide nutrition counseling for carbohydrate-controlled diet (at least 175 g/day).
- Ensure consumption of multivitamin/mineral supplement to prevent nutritional deficiencies.
- Counsel regarding indoor exercise for 30 minutes daily with physician approval.

- Refer to WIC and Maternal Infant Health Programs for assistance with food availability and transportation.
- Refer to Certified Diabetes Educator for self-blood glucose monitoring instruction.

Monitoring and Evaluation

- Body weight
- PO intake
- SBGM diaries (BGL—fasting and postprandial)
- Exercise logs
- Verification of enrollment in social support programs
- Attendance at prenatal appointments

Preeclampsia (Pregnancy-Induced Hypertension)

Preeclampsia is a form of hypertension that arises during pregnancy, typically in the 3rd trimester, and complicates approximately 3%–5% of pregnancies.[30] The term is derived from the potential culminating event of eclampsia, or convulsions, which represents an obstetric emergency. Preeclampsia poses significant risks for the pregnant person and infant. In addition to a spike in blood pressure, (systolic blood pressure of 140 mm Hg or more or diastolic of 90 mm Hg, or more, on two occasions ≥4 hours apart), a person with preeclampsia may also exhibit proteinuria 300 mg or more per 24-hour urine collection), usually after the 20th week of gestation. If proteinuria is not detected, additional diagnostic criteria also include new-onset hypertension with new onset of: thrombocytopenia, renal insufficiency, impaired liver function, pulmonary edema and headache unresponsive to medication.[31]

The condition is thought to result from impaired placental perfusion and subsequent oxidative stress in the presence of systemic inflammation and other maternal risk factors.[32] Early identification of preeclampsia and careful management are essential to prevent adverse outcomes. There are several risk factors for preeclampsia to consider early in pregnancy, several of which are moderated by parity status and preexisting conditions (**Box 2.9**).

Box 2.9 Risk Factors for Preeclampsia

High Risk	Moderate Risk
Preeclampsia in a prior pregnancy	First pregnancy (nulliparity)
Renal disease	Age 35 years or older
Autoimmune disease such as systemic lupus erythematosus or antiphospholipid antibody syndrome	Pregnancy interval of more than 10 years
Pregestational diabetes	BMI of ≥30 kg/m^2 at first visit
Chronic hypertension	Family history of preeclampsia
Multifetal gestation	Low socioeconomic status

Data from Wheeler SM, Myers SO, Swamy GK, Myers ER. Estimated Prevalence of Risk Factors for Preeclampsia Among Individuals Giving Birth in the US in 2019. *JAMA Netw Open*. 2022;5(1):e2142343. doi:10.1001/jamanetworkopen.2021.42343.

In the past, sodium restrictions were standard practice. However, there is insufficient evidence to determine the effectiveness of sodium restrictions or supplements such as vitamins C, D, and E, folic acid, fish oil, and garlic in reducing the risk of preeclampsia.[31]

Since risk factors for preeclampsia include high BMI, hypertension and pregestational diabetes, a healthy balanced diet could help reduce the risk of preeclampsia.[30] The World Health Organization (WHO) recommends a diet rich in fruits and vegetables (>400 g/day), whole grains, legumes, nuts, olive oil, consumption of 8 oz of seafood per week and limiting intake of red and processed meats, and foods high in fat, salt, and sugar including sugar sweetened beverages.[30] The Dietary Guidelines for Americans also provides a healthy U.S.-style dietary pattern for those who are pregnant or lactating.[9]

Pharmacologic management of preeclampsia involves the use of low-dose aspirin (prior to 12 weeks gestation until 36 weeks) to reduce the risk of developing preeclampsia in high-risk women. Oral antihypertensive medications such as labetalol or methyldopa are commonly used to treat pregnancy hypertension. Delivery of the fetus is the only known cure for preeclampsia, although people with chronic hypertension should continue with their recommended treatment postnatally.[32]

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CHAPTER 3

Assessing Pediatric Patients

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The nutrition assessment of pediatric patients is unique in many ways. Nutrition screening is most useful in identifying high-risk infants, children, and adolescents in all settings of health care. **Box 3.1** lists some "red flags" that warrant nutrition intervention in inpatient units, outpatient clinics, or community settings. **Box 3.2** lists some common classifications used when assessing infants.

Box 3.1 Indicators of Need for Nutrition Assessment

Infants/NICU	Toddler/Child/Adolescent
≤37 weeks gestational age	Weight for length <10th percentile or BMI <10th percentile
Very low birth weight	Weight for length >95th percentile or BMI >95th percentile
Insufficient weight gain	Significant weight loss or gain
Nonstandard formula	Enteral or parenteral feeding
Formula concentrated to nonstandard dilution	Special diet
Food allergies/intolerances	Food allergies/intolerances

Infants/NICU	Toddler/Child/Adolescent
Poor/inappropriate intake	Eating disorder
Malnutrition or at risk for malnutrition owing to diagnosis, comorbidities, social or economic status, etc.	Malnutrition or at risk for malnutrition owing to diagnosis, comorbidities, social or economic status, etc.
Inborn errors of metabolism	Omitting entire food groups for religious reasoning
Homemade formula	Adolescent pregnancy
Donor breastmilk	Homemade formula/homemade blenderized tube feeding

Box 3.2 Infant Classification and Associated Terms

Classification	Parameters
Chronological or birth age	Time since birth (days, weeks, months)
Gestational age	Estimated time since conception or postconceptional age
Corrected age	Age adjusted for prematurity
Preterm infant	<37 weeks gestation
Full-term infant	37 to 42 weeks gestation
Post-term infant	>42 weeks gestation
Low birth weight	<2,500 g

Classification	Parameters
Very low birth weight	<1,500 g
Extremely low birth weight	<1,000 g
Small for gestational age	Weight <10th percentile
Appropriate for gestational age	Weight ≥10th percentile and ≤90th percentile
Large for gestational age	Weight >90th percentile

Data from The Academy of Nutrition and Dietetics. Nutrition Care Manual. Preterm and High-Risk Infants: Anthropometric Measurements. https://www.nutritioncaremanual.org. Accessed May 14, 2023.

Anthropometric Assessment

Measurement of growth is essential for assessing the health and nutritional status of the pediatric patient. Serial measurements are the best measures of growth and are more conclusive than isolated measurements. Below are the recommended anthropometric measurements to use based on age:

- **0–24 months**
  - Body weight
  - Recumbent length
  - Head circumference
  - Weight for length
- **2–18 years**
  - Body weight
  - Height
  - Waist circumference
  - Body mass index (BMI)

Weight

Weight should be obtained using consistency in technique and scales. Infants should be nude and without a diaper. Children and adolescent patients should wear minimal clothing. Weight should be obtained to the nearest 0.1 kg.

Length

Length is measured from crown to heel. Recumbent length should be measured by use of a length board in infants aged birth to 24 months. Two people may be needed for this

process. Using a perpendicular stadiometer, standing height or stature should be measured in children aged 24 months and older who can stand. Measurement should be taken to the nearest 0.5 cm. Various other types of measurements and equations have been developed for use in condition-specific populations such as cerebral palsy, spina bifida, etc. which may be used at more specialized pediatric facilities (**Table 3.1**).

TABLE 3.1 Knee/Heel Measurement Equations

Stevenson method	Height = (2.69 × knee height) + 24.2 *Based on data obtained from children under 12 years of age with cerebral palsy*
Chumlea method	Male: Height = 64.19 − (0.04 × age) + (2.02 × knee height) Female: Height = 84.88 − (0.24 × age) + 1.83 × knee height) *Equations derived from recumbent knee height in the elderly*

Data from Delgado NB, Hastings E, Kommes K, Ling H, Spoede E, Wrobel M. *Texas Children's Hospital Pediatric Nutrition Reference Guide*, 13th edition. Texas Children's Hospital, 2022.

Head Circumference

Head circumference is measured at the largest frontal–occipital plate. Head circumference is a useful tool until about 3 years of age when head growth slows dramatically. Measurement should be taken to the nearest 0.5 cm.

Body Mass Index

The BMI for children (starting at age 2) and adolescents is unique, and using the adult BMI categories to interpret the value is not appropriate. BMI-for-age must be interpreted

using the growth charts to compare age- and sex-specific percentiles (see “Relevant Websites Relating to Growth Charts” later in this section.)

Waist Circumference

Waist circumference is measured midway between the top of the iliac crest and the lowermost portion of the rib cage. A non-stretchable measuring tape should be used. This measurement is most accurate if the patient has not recently consumed a large meal and is demonstrating normal expiration (not “sucking in”).

Mid-Upper Arm Circumference

Mid-upper arm circumference (MUAC) is a valuable tool in the serial assessment of pediatric patients since, unlike other indicators, it can be used as an independent anthropometric assessment tool because normal values change little during early years. MUAC is especially helpful in children without generalized edema but whose acute or chronic conditions affect their weight measurements due to ascites or genetic factors. Measurements can be compared with the standards developed by the Center for Disease Control for ages 2 months–18 years or the World Health Organization (WHO) validated in ages 5–19 years. Additionally, serial measurements can be used to monitor changes using the patient as his or her own control. The WHO standards can be found at https://www.who.int/tools/child-growth-standards/standards/arm-circumference-for-age

Nutrition-Focused Physical Examination

Similar to the adult patient population, pediatric practitioners should conduct a nutrition-focused physical

exam in addition to using data from the physical examination assessments conducted by other healthcare practitioners. Fat and muscle wasting may also be seen in infants, children, and adolescents but care must also be taken to assess patients appropriately for their age as well as underdeveloped patients. Pediatric patients with chronic rather than acute malnutrition often appear older than their actual age if they have wasted or underdeveloped fat mass as normal body build changes dramatically during infancy, childhood and adolescence.

Evaluation of Growth

Measurements should be plotted on growth charts and trends monitored. In 2010, the Centers for Disease Control and Prevention (CDC) adopted the WHO growth charts for children of fewer than 24 months. The WHO growth charts are based on an international sample of healthy children living under optimal health conditions to support growth (single births, breastfed, nonsmoking environment, etc.). Therefore, the WHO growth charts should be considered standard curves (or prescriptive) rather than reference (or descriptive) as were the CDC 2000 curves. The appropriate chart for age and gender should be used. In addition to standard growth charts, there are specialty growth charts for some select conditions. **Tables 3.2** and **3.3** list interpretations of percentiles on the growth charts.

TABLE 3.2 Interpretation of Height-for-Age and Weight-for-Age Plotted on Standard CDC Growth Charts

Percentile	Interpretation[a]
50th	Average for age
10th–90th	Healthy for most pediatric patients
3rd–10th or 90th–97th	Further investigation needed
<3rd or >97th	Unhealthy until proven otherwise

[a]If the patient plots higher or lower than expected potential, or if there are large changes in measurements, pathologic or nutritional factors should be

Percentile	Interpretation[a]
considered. Further assessment may include consideration of parental height, growth velocity, bone age, pubertal status, and development.	

TABLE 3.3 Interpretation of BMI-for-Age Plotted on Standard CDC Growth Charts

Classification	Body Mass Index-for-Age Percentile
Underweight	< 5th percentile
Healthy weight	5th to 84th percentile
Overweight	85th to < 95th percentile
Obesity or class I obesity	BMI ≥ 95th percentile to < 120% of the 95th percentile
Class II obesity	BMI ≥ 120% to < 140% of the 95th percentile, or BMI ≥ 35 kg/m^2
Class III obesity	BMI ≥ 140% of the 95th percentile, or BMI ≥ 40 kg/m^2

Data from The Academy of Nutrition and Dietetics. Nutrition Care Manual. Weight Management: Nutrition Assessment: Anthropometric Measurements. Accessed May 14, 2023.

If using a standard growth chart for a preterm infant, postnatal age (age of infant calculated, in weeks, from the date of birth) needs to be corrected for gestational age (age of infant calculated, in weeks, from the date of conception, determined by ultrasound examination) until at least 24 months of age.[1] **Box 3.3** gives an example of calculating gestation-adjusted age.

Box 3.3 Gestation-Adjusted Age for Preterm Infants

Adjustment for Prematurity = 40 weeks (full term) − gestational age at birth (in weeks)
Gestation-Adjusted Age = chronological age − adjustment for prematurity
Example: Michael was born on March 1, 2023. His gestational age at birth was determined to be 30 weeks based on ultrasound examination. At the time of his admission into the hospital on May 24, 2023, his chronological age was 12 weeks. Based on the previous equations, what is his gestation-adjusted age?

Adjustment for Prematurity = 40 weeks − 30 weeks = 10 weeks
Gestation-Adjusted Age = 12 weeks − 10 weeks = 2 weeks
If this were plotted on a standard WHO growth chart, anthropometric measurements would be plotted for a 2-week-old infant.

As the mode of feeding (breastfed vs. formula-fed) influences trends, it is important to note that formula-fed infants will plot differently on the WHO charts. Whereas breastfed infants have a higher growth velocity in the first 3 months of life, more formula-fed infants may be identified as low weight-for-age during this time. After this time, there is a slower rise in weight, and so fewer 6- to 23-month-old children may be identified as low weight. Differences in length for age are small, and so significant clinical differences are not expected.[2]

Relevant Websites Relating to Growth Charts

- Standard WHO growth charts for infants and children under the age of 2 and CDC growth charts (including BMI-for-age) can be accessed at http://www.cdc.gov/growthcharts.
- The WHO multicultural growth charts for ages 0 to 5 years can be accessed at http://www.who.int/childgrowth/standards/en/.
- Premature infant growth charts can be found at the following website: University of Calgary, Fenton Preterm

Growth Charts. Available at https://www.ucalgary.ca/resource/preterm-growth-chart/preterm-growth-chart

- Growth chart for children with cerebral palsy can be accessed at http://www.lifeexpectancy.org/Articles/NewGrowthCharts.shtml.
- Growth chart for children with Down syndrome can be accessed at https://www.cdc.gov/ncbddd/birthdefects/downsyndrome/growth-charts.html.

Assessment of Weight Changes

Newborn weight loss is expected after birth secondary to body composition shifts and total body water decrease. Normal weight loss is 7 to 10% of birth weight in term infants. A preterm infant may lose ≤15% of birth weight. An infant born at extremely low birth weight may lose ≤20% of birth weight. Term and preterm infant birth weight should be regained by the second week of life. Weight typically doubles by 5 to 6 months of age and triples by 12 months. Weight is typically affected during acute periods of undernutrition and will be examined later in this chapter.[3]

Usual Body Weight

Usual body weight (UBW) is useful in assessing weight status:

$$\% \text{ UBW} = (\text{actual weight}/\text{usual weight}) \times 100$$

$$\% \text{ Weight change} = [(\text{actual} - \text{usual weight})/\text{usual weight}] \times 100$$

Significant weight loss:

- >2% in 1 week
- >5% in 1 month
- >7.5% in 3 months
- >10% in 6 months

Growth Velocity

Height or weight velocity measures the change in measurement over time. Alterations in normal growth velocity indicate a need for further investigation.

$$\text{Velocity} = \frac{\begin{array}{c}\text{height (cm) or weight (g) at Time 2} - \\ \text{height (cm) or weight (g) at Time 1}\end{array}}{\text{Time 2 (month or day)} - \text{Time 1 (month or day)}}$$

Example: At her 4-month well-baby visit, Stephanie weighed 5.8 kg and was 60 cm long. At her 6-month well-baby visit, she weighed 6.5 kg and was 64 cm long. Based on this data, what is Stephanie's weight and height growth velocity?

Weight velocity in grams per day =

$$\frac{6{,}500\text{ g} - 5{,}800\text{ g}}{180\text{ days old (6 months)} - 120\text{ days old (4 months)}}$$

$$= \frac{700\text{ g}}{60\text{ days old}}$$

$$= 11.67\text{ g/day}$$

Height velocity in centimeters per week

$$= \frac{63.5\text{ cm} - 60\text{ cm}}{24\text{ weeks} - 16\text{ weeks}}$$

$$= \frac{3.5\text{ cm}}{8\text{ weeks}}$$

$$= 0.44\text{ cm/week}$$

According to **Table 3.4**, both weight and height growth velocity are within the ideal range of average growth between 4 and 8 months of age. **Table 3.5** lists normal growth rates for toddlers aged 1–3 years.

TABLE 3.4 Average Normal Growth Velocity for Children Aged 0–24 months

Age	Weight (g/day)	Length (cm/wk)
Preemie <2 kg	15–20 g/kg/day	0.8–1.1
Preemie >2 kg	20–30	0.8–1.1
<4 mo	23–34	0.8–0.93
4–8 mo	10–16	0.37–0.47
8–12 mo	6–11	0.28–0.37
12–16 mo	5–9	0.24–0.33
16–20 mo	4–9	0.21–0.29
20–24 mo	4–9	0.19–0.26

Data from (i) Delgado NB, Hastings E, Kommes K, Ling H, Spoede E, Wrobel M. *Texas Children's Hospital Pediatric Nutrition Reference Guide*, 13th edition. Texas Children's Hospital, 2022 and (ii) World Health Organization, Department of Nutrition for Health and Development. WHO Child Growth Chart Standards. Growth velocity based on weight, length, and head circumference. World Health Organization, 2009. Gevea Switzerland. ISBN 978 92 4 154763 5.

TABLE 3.5 Average Growth Rates Per Year for Toddlers Aged 1–3

	Age 12–23 Months	
Growth Parameter (per year)	**Girls**	**Boys**
Average weight gain (kg)	2.4	2.3
Average height gain (cm)	12.3	12
Average head circumference gain (cm)	2.3	2.2
	Age 2–3 Years	
Growth Parameter (per year)	**Girls**	**Boys**
Average weight gain (kg)	1.8	1.6
Average height gain (cm)	8.9	8.5
Average head circumference gain (cm)	1.09	0.96

Data from The Academy of Nutrition and Dietetics. Nutrition Care Manual. Normal Nutrition: Toddlers; Anthropometric Measurements.

Estimating Nutrient Needs

Fluid Needs

Fluid needs may be estimated using the formula in **Table 3.6**, or by using body surface area.[4]

$$\text{Fluid needs} = 1{,}500 \text{ mL fluid/m}^2\text{/day}$$

TABLE 3.6 Baseline Fluid Requirements

Weight (kg)	Fluid Needs
1–10	100 mL/kg
11–20	1,000 mL + 50 mL/kg for each kg >10 kg
>20	1,500 mL + 20 mL/kg for each kg >20 kg

Data from (i) Anderson CC, Kapoor S, Mark TE. *The Harriet Lane Handbook, by John Hopkins Hospital*, 23rd ed., Elsevier, 2024, and (ii) Somers MJ. Maintenance intravenous fluid therapy in children. UpToDate. Updated: August 30, 2021. Reviewed: May 2023. Accessed: April 15, 2023. https://www.uptodate.com.

Example: John weighs 15 kg. Using the equation in **Table 3.7**, what is his body surface area in m^2?

$$\text{Body surface area } (m^2) = (15 \text{ kg} \times 0.03) + 0.2$$

$$= 0.45 + 0.2 = 0.65 \text{ m}^2$$

TABLE 3.7 Calculating Body Surface Area (m^2)

Weight (kg)	Body Surface Area (m^2)
<5	kg × 0.05 + 0.05
5–10	kg × 0.04 + 0.1
10–20	kg × 0.03 + 0.2
20–40	kg × 0.02 + 0.4
>40	kg × 0.01 + 0.8

Based on the calculated body surface area, what are John's fluid requirements?

$$\text{Fluid needs} = 1{,}500 \text{ mL} \times 0.65 \text{ m}^2 = 975 \text{ mL/day}$$

Energy and Protein Needs

In the pediatric population, as in the adult, a variety of methods are used to estimate energy and protein needs. **Tables 3.8** and **3.9** provide commonly used methods. These methods are based on values determined for the pediatric population who display normal body composition, metabolism, and activity level.

TABLE 3.8 Estimated Energy and Protein Requirements

Category	Age	Reference Weight (kg)	DRI kcal/kg	Pro g/kg
Infants (mo)	0–2	N/A	—	1.52
	2–3	6	102	1.52
	4–6	6	82	1.5
	7–12	9	80	1.2
	13–35	12	82	1.08
Boys (yr)	3	12	85	1.08
	4–5	20	70	0.95
	6–7	20	64	0.95
	8	20	59	0.95
	9–11	36	49	0.94
	12–13	36	44	0.94
	14–16	61	39	0.85
	17–18	61	37	0.85
	>18	70	36	0.8
Girls (yr)	3	12	82	1.08
	4–5	20	65	0.95
	6–7	20	61	0.95
	8	20	59	0.95
	9–11	37	42	0.92
	12–13	37	40	0.92
	14–16	54	33	0.85
	17–18	54	31	0.85
	>18	57	34	0.8

Data from (i) Delgado NB, Hastings E, Kommes K, Ling H, Spoede E, Wrobel M. *Texas Children's Hospital Pediatric Nutrition Reference Guide*, 13th edition. Texas Children's Hospital, 2022 and (ii) Otten J, Pitzi H, Meyers LD. *Dietary Reference Intakes: The Essential Guide to Nutrition Requirements*. Washington, DC: National Academies Press, 2006.

Description

TABLE 3.9 Dietary Reference Intakes of Estimated Energy Requirement (EER)

Sex and Age	Calculation of Estimated Energy Requirements	Physical Activity (PA) Coefficient Based on Physical Activity Level (PAL)
All infants and toddlers		
0–3 mo	(89 × wt [kg] − 100) + 175	N/A
4–6 mo	(89 × wt [kg] − 100) + 56	N/A
7–12 mo	(89 × wt [kg] − 100) + 22	N/A
13–35 mo	(89 × wt [kg] − 100) + 20	N/A
Boys 3–8 years	EER = 88.5 − 61.9 × age [y] + PA × (26.7 × wt [kg] + 903 × ht [m]) + 20	PA = 1.0 if PAL is estimated to be ≥1 <1.4 (sedentary) PA = 1.13 if PAL is estimated to be ≥1.4 <1.6 (low active)
Boys 9–18 years	EER = 88.5 − 61.9 × age [y] + PA × (26.7 × wt [kg] + 903 × ht [m]) + 25	PA = 1.26 if PAL is estimated to be ≥1.6 <1.9 (active) PA = 1.42 if PAL is estimated to be ≥1.9 <2.5 (very active)
Girls 3–8 years	EER = 135.3 − 30.8 × age [y] + PA × (10 × wt [kg] + 934 × ht [m]) + 20	PA = 1.0 if PAL is estimated to be ≥1 <1.4 (sedentary)
Girls 9–18 years	EER = 135.3 − 30.8 × age [y] + PA × (10 × wt [kg] + 934 × ht [m]) + 25	PA = 1.16 if PAL is estimated to be ≥1.4 <1.6 (low active) PA = 1.31 if PAL is estimated to be ≥1.6 <1.9 (active) PA = 1.56 if PAL is estimated to be ≥1.9 <2.5 (very active)

Data from Butte NF. Estimation of dietary energy requirements in children and adolescents. UpToDate. Updated October 18, 2022. Accessed April 15, 2023. https://www.uptodate.com.

The use of basal energy metabolism is useful in estimating the energy needs of compromised infants and children.

kcal/day = Basal metabolic rate × Activity factor × Stress factor

See **Tables 3.10–3.12** for equations to estimate basal metabolic rate (BMR), as well as common activity and stress factors.

TABLE 3.10 Schofield Method to Estimate BMR

Age (yr)	Male	Female
0–3	(0.167 × wt) + (15.174 × ht) – 617.6	(16.252 × wt) + (10.23 × ht) – 413.5
3–10	(19.59 × wt) + (1.303 × ht) + 414.9	(16.969 × wt) + (1.618 × ht) + 371.2
10–18	(16.25 × wt) + (1.372 × ht) + 515.5	(8.365 × wt) + (4.65 × ht) + 200
>18	(15.057 × wt) − (1.004 × ht) + 705.8	(13.623 × wt) + (2.83 × ht) + 98.2

Data from Delgado NB, Hastings E, Kommes K, Ling H, Spoede E, Wrobel M. *Texas Children's Hospital Pediatric Nutrition Reference Guide*, 13th edition. Texas Children's Hospital, 2022.

TABLE 3.11 Activity Factors

Condition	Factor
Ambulatory	1.2–1.3

Condition	Factor
Confined to bed	1.1
Paralyzed	1.0

TABLE 3.12 Stress Factors

Condition	Factor
Burn	1.5–2.5
Growth failure	1.5–2.0
Infection	1.2–1.6
Starvation	0.70
Surgery	1.2–1.5
Trauma	1.1–1.8

Data from Page C, et al., *Nutritional Assessment and Support*. Baltimore, MD: Williams & Wilkins, 1994.

Estimating Energy Needs for Catch-up Growth

For patients who are malnourished or for those whose growth is compromised, the nutritional goal is to accelerate growth. This increase in normal weight and height velocity is referred to as “catch-up growth.” Catch-up growth is optimal to facilitate normalized weight and height. Total energy needs for catch-up growth may be as high as 150% of expected needs.

General Method of Estimating Energy Needs for Catch-up Growth

Before calculating catch-up growth, you must determine the ideal body weight (IBW) in kg for the equation.

- From birth to 24 months of age use the WHO weight-for-length growth chart and find the weight at the 50th percentile for the infant's actual length. This is done by finding the length along the bottom axis on the chart and following that line up to the 50th percentile curve to find the weight on the left vertical axis.
- For those aged 2–20 years, use the standard CDC BMI-for-age growth chart and find the BMI (kg/m^2) at the 50th percentile for the individual's age. This is done by finding the age along the bottom axis on the chart and following that line up to the 50th percentile curve to find the BMI on the left vertical axis. Multiply the BMI by the actual height of the subject in $meters^2$ to obtain the weight.

$$\text{kcal/kg/day}^* = \frac{\text{IBW in kg (50th percentile wt/ht)} \times \text{kcal/kg/day (DRI for age)}}{\text{Actual weight (kg)}}$$

where, IBW, *ideal body weight*; DRI, *dietary reference intake*.

**Note* that protein needs for catch-up growth are calculated using the same formula by substituting DRI of kcal (kcal/kg/day) with DRI of protein (g/kg/day).

Example: Andrea is 5 years and 6 months old. She weighs 13 kg (<5th percentile) and is 106 cm tall (between the 10th and 25th percentiles).

Using the standard CDC BMI-for-age growth chart for girls aged 2 to 20 years, what is Andrea's ideal body weight for her height?

$$\text{IBW in kg} = \text{50th percentile BMI} \times \text{height (in meters)}^2$$

$$= 15.2 \times 1.06^2$$

$$= 17.1 \text{ kg}$$

Using the general equation for catch-up growth, how many kilocalories per kilogram per day does Andrea require? Refer to Table 3.8 for DRI values.

$$\text{kcal/kg/day} = \frac{17.1 \text{ kg (IBW for height)} \times 65 \text{ (DRI for girls age 4–5)}}{13 \text{ kg (actual weight)}}$$

$$= 86 \text{ kcal/kg/day}$$

How many total kilocalories per day does Andrea require?

$$86 \text{ kcal/kg} \times 13 \text{ kg} = 1118 \text{ kcal/day for catch-up growth}$$

Children with Developmental Disabilities

Table 3.13 lists methods for estimating energy needs of children with developmental disabilities.[5]

TABLE 3.13 Estimating Energy Needs for Children with Developmental Disabilities

Diagnosis	Calorie Needs
Cerebral Palsy (Age 5–11 Years Old)	
Mild to moderate activity	13.9 kcal/cm ht
Severe physical restrictions	11.1 kcal/cm ht
Athetoid cerebral palsy	Up to 6,000 kcal/day
Down Syndrome (Age 5–12 Years Old)	
Males	16.1 kcal/cm ht
Females	14.3 kcal/cm ht

Diagnosis	Calorie Needs
Myelomeningocele (Spina Bifida)	
Weight maintenance requirements	9–11 kcal/cm ht
Weight loss requirements	7 kcal/cm
>1 year old	~50% DRI/RDA for age
Prader–Willi Syndrome	
Weight maintenance requirements	10–11 kcal/cm ht
Weight loss requirements	8.5 kcal/cm ht

DRI, dietary reference intake.
Konek S, Becker P. *Pediatric Nutrition in Clinical Care*. 5th edition. Jones & Bartlett Learning, 2022 (Chapter 12).

Vitamins and Minerals

A few key micronutrients require special attention in the assessment of the pediatric population.

- Iron: The combination of rapid growth and relatively low iron intake places children at risk for iron-deficiency anemia. Adolescent females and those who partake in high levels of activity are also at risk. This can be prevented with appropriate feeding choices. If iron deficiency is suspected, it should be confirmed with laboratory testing. The following are laboratory values to consider: hemoglobin, hematocrit, mean corpuscular volume, ferritin, serum iron, and total iron-binding capacity.[6] Iron is not always an ingredient in multivitamins, so care should be taken to review supplement options.

- Vitamin D: Exclusively breastfed infants should receive a vitamin D supplement of 400 international units (IU) per day in the first few days of life. Supplementation for compromised children and adolescents who do not get regular exposure to sunlight may also be indicated, and needs may be above the recommended dietary allowance (RDA) of 600 IU.[7]
- Zinc: Some studies show that infants and children are at risk for zinc deficiency because of altered absorption of the nutrient. It is important to assess dietary intake of sources of zinc. These sources are meat, eggs, legumes, and whole grains.
- Fluoride: Fluoride is necessary for reduction of dental decal. If using bottled water, it may or may not contain fluoride; therefore, individual products need to be investigated. Recommendation of a supplement depends on the assessment of a child's total daily fluoride intake based on fluid sources. From 0 to 6 months of age, no supplementation is required. After 6 months of age, supplementation amount should be based on the child's total fluoride exposure.[6]
- Calcium: Adequate calcium intake in all children and adolescents is required for optimum growth, prevention of future skeletal abnormalities, and bone weakness. Dairy and nondairy sources of calcium need to be evaluated. For those whose intake is deficient, education regarding sources of calcium should be complete. Supplementation may be considered if deemed necessary by clinical judgment.[6] A comprehensive list of standard DRIs for vitamins and elements can be found at https://ods.od.nih.gov/Health_Information/Dietary_Reference_Intakes.aspx.

Supplementation

Routine vitamin and mineral supplementation is unnecessary for the normal, healthy child older than 1 year. The following is a list of individuals who are at risk. Supplementation should be considered for these patients (see **Table 3.14**).

TABLE 3.14 Common Vitamin/Mineral Supplements[a]

Supplement	Age Group (yr)	Daily Dose
Poly-Vi-Sol (with or without iron)	0–3	1 mL
Tri-Vi-Sol (with or without iron)	0–3	1 mL
Nano VM	1–3	2 packets
Nano VM	4–8	2 packets
Nano VM T/F	9–18	41 mL
Centrum kids (complete)	2–3	½ tablet
	≥4	1 tablet
Flintstones (complete)	2–3	½ tablet
	≥4	1 tablet
Vitamax pediatric liquid	<1	1 mL
	1–3	2 mL
Vitamax chewable	4–11	1 tablet
	>11	2 tablets
Nature's plus animal parade multivitamin	1+	2 chewable tablets

Supplement	Age Group (yr)	Daily Dose

[a]Check hospital formulary for available supplements. Please note that mineral and vitamin contents vary among products. Refer to manufacturer for complete listing of contents. Many generic MVIs are available and chosen by consumers; these are typically equal to national brand unless found otherwise.
Data from Delgado NB, Hastings E, Kommes K, Ling H, Spoede E, Wrobel M. *Texas Children's Hospital Pediatric Nutrition Reference Guide*, 13th edition. Texas Children's Hospital, 2022.

- Infants with malabsorption and liver or pancreatic disease
- Children and adolescents from deprived families or children who experience neglect or abuse
- Children and adolescents with anorexia or inadequate appetites
- Children with chronic disease
- Children following dietary regimens for weight loss
- Pregnant teenagers
- Children or adolescents who omit any one food group from their diet

Fiber

Table 3.15 lists fiber recommendations for children and adolescents.

TABLE 3.15 Fiber Requirements in Children Through Adolescents

Age (yr)	Fiber/day (g)
1–3	19
4–8	25
Girls 9–13	26
Boys 9–13	31
Girls 14–18	26
Boys 14–18	38

Data from Delgado NB, Hastings E, Kommes K, Ling H, Spoede E, Wrobel M. *Texas Children's Hospital Pediatric Nutrition Reference Guide*, 13th edition. Texas Children's Hospital, 2022.

Guidelines for Completing Dietary Assessment

In addition to any information available in the medical record, a thorough dietary history should be gathered by means of interviewing any and all caregivers and/or patients when appropriate. The accuracy of information will be highly variable and is dependent on the skill of the interviewer as well as on the data collection method. Some important areas of focus include but are not limited to the following:

- Maternal, prenatal, and postnatal history (exposure to toxic agents, medications, infections, maternal nutrition, length of pregnancy, etc.)
- Birth weight and weight history
- Feeding history (duration of breastfeeding, type of formula, age at introduction to solid foods)
- Typical eating habits, food preferences, amounts (use visual models as needed), frequency of consumption
- Chewing and swallowing function
- Past or present gastrointestinal problems (gastroesophageal reflux disease [GERD], history of feeding disorders, nausea, vomiting, diarrhea, constipation)
- Number of caregivers and households involved in child's care
- Food and/or formula preparation, including location, methods, and sanitation habits
- Sleep habits
- Food allergies/intolerances
- Necessary feeding aids or special utensils or nipples

- Access to and use of community resources

Normal Feeding Guidelines

Tables 3.16 and **3.17** list feeding guidelines for infants, toddlers, and adolescents.

TABLE 3.16 Feeding Guidelines for Infants 0–12 Months Old

Age	Breast Milk and/or Fortified Infant Formula	Cereal, Bread, Grains, and Starches	Fruits and Vegetables	Meats and Other Protein-Rich Foods
Birth–4 mo	8–12 feedings 2–6 oz/feeding (18–32 oz/day)	None	None.	None.
4–6 mo	486 feedings 4–6 oz/feeding (27–45 oz/day)	None	None.	None.
6–8 mo	3–5 feedings 6–8 oz/feeding (24–32 oz/day)	2–3 servings of iron-fortified infant cereal, soft cooked breads and starches (serving = 1–2 Tbsp)	Introduce plain, cooked, mashed, or strained infant vegetables and fruits. Avoid meat-containing dinners. 2–4 oz 100% fruit juice in cup.	Begin to offer plain, cooked, pureed meats. Avoid combination dinners.
8–10 mo	3–4 feedings 7–8 oz/feeding (24–32 oz/day)	2–3 servings of iron-fortified infant cereal, soft cooked breads and starches (serving = 1–2 Tbsp)	2–3 servings of soft, cut-up, and mashed vegetables and fruits (serving = 1–2 Tbsp). 3–4 oz 100% fruit juices in cup.	Offer plain, well-cooked, soft, finely cut, or pureed meats, cheeses, and casseroles.
10–12 mo	24–32 oz/day	4 servings of iron-fortified infant cereal, soft cooked breads and starches (serving = 1–2 Tbsp)	4 servings of soft, cut-up, and mashed vegetables and fruits (serving = 1–2 Tbsp). 3–4 oz 100% fruit juices in cup.	1–2 oz of soft, finely cut, or chopped meat or other protein foods.

Data from (i) Delgado NB, Hastings E, Kommes K, Ling H, Spoede E, Wrobel M. *Texas Children's Hospital Pediatric Nutrition Reference Guide*, 13th edition. Texas Children's Hospital, 2022 and (ii) Becker P, Carney LN, Corkins MR, et al. Consensus Statement of the Academy of Nutrition and Dietetics/American Society for parenteral and enteral nutrition: Indicators recommended for the identification and documentation of pediatric malnutrition (undernutrition). *Nutr Clin Pract.* 2015;30(1):147-61.

Description

TABLE 3.17 Feeding Guidelines for Toddlers to Adolescents

	Milk and Milk Products	Cereal, Bread, Grains, and Starches	Fruits and Vegetables	Meats and Other Protein-Rich Foods	Fats and Oils	Sweets, Jellies, Jams, Soft Drinks
12–23 mo	2 cups/day (whole milk or milk products)	2 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal)	1¾ cup/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens)	1.5 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts).	Do not restrict.	Provide small amount if desired.
2–3 years[a]	2–2.5 cups/day (1 cup milk, 1.5 oz natural cheese, ½ cup shredded cheese)	3 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal)	2 cups/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens)	2 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts).	3 Tbsp/day (1 Tbsp oil, margarine, butter, or mayonnaise; 1 Tbsp salad dressing; sour cream or light mayonnaise).	165–170 discretionary calories.
4–8 years	2.5–3 cups/day (1 cup milk, 1.5 oz natural cheese, 2 oz processed cheese, 1/3 cup shredded cheese)	4–5 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal)	2½–3 cups/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens)	3–4 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts).	4 Tbsp/day (1 Tbsp oil, margarine, butter, or mayonnaise; 1 Tbsp salad dressing; sour cream or light mayonnaise).	170–195 discretionary calories.
9–13 years	3 cups/day (1 cup milk, 1.5 oz natural cheese, 2 oz processed cheese, 1/3 cup shredded cheese)	5–6 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal)	3½–4 cups/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens)	5 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts).	5 Tbsp/day (1 Tbsp oil, margarine, butter, or mayonnaise; 1 Tbsp salad dressing; sour cream or light mayonnaise).	Limit amount, use sparingly (130–140 discretionary calories).
14–18 years	3 cups/day (1 cup milk, 1.5 oz natural cheese, 2 oz processed cheese, 1/3 cup shredded cheese)	6–7 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal)	3½–5 cups/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens)	5–6 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts).	5–6 Tbsp/day (1 Tbsp oil, margarine, butter, or mayonnaise; 1 Tbsp salad dressing; sour cream or light mayonnaise).	Limit amount, use sparingly (265–650 discretionary calories).

[a]Some foods are considered choking hazards to the developing toddler and should be avoided until the child is >3 years old. Such foods are peanut butter, hot dogs, grapes, hard pieces of fruit and vegetables, and tough or chewy meats.

Data from Delgado NB, Hastings E, Kommes K, Ling H, Spoede E, Wrobel M. *Texas Children's Hospital Pediatric Nutrition Reference Guide*, 13th edition. Texas Children's Hospital, 2022.

Description

Breastfeeding

The American Academy of Pediatrics and the Academy of Nutrition and Dietetics support breastfeeding as the preferred method of feeding for all infants, including premature newborns (**Box 3.4**). Breastfeeding is recommended for the first year of life or as desired as feasible. Exclusive breastfeeding will provide adequate nutrition until approximately 6 months of age. This is the point at which complementary foods should be offered.[8]

Box 3.4 Breastfeeding Benefits and Contraindications

Benefits of Breastfeeding

Breast milk contains the most complete nutrition composition in terms of fat, carbohydrate, and protein.
There is optimal growth associated with breastfeeding.
Protein quality differs from that of cow's milk, making human milk better tolerated.
Calcium and phosphorus are more bioavailable than those found in milk formulas.
Many factors in human milk enhance maturity of the digestive tract.
Breastfeeding has been proven to reduce the risk of infection.
Decreased risk of overfeeding.
Breastfeeding has been shown to enhance cognitive and motor skill development.
Reduced costs by eliminating formula.
Reduced risk of premenopausal breast cancer in the infant's mother.
Increased mother/infant bonding.
Aids in postpartum weight loss and uterine involution.

Contraindications of Breastfeeding[a]

Infants with galactosemia.
Infants with other inborn errors of metabolism, unless as directed by physician.
Mothers infected with HIV.
Mothers with herpetic lesion of the breast or other abscesses.
Mothers receiving chemotherapy.

Mothers with substance abuse.
Mothers with untreated syphilis.

[a]Refer to pharmaceutical reference when determining safety of maternal medications in relation to breastfeeding.

Assessment of Adequacy of Feedings

- Monitor infant's growth and weight losses and gains
- Monitor stooling and urine output
- By the third day of life, breastfed infants should produce:
  - Approximately six wet diapers/day[9]
  - Approximately three stools/day. Stool consistency should progress from meconium to greenish-yellow to yellow and seedy.[9]
- Recommended feeding schedule:
  - At least 8–12 feedings/24 hour. Typically, every 1½ to 3 hours during first few weeks of life. Feed 10–20 minutes per breast during each feeding.[9]

Human milk contains approximately 20 kcal/oz. If it is determined that the infant is not obtaining adequate kilocalories from the breast milk, the milk may be fortified to a higher caloric density using human milk fortifier or standard formula powder (**Table 3.18**).

TABLE 3.18 Increasing Caloric Density of Human Breast Milk (By Adding Powdered Formulato 100 mL of Expressed Breast Milk)

Desired Caloric Concentration (kcal/oz)	Similac Advance Powder (g)	Enfamil Infant Powder (g)	Similac NeoSure Advance Powder (g)	Enfamil EnfaCare Powder (g)
22	1.3	1.3	1.3	1.4
24	2.6	2.7	2.6	2.7
27	4.6	4.7	4.5	4.8
30	6.5	6.7	6.5	6.8

Description

Infant Formulas

Commercial infant formulas provide adequate kilocalories, protein, fat, vitamins, and minerals to those infants who are not breastfed. Infant formulas are available in ready-to-feed, concentrated, or powdered forms. Most infants thrive on a standard cow's milk-based formula. However, if intolerance or allergy occurs, alternative formulas are available. **Table 3.19** lists common types of infant formulas, and **Table 3.20** shows standard formula preparation.

TABLE 3.19 Common Types of Infant Formulas[a]

Type of Formulas	Indications	Examples[b]
Cow's milk-based	The standard type of formula traditionally used. The distribution of nutrients is similar to that of breast milk. Most are iron fortified. Contains 19–20 kcal/oz (dependent on brand).	• Enfamil Infant • Good Start Supreme DHA and ARA • Good Start Essentials • Similac Advance (all may be available in lactose-free or other versions)
Soy-based	Standard formula derived from soy protein rather than from bovine milk protein. Used in patients with milk protein allergy. It should be noted that many infants who have milk protein allergy may also present with soy protein allergy. Contains 19–20 kcal/oz (dependent on brand).	• Enfamil ProSobee • Gerber Good Start Soy • Similac Soy Isomil

Type of Formulas	Indications	Examples[b]
Formulas for low- birth-weight babies	Increased caloric density and/or mineral concentration for premature infants.	• Enfamil EnfaCare (22 kcal/oz) • Enfamil Premature (20 kcal/oz or 24 kcal/oz) • Similac Expert Care NeoSure (22 kcal/oz) • Similac Special Care Advance (20 or 24 kcal/oz)
Protein hydrolysate	Formulas containing hydrolyzed proteins that are easier to digest. Indicated for use in infants who are not tolerating standard milk or soy formulas. Many contain additional medium-chain triglyceride (MCT) oil for infants with compromised gastrointestinal function. 20 kcal/oz.	• Similac Alimentum • Nutramigen • Pregestimil • Gerber Extensive HA
Free amino acids	Indicated for infants with extreme allergies to intact proteins. 20 kcal/oz.	• Neocate • EleCare • Alfamino Infant
Follow-up formulas	Iron-fortified formulas for toddlers who may be in need of higher iron or more nutrient-dense fluid source than whole milk.	• Enfagrow Toddler Transitions • Enfamil Toddler Next Step • Good Start Graduates Gentle • Similac Go & Grow Stage 3

Type of Formulas	Indications	Examples[b]

[a] Please note that the above list is not complete and does not contain all infant formulas available. Refer to product labels or manufacturers' references for nutrient values and ingredients.

[b] The manufacturers have copyrighted the terms "Lipil" and "Advance" to indicate the inclusion of docosahexaenoic (DHA) and arachidonic acid (ARA) polyunsaturated fatty acids.

TABLE 3.20 Standard Formula Preparation 19–20 kcal/oz (Calories Vary by Brand, Preparation Does Not Vary by Brand)

Concentrate	Powder	Ready to Feed
13 fl oz concentrate (1 can) + 13 fl oz water = 26 oz total formula	1 scoop powder + 2 fl oz water = 2 fl oz formula	Do not add water

Formula Shortages

The wave of infant formula recalls and subsequent shortages beginning in 2022 caused a need for the import and emergency use approval of international formulas and increased production by small company formulas, especially for the most specialized, amino acid-based ones. Pediatric dietitians and other healthcare professionals had to quickly assess and judge appropriateness of these new to the U.S. formulas. Finally, many chain stores and pharmacies now have their own line of infant standard and specialty formulas that are equivalent to those in Table 3.19 and should be compared by trained professionals for appropriateness.

If nutrition assessment indicates the need for a higher calorie formula, the formula recipe can be modified to concentrate more kilocalories into the same amount of total formula. When concentrating formulas, symptoms of intolerance may occur because of increased osmolality and renal solute load. **Table 3.21** shows methods of increasing kilocalories, protein, and other nutrients. The patient's goals need to be considered before making any changes to a formula. When formulas are concentrated to >20 kcal/oz, all nutrients are concentrated unless a modular supplement is added.

TABLE 3.21 Increasing Formula Calories Based on Patient Goals

Goal	Adjustment to Formula
Increase overall nutrients	Concentrate and/or add carbohydrate or fat modular
Increase caloric density only	Add carbohydrate and/or fat modular
Increase protein density only	Add modular protein supplement

Pediatric Formulas

There are many formulas designed to meet the needs of children >1 year (**Table 3.22**). Many products can be used as both enteral feeding and oral supplementation, when required.

TABLE 3.22 Common Pediatric Formulas[a] by Brand

Formula	Route	Amount Required to Meet DRIs	Caloric Strength (kcal/oz)	Comments
EleCare Junior	Oral or enteral	NL	30	Elemental formula, lactose free, gluten free, soy free, milk protein free, fructose free, galactose free
Neocate Junior	Oral or enteral	NL	30	Elemental formula for children with multiple protein allergies, lactose free, gluten free
Nutren Junior (with or without fiber)	Oral or enteral	1,000 mL for 1–8-year-olds 1,500 mL for 9–13-year-olds	30	Lactose free, gluten free, kosher, low residue
PediaSure (with or without fiber)	Oral and enteral	1,000 mL for 1–8-year-olds 1,500 mL for 9–13-year-olds	30	Lactose free, gluten free, kosher
Peptamen Junior	Oral and enteral	1,000 mL for 1–8-year-olds 1,500 mL for 9–13-year-olds	30	Elemental formula designed for children with severe GI tract disturbances
Vivonex pediatric	Oral or enteral	1,000 mL for 1–8-year-olds 1,500 mL for 9–13-year-olds	24	Elemental, lactose-free, gluten-free, low-residue formula

GI, gastrointestinal; NL, not listed by manufacturer.

[a]The above list is not complete and does not contain all pediatric formulas available. Refer to product labels or manufacturers' references for most current nutrient values and ingredients. In some cases, it is appropriate to utilize an adult formula for nutrition supplementation. Refer to the guidelines for use provided by the manufacturers of those products.

Description

It should be noted that it is preferable to increase energy intake with common nutrient-dense foods rather than by formula supplementation (e.g., fortifying the child’s meals with peanut butter, avocado, oils, whole milk, heavy cream, and addition of evaporated milk to casseroles and soups). This method encourages healthful feeding practices, lowers cost, and improves taste.

Blenderized Formulas

Consumer preference and improved clinical outcomes have recently increased demand for both homemade and commercially prepared blenderized tube feeding formulas (see **Table 3.23**). Home prepared blenderized tube feeding products have been shown to improve clinical outcomes such as increasing calorie and micronutrient provision, and decreased prevalence of vomiting and acid-suppressive agent use, as well as increased intestinal bacterial diversity.[10]

TABLE 3.23 Common Blenderized Formulas[a] by Brand

Formula	Route	Amount Required to Meet DRIs	Caloric Strength (kcal/oz)	Comments
Compleat Pediatric	Enteral	1,400 mL for 1–3-year-olds	30	Lactose free, gluten free
Compleat Pediatric Reduce Calorie	Enteral	1–8 years 1,000 mL 9–13 years 1,200 mL	18	Lactose free, gluten free, no corn or soy ingredients
Compleat Organic Blends	Enteral	NL	38	Lactose-free, Gluten-free No dairy, soy or corn ingredients. Ingredients vary, plant based available.
Pediasure Harvest	Enteral	1,000 mL for children 1–8 years of age 1,500 mL for children 9–13 years of age.	30	Made with no milk-, gluten- or lactose-containing ingredients
Real Food Blends	Enteral	NL	Varies	Suitable for those with nut, soy, gluten, or dairy intolerances
Nourish	Enteral or oral	1,065 mL of Nourish meets 100% of the DRI for 26 key vitamins and minerals for children 4–8 years old	33	Dairy free, tree nut free, gluten free, soy free, and corn free
Kate Farms Pediatric Standard 1.2	Enteral or oral	1–3 years: 3.6 cartons or 900 mL (1,080 calories) 4–8 years: 4 cartons or 1,000 mL (1,200 calories) 9–13 years: 6 cartons or 1,500 mL (1,800 calories)	36	Does not contain whey, lactose, casein, soy, gluten, nuts or corn. Appropriate for the following diets: lactose free, vegan, plant based, gluten free
Kate Farms Pediatric Peptide 1.5	Enteral or oral	1–3 years: 2.6 cartons or 650 mL (975 calories) 4–8 years: 3 cartons or 750 mL (1,125 calories) 9–13 years: 4 cartons or 1,000 mL (1,500 calories)	45	Does not contain whey, lactose, casein, soy, gluten, nuts or corn. Appropriate for the following diets: lactose free, vegan, plant based, gluten free

NL, not listed by manufacturer.

[a]The above list is not complete and does not contain all pediatric formulas available. Refer to product labels or manufacturers' references for most current nutrient values and ingredients. In some cases, it is appropriate to utilize an adult formula for nutrition supplementation. Refer to the guidelines for use provided by the manufacturers of those products.

Description

Biochemical Evaluation of Nutritional Status in the Pediatric Patient

See **Table 3.24**.

TABLE 3.24 Normal Laboratory Values[a]

Measure	Range
Albumin (serum), g/dL	
Infants	2.9–5.5
Other children	3.7–5.5
Calcium	
Serum total calcium, mg/dL	
Preterm infants	6–10
Term infants	7–12
Other children	8–10.5
Serum ionized calcium, mg/dL	4.48–4.92
Folate (serum)	>6–7 ng/mL
Hemoglobin (blood), g/dL	
Newborns	≥14.0
12–35-month-old children	≥11.0
3.0–4.9-year-old children	≥11.0

Measure	Range
5.0–7.9-year-old children	≥11.5
8.0–11.9-year-old children	≥11.9
12.0–15.0-year-old boys	≥12.5
12.0–15.0-year-old girls	≥11.8
>15-year-old boys	≥13.0
>15-year-old girls	≥12.0
Iron (serum), mg/dL	
Neonates	>30
Infants	>40
Children <4 years	>50
Other children	>60
Phosphorus (serum), mg/dL	
Newborns	4.0–8.0
1 year	3.8–6.2
2- to 5-year-old child	3.5–6.8
Other children	2.9–5.6
Prealbumin (serum), mg/dL	
Premature infants	4–14
Term infants	4–20
6–12-month-old children	8–24

Measure	Range
1–6-year-old children	17–30
Other children	17–42
Blood urea nitrogen, mg/dL	7–22
Transferrin, mg/dL	170–440
Vitamin A (plasma retinol), µg/dL	
Infants	13–50
Other children	20–72
Vitamin D	
25-Hydroxyvitamin-D, ng/mL (preferred)	20–30
1,25-(OH)2D3, ng/mL	15–60
Vitamin E	
Plasma alpha-tocopherol, mg/dL	
Preterm infants	0.5–3.5
Other children	0.7–10
Red blood cell hemolysis test, %	10
Vitamin K	
Prothrombin time, sec	11–15
PIVKA-II, ng/mL	≤3
Thiamin	
Red blood cell transketolase stimulation, %	<15

Measure	Range
Vitamin B_{12}, pg/mL	200–900
Absorption test	Excretion of more than 7.5% of ingested labeled vitamin B_{12}
Vitamin C (plasma), mg/dL	0.2–2.0
Magnesium (serum), mEq/L	1.5–2.0
Zinc (serum), µg/dL	60–120

PIVKA, Proteins induced by vitamin K absence.

[a]Results of a single lab test should be interpreted with caution and normal values may vary among laboratories.

Data from (i) University of Calgary, Fenton Preterm Growth Charts. Available at https://www.ucalgary.ca/resource/preterm-growth-chart/preterm-growth-chart. Accessed April 19, 2023, (ii) Fenton TR, Kim JH. A systematic review and meta-analysis to revise the Fenton Growth Chart for preterm infants. *BMC Pediatrics*. 2013;13:59, and (iii) Barlow SE and the Expert Committee. Expert Committee recommendations regarding the prevention, assessment and treatment of child and adolescent overweight and obesity: summary report. *Pediatrics*. 2007;120:S164-S192.

Common Nutritional Disturbances

Anemia

Iron deficiency is the most prevalent single nutrient deficiency in children in the United States. As discussed previously, true iron-deficiency anemia needs to be determined to properly treat the condition. An iron-rich diet may be indicated for infants, children, and adolescents at risk for iron-deficiency anemia.

Constipation

Nutrition intervention is warranted for patients who experience pain associated with the passage of stool, the hard nature of stool, or the failure to pass at least three stools per week. Painful stools, despite frequency, may also be considered constipation. Major causes include inadequate fluid and fiber, abnormal muscle tone, motor skill impairments, medications, or debilitating disorders. Therapy for children 1 year and older includes modifications in fiber, fluid, and activity level.

Failure to Thrive

Failure to thrive (FTT) is an outdated term traditionally used to describe a child's growth that is significantly lower than normal for the child's age and gender.[11] It is now recognized that the cause of FTT is malnutrition due to inadequate caloric intake, inadequate caloric absorption, or excessive caloric expenditure. The reasons for malnutrition may stem from underlying medical conditions and/or non-medical reasons such as environmental, economic, psychosocial, or behavioral factors. Regardless of the reasons, since undernutrition is the proximate cause of faltering weight gain and growth, the goals of medical nutrition therapy should be centered on assessment and therapy for malnutrition.

Gastroesophageal Reflux

Gastroesophageal reflux (GER) is a common occurrence in infants. It is typically resolved by 18 months of age and rarely requires corrective surgery. Medical nutrition therapy for GER is usually used in

combination with drug therapy. Recommendations should be individualized based on the child's age and developmental status. Commercially prepared thickened formulas are available, and feeds thickened with infant cereal remain common along with positioning techniques and avoidance of known irritants.

Lead Poisoning

Rates of lead poisoning in children are highest in older cities and among low-income communities. Medical nutrition therapy for lead poisoning consists of a high-iron, high-fiber, and fat-modified diet. A multivitamin may also be recommended. Iron deficiency increases the absorption and storage of lead in the body. Fiber speeds the transport of food through the body and thus decreases the length of stay of lead in the gut, whereby decreasing its absorption. High-fat foods should be limited in the diet because they enhance the absorption and storage of lead in the body.

Obesity

Weight management intervention should be completed in children whose BMI is greater than the 85th percentile for age with comorbidities and/or complications, and also in those whose BMI is above the 95th percentile for age. Basic framework for intervention should include nutrition education, an exercise plan, and behavior modifications (see **Table 3.25**). In 2023, The American Academy of Pediatrics published a controversial first edition clinical practice guideline related to weight management which lowered the age previously recommended for referral to treatment programs and included weight loss medication and bariatric surgery recommendations starting at age of 12.[12]

TABLE 3.25 Expert Panel Intervention Stages According to Age and BMI-for-Age Percentile

Counseling Stage	Age (yr)	BMI-for-Age Percentile	Presence of Health Risk	Goal of Intervention
Stage 1	<18	<94th	No	Weight maintenance until BMI-for-age is <85th percentile or a slower rate of weight gain
Stages 1–2	2–18	85th–94th	Yes	Weight maintenance until BMI-for-age is <85th percentile or a slower rate of weight gain
Stages 1–2	6–18	95th–98th	Yes	Either weight maintenance until BMI-for-age is <85th percentile or gradual weight loss of 1 lb/mo
Stages 1–3	2–5	95th–98th	Yes	Weight maintenance until BMI-for age is <85th percentile or a slower rate of weight gain; weight loss should be <1 lb/mo
Stages 1–3	2–5	≥99th	Yes	Gradual weight loss of ≤1 lb/mo
Stages 1–3	6–11	>99th	Yes	Weight loss of 2 lb/mo
Stages 1–4	12–18	>95th	Yes	Weight loss of ≤2 lb/wk until BMI-for-age is <85th percentile

Data from (i) Mehta N, et al. Defining pediatric malnutrition: A paradigm shift towards etiology-related definitions. *J Parenter Enteral Nutr.* 2013;37(4):460-481, and (ii) Barlow SE and the Expert Committee. Expert Committee recommendations regarding the prevention, assessment and treatment of child and adolescent overweight and obesity: summary report. *Pediatrics.* 2007;120:S164-S192.

Description

Pediatric Malnutrition

Pediatric malnutrition is typically examined in the context of its chronicity, severity, and etiology. Acute versus chronic malnutrition is determined by the National Center for Health Statistics (NCHS) definition of a chronic disease as one that lasts 3 months or longer. Stunting (decreased height velocity) is the characteristic sign of chronic malnutrition, whereas weight is usually affected during acute periods of malnutrition. Use of z-scores (standard deviation comparisons) of growth measurements have replaced previously used criteria such as the Gomez Classification, Waterlow criteria, or McClaren criteria to define severity. Pediatric malnutrition criteria and various online tools available to easily determine z-scores are shown in **Tables 3.26** through **3.28**).

TABLE 3.26 Resources for Determining Z-Scores for Anthropometrics

CDC Growth Charts	WHO Growth Charts
Epi Info 7 (available for download on computers, tablets, and smartphones): https://www.cdc.gov/epiinfo/	WHO z-score charts: https://www.who.int/tools/child-growth-standards/standards/weight-for-age
CDC website: z-score data files available as tables: https://www.cdc.gov/growthcharts/zscore.htm	WHO Multicenter Growth Study website: http://www.who.int/childgrowth/software/en/
Clinical tools for pediatric providers: growth charts, calculators, mobile apps including STAT GrowthCharts WHO and Child Growth Tracker, PediTools at www.peditools.org	

TABLE 3.27 Pediatric Malnutrition Criteria (Single Data Point)

Primary Indicators	Mild Malnutrition	Moderate Malnutrition	Severe Malnutrition
Weight for height z-score	–1 to –1.9 z-score	–2 to –2.9 z-score	–3 or greater z-score
BMI-for-age z-score	–1 to –1.9 z-score	–2 to –2.9 z-score	–3 or greater z-score
Length/height z-score	No data	No data	–3 z-score
Mid-upper arm circumference	Greater than or equal to –1 to –1.9 z-score	Greater than or equal to –2 to –2.9 z-score	Greater than or equal to –3 z-score

Data from (i) University of Calgary, Fenton Preterm Growth Charts. Available at https://www.ucalgary.ca/resource/preterm-growth-chart/preterm-growth-chart. Accessed April 19, 2023, and (ii) Fenton TR, Kim JH. A systematic review and meta-analysis to revise the Fenton Growth Chart for Preterm Infants. *BMC Pediatrics*. 2013;(13):59.

Description

TABLE 3.28 Pediatric Malnutrition Criteria (Two or More Data Points)

Primary Indicators	Mild Malnutrition	Moderate Malnutrition	Severe Malnutrition
Weight gain velocity (<2 years)	<75% of the norm for expected weight gain	<50% of the norm for expected weight gain	<25% of the norm for expected weight gain
Weight loss (2–20 years)	5% usual body weight	7.5% usual body weight	10% usual body weight
Deceleration in weight for length/height z-score	Decline of 1 z-score	Decline of 2 z-score	Decline of 3 z-score
Inadequate nutrient intake	51–75% estimated energy/protein need	26–50% estimated energy/protein need	25% estimated energy/protein need

Data from (i) University of Calgary, Fenton Preterm Growth Charts. Available at https://www.ucalgary.ca/resource/preterm-growth-chart/preterm-growth-chart. Accessed April 19, 2023, and (ii) Fenton TR, Kim JH. A systematic review and meta-analysis to revise the Fenton Growth Chart for Preterm Infants. *BMC Pediatrics*. 2013;(13):59.

Description

Preterm and Neonatal Malnutrition

Additionally, criteria have been established to diagnose and document malnutrition related to undernutrition in the highest risk populations in pediatrics: preterm and neonatal infants.[3] The recommended indicators in **Tables 3.29** and **3.30** are intended for use in infants who remain younger than 37 weeks of corrected gestation/postmenstrual age, whether they are in neonatal critical care units, other inpatient hospital settings, or after discharge.

TABLE 3.29 Primary Indicators of Neonatal Malnutrition

Indicator	Mild Malnutrition	Moderate Malnutrition	Severe Malnutrition	Use of Indicator
Primary Indicators Requiring One Indicator				
Decline in weight-for-age z-score	Decline of 0.8 to 1.2 SD[b]	Decline of >1.2–2 SD	Decline of >2 SD	Not appropriate for first 2 wk of life
Weight gain velocity[a]	<75% of expected rate of weight gain to maintain growth rate	<50% of expected rate of weight gain to maintain growth rate	<25% of expected rate of weight gain to maintain growth rate	Not appropriate for first 2 wk of life
Nutrient Intake	3–5 consecutive days of protein/ energy intake 75% of estimated needs	5–7 consecutive days of protein/ energy intake 75% of estimated needs	>7 consecutive days of protein/ energy intake 75% of estimated needs	Preferred indicator during the first 2 wk of life

[a]Expected weight gain velocity, expected linear growth velocity, and z-scores can be determined using the online calculator PediTools (www.peditools.org).
[b]SD = standard deviation.

Reproduced from Goldberg D, Becker PJ, Brigham K, et al. Identifying malnutrition in preterm and neonatal populations: Recommended indicators. *J Acad Nutr Diet.* 2018;118(9):1575.

Description

TABLE 3.30 Primary Indicators of Neonatal Malnutrition

Indicator	Mild Malnutrition	Moderate Malnutrition	Severe Malnutrition	Use of Indicator
Primary Indicators Requiring 2 Indicators				
Days to regain birth weight	15–18	19–21	>21	Use in conjunction with nutrient intake.
Linear growth velocity[a]	<75% of expected rate of linear gain to maintain expected growth rate	<50% of expected rate of linear gain to maintain expected growth rate	<25% of expected rate of linear gain to maintain expected growth rate	Not appropriate for first 2 wk of life. May be deferred in critically ill, unstable infants. Use in conjunction with another indicator when accurate length measurement available.
Decline in length-for-age z-score	Decline of 0.8–1.2 SD[b]	Decline of >1.2–2 SD	Decline of >2 SD	Not appropriate for first 2 wk of life. May be deferred in critically ill, unstable infants. Use in conjunction with another indicator when accurate length measurement is available.

[a]Expected weight gain velocity, expected linear growth velocity, and z-scores can be determined using the online calculator PediTools (www.peditools.org)
[b]SD = standard deviation.

Data from Goldberg D, Becker PJ, Brigham K, et al. Identifying malnutrition in preterm and neonatal populations: Recommended indicators. *J Acad Nutr Diet.* 2018;118(9):1575.

Description

Renal Diseases

Nutrition interventions in pediatric chronic kidney disease should be disease specific (see **Table 3.31**). Macronutrient, micronutrient, and fluid needs vary depending on stage of CKD and urine output among other factors. Polyuric kidney diseases can increase needs for fluid and electrolytes. Dialysis modality may affect protein needs. Glucose calories may be derived from peritoneal dialysis fluid.

TABLE 3.31 Pediatric Nutrition Management for Renal Dysfunction

Nutrient	Nephrotic Syndrome	AKI, No dialysis	AKI, PD or HD	AKI, CRRT	CKD stages 3–5	CKD 5, HD	CKD 5, PD
Energy	EER for age	EER for age or original disease state	EER for age or original disease state	EER for age or original disease state	EER for age	EER for age	EER for age
Protein	DRI-do not supplement to replace urinary losses	DRI or less per BUN monitoring	DRI with 0.2 g/kg increases for HD, 0.4 g/kg for PD	At least 2.5 g/kg or greater	CKD 3 100–140% DRI CKD 4–5 100–120% DRI	0–6 mo: 1.6 g/kg 7–12 mo: 1.3 g/kg 1–3 y: 1.15 g/kg 4–13 y: 1.05 g/kg 14–18 y: 0.95 g/kg	0–6 mo: 1.8 g/kg 7–12 mo: 1.5 g/kg 1–3 y: 1.3 g/kg 4–13 y: 1.1 g/kg 14–18 y: 1.0
Sodium	1–3 mEq/kg; will vary according to edema or hypertension	Varies	Varies	Typically no restriction; may need electrolyte supplementation	1–3 mEq/kg; will vary according to edema or hypertension	1–3 mEq/kg; will vary according to edema or hypertension	1–3 mEq/kg; will vary according to edema or hypertension. Na supplements should be considered for infants.
Potassium	Restriction not needed	<1–2 mEq/kg	Limit	Typically no restriction; may need electrolyte supplementation	Most will tolerate 3 mEq/kg/day	1–3 mEq/kg/ day but will vary according to serum levels and age	Generally unrestricted unless low transporter, will need to be monitored closely.
Phosphorus	Restriction not needed	Tight limit	Limit	Typically no restriction; may need electrolyte supplementation		**High PTH and Normal Phosphorus**	**High PTH and High Phosphorus**
					0–6 mo	≤100	≤80
					7–12 mo	≤275	≤220
					1–3 y	≤460	≤370
					4–8 y	≤500	≤400
					9–18 y	≤1,250	≤1,000
Fluids	Will vary according to urine output	Will vary according to urine output	Will vary according to urine output	May need additional fluid replacements	Generally unrestricted	Replace urine output, insensible losses, +dialysis ultrafiltrate.	Replace urine output, insensible losses, +dialysis ultrafiltrate.
Micronutrients	100% DRI, supplementation as needed	100% DRI, supplementation will most likely be needed	Monitor fat-soluble vitamins	100% DRI, supplement water-soluble vitamins as needed	100% DRI, supplementation as needed	100% DRI water-soluble vitamin supplement is recommended. Monitor vitamin D quarterly and supplement as needed.	

AKI, acute kidney injury; CKD, chronic kidney disease; CRRT, continuous renal replacement therapy; DRI, daily recommended intake; EER, estimated energy expenditure; HD, hemodialysis; PD, peritoneal dialysis; PTH, parathyroid hormone.

Data from (i) Nelms C, Juarez M, Warady BA. Renal Disease. *ASPEN Pediatric Core Curriculum: Renal Diseases.* 2nd ed. American Society for Parenteral and Enteral Nutrition, 2015, (ii) Nelms CL, Shaw V, Greenbaum LA, et al. Assessment of nutritional status in children with kidney diseases—clinical practice recommendations from the Pediatric Renal Nutrition Taskforce. *Pediatr Nephrol.* 2021;36(4):995-1010, (iii) Shaw V, Polderman N, Renken-Terhaerdt J, et al. Energy and protein requirements for children with CKD stages 2-5 and on dialysis–clinical practice recommendations from the Pediatric Renal Nutrition Taskforce. *Pediatr Nephrol.* 2020;35(3):519-531, (iv) Desloovere A, Renken-Terhaerdt J, Tuokkola J, et al. The dietary management of potassium in children with CKD stages 2–5 and on dialysis—clinical practice recommendations from the Pediatric Renal Nutrition Taskforce. *Pediatr Nephrol.* 2021;36(6):1331-1346, and (v) McAlister L, Pugh P, Greenbaum L, et al. The dietary management of calcium and phosphate in children with CKD stages 2-5 and on dialysis—clinical practice recommendation from the Pediatric Renal Nutrition Taskforce. *Pediatr Nephrol.* 2020;35(3):501-518.

Description

Nutrition Programs Beyond the Acute Care Setting

Several federally funded government programs have been created to address health and nutrition disparities for American children who qualify based on income. Some of these are school-based while others are limited to families with children who are not yet school-aged. These programs work to address overall access to food as well as access to healthy food choices. Clinicians in the acute care setting should provide information on these programs to patients who may qualify, as part of the continuum of care.

Women, Infants, and Children (WIC)

Women, Infants, and Children is a supplemental nutrition program funded by federal grants to provide supplemental foods, healthcare referrals, and nutrition education to low-income pregnant, breastfeeding, and non-breastfeeding postpartum women, and to infants and children up to age five who are found to be at nutritional risk. WIC serves about half of the infants born in the United States. It is administered by 90 WIC state agencies, through approximately 47,000 authorized retailers. Those eligible to be participants should apply at their state or local agency to set up an appointment.

National School Lunch and Breakfast Programs

The National School Lunch Program and National School Breakfast Program are federally assisted meal programs operating in public and nonprofit private schools and residential childcare institutions. They provide nutritionally

balanced, low-cost, or no-cost meals to children each school day. Children may be determined “categorically eligible” for free meals based on their status as a homeless, migrant, runaway, or foster child or through participation in certain Federal Assistance Programs, such as the Supplemental Nutrition Assistance Program. Children enrolled in a federally funded Head Start Program, or a comparable State-funded pre-kindergarten program, are also categorically eligible for free meals. Children can also qualify for free or reduced-price school meals based on household income and family size. Children from families with incomes at or below 130% of the Federal poverty level are eligible for free meals. Those with incomes between 130 and 185% of the Federal poverty level are eligible for reduced-price meals. Schools may not charge children more than 40 cents for a reduced-price lunch. All breakfasts and lunches must meet Federal requirements, but decisions about the specific foods to serve and the methods of preparation are made by local school food authorities.

The Fresh Fruit and Vegetable Program (FFVP)

The FFVP is a federally assisted program providing free fresh fruits and vegetables to children at eligible elementary schools during the school day. It is administered on a federal level by the Food and Nutrition Service of the United States Department of Agriculture and by State agencies, which operate the Program through agreements with local school food authorities, on a state level. Schools must operate the National School Lunch Program in order to operate the FFVP and it prioritizes schools with the highest percentage of children certified as eligible for free and reduced-price meals.

Summer Food Service Program (SFSP)

The SFSP is a federally funded, state-administered program that reimburses providers who serve free healthy meals to children and teens in low-income areas during the summer months when school is not in session.

ADIME 3.1 ADIME At-A-Glance: Pediatric Malnutrition

Assessment

- Breast- or bottle-fed, feeding regimen, appetite
- Labs (electrolytes, BUN, creatinine, glucose, albumin)
- Physical findings: GI symptoms, diarrhea, constipation, NFPE findings (loss of muscle mass/fat stores, edema)
- Anthropometrics (weight, length, BMI, head circumference, WHO growth chart percentiles, z-scores)
- Food availability

Nutrition Diagnosis

- Malnutrition (acute, mild) RT improper feeding regimen AEB weight for length z-score –1.85

Intervention

- 3 oz formula per feeding (concentrate formula to 24 kcal/oz) every 4 hours, including through the night, with goal intake of 18 oz/24 hours

Monitoring and Evaluation

- Weekly weights; goal ≥35 g/day
- Confirm enrollment in WIC program

REFERENCES

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CHAPTER 4

Older Adults

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Aging produces numerous physical and physiologic changes, which, in turn, alter nutritional requirements and affect nutritional status (**Box 4.1**). The presence of chronic disease and/or polypharmacy can enhance potential disparities between nutrient needs and dietary intake, leading to malnutrition. Indeed, research suggests that malnutrition is a common condition among the elderly, with a prevalence 1–15% of those among noninstitutionalized older adults, 25–60% of elderly in extended-care facilities, and 35–65% of older adults in hospitals.[1] Malnutrition among older adults can have many causes but are usually categorized as starvation related, chronic disease related, or acute disease or injury related.[2]

Box 4.1 Physiologic Changes in Aging and Nutrition Implications

Body System or Function	Changes in Aging	Nutrition Implications
Body composition	Increase in body fat, especially intra-abdominal; decrease in muscle (sarcopenia); bone loss (including tooth)	Increased risk for obesity, cardiovascular disease, diabetes
Cardiovascular function	Reduced blood vessel elasticity, higher peripheral resistance,	Higher risk for hypertension and other cardiovascular disease

Body System or Function	Changes in Aging	Nutrition Implications
	and blood flow to the heart	
Gastrointestinal function	Reduction in secretions, especially acid, achlorhydria or hypochlorhydria (a consequence of atrophic gastritis [AG], which occurs in 33% of elderly)	Impairment of digestion and absorption (iron, B_{12}, zinc, folate, biotin, calcium); AG causes inflammation and reduction in intrinsic factor, which can cause B_{12} deficiency; AG can also cause B_6 deficiency, dysphagia, and constipation
Immunocompetence	Reduced function, especially T-cell component	In combination with poor nutritional status, higher susceptibility to infection
Oral health	Reduction in saliva, leading to dry mouth (xerostomia); tooth loss	Problem in chewing and swallowing
Neurologic function	Reduction in neurotransmitter synthesis; less efficient nerve conduction; central nervous system (CNS) changes cause problems in balance/coordination; depression; dementia	Depression can cause loss of appetite and food intake, leading to malnutrition
Nutrient metabolism	Lower synthesis of cholecalciferol in skin and renal activation; increased retention of vitamin A owing to reduced clearance	Vitamin D deficiency; vitamin A retention could be toxic if high-dose supplement is used

Body System or Function	Changes in Aging	Nutrition Implications
Renal function	Reduction in number of nephrons (therefore, lower glomerular filtration rate [GFR] and total renal function)	Fluid balance aberrations; acid–base balance problems; metabolism of nutrient medications may be altered
Sensory losses	Decreased sensitivity in taste (dysgeusia), smell (hyposmia), sight, hearing, tactile	Dysgeusia and hyposmia cause loss of appetite and intake, and also increase risk for foodborne illness

Nutrition Assessment

Screening for Nutrition Risks in the Elderly

Nutrition assessment in elderly patients consists of the typical nutrition assessment parameters (refer to Chapter 1), with particular emphasis on risk factors in this population. The Nutrition Screening Initiative was a collaborative effort of several lead agencies whose goal was to identify and treat nutritional problems in the elderly and has been in widespread use for decades.[3] Simple screening tools from both that project and others focusing on all aspects of elderly health, including psychosocial and environmental factors, were developed (Determine and Meals-on-Wheels),[4] and continue to be used by healthcare providers working with the elderly (**Boxes 4.2** and **4.3**).

Box 4.2 Determine Your Nutrition Health Checklist

D Presence of chronic disease or condition
E Eating poorly; too little or poor quality of diet
T Tooth loss or mouth pain
E Economic hardship
R Reduced social contact
M Multiple medicines
I Involuntary weight loss or gain
N Needing assistance in self-care
E Elder years; older than age 80

Box 4.3 Meals-on-Wheels: Assess Malnutrition Risks

M Medications
E Emotional problems
A Anorexia nervosa and other eating behavior problems

L Late-life paranoia
S Swallowing problems
O Oral problems
N No money
W Wandering and other dementia behaviors
H Hyperthyroidism, hyperparathyroidism
E Entry problems (malabsorption)
E Eating problems (both physical and cognitive)
L Low-salt, low saturated-fat/cholesterol diets
S Shopping (lack of food availability, access to foods)

Anthropometric Measurements

In adults over the age of 65, body mass index (BMI) is a valuable assessment tool. Older adults whose BMI values fall <25 and >35 are more likely to experience decreased functional capacity, gait and balance difficulties, increased fall risk, decrease in muscle strength, and malnutrition, with apparent optimal values being 31–32 for females and 27–28 for males.[5] In addition, regardless of usual body weight, one of the most significant anthropometric risk factors in the elderly is involuntary or unintentional weight loss (IWL, UWL). For this reason, the use of ideal body weight is not necessarily appropriate, although it continues to be widely used. A more important parameter is usual body weight and percentage of body weight lost, as described in Chapter 1.

ADIME 4.1 ADIME-At-A-Glance

Assessment

- BMI <23, UWL, decrease in appetite and food intake, dehydration
- Change in ADLs/IADLs, cognitive decline, depression
- Neurologic disease, swallowing difficulty, infection, pressure injuries
- Multiple medications, recent hospitalization

Nutrition Diagnosis

- Inadequate oral intake related to decreased appetite, as evidenced by (AEB) recent UWL of 5% × 2 weeks, and PO intake of <50%
- Inadequate fluid intake related to Alzheimer's disease, AEB dry skin and mucosal tissues, UWL of 5% × 6 days, and high blood urea nitrogen
- Swallowing difficulty related to recent cerebrovascular accident (CVA), AEB coughing when drinking thin liquids
- Unintended weight loss related to Alzheimer's disease, AEB UWL of 10% × 5 weeks
- Self-feeding difficulty related to multiple sclerosis, AEB dropping of utensils, and decreased PO intake of <75%
- Impaired ability to prepare foods/meals related to recent hip fracture, AEB reduced mobility

Intervention

- Provision of commercial beverage product
- Increased fluid diet
- Liquid consistency—nectar-thick liquids
- Insert enteral feeding tube
- Feeding assistance
- Referral to Home-Delivered Meals Program

Monitoring and Evaluation

- Body weight
- PO intake (food, fluid)

CVA, cerebrovascular accident.

Patient History

Patient history is a significant area of assessment in the elderly, as a change in various functional abilities is correlated with morbidity and mortality. The typical daily functions are described as activities of daily living (ADLs) and instrumental activities of daily living (IADLs) (**Box 4.4**). Healthcare practitioners should ask questions regarding any change in the ADLs and IADLs of both the patient and the significant others. In addition, patient history risk factors include cognitive decline, depression, neurologic disease, dehydration, presence of infection and/or pressure injuries, and recent hospitalization.

Box 4.4 Activities of Daily Living and Instrumental Activities of Daily Living

ADLs	IADLs
Bathing	Doing light housework
Dressing	Preparing meals
Eating	Using the telephone
Maintaining continence	Managing money
Mobility indoors and outdoors	Shopping
Moving into and out of bed and chairs	Traveling
Toileting	Taking medications

Data from A Place for Mom. Retrieved from https://www.aplaceformom.com/blog/adls-and-iadls/. Accessed on June 2, 2023.

Estimation of Energy and Protein Needs

The use of energy expenditure equations, as described in Chapter 1, to determine energy needs is varied and depends on factors such as body weight and disease or condition. While these formulas can represent an easy starting point for estimation of energy needs, especially those that take into account age,[6] care must be taken when trying to incorporate body composition into estimation of resting energy expenditure as this can lessen the accuracy of the predictive equations.[7]

There is growing consensus that the elderly, those not affected by chronic kidney disease, may require higher protein intake to maintain "metabolic, physical, and functional status,"[8] with levels of 1.0 to 1.2 g/kg of body weight commonly recommended and used.[9] Research has also shown that protein intake can be low in nursing home residents at risk of malnutrition,[10] at which point dietary supplements may be considered.

Essential Nutrient Recommendations

The physiologic changes associated with aging affect the requirement for several essential nutrients. In general, the requirement for many nutrients decreases, concomitant with the decrease in energy needs. However, some nutrients are needed in higher amounts. Additionally, various psychosocial and socioeconomic changes that often accompany aging may also alter dietary intake (**Box 4.5**).[11]

Box 4.5 Psychosocial and Socioeconomic Problems Affecting Dietary Intake with Aging

Problem	Approach
Limited income	1. Buy low-cost food, such as dried beans/peas, rice, and pasta. 2. Use coupons for money off on foods used, and check store weekly circular to buy foods on sale. 3. Buy store-brand foods. 4. Check with local place of worship for free or low-cost meals. 5. Participate in local senior nutrition programs,[a] offered at senior congregate feeding sites or home-delivered meals. 6. Check for eligibility for the Food Stamp Program.[a] 7. Contact local food banks or emergency food program.[a]
Inability to grocery shop	1. Ask a friend or relative to grocery shop. 2. Ask a friend or relative to grocery shop. Contact local grocery store to bring groceries to your home. 3. Contact food delivery companies. 4. Check with local place of worship or senior center for volunteers who will shop. 5. Hire a home health worker (listed under “Home Health Services” in phone book).
Inability to prepare food	1. Use a microwave oven to cook frozen meals and foods. 2. Buy easily prepared nutritious foods (fresh fruits, whole grain breads, peanut butter, tuna in foil pouch). 3. Participate in local senior nutrition programs,[a] offered at senior congregate feeding sites or home-delivered meals. 4. Hire a home health worker (listed under “Home Health Services” in phone book) to cook meals

Problem	Approach
	(also make-ahead meals that can be frozen).
Psychological changes that cause poor appetite	1. The following can cause loss of appetite: living alone, having lost a spouse, feeling depressed—participate in senior meal programs, invite family or friends to share a meal; check with a doctor if depression continues. 2. If cooking for just one, you may not feel like making meals; invite family or friends for a meal. 3. Food may not have much taste, which could be psychological, physiologic, or because of medications; it may help to: a. Eat with family and friends or participate in senior meal programs. b. Ask the doctor if drugs could be affecting appetite or taste changes. c. Increase the flavor of food by adding spices and herbs.

[a]Programs are listed under “County Government” in blue pages of phone book; elder care locator: (800) 677–1116.

Data from Melissa Bernstein, Nancy Munoz, and Academy of Nutrition and Dietetics, “Position of the Academy of Nutrition and Dietetics: Food and Nutrition for Older Adults: Promoting Health and Wellness,” *Journal of the Academy of Nutrition and Dietetics* 112, no. 8 (2012): 1255–1277.

The age category for older adults is divided into two separate categories in the dietary reference intakes (DRIs) (**Tables 4.1–4.3**).[12] The adult age groups are 51–70 years and 70 and above, although the nutrient levels are the same for both adult categories for essential nutrients. One exception is the tolerable upper intake level (UL) for phosphorus, which decreases from 4,000 to 3,000 mg for both males and females at the higher age group. Two

nutrients of particular concern in the elderly are B_{12} and vitamin D.

TABLE 4.1 Dietary Reference Intakes for Older Adults for Macronutrients

Nutrient/Units	Males, 51 + yr	Females, 51 + yr
Carbohydrate, g	130	130
Protein, g	56	46
Total fat, g	ND; AMDR: 20–35% of total calories	ND; AMDR: 20–35% of total calories
Linoleic acid, g	14	11
α-Linolenic acid, g	1.6	1.1
Saturated fat, g	As low as possible while consuming a nutritionally adequate diet; dietary guidelines: <10% of total calories	As low as possible while consuming a nutritionally adequate diet; dietary guidelines: <10% of total calories
Cholesterol, mg	As low as possible while consuming a nutritionally adequate diet; DV: <300	As low as possible while consuming a nutritionally adequate diet; DV: <300
Fiber, g	30	21

ND, not determined. AMDR, Acceptable macronutrient distribution range.

TABLE 4.2 Dietary Reference Intakes for Older Adults for Vitamins

Nutrient/Units	Males				Females			
	DRI[a]		Tolerable UL		DRI		UL	
	51–70 yr	70+ yr	51–70 yr	70+ yr	51–70 yr	70+ yr	51–70 yr	70+ yr
Vitamin A, μg	900	900	3,000	3,000	700	700	3,000	3,000
Vitamin D, μg	15	20	100	100	15	20	100	100
Vitamin E, mg	15	15	1,000	1,000	15	15	1,000	1,000
Vitamin K, μg	120	120	ND	ND	90	90	ND	ND
Vitamin C, mg	90	90	2,000	2,000	75	75	2,000	2,000
Vitamin B_6, mg	1.7	1.7	100	100	1.5	1.5	100	100
Vitamin B_{12}, μg	2.4	2.4	ND	ND	2.4	2.4	ND	ND
Biotin, μg	30	30	ND	ND	30	30	ND	ND
Choline, mg	550	550	3,500	3,500	425	425	3,500	3,500
Folate, μg	400	400	1,000	1,000	400	400	1,000	1,000
Niacin mg	16	16	35	35	14	14	35	35
Pantothenic acid, mg	5	5	ND	ND	5	5	ND	ND
Riboflavin, mg	1.3	1.3	ND	ND	1.1	1.1	ND	ND
Thiamin, mg	1.2	1.2	ND	ND	1.1	1.1	ND	ND

ND, not determined.

[a]DRIs represent RDAs except for vitamin K, biotin, choline, and pantothenic acid (AI values).

Description

TABLE 4.3 Dietary Reference Intakes for Older Adults for Minerals

Nutrient/Units	Males				Females			
	DRI[a]		Tolerable UL		DRI		UL	
	51–70 yr	70+ yr	51–70 yr	70+ yr	51–70 yr	70+ yr	51–70 yr	70+ yr
Calcium, mg	1,000	1,200	2,000	2,000	1,200	1,200	2,000	2,000
Chromium, μg	30	30	ND	ND	20	20	ND	ND
Chloride, mg	2,000	1,800	3,600	3,600	2,000	1,800	3,600	3,600
Copper, μg	900	900	10,000	10,000	900	900	10,000	10,000
Fluoride, mg	4	4	10	10	3	3	10	10
Iodine, μg	150	150	1,100	1,100	150	150	1,100	1,100
Iron, mg	8	8	45	45	8	8	45	45
Magnesium, mg[a]	420	420	350	350	320	320	350	350
Manganese, mg	2.3	2.3	11	11	1.8	1.8	11	11
Molybdenum, μg	45	45	2,000	2,000	45	45	2,000	2,000
Nickel, mg	ND	ND	1	1	ND	ND	1	1
Phosphorus, mg	700	700	4,000	3,000	700	700	4,000	3,000
Potassium, mg	3,400	3,400	ND	ND	2,600	2,600	ND	ND
Selenium, μg	55	55	400	400	44	44	400	400
Sodium, mg[b]	1,500	1,500	2,300	2,300	1,500	1,500	2,300	2,300
Vanadium, mg	ND	ND	1.8	1.8	ND	ND	1.8	1.8
Zinc, mg	11	11	40	40	8	8	40	40

ND, not determined. DRIs represent RDAs except for chromium, chloride, fluoride, manganese, potassium, and sodium (AI values).
[a]For magnesium, the UL applies to synthetic forms obtained from drugs or supplements only.
[b]For sodium, the upper lever (UL) is replaced with the Chronic Disease Risk Reduction (CDRR) intake level.

Description

Data from large population surveys indicate that between 5 and 40% of elderly may be deficient in vitamin B_{12}, depending on the deficiency definition that is used, with long-term care residents having a slightly higher risk. One reason is that malabsorption from food sources is often the result of the reduction in the production of hydrochloric acid, achlorhydria, as it occurs in atrophic gastritis, a common problem in the elderly. Other causes of malabsorption

include long-term consumption of proton pump inhibitors (PPIs), histamine H_2 blockers, and chronic alcohol consumption.[13] The use of PPIs is also associated with an increased risk for fractures.[14] Vitamin D deficiency is more likely in aging due to low sun exposure, malabsorption related to chronic diseases such as inflammatory bowel disease, poor dietary intake, smoking, and exposure to certain drugs. Supplementation can reduce the risk of deficiency, especially in those residing in long-term care facilities.[15] Low serum vitamin D concentrations are associated with increased fracture risk and sarcopenia[16] and because of the concern for fractures, the Institute of Medicine revised the DRI for this nutrient in 2011 (Table 4.2).

Common Problems in Aging

Several problems common in aging affect key aspects of either dietary intake or nutritional needs and may adversely affect nutritional status. The most notable of these include dysphagia and pressure injuries, which, although not unique to the elderly, are highest in this population. The profoundly negative effects of these conditions on an elderly patient's nutritional status make nutritional assessment for their presence and/or severity imperative.

Dysphagia

Disease Process

Dysphagia represents both a symptom and a disorder, which affects one or all stages of the swallowing mechanism, making swallowing difficult. It affects 10 to 33% of older adults with the highest prevalence among the hospitalized and residents of long-term care facilities.[17] The possible causes are varied (**Box 4.6**), as are the symptoms (**Box 4.7**).[18] Depending on the cause, dysphagia may be an acute condition that resolves or it may be chronic. Early identification and intervention are critical not only because of the effect on the quality of life but also because of the increase in risk of other serious illnesses which may lead to longer hospital stays.[17]

Box 4.6 Causes of Dysphagia

Achalasia
Aging
Alzheimer's disease
Cancer, chemotherapy, radiation
Dementia
Eosinophilic esophagitis

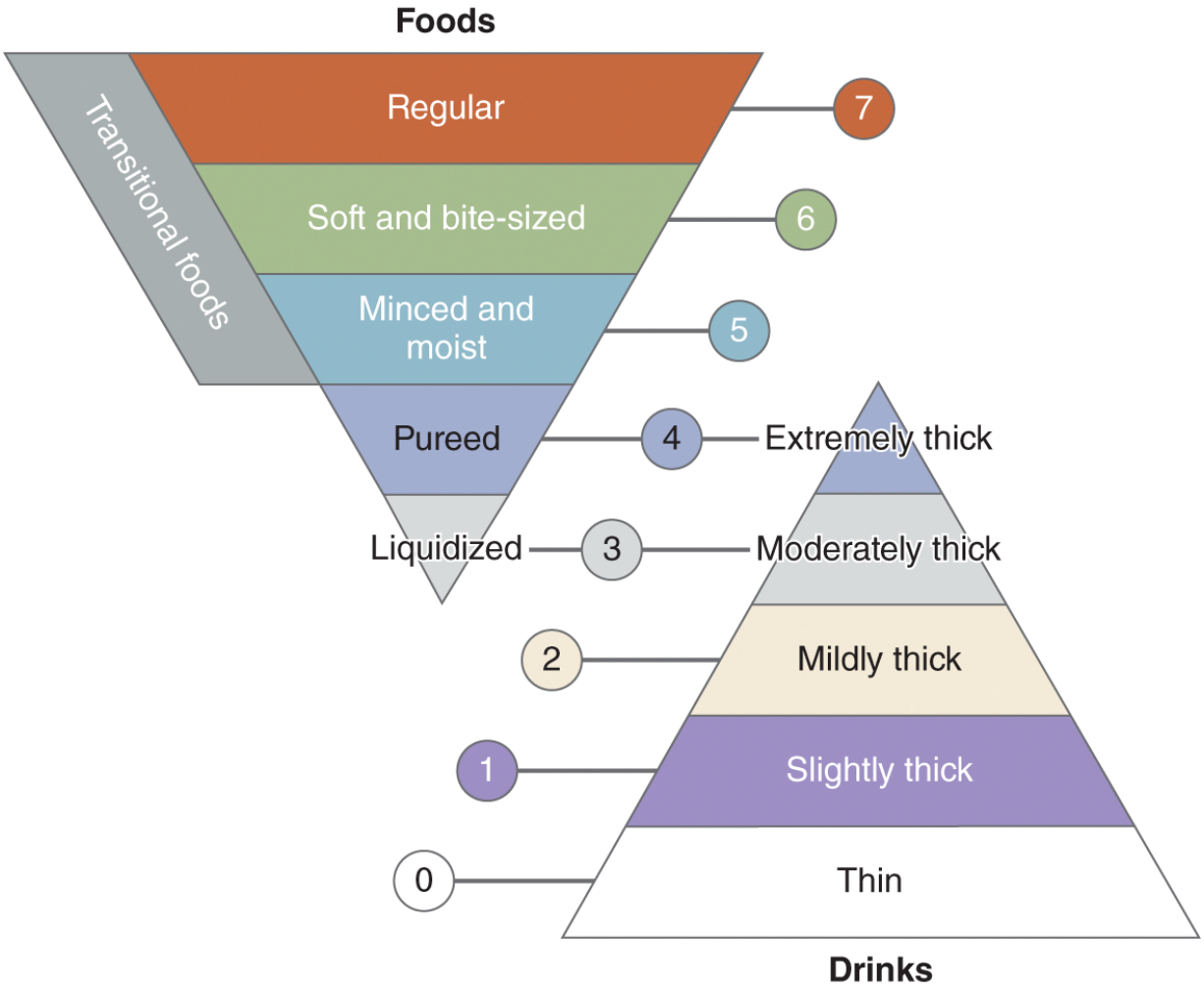
Head and neck surgery
Intubation
Multiple sclerosis
Neurologic disorders
Parkinson disease
Stroke
Diabetic neuropathy
Trauma to esophagus

Box 4.7 Symptoms of Dysphagia

- Pain while swallowing
- Inability to swallow
- A sensation of food getting stuck in the throat or chest
- Hoarseness
- Drooling, excessive secretions
- Food coming back up (regurgitation)
- Frequent heartburn
- Coughing or gagging when swallowing
- Weight loss

Treatment and Nutrition Intervention

The team approach is important in both assessment and intervention, and can include a swallow therapist, a primary care provider, and a registered dietitian nutritionist (RDN).[17] The International Dysphagia Diet Standardisation Initiative (IDDSI) is the most current medical nutrition therapy for the disorder.[19] It consists of a continuum of numbered, text-labeled, color-coded levels corresponding to the variations in thickness for liquids and textures for foods (**Figure 4.1**).



Description

Figure 4.1 IDDSI Dysphagia Diet Continuum.

Used with permission from IDDSI.

The IDDSI viscosity levels are established through highly objective testing methods using only basic equipment such as forks, spoons, and syringes. Avoided is the use of more sophisticated equipment such as rheometers and viscometers that are unavailable in most kitchens. Implementation is currently underway and is being achieved through the use of carefully planned IDDSI-provided schedules and in-service sessions by adopting institutions. The IDDSI standards are now recognized by the Academy of Nutrition and Dietetics and are included in their Nutrition

Care Manual along with transition instructions.[20] Transition to the IDDSI standards from those previously used is proceeding effectively, especially in long-term care facilities.[21]

Pressure Injuries

Disease Process

Pressure injuries, also known as pressure ulcers, decubitus ulcers, or bedsores, represent a point of skin breakdown from continual contact with a surface, such as a bed or wheelchair. This is significant to healthcare practitioners because up to 11% of patients admitted to long-term care facilities can develop a pressure injury,[22] and of particular significance is the fact that the treatment of pressure injuries is estimated to be as high as $22 billion annually in the United States.[23]

Treatment and Nutrition Intervention

Pressure injuries are assessed as being at one of four stages, relative to depth of tissue involvement and, therefore, severity (**Box 4.8**).[24]

Box 4.8 Pressure Ulcer Staging

Stage 1: Intact skin with non-blanchable redness of a localized area, usually over a bony prominence.
Stage 2: Partial-thickness loss of dermis presenting as a shallow, open ulcer with a red-pink wound bed, without slough. May also present as an intact or open/ruptured, serum-filled blister.
Stage 3: Full-thickness tissue loss. Subcutaneous fat may be visible, but bone, tendon, or muscle is not exposed. Slough may be present but does not obscure the depth of tissue loss and may include undermining and tunneling.
Stage 4: Full-thickness tissue loss with exposed bone, tendon, or muscle. Slough or eschar may be present on some parts of the wound bed. Often includes undermining and tunneling.

Data from National Pressure Ulcer Advisory Panel 2016. Available at https://www.npuap.org/national-pressure-ulcer-advisory-panel-npuap-announces-a-change-in-terminology-from-pressure-ulcer-to-pressure-injury-and-updates-the-stages-of-pressure-injury.

Inadequate dietary intake and poor nutritional status are major risk factors in both the development and prolonged healing of pressure injuries. The goals of nutrition intervention of pressure injuries are as follows:[25]

1. Provide adequate energy to maintain or regain lost weight: 30–35 kcal/kg increasing to 35–40 kcal/kg per day for people who are underweight or losing weight.
2. Provide adequate protein for positive nitrogen balance and to spare energy: 1.5–2.0 g/kg per day.
3. If vitamin and mineral deficiencies are confirmed or suspected, provide a multivitamin/mineral supplement that contains the DRI for micronutrients needed for wound healing.
4. The routine supplementation of zinc in the absence of confirmed or suspected deficiency is not recommended.
5. Fluid intake of 1 mL per calorie per day is recommended.

Greater success in both treating and preventing pressure injuries is more likely when a multidisciplinary approach is taken, especially in long-term care facilities and acute care settings. This can involve increasing awareness and education among clinical staff and patients by incorporating screening practices into routine patient care.[26]

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CHAPTER 5

Nutrition Support

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Patients who are unable to meet nutrition requirements with a conventional diet may need aggressive therapies and alternative means of nutrition. Alternative means of nutrition are enteral nutrition and parenteral nutrition.

Enteral Nutrition

Indications and Patient Selection

Enteral nutrition (EN) is the preferred route if the gut is functional because of fewer complications, fewer costs, and improved outcomes. Benefits of EN include increased nutrient utilization, maintenance of normal gut pH and flora, inhibiting opportunistic bacterial overgrowth, and support of gut mucosa's immunologic barrier function, which may decrease the risk of gut-related sepsis. Examples of clinical situations in which oral intake is deemed unsafe, insufficient, or impossible are patients with altered mentation, severe dysphagia, poor appetite, and respiratory failure requiring vent via endotracheal tube.[1,2,3] Indications and contraindications for EN can be found in **Box 5.1**.

Box 5.1 Indications and Contraindications for Enteral Nutrition Support

Indications

- Malnourished patient expected to be unable to eat for >5–7 days
- Normally nourished patient expected to be unable to eat for >7–9 days
- Functional or partially functional gut
- Adaptive phase of short bowel syndrome
- Following severe trauma or burns
- EN initiation within 24–48 hours in critically ill patients who are unable to maintain volitional intake
- Patients in the intensive care unit (ICU) setting with acute respiratory distress syndrome/acute lung injury expected to have a duration of mechanical ventilation ≥72 hours

Contraindications

- Aggressive intervention not warranted or not desired
- Severe short bowel syndrome
- Nonoperative mechanical gastrointestinal (GI) obstruction

- Intractable vomiting and diarrhea refractory to medical management
- Distal high-output fistulas (too distal to bypass with feeding tube)
- GI ischemia
- Paralytic ileus
- Severe GI bleed
- EN not required for patients who are at low nutrition risk with normal baseline nutrition status, have low disease severity in the ICU setting, and do not require specialized nutrition therapy over the first week of hospitalization
- Unable to gain enteral access
- Severe coagulopathies (disease related or medication induced) may have potential complications but generally do not preclude placement

Data from (i) Mueller C (ed.) *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017; (ii) Compher C, Bingham AL, McCall M, et al. Guidelines for the provision of nutrition support therapy in the adult critically ill patient: The American Society for Parenteral and Enteral Nutrition. *J Parenter Enteral Nutr*. 2022;46:12-41. https://doi.org/10.1002/jpen.2267; and (iii) Koseniecki M, Rebecca F. Enteral nutrition for adults in the hospital setting. *Nutr Clin Prac*. 2015;30(5):634-651.

Enteral Feeding Devices

Route of EN is the next step after determining whether the patient would benefit from nutrition support (see **Table 5.1**). Long-term access should be considered if the anticipated need will be >4–6 weeks. Enteral access devices include gastrostomy, jejunostomy, or gastrojejunostomy tubes.

TABLE 5.1 Enteral Access Devices

	Description	Pros	Cons and Complications
Short-term (<4 wk)			
Nasogastric tube (NGT): prepyloric tube	Used for feeding, decompression of stomach, administration of medications, measurement of gastric pH and residuals. At least 10F needed to avoid clogging from enteral formulas.	■ Variety of sizes available (8–24F) ■ Generally easy placement ■ Large-bore tube allows more versatility in feeding regimen ■ Gastric phase of digestion and nutrient metabolism	■ Contraindicated if nasal/facial fractures, severe coagulopathy, severe thrombocytopenia, esophageal obstruction ■ Complications include clogging, esophageal or sinus perforation, nasal mucosal ulceration, pneumothorax, epistaxis, pulmonary aspiration
Orogastric tube: prepyloric tube	Optional route when tube cannot be placed nasally (facial fracture or head injury).	■ Lower incidence of sinusitis	■ Tolerated for short periods of time ■ Complications are the same as with NGT, with exception of nasal-related events
Nasoenteric tube: postpyloric tube	Tip of tube placed past the pyloric sphincter. Radiologic verification or electromagnetic image of placement needed before use. Not appropriate to check residuals.	■ Small-bore tubes are more flexible and more comfortable ■ Less risk of aspiration as patient is able to lay flat, esophageal reflux, delayed gastric emptying if placement past distal third of duodenum	■ Infusion pumps necessary ■ Difficulty of postligament of Treitz placement ■ Difficulty of administering some medications
Long-term (>4 wk)			
Gastrostomy tube: prepyloric	Placed surgically, endoscopically (PEG), or radiologically into the stomach. Available sizes from 10 to 28F.	■ Allows bolus feeding ■ Allows gastric phase of digestion and nutrient metabolism ■ Low-profile tubes available	■ Complications may include dislodgment, bleeding, wound infection, tube occlusion, pneumoperitoneum, stomal leakage
Jejunostomy tube: postpyloric	Placed surgically, endoscopically (PEJ), or radiologically into the jejunum. Size of 9–12F tube. Not able to check residuals to assess tolerance. Requires infusion via a pump.	■ Reduces aspiration risk if the patient is required to lay flat (several studies have demonstrated no difference in aspiration risk between intragastric and small bowel feedings)[3] ■ Low-profile tubes available	■ Does not allow bolus enteral feeding ■ Complications may include wound dehiscence or infection, bowel obstruction, occlusion, bleeding, dislodgment, volvulus
Transgastric jejunostomy	Placed surgically, endoscopically, or radiologically. A jejunal	■ Can be converted to gastrostomy feeding as tolerated	■ Does not allow bolus enteral feeding ■ Complications of both

	Description	
	port is placed through gastrostomy tube, allowing infusion of enteral feeding into the small bowel while also suctioning gastric contents	jejunostomy and gastrostomy tubes

PEJ, percutaneous endoscopic jejunostomy.

Data from (i) Mueller CM, Ohman EM, *ASPEN Adult Nutrition Support Core Curriculum*, 3rd ed. American Society of Parenteral and Enteral Nutrition; 2018; (ii) Kreymann KG, Berger MM, Deutz NE, et al. ESPEN Guidelines on Enteral Nutrition: Intensive care. *Clin Nutr*. 2006;25(2):210-23; (iii) Bingham AL, McCall M, et al. Guidelines for the provision of nutrition support therapy in the adult critically ill patient: The American Society for Parenteral and Enteral Nutrition. *J Parenter Enteral Nutr*. 2022;46:12-41. https://doi.org/10.1002/jpen.2267; (iii) Kosenieckl M, Rebecca F. Enteral nutrition for adults in the hospital setting. *Nutr Clin Pract*. 2015;30(3):306-15; and (iv) Taylor KR, Etal, Victoria. Enteral feeding in critically ill patients in the transport setting. *J Parenter Enteral Nutr*. 2014;38(2):208-15.

ENFit Connection

Enteral tubing misconnections have led to several deaths and increased morbidity in survivors. Examples of enteral tube misconnections include enteral feedings infused into intravenous (IV) tubing, tracheal tubes, and nasal cannulas. In response to this preventable harm, the International Organization for Standards led to the Global Enteral Device Supplier Association (GEDSA) developing ENFit (ISO 80369-3 standard). ENFit can be described as a feeding tube connector, which can only fit with other enteral devices such as tubing on feeding pumps and syringes for medication administration. ENFit connectors have a female connector that fits around the male connector with a screwing motion, unlike the male “Christmas tree” connection into a female port, which can be connected to IV tubing.[4,5]

Enteral Formula Selection

Because of the large number of enteral formulas that are available commercially, a comprehensive list is not appropriate for a resource book of this size. A listing of contact information for enteral formula manufacturers is available at the end of this chapter.

Enteral Nutrition Delivery Methods

Once the route of access for EN has been established, dosing and method of delivery can be determined. **Box 5.2** shows a sample calculation for determining volume goal and administration methods. Gravity-controlled and pump methods are available. Enteral formulas are provided at full strength because of contamination risk. In critical care patients who are at high nutrition risk, enteral feeding is

usually started at a low rate and advanced by 10–20 mL/hr every 8–12 hours, a method proven to contribute to calorie and protein deficits. Many critically ill patients can tolerate an advancement to goal rate within 24 to 48 hours and studies have demonstrated no difference in tolerance when started at goal rate rather than advancing more slowly.[1]

Box 5.2 Sample Calculation for Determining Goal Enteral Nutrition Volume and Method of Administration

Patient's daily requirements based on nutrition assessment are 80 g protein, 2,000 kcal, and 2,000 mL fluid.

A standard polymeric formula provides 1.2 kcal/mL, 55.5 g protein/L, 820 mL free H_2O/L, and meets RDI for vitamins and minerals in 1,000 mL of formula.

- 2,000 kcal needed/1.2 kcal per mL = 1,667 mL formula required (1.7 L)
- 1.7 L × 55.5 g protein/L = 94 g protein provided with formula
- 1.7 L × 820 = 1,394 mL free H_2O provided with formula
- 2,000 mL daily fluid needs − H_2O provided in formula = 606 mL additional H_2O required

If formula is to be infused continuously via pump, divide the total formula needed by the desired duration of therapy: 1,667 mL formula/24 hrs = 69.5 mL/hr, which can round to 70 mL/hr ATC (around the clock) plus additional 200 mL H_2O flush 3×/day to meet daily requirements. Frequent interruptions of enteral feedings in the hospital setting have resulted in delivery of 50–60% prescribed daily enteral formula, which has led some institutions to prescribe formula by total volume needs instead of hourly rate.[6,7,8,9] An example would be infusing an enteral formula at 70–80 mL/hr until goal volume of 1,667 mL/day is reached.

If the formula is to be infused via intermittent or bolus method, divide total formula needed by desired number of daily feedings: 1,667 mL formula/4 feedings per day = 417 mL formula per feeding. Additional 75 mL H_2O flush before and after each feeding is needed to meet daily fluid requirements. Determine duration of each feeding from 15 to 45 minutes, which depends on individual tolerance.

RDI, Reference Daily Intake.

Bowel sounds do not need to be heard before initial feeding. Fluid and air must be present in the intestinal lumen to hear bowel sounds, which may not be heard if there is a nasogastric or percutaneous endoscopic gastrostomy (PEG) tube for suction or decompression. Delayed enteral nutrition will increase the risk of compromising the GI mucosal barrier and immune function.[1] If refeeding syndrome is a risk, EN should be initiated at 25% of goal rate and slowly advanced to the goal over 3–5 days, with daily monitoring of potassium, magnesium, and phosphorus.

Water flushes should also be added to the EN prescription to provide adequate hydration and decrease constipation. Water flushes can be provided by the bolus method, although some infusion pumps can be programmed to provide a timed dose.

Bolus or Intermittent

- Gravity-controlled delivery
- Best suited for gastric feeding
- Appropriate for patients who can protect their airway and are neurologically intact
- Most often used in the nonacute care or home care setting
- Advantages include the following: no pump required so more economical; more physiologic because the regimen closely mimics normal meals; allows for increased patient mobility
- Disadvantages include the following: may be poorly tolerated with diagnosis of gastroparesis, uncontrolled gastroesophageal reflux disease, or gastric outlet obstruction
- Administration:

- Bolus feeding: infused over approximately 15 minutes via gravity assist or syringe.[3] Tolerated volumes typically range from 240 to 720 mL per bolus, 2–6 times daily depending on individual tolerance. Initial bolus of 120 mL is generally tolerated, with advancement by 120 mL increments until goal volume is attained.
- Intermittent feeding: similar to bolus in total administration volume and frequency, with formula delivery over 30–45 minutes by gravity or pump assist[3].

Cyclic

- Pump-assisted delivery
- Can be used in the acute care or home care setting, especially when transitioning to an oral diet
- Advantages include the following: allows for maximal nutrient absorption; allows for gut rest; allows for increased mobility and time away from the pump
- Disadvantages include the following: requires high infusion rates of typically 50–250 mL/hr over 8–20 hours daily to meet full nutrition and fluid requirements, which can be poorly tolerated by some patients
- Administration: typically runs for 8–20 hours per day, which can be infused either during daytime or nocturnally
- Cyclic feedings via the jejunum may be limited to ≤90–100 mL/hr depending on individual tolerance

Continuous

- Pump-assisted delivery
- Can be used for gastric and transpyloric feedings

- Commonly used for patients who cannot tolerate bolus or intermittent feedings, for those requiring mechanical ventilation, and for critically ill patients
- Advantages include the following: possible decreased risk of distention compared with bolus/intermittent infusion
- Disadvantages include the following: requires pump delivery, which is more costly and limits patient mobility
- Administration: infuses for 24 hours per day, commonly referred to as “around the clock,” or ATC; can be initiated at 10–40 mL/hr, with advancement by 10–20 mL/hr every 8–12 hours as tolerated, until goal is reached

Monitoring of Enteral Feeding

Monitoring of GI tolerance, hydration status, and nutritional status of patients receiving EN is important. Protocol for monitoring EN may vary by institution; however, **Table 5.2** lists some common guidelines for monitoring EN.

TABLE 5.2 Enteral Nutrition Monitoring Guidelines

Parameter	Frequency
Weight	Before initiation and at least twice a week
Intake and output (I/O)	Daily
Stool output and consistency	Daily
Signs/symptoms of edema	Daily
Signs/symptoms of dehydration	Daily
Gastric residuals	SCCM/ASPEN no longer recommends checking GRV as standard practice for all patients receiving enteral feeding

Parameter	Frequency
Abdominal exam: if soft, firm, or distended	Daily
Serum electrolytes, blood urea nitrogen (BUN), creatinine	Daily until stable, then 2–3 times/wk
Calcium, magnesium, phosphorus	Daily until stable, then weekly
Glucose	Patients with diabetes: q6h Patients without diabetes: daily until stable, then weekly
Nitrogen balance	Weekly, if appropriate

Gastric Residuals

Monitoring gastric residual volume (GRV) has historically been done as an aid in assessing EN tolerance as well as aspiration risk. Gastric residuals are checked by withdrawing and measuring fluid and formula with a syringe via nasogastric tube (NGT) or gastrostomy tube. The fluid should be reinstilled into the stomach to prevent acid loss. Normal gastric secretions range from 3,000–4,000 mL/day, and residuals of 400–500 mL have shown to be tolerated without aspiration.[10] The SCCM/ASPEN guidelines do not recommend checking GRV. They also suggest if GRV is checked to avoid holding EN if GRV is ≤500 mL in absence of other signs and symptoms of intolerance.[11] Delayed gastric emptying can be caused by medications like narcotics or paralytics in which initiating a promotility agent such as metoclopramide or erythromycin may be beneficial. Maintaining the head of bed (HOB) >30–45 degrees may also aid with enteral feeding tolerance. If high residuals persist, verifying tube placement may be needed. Aspirating syringes must be at least 50 mL for small-bore tubes of 12F

or less because of pressure in which the tube may collapse. Residuals should not be checked when feeding via the jejunum.

GI Complications

Tube feeding or GI intolerance may be indicated by abdominal distension, fullness, and pain or cramping. Decreasing infusion rate, ensuring formula is at room temperature, verifying tube placement, and assessing formula osmolality can help with tolerance. If the patient has delayed gastric emptying or is on medications that slow peristalsis, high-fiber, and/or high-fat formulas may not be tolerated and should be adjusted. Nausea and vomiting may indicate tube migration, such as a balloon gastrostomy causing a gastric outlet obstruction.[1] While there is no universally accepted definition of diarrhea, a clinically relevant definition is any abnormal volume or consistency of stool. It has also been defined as more than 500 mL stool output every 24 hours or more than three stools per day for at least 2 consecutive days.[12,13] Potential causes of diarrhea are medications, fecal impactions, pathogenic bacteria, pancreatic insufficiency, short bowel syndrome, gut atrophy, and inflammatory bowel disease. If disease-related causes, medications, or pathogen-induced diarrhea have been ruled out, soluble fiber or antimotility agents may be beneficial. Elemental EN formulas may also aid nutrient absorption.

Transitioning to Discontinue Enteral Nutrition

Temporary EN support is often needed during the critical care process and should be discontinued as patients can adequately tolerate an oral diet. As oral diets are started, enteral feedings should be infused nocturnally via pump, infused between meals via bolus, or stopped more than 1 hour before meals to aid with appetite. When the patient is consuming 60–75% of energy and protein needs, the

clinician should consider stopping EN and removing the temporary enteral access device.[3] If there is long-term enteral access and suspect of future necessity, the feeding tube may be left in place.

Parenteral Nutrition

Indications and Patient Selection

Parenteral nutrition (PN) is a method of nutrition support in which provision of macronutrients, micronutrients, and some medications is infused directly into the bloodstream via a peripheral or central vein. PN is indicated when patients cannot meet their nutrient needs enterally, via either oral intake or enteral tube feedings, because of the GI tract being compromised. Indications/contraindications for PN are outlined in **Box 5.3**.

Box 5.3 Indications and Contraindications for Parenteral Nutrition Support

Indications for PN

- Ischemic bowel
- Paralytic ileus
- Short bowel syndrome with malabsorption
- Bowel obstruction
- High-output enterocutaneous fistula with inability to place EN tube distal of the fistula
- Intractable vomiting or diarrhea
- Chylous effusion in which very-low-fat diet/EN not feasible or has failed
- Persistent EN intolerance or inability to gain enteral access

Contraindications for PN

- Catabolic patients expected to have usable GI tract within 5–7 days
- Well-nourished patient expected to resume EN/oral diet within 7–10 days
- Duration of therapy expected <5–7 days
- Aggressive nutrition support not desired by the patient
- Patient’s prognosis does not warrant aggressive nutrition support
- Functional GI tract
- Anorexia or inability to ingest enough nutrients orally

Data from Mueller C (ed.) *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017.

Patient selection for who may benefit from PN relies on thorough nutrition assessment, determination of nutritional status, and the overall clinical condition and prognosis of the patient. Benefits of PN should outweigh the risks. For severely malnourished patients, PN is indicated when an impairment of the GI tract occurs. Patients who are not severely malnourished may tolerate a period of no nutrition for up to 7–10 days without adversely affecting their outcome. Postoperative patients who receive PN for >7–10 days receive maximal benefit.[14] Many risk factors are related to PN. Intravenous dextrose, lipid, and amino acids provide an optimal host for bacterial and/or fungal infections. Patients are also at risk for catheter-related infections and complications. Macronutrient, electrolyte, and mineral and fluid abnormalities are risk factors as are hyperglycemia, hepatic steatosis, and compromised renal clearance.

Parenteral Nutrition Access

Central Venous Access

Central venous access allows hypertonic, hyperosmolar medications, and nutrition therapy infusion into a large-diameter central vein via a central venous catheter (CVC). Subclavian, cephalic, jugular, femoral, and basilica veins are the most common sites for a CVC. Parenteral nutrition via CVC is indicated if anticipated need is >10–14 days and/or peripheral PN (PPN) would not be adequate or medically feasible. Types of CVCs are outlined in **Table 5.3**.

TABLE 5.3 Central Venous Catheter Types

Type of Access	Definition/Description	Length of Therapy
Nontunneled CVC	Single or multilumen catheter inserted preferably into the subclavian vein. Ease of removal and exchange for short-term therapy in an acute care setting.	Weeks
Tunneled CVC	Single or multilumen catheter inserted into the jugular, subclavian, or cephalic vein, then is tunneled in the subcutaneous tissue (i.e., Hickman/Broviac/Groshong). Secured CVC for long-term use, less infection risk than nontunneled CVC, ease of care.	Months to years
Peripherally inserted central catheter (PICC)	Single or multilumen catheter inserted from peripheral vein into large central vein. Use in acute care or outpatient settings. Routine heparin flushes and site care needed, not ideal for long-term home care.	Several weeks to months
Port	Single or dual lumen subcutaneous port with silicone septum, most commonly placed in anterior chest wall. Venous access via port with noncoring needle.	Months to years

Data from Mueller C (ed.) *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017.

Peripheral Venous Access

PPN is administered into a peripheral vein and is indicated for short-term therapy of up to 14 days. Standard peripheral cannulas require site rotation every 72–96 hours to decrease catheter-related complications.[15] Adequate veins are necessary, as well as ensuring that PPN solutions do not exceed 900 mOsm/L because of the risk of thrombophlebitis. Formulations for PPN require higher volume and lipid tolerance than central PN to lower osmolarity and more closely meet caloric and protein

requirements. See **Box 5.4** for calculating osmolarity of PPN solutions.

Box 5.4 Calculating Osmolarity of PPN Solutions[a]

1 g amino acid/L = 10 mOsm
1 g dextrose/L = 5 mOsm
1 g lipid (20% stock solution)/L = 1.3 mOsm
1 mEq − calcium gluconate/L = 1.4 mOsm
  − magnesium sulfate/L = 1.0 mOsm
  − potassium and sodium/L = 2 mOsm

Example calculation: PPN solution provides 1,790 kcal, 80 g lipid, 120 g amino acid, 150 g dextrose, 200 mEq sodium chloride, 8 mEq magnesium sulfate, 40 mEq potassium chloride, 5 mEq calcium gluconate for total volume of 3,000 mL daily.

1. 120 g amino acid ÷ 3.0 L = 40 g/L × 10 = **400 mOsm/L**
2. 150 g dextrose ÷ 3.0 L = 50 g/L × 5 = **250 mOsm/L**
3. 80 g lipid ÷ 3.0 L = 26.7 g/L × 1.3 = **35 mOsm/L**
4. 8 mEq magnesium sulfate ÷ 3.0 L = 2.7 mEq/L × 1 = **2.7 mOsm/L**
5. 5 mEq calcium gluconate ÷ 3.0 L = 1.67 mEq/L × 1.4 = **2.3 mOsm/L**
6. 240 mEq sodium chloride and potassium chloride ÷ 3.0 L = 80 mEq/L × 2 = **160 mOsm/L**

Total osmolarity = 850 mOsm/L

[a] Osmolarity of nutrients may vary slightly among institutions.

Nutrition Requirements: Indirect Calorimetry

Indirect calorimetry is one of several methods in which metabolic requirements of macronutrients are determined in the critically ill. Predictive equations such as those of Penn State, Mifflin–St. Jeor, and Harris–Benedict are useful but can have significant variance in results. Indirect calorimetry measures oxygen (O_2) and carbon dioxide (CO_2) and calculates both resting energy expenditure (REE) and macronutrient utilization or respiratory quotient (RQ), which is the ratio of CO_2 produced to O_2 consumed. Although

changes in RQ do correlate with the percent calories provided, poor sensitivity and specificity limit its use as an indicator of under- or overfeeding. The physiologic range for RQ in humans is 0.67–1.3; therefore, an RQ outside this range may indicate an invalid study.[14] **Box 5.5** reviews technical aspects, which may alter indirect calorimetry results.[15]

Box 5.5 Aspects That May Alter Indirect Calorimetry Results

- Mechanical ventilation with F_{io_2} ≥60% and/or positive end-expiratory pressure (PEEP) >12 cm H_2O
- Acute changes of ventilation (if vent changes, wait 90 minutes to complete study)
- Leak in sampling system
- Inability to collect all expiratory flow (example of air leak via chest tube or bronchopleural fistula)
- Hemodialysis in progress (wait 3–4 hours after dialysis to complete the study)
- Error in calibration of indirect calorimeter
- General anesthesia given within 6–8 hours prior to the study
- Painful procedure recently completed (wait 1 hour after procedure to complete the study)

Data from Mueller C (ed.) *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017.

Parenteral Formulation: Macronutrients

Carbohydrate

Carbohydrate (CHO) is the primary source of energy for the body, including the brain and central nervous system. Minimum CHO requirements recommended per dietary reference intakes (DRIs) are 130 g/day for healthy adults and children.[16] CHO infusion should not exceed 4 mg/kg/min in critically ill patients and 7 mg/kg/min in stable patients

(**Box 5.6**). Risks of excessive CHO infusion include hyperglycemia, glucosuria, lipogenesis, hepatic steatosis, and hyperinsulinemia.

Box 5.6 Calculating Maximum CHO (g) Oxidative Capacity

To calculate maximum dextrose (g):

1. 4–7 mg × wt (kg) × 1,440 min[a] = mg/day
2. mg/day ÷ 1,000 = g dextrose/day

Example: A critically ill patient weighs 73 kg.

1. 4 mg × 73 kg × 1,440 min = 420,480 mg/day
2. 420,480 mg/day ÷ 1,000 = **420 g/day dextrose**

[a] 60 min/hr × 24 hr/day = 1,440 min/day.

Dextrose monohydrate is the CHO source used for PN, which provides 3.4 kcal/g. Stock or base solutions of dextrose range from 5 to 70%. Percentage dextrose concentration is grams of solute per 100 mL of solution. A 10% dextrose solution contains 10 g of dextrose per 100 mL solution, thereby providing 100 g dextrose/L.

Protein

Protein in PN is provided in the form of a crystalline amino acid solution in which standard solutions contain a physiologic mixture of essential and nonessential amino acids. Specialty amino acid formulations should be reserved for patients expected to benefit clinically from their use; indications for these formulations are very limited.[14] Examples of disease-specific solutions are NephrAmine for renal failure and HepatAmine for liver disease. Amino acids are required in PN to minimize lean body mass losses, promote tissue repair, and maintain oncotic pressure in blood plasma. Protein requirements range from 1.2 to 2.0

g/kg/day in critically ill patients and 0.8 to 1.0 g/kg/day in stable patients. Protein needs may exceed >2.0 g/kg if there are extreme losses such as in weeping wounds or large body surface area burns. Amino acids provide 4 kcal/g, with stock or base solutions ranging from 3 to 20%.

Lipid

Intravenous fat emulsions (IVFEs), specifically long-chain fatty acids (LCFAs), are required to prevent essential fatty acid deficiency (EFAD), which can occur within 1–3 weeks of lipid-free nutrition administration. LCFAs are available in concentrations of 10, 20, and 30%. IVFEs contain egg phosphatides as an emulsifier and glycerol for stability. A 10% IVFE concentration provides 1.1 kcal/mL, whereas a 20% concentration provides 2.0 kcal/mL or 10 kcal/g. Minimum LCFA requirements are 3 to 4% total kcal, with a DRI equating to about 10% total kcal to prevent EFAD. Risks of excessive or too rapid infusion of IV lipids include impairment of clearance as well as compromised reticuloendothelial or immune function. Limiting IV lipid to 1 g/kg/day or 30% or total kilocalorie requirements is recommended. IV lipids in PN can be increased and are tolerated if serum triglycerides are ≤400 mg/dL. Alternative IVFE, which includes fish oil, olive oil, and medium chain triglycerides in addition to long chain triglycerides were FDA approved on 7/13/2016 and are now available in the United States. These alternative lipids may potentially improve anti-inflammatory responses and decrease parenteral nutrition-associated liver disease.[17,18]

Parenteral Formulation: Total Volume

Formulation of PN includes total volume required to meet estimated fluid needs of 25–40 mL/kg/day. Fluid requirements in the critically ill are dependent on total fluid status and organ function. Minimum volume of PN in the

critically ill is often required, as there is usually a simultaneous mode of IV fluids that can be adjusted pending the patient's fluid status. Fluids provided by separate IV fluids, medications, and drips should be subtracted from total fluid requirements in determining total volume requirements for PN. Additional fluids may be required if there are excessive losses via diarrhea, vomiting, or fistula drainage. Estimation of total body water (TBW) and TBW deficit can assist with estimating fluid needs in PN formulation (**Box 5.7**).[14,19]

Box 5.7 Estimating Total Body Water and Total Body Water Deficit

TBW Estimation

TBW = 0.6 × wt (kg) males; 0.5 × wt (kg) for males ≥80 years of age
= 0.5 × wt (kg) for females; 0.4 × wt (kg) for females ≥80 years of age
Subtract 10% for obese, 20% for very obese

TBW Deficit Estimation

Water deficit (L) = TBW × [Na^1/Na^2) – 1]
Na^1 = actual serum sodium
Na^2 = desired serum sodium (140 mEq/L typically used)

Data from (i) Mueller C (ed.) *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017, and (ii) Kingley J. Fluid and electrolyte management in parenteral nutrition. *Support Line*. 2005;27(6):13-22.

Types of Parenteral Nutrition

PN solutions comprise protein, CHO, electrolytes, vitamins, minerals, medications, and sterile water. IVFEs infused separately, or "piggy-backed," are referred to as "2-in-1" solutions. IVFEs admixed with other nutrients and additives are "3-in-1" solutions, or total nutrient admixtures (TNAs). Advantages of TNAs are decreased contamination, decreased nursing time, decreased pharmacy preparation

time, overall decreased cost, and better fat utilization. Disadvantages of TNAs include decreased stability of the fat emulsion and compatibility with other components. Components of PN must be compounded in a specific sequence for optimal stability.[15]

Sample calculations for macronutrients in both 2-in-1 and TNAs are given in **Box 5.8**. Specifying quantity of needed amino acid (AA), dextrose, and lipid can be ordered in grams or percentage of solution depending on the facility, however some publications recommend using grams as a safer method. Ordering nutrients and additives either per day or per liter also varies between facilities, although safe practice guidelines are standardized PN formulations in which nutrients should be in amounts per day.[20] An exception to ordering per day is if the facility has admixed PN solutions in 1-L volumes in which using quantity per liter is supported. **Table 5.4** may aid with calculating the PN formulation, as it displays concentrations of dextrose, amino acids, and lipids, as well as what they provide.

Box 5.8 Sample Calculation for 2-in-1 and TNA PN Formulations

Patient's daily requirements based on nutrition assessment are 80 g protein, 2,000 kcal, 2,000 mL fluid with maximum CHO oxidative capacity of 385 g dextrose/day.
2-in-1: Facility options are:

AA solutions: 10% or 15%

Dextrose solutions: 20%, 30%, 40%, 50%, or ___%
Lipid: 10% (500 mL) or 20% (500 mL) ___ daily or ___ × per week

1. Determine needed lipids/day and what the solution provides:
   a. **10% lipid/day** would provide 500 mL volume, 50 g lipid, and 550 kcal.
2. Assess the best AA volume needed to meet AA (g) requirements:
   a. 80 g AA ÷ **10% AA concentration** = 800 mL volume and 320 kcal.
3. Calculate needed dextrose (g) by subtracting kcal from daily lipids and AA solutions:

a. 2,000 kcal − 550 kcal (lipid) − 320 kcal (AA) = 1,130 kcal from dextrose needed.
b. 1,130 kcal ÷ 3.4 = 332 g dextrose needed/day.

4. Assess the best dextrose volume needed to meet dextrose (g) requirements:
   a. 332 g dextrose ÷ **50% dextrose concentration** = 664 mL volume.
   b. Round to 700 mL volume to provide 350 g dextrose and 1,190 kcal.
5. Calculate infusion rate per hour from total AA and dextrose volumes:
   a. 800 mL (AA) + 700 mL (dextrose) = 1,500 mL volume.
   b. 1,500 mL ÷ 24 hours = **62.5 mL/hr** AA and dextrose.
   c. Separate 10% lipid infusion (500 mL)* 12 hours = 41.6 mL/hr of lipids.
6. PN goal = 62.5 mL/hr* 24 hours from 800 mL of 10% AA and 700 mL of 70% dextrose. Piggy back 500 mL of 10% lipid at 41.6 mL/hr* 12 hours. This provides daily total 2060 kcal, 80 g AA 350 g dextrose, 50 g lipid, and 2,000 mL volume.

TNA: Macronutrients are ordered in grams per day for this facility.

1. Calculate kilocalories provided by goal AA.
   a. **80 g AA** × 4 = 320 kcal
2. Calculate kilocalories provided by desired lipid (g). Typically, initial lipid dose is 30% total kcal requirements.
   a. 2,000 kcal × 30% (.30) = 600 kcal from lipid
   b. 600 kcal ÷ 10 kcal/g = **60 g lipid**
3. Determine goal dextrose (g) needed by subtracting kilocalories from AA and lipids.
   a. 2,000 kcal − 320 kcal (AA) − 600 kcal (lipid) = 1,080 kcal needed from dextrose
   b. 1,080 kcal ÷ 3.4 kcal/g = 317 g dextrose (round to **320 g dextrose** for 1,088 dextrose kcal)
4. PN goal: 80 g AA, 320 g dextrose, 65 g lipid to provide 2,008 total kcal in 2,000 mL volume.

TABLE 5.4 Macronutrient Composition of Solutions

Macronutrient	Concentrations (%)	Grams per Liter	Kilocalories per Liter
Dextrose	5	50	170
	10	100	340
	20	200	680
	30	300	1,020
	40	400	1,360
	50	500	1,700
	70	700	2,380
Amino acids	8.5	85	340
	10	100	400
	15	150	600
	20	200	800
	Concentration	**Kilocalories per Milliliter**	**Kilocalories per Liter**
Lipid emulsions	10	1.1	1,100
	20	2	2,000
	30	3	3,000

Description

Premixed PN is another method of parenteral compounding, which is becoming popular at many institutions. Dual or triple compartment premixed PN products provide fixed nutrient profiles of dextrose, amino acids, lipid, and micronutrients, while dual compartment premixed PN products do not contain lipids. Benefits of premixed PN include decreased compounding time, decreased delay of delivery and therapy initiation, and decreased risk of blood stream infections due to decreased manipulation of additives. Standardized compounding also results in decreased cost while providing a longer shelf life of 2 years at room temperature if the product remains in overwrap. Disadvantages of premixed solutions include potential inability to meet individual patient needs when high amino acids, low volume, and/or nonstandard

electrolyte profiles are required. Premixed PN labeling identifies the amount of nutrients per bag. After assessing the patient's nutrient needs, the clinician should calculate the total mL in the required number of "bags," then divide by the number of hours infused, which provides the goal infusion rate (mL/hr). Various options of macro- and micronutrients can be compounded based on the individual institution's patient population needs; therefore, dosing of premixed PN varies by the institutions' ordering process.[21,22,23]

Parenteral Formulation: Micronutrients and Additives

Electrolytes

Altered serum electrolyte values are common in the acutely ill, and PN formulations can aid with correcting these abnormalities. Assessing current IV electrolyte administration is needed before developing the PN formulation. **Table 5.5** shows the composition of intravenous solutions. **Table 5.6** reviews daily electrolyte requirements, what factors may alter electrolyte values, and dosage form.[14,15] Formulation of PN is to maintain electrolyte balance; therefore, repletion of an electrolyte deficiency should be treated with a separate IV bolus. Acetate forms of potassium and sodium are useful when serum CO_2 is low and/or serum Cl^- is elevated in which NaCl or KCl would not be beneficial. Acetate is the precursor to bicarbonate, which is then converted to CO_2 by the liver. Calcium phosphate solubility is a compatibility concern because these precipitated salts have been associated with respiratory distress and death.[14] Various factors influence calcium phosphate solubility (i.e., amino acid type and final pH of the solution); therefore, it is crucial for communication with

the compounding pharmacist when formulating a PN solution.

TABLE 5.5 Standard IV Solutions

IV solution	Glucose (g/dL)	Na^+ (mEq/L)	Cl^- (mEq/L)	K^+ (mEq/L)	CA^{2+} (mEq/L)	Lactate (mEq/L)	Osmolarity (mOsm/L)
5% dextrose in water	5	—	—	—	—	—	252
10% dextrose in water	10	—	—	—	—	—	505
0.45% NaCl	—	77	77	—	—	—	154
0.9% NaCl (normal saline)	—	154	154	—	—	—	308
5% dextrose in 0.45% NaCl	5	77	77	—	—	—	406
10% dextrose in 0.9% NaCl	5	154	154	—	—	—	560
Lactated Ringer's	—	130	109	4	3	28	273

Data from Mueller C (ed.) *The A.S.P.E.N. Adult Nutrition Support Core Curriculum.* 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017.

Description

TABLE 5.6 Daily Electrolyte Requirements in Formulation of Parenteral Nutrition

Electrolyte	Daily Requirement (Adult)	Causes of Elevated Levels	Causes of Decreased Levels	Dosage Form
Calcium	10–15 mEq	Vitamin D excess, renal failure, tumor lysis syndrome, hyperparathyroidism, prolonged immobilization and stress, bone cancer	Decreased vitamin D intake, hypoparathyroidism, hypoalbuminemia, hypomagnesemia, citrate binding of calcium with blood product administration	Ca gluconate
Magnesium	8–20 mEq	Excessive Mg intake in renal insufficiency	Refeeding syndrome, alcoholism, diuretic use, nasogastric suction, diabetic ketoacidosis, elevated stool output, Mg wasting medications	Mg sulfate
Phosphorus	20–40 mmol	Excessive phosphate administration, renal failure	Refeeding syndrome, alcoholism, inadequate intake	Na phosphate K phosphate
Sodium	1–2 mEq/kg	Inadequate free water, excessive water loss, excessive sodium intake, hyperaldosteronism	Excessive hypotonic fluid, nephritis, adrenal insufficiency, congestive heart failure, syndrome of inappropriate antidiuretic hormones (SIADHs), cirrhosis with ascites	Na chloride Na acetate Na lactate
Potassium	1–2 mEq/kg	Renal dysfunction, excessive K^+ intake, metabolic acidosis K^+-sparing medications	Refeeding syndrome, inadequate K^+ intake, excessive losses with diarrhea or intestinal fluids, diuretic medications, hypomagnesemia, metabolic alkalosis	K phosphate K acetate K chloride

Data from (i) Merritt R. (ed) *The A.S.P.E.N. Nutrition Support Practice Manual*, 2nd ed. American Society of Parenteral and Enteral Nutrition, 2005, and (ii) Mueller C (ed.) *The A.S.P.E.N. Adult Nutrition Support Core Curriculum*. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017.

Description

Vitamins, Minerals, and Trace Elements

Standard parenteral multivitamin and multiple–trace element supplements are included in the PN admixture. Multivitamin solutions are based on the U.S. Food and Drug Administration (FDA) daily requirements. Multivitamin formulations are available with or without vitamin K. Once the multivitamins are added, the solution is stable for 24

hours. Trace elements contain a minimum of chromium, copper, manganese, and zinc. Some trace element additives also contain iodine, molybdenum, and selenium. Trace elements should be held if conjugated bilirubin exceeds >2.0, as copper and manganese toxicity can result. Iron dextran is incompatible with IVFE and can only be added to 2-in-1 PN solutions with caution. Separate IV infusion of iron dextran is a preferred method of administration, if needed.

Other Additives

Additional additives in PN formulation can also include Sandostatin, insulin, GI prophylaxis such as Famotidine, and heparin. Regular insulin can be added starting at 0.1 unit (U) per gram of dextrose per liter of PN solution. Increasing insulin by 0.05 U per gram of dextrose per day may be needed if hyperglycemia continues, until 0.25 U per gram of dextrose is reached.[14,15]

Not all insulin provided in PN is utilized, as some will adhere to the plastic bag and tubing. Heparin is often added to PPN in 1 U/mL dose as prophylaxis against peripheral vein thrombophlebitis.

National Nutrient Shortages

Various national IV nutrient shortages have posed a challenge when altered bowel function does not allow the provision of nutrients by an alternative mode. Premixed PN solutions may be of benefit during periods of a national shortage due to the ability to compound the nutrients prior to the shortage, and the fact that they are shelf-stable for 2 years. Communication with the infusion company or compounding pharmacist is crucial to improvise the PN solution based on patient needs and alternate IV nutrient availability. If specific nutrients are not available to compound into the PN solution, and premixed PN is not an option, regular blood level monitoring with separate IV bolus is another option.

Initiation, Monitoring, and Discontinuation of PN

Initiation

PN can be initiated in adult patients when they are hemodynamically stable and, ideally, if they have satisfactory hydration, electrolyte, and acid–base status.[15] Macronutrients of PN can be advanced to goal by day 2 or 3 if serum glucose is consistently ≤180 mg/dL and electrolytes are satisfactory (**Table 5.7**).

TABLE 5.7 Initiation of Parenteral Nutrition

Macronutrient	Initiation Amount	Precautions
Amino acid	• 60–70 g/L	• Infusion of PN should be completed within 24 hours of initiation.
Dextrose	• 100–150 g/L or 10–15% final concentration with glucose intolerance, risk for hyperglycemia as with steroid therapy, or diabetes • 200 g/L or 15–20% final concentration as maximum initial amount	• Initiate with caution because of metabolic side effects, including reactive hyperglycemia, hyponatremia, hyperinsulinemia, glucosuria • Infusion of PN should be completed within 24 hours of initiation.
Lipid (IVFE)	• Can initiate full concentration as long as precautions are met	• Administer if baseline serum triglycerides are <200 mg/dL. • When infused separately as in a 2-in-1, infusion should be

Macronutrient	Initiation Amount	Precautions
		completed within 12 hours.

Monitoring

Monitoring PN tolerance is necessary for achieving caloric and protein goals as well as prevention of metabolic complications. **Table 5.8** provides appropriate frequency of lab monitoring.[14] Hypertriglyceridemia >400 mg/dL or a rise of ≥50 mg/dL indicates compromised clearance in which temporary discontinuation or decreased infusion rate of IVFE is warranted. Withholding IVFE is appropriate if triglycerides are >500 mg/dL. Topical linoleic acid via soybean or safflower oil can be an alternative method of preventing essential fatty acid deficiency without exacerbating hypertriglyceridemia.

TABLE 5.8 Monitoring for Adult Patients on Parenteral Nutrition

Parameter	Baseline	Critically Ill Patients	Stable Patients
Electrolytes: Na, K, Cl, CO_2, phosphorus, BUN, Cr	Yes	Daily	1–2 × per week
ALT, AST, ALP, total bilirubin	Yes	Weekly	Monthly
Serum triglycerides	Yes	Weekly	Weekly
Complete blood count with differential	Yes	Weekly	Weekly
INR, Prothrombin time (PT), partial thromboplastin time (PTT)	Yes	Weekly	Weekly
Capillary glucose	3×/day	3×/day (until consistently <150 mg/dL)	As needed
Weight	If possible	Daily	2–3×/wk
Intake and output	Daily	Daily	Daily unless fluid status is assessed by physical exam
Nitrogen balance	As needed	As needed	As needed
Indirect calorimetry	As needed	As needed	As needed

Data from Mueller C (ed.) *The A.S.P.E.N. Adult Nutrition Support Core Curriculum.* 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017.

Description

Elevated or increasing liver function tests (LFTs) from baseline can indicate hepatic steatosis in which provision requires infusion at 50% goal for the first and last hour of infusion. PN can be initially decreased to an 18- to 20-hour infusion and further decreased to a 12-hour infusion if there is good glycemic control. Glucose should be monitored before, during, and after PN cycle until glucose tolerance is established.

Refeeding Syndrome

Metabolic side effects of PN can occur, which may delay reaching nutrition goals. Refeeding syndrome can occur with malnourished patients who have anorexia nervosa, have had extreme weight loss, or have been without nutrition for 7–10 days. Refeeding syndrome occurs as the primary fuel

source converts from stored fat to CHO because energy is provided after starvation. CHO as the fuel source causes insulin levels to rise, which leads to intracellular shifts of potassium, magnesium, and phosphorus. Rapid decrease of serum potassium, magnesium, and phosphorus may lead to respiratory distress, tetany, cardiac arrhythmias, paresthesia, cardiac arrest, and sudden death. Prevention of refeeding syndrome can be accomplished by correcting electrolyte abnormalities before PN administration, initiating PN at half energy requirements, 15 to 20 kcal/kg, or 1,000 kcal/day.[1,15] Gradually increasing PN to goal nutrients over 5 to 7 days, as well as estimating caloric and protein goals based on actual weight, may also prevent initial overfeeding and refeeding syndrome.

Discontinuation of PN

Discontinuation of PN is the ultimate goal as patients are able to tolerate an oral diet or EN support. Well-nourished individuals prior to PN therapy, who are free of malignancy, not debilitated, or without oral intake for <2 weeks, can have PN stopped as soon as diet tolerance is established.[1] Transitional weaning of PN is necessary in patients who are at higher risk of oral or EN intolerance or suboptimal nutrition intake. PN infusion can suppress appetite if providing >25% of caloric needs. PN can begin to be decreased in comparable amounts as soon as patients are eating 500 kcal daily, with discontinuation of PN when oral or enteral provisions equal >60% of nutrition requirements. Calorie counts can be beneficial to estimate intake more accurately. EN support should be considered if oral intake is inadequate in meeting nutrition requirements within a few days. Transition from PN to EN support should be done gradually. For example, once EN therapy is tolerated and providing approximately 50% of estimated daily nutrition needs, PN therapy can be decreased by 50%. When EN is providing >75% of the patient’s requirements, PN can be

discontinued. Rebound hypoglycemia can occur if PN is stopped abruptly; therefore, decreasing infusion rate by 50% for 1 to 2 hours before discontinuation is suggested. Blood glucose levels can be checked 30 to 60 minutes after central PN cessation if patient is at risk or has signs of hypoglycemia.

Nutrition Support in the Home

Home parenteral nutrition (HPN) and home enteral nutrition (HEN) may be indicated, provided that patients receive thorough education on care of access devices, nutrition delivery, support contacts, and an emergency plan. Coordination from multiple disciplines including nurses, dietitians, physicians, and medical social workers help ensure education has been provided and that the patient and/or caregivers are able to follow safe nutrition infusion to help prevent complications and hospital re-admissions. Insurance coverage, therapy cost, along with necessary documentation for insurance coverage should be evaluated prior to discharge to the home setting.[24]

TABLE 5.9 Enteral Formula Manufacturers

Company Name	Phone Number	E-mail/Website
Abbott Nutrition	800-227-5767	http://www.abbottnutrition.com
Hormel Health Labs	888-617-3482	http://www.hormelhealthlabs.com
Nestle Nutrition	800-422-2752	http://www.nestlehealthscience.us

ADIME 5.1 ADIME At-A-Glance

Assessment

- Admission Dx and pertinent PMH, home diet or EN regimen PTA and what nutrients EN was providing, intake from PO diet
- If EN support, tube access and tube patency
- Medications (vitamin/mineral supplements, antinausea, promotility agents, medications in liquid or crushable tablet form)
- Labs (electrolytes, albumin, prealbumin, CRP, hemoglobin A1C)
- Bowel function (Last BM, R/O constipation, diarrhea, ileus or obstruction)
- Physical findings: edema, skin integrity (pressure ulcers, skin around feeding tube site), NFPE findings (loss of muscle mass/fat stores); vitals, functional status
- Anthropometrics (wt history, BMI)

Nutrition Diagnosis

- Inadequate EN infusion RT dislodged gastrostomy feeding tube AEB EN on hold × 2 days with plan for new enteral access within 24–48 hours
- Inadequate EN infusion RT increased nutrient requirements with sepsis and skin breakdown AEB current EN regimen meets 80% estimated kcal and 60% estimated protein requirements
- Swallowing difficulty RT oropharyngeal dysphagia s/p CVA AEB + S/S aspiration on all food consistencies per speech pathology findings and recommendation for long-term EN access

Intervention & Recommendations

- State calculated estimated nutrient daily requirements
- EN recommendations
- Addition of multivitamins and minerals
- Nutrition-related medication management (fiber modular, probiotic, promotility agents, anti-emetic agents)
- Instruction for EN administration and requirements
- Communication with multiple medical disciplines (Speech Language Pathology, social work and discharge planning, physicians)

Monitoring and Evaluation

- Patient able to tolerate 100% estimated EN requirements within 3–5 days
- No signs/symptoms of refeeding syndrome
- Consistent BM without abdominal distention, constipation, or diarrhea
- Enteral tube remains functional
- Gradual weight gain of 0.5–1 lb weekly, BMI to improve
- Labs to return to normal limits
- Wound healing

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PART 2

Nutrition Considerations for Specific Diseases



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CHAPTER 6

Bariatric Surgery

Lindsey Battistelli, MS, RDN

Obesity has been recognized as a disease entity by the American Medical Association and not simply an issue of overeating and lack of control. The World Health Organization (WHO) and the Institute of Medicine, as well as other scientific organizations, recognize the metabolic consequences of obesity in high-risk individuals for whom, depending on the severity of the obesity, they may be life-threatening. Given the strong evidence that a high percentage of people who lose weight using conventional methods will regain it, bariatric surgery is considered a safe and effective treatment for long-term weight loss and maintenance.[1]

Bariatric Procedures

When nonsurgical methods have failed to produce weight loss and sustained maintenance, bariatric surgery may be indicated for patients with class III obesity (body mass index [BMI] ≥40) without comorbidities, or class II obesity (BMI ≥35) with at least two specified comorbidities: type 2 diabetes, heart disease, hypertension, dyslipidemia, obstructive sleep apnea, or other respiratory disorders, nonalcoholic fatty liver disease, gastrointestinal (GI) disorders, or osteoarthritis. The guidelines include the surgical option for patients with BMI of 30–34.9, or class I obesity, who have not had success with long-term weight management through nonsurgical methods, and whose comorbidities may be exacerbated, such as diabetes.[2]

Bariatric surgery procedures have evolved and improved over time. The most common procedures performed today include the Roux-en-Y gastric bypass (RYGB), the sleeve gastrectomy (SG), the adjustable gastric band (AGB), and the biliopancreatic diversion with or without duodenal switch (BPD-DS).[3] Since 2008, the SG has advanced from being an investigative procedure to being comparable to the gastric bypass or gastric band. Most bariatric procedures are performed laparoscopically rather than through a large open incision. This decreases early postoperative complications and enhances recovery. Bariatric surgery is considered a safe and effective treatment for those who meet the criteria. Several procedures are in use, and they vary with regard to the potential nutrient deficiencies that may arise from the specific procedure (see **Table 6.1**).

TABLE 6.1 Bariatric Surgery Procedures

Procedure	Also Known As	How the Procedure Works	Potential Nutrient Deficiencies
Roux-en-Y gastric bypass	"Roux-en-Y" or "Gastric Bypass," "the Bypass"	Creating a small stomach pouch approximately 30 cc (30 mL) in size and thus bypassing the majority of the stomach and the first portion of the small intestine, including the duodenum.	Iron, vitamin B_{12}, calcium, folate, vitamin D, thiamin
Biliopancreatic diversion with (or without) duodenal switch	"the Switch," "BPD-DS" "BPD"	Majority of stomach is removed, and the distal part of the small intestine is connected to the pouch, bypassing the duodenum and jejunum. The bypassed small intestine, which carries the bile and pancreatic enzymes, is reconnected to the last portion of the small intestine to eventually mix with the food. The DS keeps the pylorus intact.	Vitamins A, D, E, K, zinc, iron, calcium, protein
Sleeve gastrectomy	"Gastric Sleeve," "the Sleeve," "Vertical Sleeve Gastrectomy (VSG)"	Approximately 80% of the stomach is removed to leave a long "sleeve" or banana-shaped pouch.	Vitamin B_{12}, calcium, iron, thiamin
Adjustable gastric band	"the Band," "Lap-Band"	Silicone band is placed around the top of the stomach, creating restriction; saline may be injected into the band, making it tighter.	Folate, vitamin B_{12}, calcium, thiamin
Single anastomosis duodenal-ileal bypass with sleeve gastrectomy	SADI	A sleeve gastrectomy, then the first part of the small intestine is divided, a loop of intestine is measured several feet and connected to the stomach. Food goes through the pouch and into the latter portion of the small intestine.	Vitamins A, D, E, K, zinc, iron, calcium, protein

Data from Bariatric Surgery Procedures. American Society for Metabolic and Bariatric Surgery. Updated May 2021. Accessed April 26, 2023. http://asmbs.org/patients/bariatric-surgery-procedures.

Description

Preoperative Care

All potential bariatric surgery candidates should undergo preoperative evaluation by a multidisciplinary team, including a physician, bariatric surgeon, registered dietitian (RD), psychologist, and exercise specialist. **Box 6.1** includes a preoperative checklist for bariatric surgery.[4]

Box 6.1 Preoperative Checklist for Bariatric Surgery

- Complete history and physical examination (H&P) for obesity-related comorbidities, causes of obesity, weight/BMI, weight loss history, commitment, and exclusions related to surgical risk.
- Routine labs including fasting blood glucose and lipid panel, kidney function, liver profile, urine analysis, prothrombin time/INR, blood type, CBC.
- Nutrient screening with thiamin, iron studies, B_{12}, and folic acid, and 25-vitamin D (vitamins A and E optional).
- Consider more extensive testing in patients undergoing malabsorptive procedures based on symptoms and risks.
- Cardiopulmonary evaluation, sleep apnea screening, venous thromboembolism (VTE) risk screening as clinically indicated, ECG, chest x-ray (CXR), echocardiography if cardiac disease or pulmonary hypertension suspected, deep vein thrombosis (DVT) evaluation if clinically indicated.
- GI evaluation (EGD, *Helicobacter pylori* screening, gallbladder evaluation and endoscopic and radiologic testing, if clinically indicated).
- Endocrine evaluation: A1C with suspected or diagnosed prediabetes or diabetes, with improvement target of <8%, thyroid-stimulating hormone (TSH) with symptoms or increased risk of thyroid disease, androgens with polycystic ovary syndrome suspicion (total/bioavailable testosterone, DHEAS, D4-androstenedione), screening for Cushing syndrome if clinically suspected.
- Clinical nutrition evaluation by RD.
- Psychosocial–behavioral evaluation: document medical necessity for bariatric surgery.
- Informed consent.
- Provide relevant financial information.

- Continue efforts for preoperative weight loss.
- Optimize glycemic control.
- Pregnancy counseling.
- Smoking cessation counseling.
- Verify cancer screening by primary care physician.

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Preoperative Nutrition Assessment

A complete nutrition assessment should be completed for all patients before any bariatric surgery procedure.[5] This should include assessment of the patient's ability to follow and comply with all pre- and postoperative dietary, behavior, and lifestyle change recommendations, current nutrition intake and history of weight loss attempts, educational needs, and micronutrient screening.[4] Components of a long-term lifestyle change include the patient's motivation or a readiness to change, goal setting, nutrient knowledge, and other factors affecting weight status including behavioral, psychological, cultural, and economic influences.

Correction of any insufficient or deficient micronutrient levels should be a primary intervention before bariatric surgery because obesity is a risk factor for nutrient deficiencies, particularly.[5]

- Iron
- Vitamin B_{12}
- Zinc
- Vitamin D

The following laboratory studies should be assessed in the presurgical patient:

- Liver function tests
- Lipid profile

- CBC with differential
- Hgb A1C
- Serum iron, ferritin, and TIBC
- Serum calcium, alkaline phosphatase
- Serum vitamin B_{12}
- Serum vitamin B-1 (thiamin)
- Serum folate
- PTH, 25-hydroxyvitamin D

The Mifflin-St. Jeor formula for estimating resting metabolic rate (RMR) is recommended for estimating energy requirements in the obese patient using actual body weight.[6]

Men: RMR (kcal/day) = 10 × weight (kg) + 6.25 × height (cm)

– 5 × age (years) + 5

Women: RMR (kcal/day) = 10 × weight (kg) + 6.25 × height (cm)

– 5 × age (years) – 161

Preoperative Diet and Weight Loss

Most programs require that the patient lose weight before bariatric surgery. A weight loss of 5–10% is usually recommended to decrease total liver volume and improve ease of a laparoscopic procedure and surgical outcomes. A high-protein and/or liquid diet may be recommended 2 days or more before the scheduled bariatric surgery, according to program protocols. This allows for adequate protein intake and hydration before surgery, and also gives time for the patient to practice using protein supplements. Presurgery is

also an opportune time to include education on lifestyle modification to improve success after surgery.

All patients are required to be NPO (nothing by mouth) after midnight the morning of their procedure to clear the stomach and bowel before surgery. It is also recommended that patients who smoke should stop at least 6 weeks, or more, before bariatric surgery.

Postoperative Care

Acute Postoperative Period (Up to 3 Months Following Surgery)

The early postoperative period, typically from immediately after surgery until 3 months later, is a crucial time for meeting nutritional needs that result from the stress of surgery and healing involved (see **Box 6.2**).[7] Protein needs should be met to allow the stomach to heal from surgery and to prevent lean tissue loss. Some patients may require more support and guidance with the progression of their diet, and frequent nutritional follow-up is critical for healthy weight loss. The length and variation of postoperative diet progression depend on program preference but generally follow the transition listed in **Table 6.2**.

Box 6.2 Early Postoperative Care Checklist

- Monitored telemetry at least 24 hours if high risk for cardiopulmonary complication.
- Protocol-derived staged meal progression supervised by RD.
- Healthy eating education by RD.
- Multivitamin plus minerals (# tablets for minimal requirement).
- Calcium citrate, 1,200–1,500 mg/day.
- Vitamin D, at least 3,000 units/day, titrate to >30 ng/mL.
- Vitamin B_{12} as needed for normal range levels; 350–500 µg daily oral sublingual, nasal spray, or monthly intramuscular injection
- Maintain adequate hydration (usually >1.5 L/day PO).
- Monitor blood glucose with diabetes or hypoglycemic symptoms.
- Pulmonary toilet, spirometry, DVT prophylaxis, early ambulation.
- If unstable, consider pulmonary embolus (PE), intestinal leak (IL).
- If rhabdomyolysis is suspected, check CPK.

Data from Mechanick JI, Apovian C, Brethauer S, et al. *Endocr Pract* 2019 update 2019;25(12):1346-1359. Epub 2019 Nov 4; doi:10.4158/GL-2019-0406

TABLE 6.2 Diet Progression Following Bariatric Surgery

Diet	Duration	Goals
Clear liquid	Short-term: 1–2 days; longer requires nutritional supplementation	Adequate hydration and protein intake, low sugar
Full liquid	7–14 days	Adequate hydration and increased protein intake, low sugar
Pureed/soft foods	7–14 days	Adequate hydration, increased protein intake, addition of fruits and vegetables, low sugar
Modified regular	Ongoing	Adequate hydration, protein, fruits, vegetables, and small amounts of whole grains, low sugar

Data from Isom KA, Majumdar MC. *Pocket Guide to Bariatric Surgery*. 3rd ed. Academy of Nutrition and Dietetics, 2023.

Patients typically begin a low-sugar, clear liquid diet within 24 hours of surgery.[4] It is important to differentiate between a standard clear liquid diet and a bariatric clear liquid diet related to sugar content. A standard clear liquid diet could cause a postop patient to experience dumping syndrome. Straws, carbonation, and caffeine are not recommended in the immediate postop stage. A patient's new stomach can hold only very small amounts owing to the size of the newly created pouch and surgical swelling. As healing progresses, the patient will be able to tolerate thicker consistencies of

food. A liquid diet will also allow the stomach to heal properly without added tension to the staple line or stitches. Complete stomach healing is achieved approximately 6 weeks after surgery.

It is helpful for the dietitian to provide postop discharge instructions to the patient so that questions can be answered. In addition to diet instructions, the patient should be encouraged to walk regularly as part of the treatment plan to lower the risk of DVT. Patients should be restricted from lifting heavy weights until cleared by the surgeon, usually 2–4 weeks after surgery. Laparoscopic surgeries usually only require brief in-hospital stay before discharge.

Most patients will indicate that they are not hungry after surgery, because of both the surgery anesthesia and the decrease in the hunger hormone, ghrelin. Pancreatic peptide YY (PYY) and glucagon-like peptide-1 (GLP-1), which decrease appetite through different mechanisms, are also shown to be increased in the postprandial period after SG and RYGB procedures.[8] It is important to adhere to the nutrition prescription so as to preserve lean body tissue and to meet nutritional needs. Small, frequent meals are recommended over the course of the day, as is teaching the patient to identify the difference between snacking and grazing on empty calories versus high-quality food sources. **Table 6.3** lists specific foods that should be avoided or delayed for reintroduction into the diet.

TABLE 6.3 Recommended Foods to Avoid or Delay Reintroduction

Food Type	Recommendation
Sugar, sugar-containing foods, concentrated sweets	Avoid

Food Type	Recommendation
Carbonated beverages	Avoid/delay[a]
Fruit juice	Avoid
High-saturated fat, fried foods	Avoid
Soft “doughy” bread, pasta, rice	Avoid/delay[a]
Tough, dry, red meat	Avoid/delay[a]
Nuts, popcorn, other fibrous foods	Delay[a]
Caffeine	Avoid/delay[a] in moderation
Alcohol	Avoid

[a]Foods to delay should be a minimum of 6 weeks, but reintroduction varies by bariatric facility protocol.
Data from Parrott J, Frank L, Rabena R, Craggs-Dino L, Isom KA, Greiman L. American Society for Metabolic and Bariatric Surgery integrated health nutritional guidelines for the surgical weight loss patient 2016 update: Micronutrients. *Surg Obes Relat Dis*. 2017;13(5):727-741. doi: 10.1016/j.soard.2016.12.018.

Dumping Syndrome

Patients are advised to limit the total amount of sugar intake because of the effect of dumping syndrome that can be experienced after bariatric surgery. Dumping syndrome is considered a complication of surgery where food, particularly food with high sugar content, is quickly released or “dumped” into the small bowel owing to the absence of the pyloric sphincter in the bypassed portion of the stomach in a Roux-en-Y gastric bypass. Consequently, a hypertonic solution is created in the jejunum that causes distension.[5] Early dumping symptoms include rapid heartbeat, sweating, dizziness, nausea, stomach cramps, and diarrhea. Late

dumping occurs 1–3 hours afterward with the quick uptake of glucose triggering an insulin release, causing reactive hypoglycemia. Dumping syndrome can present in various types of bariatric surgery procedures, including gastric bypass, sleeve gastrectomy, and biliopancreatic diversion (with or without duodenal switch).[8]

Not all patients will experience dumping syndrome, but as a precaution, bariatric patients should receive education on how to avoid excess sugar intake by reading and interpreting nutrition facts labels. There is no specific amount of sugar intake that may cause dumping syndrome, but generally, no more than 25 g of sugar per serving is recommended. Patients are advised to keep a food diary to track any dumping-related symptoms. It is recommended that significant amounts of fluid intake be avoided for 30 minutes before, during, and 30 minutes after eating, to avoid the risk of dumping syndrome posed by quick gastric emptying, and to improve satiety. However, most patients are able to drink up to the point of eating without an issue. As patients heal after surgery and swelling in the pouch decreases, hydration should continue to be encouraged as patients resume busy and active lifestyles, to avoid dehydration.

Protein

Protein consumption should be individualized and assessed by the dietitian on the basis of age, gender, and body weight of the patient.[4] A factor of 1.5 g/kg of ideal body weight per day (with a minimum of 60 g/day) should provide adequate protein. Owing to the small size of the postoperative stomach, protein needs should be divided throughout the day, with an average of 15–30 g of protein per serving to allow for adequate digestion and absorption. Liquid protein supplements are used in the immediate postop period to meet protein requirements, and the patient should be encouraged to sip his or her protein drinks slowly.

Complete protein supplements containing all nine essential amino acids are required. Patients should be given guidance before surgery on the types of high-bioavailability protein supplements to purchase, such as whey, casein, egg white, milk, and soy-based products.

As the diet progresses, some of the liquid protein supplements may be replaced with high-protein food choices such as poultry, fish, meat, low-fat dairy, and soy. One to two ounces may be tolerated at one time, and protein foods must be eaten slowly and chewed well. Behavior modification to support slow eating should be discussed, as eating too much or too quickly, or not chewing food well, may cause nausea and vomiting. Moist, tender, and juice-containing meats are recommended over dry, rubbery, or dense textures. Patients should be instructed on proper food preparation techniques when cooking foods to avoid rubbery and dry textures. In addition, they should be encouraged to consume protein in small and frequent meals, along with a regular supplement, to consistently meet daily needs.

Alcohol and Tobacco

Alcohol is not recommended after bariatric surgery. Patients are to avoid alcohol intake and continue abstinence postoperatively owing to the high-calorie content and low nutritional value. Alcohol may also increase the risk for ulcers and the risk for alcohol abuse because the patient's tolerance of alcohol is altered. Patients should avoid tobacco use after bariatric surgery because of the increased risk of anastomotic ulcer and poor wound healing.

Micronutrients

Initially, patients are to consume liquid or chewable vitamin and mineral supplements for maximum digestion and absorption in the early postoperative period. Specific bariatric formulas are now available in specialized capsule

form that are made to dissolve without a large amount of gastric acid, allowing patients an alternative to liquid or chewable formulas. Daily chewable, liquid or specific bariatric formulated capsules of multivitamins can usually be started when any postsurgical nausea or vomiting has subsided.[4] Supplements are usually recommended to be taken with food because of the highly concentrated formula. Other supplements required may include sublingual vitamin B_{12}, calcium citrate with vitamin D, and iron in the form of ferrous fumarate. Specific micronutrient deficiencies or insufficiencies targeted preop are to continue to be treated depending on the individual's lab results. **Table 6.4** lists the suggested vitamin supplementation after specific types of bariatric procedures.

TABLE 6.4 Suggested Postoperative Vitamin Supplementation

Supplement	AGB	RYGB & SG	BPD/DS	Comment
Multivitamin–multimineral supplement ■ High potency containing at least 100% DV for 2/3 of nutrients, and 18 mg iron, 400 μg folic acid, at least 50 mg thiamine, and containing selenium and zinc (at least 8 mg) in each serving. ■ Start with chewable or liquid, and progress to whole tablet/capsule as tolerated. ■ Avoid children's or gummy formulas that are not complete. ■ May separate dosage. ■ May improve GI tolerance when taken close to food intake. ■ Do not mix multivitamin containing iron with calcium, take 2 hr apart.	100% of daily value (DV)	200% of DV	200% of DV	Begin as tolerated
Additional cobalamin (B_{12}) ■ Sublingual tablets, liquid drops, mouth spray, or nasal gel/spray (oral). ■ Intramuscular (IM) injection. ■ Supplementation after AGB and BPD/DS may be required.	None	350–500 μg/day (oral), 1,000 μg/mo (IM)	None	Begin 0–3 mo after surgery
Additional elemental calcium ■ Calcium citrate and vitamin D_3. ■ Split into 500–600 mg doses; be mindful of serving size on supplement label. ■ Suggest a brand that contains magnesium, especially for BPD/DS. ■ Do not combine calcium with iron. ■ Promote intake of dairy beverages and/or foods that are significant sources of dietary calcium in addition to recommended supplement, up to 3 servings daily. ■ Combined dietary and supplemental calcium intake >1,700 mg/day may be required to prevent bone loss during rapid weight loss. ■ 3,000 IU D3 daily until blood levels of 25(OH)D are >30 ng/mL.	1,500 mg/day	1,500–2,000 mg/day	1,800–2,400 mg/day	May begin as tolerated or within 1 mo after surgery
Additional elemental iron (above that provided by multivitamin) ■ Recommended for menstruating women and those at risk for anemia (total cumulative intake = 45–60 mg elemental iron/day); males and patients without history of anemia should receive at least 18 mg iron. ■ No enteric coasting. ■ Avoid excessive intake of tea owing to tannin interaction. ■ Encourage foods rich in heme iron. ■ Vitamin C may enhance absorption of nonheme sources.	None	Add a minimum of 27–60 mg/day elemental	Add a minimum of 27–60 mg/day elemental	Begin as tolerated
Fat-soluble vitamins ■ With all procedures, higher maintenance doses may be required for those with a history of deficiency. ■ Water-soluble preparations of fat-soluble vitamins are available. ■ Retinol sources of vitamin A should be used to calculate dosage.	Vitamin A 5,000 IU/day and Vitamin K 90–120 μg/day	Vitamin E should be taken at a dosage of 15 mg/day	10,000 IU of vitamin A, 3,000 IU of vitamin D, and 300 μg of vitamin K	May begin 2–4 wk after surgery
■ Most supplements contain a high percentage of beta carotene, which does not contribute to vitamin A toxicity. ■ Intake of 3,000 IU vitamin D_3 may be achieved with careful selection of multivitamin and calcium supplements. ■ No toxic effect known for vitamin K_1, phytonadione (phyloquinone). ■ Vitamin K requirement varies with dietary sources and colonic production. ■ Caution with vitamin K supplementation for patient receiving coagulation therapy. ■ Vitamin E deficiency has been suggested but is not prevalent in published studies.		Vitamin A 5,000–10,000 IU/day and Vitamin K 90–120 μg/day		
Optional B complex B-50 dosage ■ Liquid form is available. ■ Avoid time-released tablets. ■ No known risk of toxicity. ■ May provide additional prophylaxis against B-vitamin deficiencies, including thiamin, especially for BPD/DS procedures as water-soluble vitamins are absorbed in the proximal jejunum. ■ Note: >1,000 ng of supplemental folic acid, provided in combination with multivitamins, could mask B_{12} deficiency.	1 serving/day	1 serving/day	1 serving/day	May begin as tolerated

AGB, adjustable gastric band; BPD/DS, biliopancreatic diversion with or without duodenal switch; RYGB, Roux-en-Y gastric bypass; SG, sleeve gastrectomy.

Data from (i) Linda Aills, et al., "ASMBS Allied Health Nutritional Guidelines for the Surgical Weight Loss Patient," *Surgery for Obesity and Related Diseases* 4, no. 5 (2008): S73-S108, © 2013 by Elsevier, and (ii) Updated with data from Parrott J, Frank L, Rabena R, Craggs-Dino L, Isom KA, Greiman L. American Society for Metabolic and Bariatric Surgery integrated health nutritional guidelines for the surgical weight loss patient 2016 update: Micronutrients. *Surg Obes Relat Dis.* 2017;13(5): 727-741. doi: 10.1016/j.soard.2016.12.018, and (iii) Updated with data from Mechanick JI, Apovian C, Brethauer S, et al. *Endocr Pract* 2019 update 2019;25(12):1346-1359. Epub 2019 Nov 4; doi:10.4158/GL-2019-0406

Description

Nutrition Complications in the Postoperative Patient

Bariatric surgery has proven to be safe and effective. However, every surgery has its own risk for complications, with bariatric surgery at 2–4%.[1] Acute medical complications following bariatric surgery can typically arise within 30 days of surgery; however, regular follow-up visits with an RD, and bariatric team, for lifestyle modification are important during the first year after surgery.[7]

Acute complications of a bariatric procedure may be anastomotic leak, bleeding, DVT, dysphagia, abscess, stricture, reflux, nausea, vomiting, infection, pulmonary embolism, and cardiopulmonary complications. Long-term complications include gastrojejunostomy stenosis, ulcer, hernia, gallstones, dumping syndrome, vitamin and/or mineral deficiency, and weight regain.

Protein malnutrition is not commonly seen in bariatric surgery but needs to be considered because of the restriction of food and decreased appetite, and when there are complications. Enteral and parenteral nutrition should be considered for those at high nutritional risk.[4] Noncritical bariatric surgery patients who are unable to use their GI tract to meet their needs at 5–7 days should be considered for parenteral nutrition and at 3–7 days in the critical patient, or if severe protein malnutrition is present and enteral nutrition is not shown to be effective. **Table 6.5** lists the most common nutritional complications of bariatric surgery along with suggested interventions.

TABLE 6.5 Common Nutritional Complications After Bariatric Surgery

Complication	Suggested Interventions
Dehydration	Assess fluid intake, IV hydration should be recommended if severe with adequate thiamin (B_1)

Complication	Suggested Interventions
	(see thiamin deficiency).
Nausea/vomiting	Assess PO intake for possible contributors; separate eating/drinking; decrease portion size; sit upright when eating/drinking; take small bites/sips and eat slowly; limit fat, sugar, and highly acidic foods; avoid extreme food temperatures; rule out offending foods; regress diet progression as tolerated.
Dysphagia	Assess PO intake for possible contributors including dry, tough, rubbery, doughy, or sticky foods; assess cooking techniques, regress diet progression as tolerated.
Diarrhea	Assess PO intake for possible contributors including lactose, whey protein concentrate; high-fat and high-sugar foods.
Flatulence	Assess PO intake for possible contributors including lactose, whey protein concentrate; carbonated beverages and straw use.
Constipation	Assess PO intake for fluids, fiber.
Dumping syndrome	Assess PO intake for concentrated sweets, sweetened beverages, eating and drinking at the same time.
Food intolerance/lactose intolerance	Assess PO intake for lactose, high-fat foods, gas-containing foods. Discontinue offending food, and wait to reintroduce until later; regress diet progression as tolerated.
Hair loss	Assess PO intake for protein, fluid, vitamins, and minerals; determine months postop for telogen effluvium.

Data from Isom KA, Majumdar MC. *Pocket Guide to Bariatric Surgery*. 3rd ed. Academy of Nutrition and Dietetics, 2023.

Troubleshooting in the Postoperative Period

Problems can arise in the longer-term postoperative period of more than 3 months, and regular follow-up is imperative (see **Box 6.3**). Common problems can include hair loss and various micronutrient deficiencies. Hair loss after bariatric surgery may or may not be related to nutrition. Stress on the body from a major surgery and rapid weight loss are the common causes of hair loss post bariatric surgery, resulting in telogen effluvium.[9] Hair follicles in the anagen phase are changed into the telogen phase, which only last for about 100–120 days before falling out. This is seen most in patients who are 3–4 months post-surgery; however, this is temporary and in the absence of a nutritional cause, will grow back normally once the hairs have fallen out. Most patients may not notice or may only have slight thinning. Patients with severe hair loss may have had thin or receding hair before bariatric surgery.

Box 6.3 Follow-Up Postoperative Care Checklist

- Initial, interval until stable, once stable (months).
- Monitor progress with weight loss and evidence of complications each visit.
- SMA-21, CBC/platelet with each visit (and iron at baseline and after as needed).
- Avoid nonsteroidal anti-inflammatory drugs.
- Adjust postoperative medications.
- Consider gout and gallstone prophylaxis in appropriate patients.
- Need for antihypertensive therapy with each visit.
- Lipid evaluation every 6–12 months based on risk and therapy.
- Monitor adherence with physical activity recommendations.
- Evaluate need for support groups.
- Bone density (DXA), if indicated.
- 24-hour urinary calcium excretion, if indicated.
- B_{12} (annually; MMA and HCy optional; then q 3–6 months if supplemented).
- Folic acid (RBC folic acid optional), iron studies, 25-vitamin D, iPTH.
- Vitamin A (initially and q 6–12 months thereafter).
- Copper, zinc, and selenium evaluation with specific findings.

- Thiamin evaluation with specific findings.
- Consider eventual body contouring surgery in appropriate patients

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Hair loss related to a nutritional deficiency after bariatric surgery is usually seen more than 6 months postoperatively and beyond. Nutritional deficiencies can also contribute to telogen effluvium. Patients whose oral intake is not meeting requirements or whose weight loss is more than what was expected will be at risk for hair loss related to nutritional needs not being met. Iron, zinc, and protein are the nutrients most often correlated with hair loss and should be assessed (see **Table 6.6**).

TABLE 6.6 Hair Loss After Bariatric Surgery

Time Frame	Possible Causes	Interventions
<6 mo since surgery	Most likely related to stress from major surgery and rapid weight loss; temporary	Confirm that nutritional needs are being met, and continue to follow up to access patient >6 mo postop.
>6 mo since surgery	Most likely related to nutrient deficiency, disease state, or genetics	Confirm protein needs are being met, as well as zinc, iron, and essential fatty acids.

Once the early postop phase is completed, regular biochemical monitoring is recommended for gauging a patient's micronutrient levels. This also allows the clinician to determine whether current supplementation is meeting the patient's needs, and whether further supplements are required. **Table 6.7** suggests biochemical monitoring tools

for nutrition status in the post bariatric patient. Certain nutrients can be particularly problematic if careful attention is not paid to ensuring adequate intake, including thiamin, vitamin B_{12}, iron, calcium, and vitamin D.

TABLE 6.7 Biochemical Monitoring Tools for Nutrition Status in the Post–Bariatric Surgery Patient

Vitamin/ Mineral	Screening	Additional Laboratory Indexes	Normal Range	Critical Range	Postoperative Deficiency	Comment
B_1 Thiamin	Serum thiamin	(↓)RBS transketolase (↑)Pyruvate	10–64 ng/mL	Transketolase activity >20% Pyruvate >1 mg/dL	15–29% more common in AA and Hispanics; often assoc. with poor hydration	Serum thiamin responds to dietary supplementation, but is poor indicator of total body stores.
B_6 Pyridoxine	PLP	RBC glutamic pyruvate oxaloacetic transaminase	5–24 ng/mL	<3	Unknown	Consider with unresolved anemia; diabetes could influence values.
B_{12} Cobalamin	Serum B_{12}	(↑)Serum and urinary MMA, (↑)Serum tHcy	200–1,000 pg/mL	<200	10–13% may occur with older patients and those taking H_2 blockers and PPIs	When symptoms are present and B_{12} 200–250 pg/mL, MMA and tHcy are useful; serum B_{12} may miss 25–30% of deficiency cases.
Folate	RBC folate, serum folate	Urinary FIGLU Normal serum and urinary MMA (↑)Serum tHcy	280–791 ng/mL	<305 nmol/L	Uncommon	Serum folate reflects recent dietary intake rather than folate status; RBC folate is a more sensitive marker; excessive supplementation can mask B_{12} deficiency in CBC; neurologic symptoms will persist.
Iron	Ferritin	(↓)Serum iron (↑)TIBC	Males: 15–200 ng/mL Females: 12–150 ng/mL	<20	9–16% of adult women in general population are deficient	Low Hgb and Hct are consistent with iron deficiency anemia in stage 3 or stage 4 anemia; ferritin is an acute phase reactant and will be elevated with illness and/or inflammation; oral contraceptives reduce blood loss for menstruating females.
Vitamin A	Plasma retinol	RBP	20–80 μg/dL	<10	Uncommon, up to 7% in some studies	Ocular finding may suggest diagnosis.
Vitamin D	25(OH)D	(↓)Serum phosphorus (↑)Alkaline phosphatase (↑)Serum PTH (↓)Urinary calcium	25–40 ng/mL	<20	Common; 60–70%	With deficiency, serum calcium may be low or normal; serum phosphorus may decrease, serum alkaline phosphatase increase; PTH elevated.
Vitamin E	Plasma alpha tocopherol	Plasma lipids	5–20 μg/mL	<5	Uncommon	Low plasma alpha tocopherol to plasma lipids (0.8 mg/g total lipid) should be used with hyperlipidemia.
Vitamin K	PT	(↑)DCP (↓)Plasma phylloquinone	10–13 seconds	Variable	Uncommon	PT is not a sensitive measure of vitamin K status.
Zinc	Plasma zinc	(↓)RBC zinc	60–130 μg/dL	<70	Uncommon, but increased risk of low levels associated with obesity	Monitor albumin levels and interpret zinc accordingly, albumin is primary binding protein for zinc; no reliable method of determining zinc status is available; plasma zinc is method generally used; studies cited in this report did not adequately describe methods of zinc analysis.
Protein	Serum albumin Serum total protein	(↓)Serum prealbumin (transthyretin)	4–6 g/dL 6–8 g/dL	<3 <20 mg/dL	Uncommon	Half-life for prealbumin is 2–4 days and reflects changes in nutritional status sooner than albumin, a nonspecific protein carrier with a half-life of 22 days.

Data from Parrott J, Frank L, Rabena R, Craggs-Dino L, Isom KA, Greiman L. American Society for Metabolic and Bariatric Surgery integrated health nutritional guidelines for the surgical weight loss patient 2016 update: Micronutrients. *Surg Obes Relat Dis.* 2017;13(5):727-741. doi: 10.1016/j.soard.2016.12.018.

Description

Thiamin (Vitamin B_1): Part of B-Complex Vitamins

- All post-bariatric surgery patients should take at least 12 mg thiamin. An additional 50 mg from b-complex or in multivitamin is preferred.[5] Primary absorption sites are bypassed with some bariatric procedures, and dietary sources may be diminished.
- Include in the multivitamin; gummy vitamins typically do not include thiamin.
- Dietary sources include fortified grains, legumes, nuts, and pork.
- Drug interactions: Lasix and Dilantin.
- Deficiency: Beriberi (dry, wet, cerebral, bariatric).
- Risk factors: poor PO intake, intestinal loss through vomiting, ethyl alcohol (ETOH) intake, eating disorders, dialysis.
- Signs/symptoms: vomiting, loss of appetite, weakness, sleepiness, burning feet, calf and leg pain, abdominal pain, constipation, headache, cramping, peripheral polyneuropathy.
- Treatment: 100 mg of oral thiamin (preferred as thiamin mononitrate), 50–100 mg intramuscularly (IM) or intravenously (IV) for several days with emesis, 10–20 mg as emesis subsides. Adequate levels of other vitamins and minerals are required such as magnesium and B-vitamins.
- With bariatric surgery patients, it is most frequently seen in the early postop phase related to vomiting.

Vitamin B_{12} (Cobalamin): Part of B-Complex Vitamins

- Include in the multivitamin; separate supplementation may also be required through oral, nasal, or intramuscular supplementation.

- Loss of absorption that results from decrease in gastric acid, HCl, intrinsic factor.
- Dietary sources include meat, eggs, dairy.
- Drug interactions: tetracycline, PPI, and H_2 blockers can reduce B_{12} absorption.
- Deficiency: B_{12} deficiency, pernicious anemia.
- Risk factors: intestinal surgery—loss of HCl, gastric churning, pepsin, and intrinsic factor (IF), atrophic gastritis, *H. pylori* infection, veganism, alcoholism, fad dieting, poor nutrient intake.
- Signs/symptoms: fatigue, weakness, shortness of breath, loss of appetite, paresthesia, sore tongue. Numbness, coldness, cramping and shooting pains, clumsiness and gait abnormality are most common complaints.
- Treatment: oral or sublingual dose of 2,000 µg/day for 2–4 weeks; or 1,000 µg IM injection daily for 1–2 weeks. Transition to maintenance dose.

Iron

- Consider including in the multivitamin of at least 18 mg, and additional supplement is recommended in high-risk patients of 45–60 mg of total elemental iron from all sources.
- Dietary sources: beef, sardines, turkey, mussels, oysters (heme), lentils, beans, and leafy greens (nonheme).
- Drug interactions: tetracycline, thyroid, bisphosphonates, levodopa, methyldopa, fluoroquinolones, penicillamine. Iron absorption is decreased with acid-reducing medications, calcium, and some fiber supplements. Consider absorption of copper, zinc, selenium, and vitamin A with high iron dosages.
- Deficiency: iron deficiency, iron deficiency anemia.

- Risk factors: menstruating women and those who become pregnant. Preferential absorption site is bypassed in RNYGB and DS (proximal duodenum). Absorption is also dependent upon iron stores. Reduced hydrolysis in the stomach.
- Signs/symptoms: fatigue, leg cramping, pagophagia, pica, hair loss, shortness of breath, pallor, irritability, difficulty swallowing, loss of appetite, sore/swollen tongue, angular cheilitis, brittle nails, koilonychias, headache, rapid pulse, tachycardia, difficulty with concentration, coldness, and decreased immune function.
- Treatment: oral therapy of 100–200 mg elemental iron per day in divided doses. Vitamin C is shown to positively influence absorption. Do not include calcium supplementation with iron; keep at least 2 hours apart because of the competition for the same absorption site. Iron dextran can also be given intravenously, but monitoring for allergic reaction is very important. Refer to hematology.

Calcium

- Separate supplementation is required for adequate dosage.
- Dietary sources include dairy products, leafy greens, soybeans, salmon, sardines, and calcium-fortified foods.
- Drug interactions: antibiotics and thyroid hormones and cardiac medications. Other interactions include magnesium, phytate, oxalate, large amounts of sodium and protein, iron, phosphorus, and large amounts of caffeine.
- Deficiency: hypocalcemia, metabolic bone disease (osteoporosis, osteomalacia, hyperparathyroidism).

- Risk factors: low intake, low protein, malabsorption, hypo- or achlorhydria, hypomagnesemia, vitamin D deficiency, steatorrhea.
- Signs/symptoms: paresthesia, numbness, muscle cramping, loss of sensation around the mouth, unexplained GI cramping.
- Treatment: orally 1,000–1,500 mg of elemental calcium in divided doses with vitamin D for prevention and maintenance, magnesium may be required. Up to 2,000 mg/day for treatment of chronic or mild hypocalcemia. Low protein levels should be addressed. Calcium citrate is preferred for both prevention and treatment. Bone, dolomite, and shell products should be avoided for possible lead or aluminum.

Vitamin D

- Include with calcium supplementation. Preoperatively, deficiency is seen in 61% of patients.[10]
- Dietary sources are not considered necessary if adequate sunlight is provided. Dietary sources include sardines, dairy products.
- Drug interactions: verapamil, cimetidine, orlistat, bile acid sequestrants, corticosteroids
- Risk factors: fat malabsorption in RNYGB and DS procedures.
- Signs/symptoms: most are related to subsequent calcium deficiency, increased head sweating, burning in the mouth, dysmenorrhea, seasonal affective disorder (SAD), cardiac arrhythmias, seizures, fatigue, loss of balance/falling, hypertension, and poor immune function.
- Treatment: 50,000 IU of D_2 is the typical treatment taken weekly, or 3,000 IU D3 taken daily to achieve an adequate level of 30 ng/mL is considered for those with levels <8 ng/mL, and in the presence of hypocalcemia.[5]

ADIME 6.1 ADIME At-A-Glance: Bariatric Surgery

Assessment

History of multiple, unsuccessful diets, overweight her whole life. Skips meals and overeats at bedtime (HS). No regimented physical activity reported, and has a sedentary job where she works long hours. Height 65 inches, weight 345 lb.

Nutrition Diagnosis

Excessive energy intake related to unsuccessful attempts at reducing caloric intake as evidenced by BMI of 57.

Intervention

- Increase food frequency to a regular, structured eating pattern of three meals and one to two snacks.
- Avoid skipping meals. Increase physical activity by including a regimented exercise plan of at least 3 days/week of 45 minutes, and increase total steps walked per day by taking regular activity breaks at work for a total of 8,000–10,000 steps/day.

Monitoring and Evaluation

Regular follow-up intervals with the RD every 2 weeks to evaluate adherence to recommendations, reports of PO intake, and regular weigh-ins. Will monitor the following:

- Energy intake: caloric intake from food and beverages: food selection and portion consumed.
- Energy expenditure: caloric output from overall physical activity (steps) and regimented exercises: days per week of exercises and steps per day.

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CHAPTER 7

Cancer

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Cancer is a leading cause of death worldwide, accounting for nearly 10 million deaths in 2020.[1] In 2023, 1,958,310 new cancer cases and 609,820 cancer deaths are projected to occur in the United States.[2] The five most common types of new cancer cases in the United States estimated among men in 2023 are (in order of prevalence): prostate, lung, colorectal, bladder, and melanoma. Among women, the estimated top five most commonly diagnosed cancers in 2023, in order of prevalence are breast, lung, colorectal, uterine, and melanoma.[3] Around one-third of deaths from cancer are due to tobacco use, high body mass index, alcohol consumption, low fruit and vegetable intake, and a lack of physical activity.[4] Studies have reported malnutrition in 30% to 85% of patients with cancer.[5-7] In addition, malnutrition increases treatment toxicities, diminishes quality of life, and accounts for 10–20% of mortality in patients with cancer.[5,8] Additionally, protein-calorie malnutrition (PCM) is the most common secondary diagnosis in individuals diagnosed with cancer, stemming from the inadequate intake of carbohydrate, protein, and fat to meet metabolic requirements and/or the reduced absorption of macronutrients.[5]

Disease Process

Cancer is not one disease; rather, it represents over 100 diseases. The common characteristics of all cancers are uncontrolled cellular proliferation and the ability of these cells to metastasize, or migrate, from the original site and spread to distant sites throughout the body.

Cancer staging is a system used by physicians and other medical professionals to describe the extent or severity of an individual's cancer. Staging is based on the extent of the primary tumor and on the extent of metastasis. Staging is important when a cancer diagnosis is made because it helps the physician plan the course of treatment, estimate the patient's prognosis, and identify the clinical trials that may be suitable for that particular patient.[9] Staging is also important for cancer registries and researchers, as it provides a common language for cancer reporting, and for evaluating and comparing clinical trial results. An understanding of cancer staging is helpful when working with oncology patients, as the stage of the patient's cancer may correlate with nutritional status and provide insight into possible nutrition interventions.

Staging systems for cancer are constantly evolving as scientists learn more about the disease. Many staging systems are currently in use. Some cover many different types of cancer, whereas others are specific to a particular type of cancer. The common elements in most staging systems include the following:

- Location of the primary tumor
- Tumor size and number of tumors
- Lymph node involvement
- Cell type and tumor grade

- Presence or absence of metastasis

The Tumor, Node, Metastasis (TNM) Staging System

The TNM staging system is the most widely used staging system.[10] This system was developed and is maintained by the American Joint Committee on Cancer (AJCC) and has been accepted by the Union for International Cancer Control (UICC). The National Cancer Institute (NCI) uses this system in their comprehensive cancer database, and most medical facilities use the TNM system as their main method for cancer reporting.[9]

The TNM system is based on the extent of the tumor (T), the extent of spread to the lymph nodes (N), and the presence of metastasis (M). A number is added to each letter to indicate the size or extent of the tumor and the extent of spread (see **Table 7.1**).

TABLE 7.1 Tumor, Nodes, Metastasis Cancer Staging System

T = Primary Tumor	
TX	Primary tumor cannot be measured
T0	Main tumor cannot be found
Tis	Carcinoma in situ (early cancer that has not spread to neighboring tissue)
T1–T4	Size and/or extent of primary tumor
N = Regional Lymph Nodes	
NX	Cancer in nearby lymph nodes cannot be measured

T = Primary Tumor	
N0	There is no cancer in nearby lymph nodes
N1–N3	Refers to the number and location of lymph nodes that contain cancer
M = Distant Metastasis	
MX	Metastasis cannot be measured
M0	Cancer has not spread to other parts of the body
M1	Cancer has spread to other parts of the body

Data from (i) Cancer Staging. National Cancer Institute, U.S. National Institutes of Health. Reviewed October 14, 2022. Accessed April 20, 2023. https://www.cancer.gov/about-cancer/diagnosis-staging/staging, and (ii) Cancer Staging Systems. American College of Surgeons. Accessed April 20, 2023, https://www.facs.org/quality-programs/cancer-programs/american-joint-committee-on-cancer/cancer-staging-systems

An example of the TNM system for staging colon cancer would be T3 N0 M0. This staging would represent a large tumor, located only in the colon, without spread to lymph nodes or any other parts of the body.

Overall Stage Grouping

Overall Stage Grouping is also referred to as Roman Numeral Staging. This system uses numerals I, II, III, and IV (plus the 0) to describe the progression of cancer. Once the TNM staging has been done, the result can then be categorized into one of the five stages (see **Table 7.2**). The criteria for stages differ for different types of cancer, and so a T3 N0 M0 bladder cancer may be a different overall stage than a T3 N0 M0 breast cancer.

TABLE 7.2 Overall Stage Grouping

Stage	Definition
Stage 0	Abnormal cells are present but have not spread to nearby tissue. Also called carcinoma in situ, or CIS. CIS is not cancer, but may become cancer.
Stages I, II, and III (may also be written as Stage 1, Stage 2, and Stage 3)	Cancer is present. The higher the number, the larger the cancer tumor and the more it has spread to nearby tissues.
Stage IV (may also be written as Stage 4)	The cancer has spread to distant parts of the body.

Data from (i) Cancer Staging. National Cancer Institute, U.S. National Institutes of Health. Reviewed October 14, 2022. Accessed April 20, 2023, https://www.cancer.gov/about-cancer/diagnosis-staging/staging, and (ii) Cancer Staging Systems. American College of Surgeons. Accessed April 20, 2023, https://www.facs.org/quality-programs/cancer-programs/american-joint-committee-on-cancer/cancer-staging-systems

Summary Staging

Another staging system that is used for all types of cancer, groups the disease into one of five main categories. This staging system is more often used by cancer registries than by doctors.

- **In situ:** Abnormal cells are present but have not spread to nearby tissue.
- **Localized:** Cancer is limited to the place where it started, with no sign that it has spread.
- **Regional:** Cancer has spread to nearby lymph nodes, tissues, or organs.
- **Distant:** Cancer has spread to distant parts of the body.
- **Unknown:** There is not enough information to figure out the stage.

Treatment and Nutrition Intervention

Treatment of cancer with chemotherapy and radiation has significant nutritional consequences. Both types of treatment contribute to nutrient alterations in the cancer patient by reducing food intake, decreasing absorption, and/or altering metabolism.

Chemotherapy

Many chemotherapy medications are used in combination, often referred to as protocols or “cocktails,” for treatment of specific cancers. **Table 7.3** lists some common antineoplastic agents and their nutritional implications.

TABLE 7.3 Chemotherapy Medications

Chemotherapeutic Agent: Generic Name (Trade Name)	Nausea and Vomiting	Diarrhea	Xerostomia	Stomatitis and Esophagitis	Anorexia	Taste Alterations
Anastrozole (Arimidex)	Mild	No	No	No	No	No
Bleomycin (Blenoxane)	Mild to moderate	No	Yes	Yes	Yes	No
Busulfan (Myleran)	Mild	No	No	No	Yes	No
Capecitabine (Xeloda)	Mild	Yes	No	No	No	No
Carboplatin (Paraplatin)	Moderate	Yes	No	No	No	No
Carmustine (BCNU)	Moderate	No	No	Yes	Yes	No
Cisplatin (CDDP)	Severe	Yes	No	No	Yes	Metallic taste
Cyclophosphamide (Cytoxan)	Severe	No	Yes	Yes	Yes	No
Cytarabine (ARA-C)	Severe	Yes	No	Yes	Yes	No
Dacarbazine (DTIC-Dome)	Severe	Yes	No	Yes	No	Metallic taste
Dactinomycin (Actinomycin-D, ACT)	Severe	Yes	Yes	Yes	No	Yes
Daunorubicin citrate (Daunomycin)	Moderate	Yes	Yes	Yes	Yes	Yes
Docetaxel (Taxotere)	Mild	No	No	No	No	No
Doxorubicin (Adriamycin)	Moderate	Yes	Yes	Yes	Yes	No
Epirubicin HCL (Ellence)	Moderate	Yes	No	Yes	No	No
Etoposide (VP-16-23)	Mild to moderate	Yes	No	Yes	Yes	No
Floxuridine (FUDR)	Mild	Yes	No	Yes	Yes	No
5-Fluorouracil (5-FU)	Moderate	Yes	No	Yes	No	Yes
Gemcitabine hydrochloride (Gemzar)	Mild	Yes	No	Yes	No	No
Hydroxyurea (Hydrea)	Mild to moderate	Yes	No	Yes	Yes	No
Irinotecan (Camptosar)	Severe	Yes	No	No	Yes	No
L-Asparaginase (Elspar)	Moderate	No	No	Yes	Yes	No
Mechlorethamine (Mustargen)	Severe	Yes	No	No	Yes	Metallic taste
Methotrexate (MTX)	Mild to moderate	Yes	No	Yes	Yes	Yes
Mitomycin (Mutamycin)	Moderate	Yes	No	Yes	Yes	No
Nivolumab (Opdivo)	Mild	Yes	No	No	Yes	No
Oxaliplatin (Eloxatin)[a]	Moderate	Yes	No	No	No	No
Paclitaxel (Taxol)	Mild	No	No	Yes	No	No
Pembrolizumab (Keytruda)	Mild	Yes	No	No	No	No
Procarbazine hydrochloride (Matulane)[b]	Severe	Yes	No	Yes	Yes	No
Streptozocin (Zanosar)	Severe	Yes	No	No	No	No
Tamoxifen citrate (Nolvadex)	Mild	No	No	No	Yes	No
Temozolomide (Temodar)	Moderate	No	No	No	Yes	No
Topotecan hydrochloride (Hycamtin)	Mild	Yes	No	No	Yes	No
Vinblastine sulfate (Velban)	Moderate	Yes	No	Yes	No	No
Vincristine sulfate (Vinscar)	Mild	Yes	No	Yes	Yes	No
Vinorelbine tartrate (Navelbine)	Mild	No	No	No	Yes	No

[a]Sensitivity to cold for 5 or more days following drug administration.
[b]To prevent hypertensive crisis, a low-tyramine diet must be followed while receiving procarbazine hydrochloride and 4 weeks after the final dose.

Data from medication product labeling.

Description

Nutrition-Related Side Effects of Chemotherapy (Effects Depend on the Agents Administered)

- Anorexia
- Nausea, vomiting
- Mucositis (stomatitis, esophagitis, enteritis, gastritis, proctitis)
- Diarrhea
- Constipation
- Weight loss
- Taste alterations (ageusia—no taste; hypogeusia—little taste; dysgeusia—distorted taste)
- Metallic taste in mouth
- Xerostomia (dry mouth)
- Lactose intolerance
- Thrush
- Fatigue

Radiation Therapy

The side effects of radiation can be acute or chronic in nature and are dependent on the area of the body that has been irradiated. Changes in taste or saliva caused by radiation to the head or neck can take months to show improvement and sometimes never return to baseline.[11]

Nutrition-Related Side Effects of Radiation Therapy

- General—anorexia, fatigue
- Brain—nausea
- Oropharyngeal
  - Taste alterations (ageusia—no taste, hypogeusia, dysgeusia)
  - Mucositis (stomatitis, esophagitis)

    - Dysphagia, odynophagia (difficult or painful swallowing)
    - Xerostomia, thick saliva
    - Dental caries
    - Loss of teeth
    - Swollen, tender gums
    - Change or loss of smell
    - Trismus (restriction or inability to open the mouth)
- Esophagus/chest/thorax
    - Esophagitis
    - Dysphagia
    - Esophageal stricture
- Abdomen/pelvis
    - Nausea, vomiting
    - Diarrhea, steatorrhea
    - Acute colitis and enteritis
    - Fistulas
    - Maldigestion, malabsorption
    - Perforations

Hematopoietic Cell Transplantation (HCT)

Diseases treated by HCT include[12]:

- Acute lymphocytic leukemia
- Acute myeloid leukemia
- Chronic myeloid leukemia/chronic lymphocytic leukemia
- Hodgkin and Non-Hodgkin lymphoma
- Multiple myeloma
- Myelodysplastic syndrome
- Myelofibrosis, essential thrombocytosis, and polycythemia vera

- Solid tumors (testicular, medulloblastoma, metastatic breast cancer, others)

Bone marrow transplant, also known as a hematopoietic stem cell transplant (HPSCT or HCT), involves the administration of healthy hematopoietic stem cells to patients with dysfunctional or depleted bone marrow.[12] The first step in HCT is to eliminate malignancy through the use of chemotherapy and, in some circumstances, total body irradiation (TBI). The second step is the intravenous infusion of stem cells using the patient's own cells (autologous), cells from an identical twin (syngeneic), or cells from a related or unrelated donor (allogenic) that are a sufficient match. The stem cells are obtained from bone marrow, peripheral blood, or umbilical cord blood. Patients often remain neutropenic for 2–3 weeks until engraftment takes place, and during this time they are best served by following a low microbial diet as well as safe food-handling guidelines (see guidelines later in this chapter). Immediately following HCT, patients may experience an increase in metabolic stress and potential fever/infection that may increase their calorie and protein needs. Graft-versus-host disease is a condition where transplanted cells (graft) attack the patient (host). The major organs impacted by graft-versus-host disease are the GI tract, liver, and skin.

Nutrition-Related Side-Effects of HCT

- Anorexia
- Nausea/vomiting
- Mucositis
- Early satiety
- Diarrhea (often high-volume, secretory diarrhea)
- Abdominal pain
- Intestinal bleeding

- Renal impairment
- Osteoporosis (long-term side-effect)
- Endocrine complications (long-term)
- Iron overload (long-term)

Management of Cancer Symptoms and Treatment of Side Effects

- Anorexia, the loss of appetite or desire to eat, is typically present in 15–25% of all patients with cancer at diagnosis and may also occur as a side effect of treatments or related to the tumor itself.[4,13] One of the most severe aspects of cancer is cachexia, which develops in 80% of patients with advanced-stage cancer and is the cause of death in up to 40% of cancer patients.[5,13,14] Cachexia is diagnosed with the presence of one of the following factors:
- Weight loss of more than 5% over the past 6 months, in the absence of simple starvation.
- BMI (body mass index) of less than 20 and any degree of weight loss greater than 2%.
- Appendicular skeletal muscle index consistent with sarcopenia (men, <7.26; women, <5.45) and any degree of weight loss greater than 2%.[15]

Cancer cachexia is most often seen with gastric, pancreatic, esophageal, lung, liver, and colorectal cancers.[16]

The etiology of cancer cachexia is complex and not well understood. Some individuals with cachexia do respond to nutrition therapy, but most will not see a complete reversal of the syndrome, even with aggressive therapy. Therefore, the most advantageous approach to cachexia is the prevention of its initiation through nutrition assessment, intervention, and monitoring.[5]

The symptoms associated with cancer, cachexia, and the side effects of the treatments used to control cancer can have devastating effects on nutritional status. Individualizing treatment plans to help maximize oral intake, and allowing patients to have flexibility in the type, quantity, and timing of their meals and snacks are essential parts of nutrition therapy. Many of these symptoms and side effects cannot be totally alleviated; however, they can be managed through the proper use of medications and through patient education.

Many medications are available to manage the symptoms of cancer and the side effects of cancer treatments. **Tables 7.4** through **7.7** list the common medications used to help alleviate anorexia, oral problems, nausea and vomiting, and diarrhea associated with cancer and its treatments.

TABLE 7.4 Medications to Treat Anorexia

Medication: Generic Name (Trade Name)	Dose	Action	Special Considerations
Dronabinol (Marinol)	2.5 mg BID (up to 20 mg/d)	Increases appetite, decreases nausea	Drug may be habit-forming
Megestrol acetate (Megace)	800 mg daily	Increases appetite, promotes weight gain	May take 8–12 wk to reach maximal weight gain
Mirtazapine (Remeron)	15–45 mg daily	Increases appetite	Take at bedtime, causes drowsiness

Data from medication product labeling.

Description

TABLE 7.5 Medications to Treat Oral Problems

Medication	Uses
Benzocaine oral spray (HurriCaine® spray)	Relief of mouth pain, mouth sores

Medication	Uses
Artificial saliva (Xero-Lube, Salivart)	Used for xerostomia
Gelclair bioadherent oral gel	Relief of mouth pain, mouth sores
Lidocaine (viscous)	Relief of sore/irritated mouth/throat
Nystatin oral suspension	Antifungal used for treating thrush
Mix of Maalox, Benadryl, and lidocaine (often called the “radiation cocktail”)	Used orally to swish and swallow for mouth pain and esophagitis
Mix of Maalox, Benadryl, and nystatin (often called “cools solution”)	Used orally to swish and swallow for mouth pain associated with, and to treat, thrush
“Magic Mouthwash”; constituents vary by formulation but usually contain a mix of antibiotic antihistamine, antifungal, corticosteroid, and Maalox (for coating purposes)	Used orally to swish and swallow for mouth pain and discomfort, to kill bacteria, to reduce fungal growth, and to treat inflammation

Data from medication product labeling.

TABLE 7.6 Medications to Treat Nausea and Vomiting

Medication: Generic Name (Trade Name)	Dose	Action	Special Considerations
Aprepitant (Emend)	Oral: 125 mg on the day of chemo; 80 mg a day after chemo; 80 mg 2 d after chemo IV: 150 mg over 20–30 min, given 30 min prior to chemo	Prevents N&V	Dose is for ≥12 year olds and ≥30 kg
Dolasetron mesylate (Anzemet)	Oral: 100 mg IV: 1.8 mg/kg over 30 sec	Decreases N&V	Give 30–60 min before chemo
Granisetron HCl (Kytril)	Oral: 1 mg tablet or 5 mL suspension BID IV: 10 µg/kg	Decreases N&V	Oral first dose 1 h before chemo and second dose 12 h later IV: 30 min before chemo
Lorazepam (Ativan)	Oral: 1–6 mg/d IV: 1.4 mg/m^2	Decreases anxiety, relaxes muscles, decreases N&V	Oral: in divided doses IV: 30 min before chemo
Metoclopramide (Reglan)	Oral: 10 mg QID IV: 1–2 mg/kg q2h	Stimulates GI motility and gastric emptying	May cause diarrhea at high doses
Ondansetron (Zofran)	Oral: 8 mg q4h for three doses, then q8h for 1–2 d IV: 32 mg bolus	Decreases N&V	Start oral and IV 30 min before chemo
Palonosetron hydrochloride (Aloxi)	IV: 0.25 mg over 30 sec, given 30 min prior to chemo	Prevents N&V	Adult dose
Perphenazine (Trilafon)	Oral: 4 mg q4–6h IV/IM: 3–5 mg bolus q4–6h	Decreases N&V	May cause dry mouth
Prochlorperazine (Compazine)	Oral: 5–10 mg QID Rectal suppository: 25 mg BID	Decreases N&V	Antacids decrease absorption of oral dose—separate by 2 h

GI, gastrointestinal.

Data from medication product labeling.

Description

TABLE 7.7 Medications to Treat Diarrhea

Medication: Generic Name (Trade Name)	Dose	Action	Special Considerations
Diphenoxylate HCl and atropine (Lomotil)	Oral: 5 mg QID	Slows peristalsis	Can cause nausea and dry mouth
Loperamide HCl (Imodium)	Oral: 4 mg, followed by 2 mg after each unformed stool	Inhibits peristalsis	Shown to be 2–3 times more potent than Lomotil

Data from medication product labeling.

Description

The following lists provide tips and techniques that the cancer patient and their family may find useful when managing some of the most common problems associated with cancer/cancer treatments.[13]

Taste Changes

- Eliminate unpleasant odors and food from sight.
- Drink nutritional supplements through a straw (placed in the back of the mouth) in a covered container to decrease odors and contact with taste buds.
- Bitter and acidic flavors are often well tolerated if stomatitis/esophagitis is not present (vinegar, pickled foods, vinegar-based salad dressings, sweet-and-sour sauce, lemon juice, mustard, ketchup, barbeque sauce).
- Drink fluids with meals and frequently throughout the day to moisten oral mucosa.
- Increase taste by adding spices and flavorings such as sugar, lemon, herbs, and wine.
- Serve foods attractively, balancing colors and textures.
- Use plastic utensils if metallic taste is a problem.
- Use temperature extremes (hot and cold) to stimulate taste.
- Use cold pineapple chunks between foods to change and stimulate taste sensation.

- Use lemon sorbet to cleanse the palate between foods.
- If foods are too sweet:
  - *Gymnema sylvestre*, an herbal tea that is often used by professional wine tasters, will deaden the taste buds to sweet tastes for about 20 minutes. This should be held in the mouth for about 5 minutes before eating.
  - Add a few drops of bitters (Angostura bitters, found in grocery stores) to drinks to cut the sweet taste.

Xerostomia

- Take several sips of water before swallowing.
- Cleanse mouth every 2–4 hours.
- Drink 2–3 L of fluid daily.
- Suck sugarless candy—lemon drops.
- Use Chapstick or Vaseline to keep lips moist.
- Avoid citrus fruits and dry foods.
- Use sauces and gravies with food to provide extra moisture.
- Use saliva substitute.

Oral Mucositis and Esophagitis

- Avoid acidic foods and juices (orange, pineapple, tomato).
- Avoid extreme temperatures and foods that are hard or irritating in texture.
- Eat food and fluids that are chilled.
- Drink nutritional supplements chilled.
- Rinse mouth with a warm saline solution after meals and at bedtime.
- Avoid alcohol-based mouthwashes.

- In mild cases, ice pops, ice water, or ice chips may help numb the area, but most cases require more intervention for relief of pain.

Nausea

- Eat small, frequent meals.
- Foods should be cold or at room temperature, soft, salty, and *not* greasy or rich.
- Separate liquids and solid food by at least 1 hour.
- Liquids should be cold.
- Try ginger ale or other ginger-containing foods.
- Stay upright 1–2 hours after eating, and keep head elevated.

Diarrhea

- Eat small, frequent meals that are warm or at room temperature.
- Avoid fatty foods (bacon, cheese, oils) and food that causes gas (broccoli, beans).
- Avoid citrus fruits and juices.
- Eat foods high in soluble fiber (oatmeal, barley, bananas, apple sauce).
- Avoid foods high in insoluble fiber (whole grains, legumes, cabbage, broccoli, fibrous fruits and vegetables with seeds, skins, or peels).
- Avoid alcohol and caffeine.
- Eat boiled white rice, tapioca, cream of rice cereal, bananas, and peeled potatoes.
- Use low-lactose dairy products such as yogurt and aged cheeses instead of milk and ice cream.

Constipation

- Drink adequate amounts of fluids.
- Increase intake of fiber-rich foods (whole grains, high-fiber cereals, wheat bran, fruits and vegetables, legumes, and popcorn).
- Eat meals at regular intervals on a daily basis.
- Drink a hot beverage in the morning to stimulate a bowel movement.
- If allowed, increase physical activity.

Kilocalorie and Protein Needs of the Adult with Cancer

The nutrition requirements of most individuals with cancer can be estimated using the following: energy, 25–35 kcal/kg body weight; protein: adults with cancer 1.0–1.5 g/kg/day, adults with cancer cachexia 1.5–2.5 g/kg/day, adults undergoing HCT 1.5g/kg/day. Individuals diagnosed with pancreatic, gastric, bile duct, kidney, adrenal, non-small cell lung, and head and neck cancers are typically hypermetabolic at the time of diagnosis.[17] These individuals should be screened for recent unintentional weight loss, and energy expenditure estimates should trend toward the higher end of the nutrient needs range. It is important to note that a patient who has a poor intake needs to consume nutritionally dense foods. Providing your patient with a high-kilocalorie, high-protein diet will help prevent rapid weight loss. The following are some common tips for patients:

- Eat small, frequent meals; keep snacks handy.
- Use nutritional supplements such as Ensure, Boost, etc.
- Add the following to foods to increase caloric and/or protein content: butter, margarine, whipped cream, half and half, cream cheese, sour cream, salad dressings,

mayonnaise, honey, jam, sugar, granola, dried fruits, cottage or ricotta cheese, whole milk, powdered milk, ice cream, yogurt, eggs, nuts, seeds, wheat germ, and peanut butter.

Neutropenic Diet

The neutropenic diet has been a mainstay in the oncology setting for decades despite a lack of evidence showing that adherence to this diet provides benefits to individuals with neutropenia. In a randomized controlled trial of 726 hematopoietic stem cell transplantation recipients, half followed a neutropenic diet and half did not. Results from this trial showed that following a neutropenic diet did not reduce infection. Interestingly, following a neutropenic diet was associated with *higher* rates of infection as well as increased rates of *Clostridium difficile* infection.[18] Many institutions have recently shifted from using a neutropenic diet to emphasizing the following safe food-handling techniques for healthy and immunocompromised individuals.

General Food Safety Guidelines

- Check expiration dates on all products before buying. Be sure nothing is past its expiration date.
- Wash the following with soap/cleanser and hot water before and after contact with food. Air-dry or use paper towels—do not use cloth towels (using a dishwasher is preferred, if available):
  - Counter tops
  - Cutting boards
  - Cooking utensils
  - Silverware
  - Pots and pans
  - Dishes

- Wash hands frequently with warm soapy water, and dry with paper towels when preparing food. This is important especially after touching raw meat, chicken, eggs, and fish.
- Keep perishable food very hot or very cold. Do not leave perishable items at room temperature for more than 10–15 minutes.
- All perishable foods should be cooked thoroughly (no raw or rare meats).
- Thaw frozen foods in the refrigerator overnight or quickly in the microwave. Do not thaw food on the counter.
- Refrigerate leftovers promptly in airtight containers.
- Use leftovers only if they have been stored properly and have been around for no more than 24 hours.

Diet Guidelines for Immunosuppressed Patients[19]

To minimize food safety concerns for immunosuppressed individuals, the following foods should be avoided:

- Unpasteurized/raw dairy products and soft cheeses (feta, Brie, Mexican queso, etc.) and cheeses made with live mold (blue, gorgonzola, etc.)
- Unwashed raw fruits/vegetables/herbs
- Raw, rare, and undercooked meats, poultry, fish/seafood, and eggs
- Raw sprouts (bean, alfalfa, mung, clover, etc.)
- Smoked and pickled fish
- Non-pasteurized and fresh salsas found in the grocery refrigerator case
- Hot dogs, luncheon/deli meats unless heated to 165 degrees F
- Raw honey

- Brewer’s yeast, if uncooked
- Avoiding salad bars/buffets/bulk bins/street vendors is recommended
- Kombucha tea
- Non-pasteurized fruit and vegetable juices
- Well water (unless tested safe each year)

Survivorship Nutrition

Any individual with a cancer diagnosis who is not in need of a specific nutrition intervention should be encouraged to follow dietary guidelines developed for cancer survivors. The American Cancer Society has established guidelines for cancer survivors to follow in order to reduce the risk of future disease.[20] Maintaining a healthy weight during one's lifetime is the second most important cancer-reducing risk factor following the avoidance of tobacco.[21] The guidelines also apply to individuals wishing to reduce dietary risk for the development of cancer. A summary of these guidelines follows:

- Body weight—achieve and maintain a healthy weight (BMI, 18.5 to 25 kg/m^2).
- Physical activity—from 150–300 minutes per week of moderate-intensity physical activity (brisk walking, yoga, leisurely bicycling) OR 75–150 minutes per week of vigorous-intensity activity (running, swimming, singles tennis) OR a combination of the two intensities; AND muscle-strength activities two or more days per week (hand weights, exercise bands, body weight activities such as push-ups or squats).[22]
- Diet—choose a mostly plant-based diet high in fruits, vegetables, legumes, and whole grains. Eat at least 2.5 cups of fruits and vegetables daily. Limit red and processed meats and avoid cooking these and other high-fat protein sources at high temperatures. Strong evidence suggests that the risk of colorectal cancer increases 12% for every 100 g red meat consumed daily and 16% for every 50 g processed meat consumed daily.[23]

- Sugar/fat—limit high-fat foods as well as foods with added sugar.
- Alcohol—advice on alcohol consumption should be tailored to each patient by his or her healthcare provider, as alcohol may increase risk of specific cancers. Alcohol consumption is one of the major modifiable risk factors for breast cancer, causing 7 of every 100 new breast cancer cases in the WHO European Region; the risk of breast cancer can be significantly reduced by simply reducing alcohol consumption.[24]
- Supplements—supplements should be considered only if a nutrient deficiency is biochemically or clinically demonstrated.

ADIME 7.1 ADIME At-A-Glance

Assessment

- Diet, appetite, food tolerance, taste alterations, intake history
- Medications (chemo, vitamin/mineral supplements, saliva substitute, antinausea)
- Labs (C-reactive protein, tumor markers, WBC counts)
- Physical findings: GI symptoms (esp. mouth sores, thrush, xerostomia, mucositis, dysphagia, trismus), N&V, diarrhea, constipation; nutrition-focused physical exam findings (loss of muscle mass/fat stores, edema); vitals, functional status
- Anthropometrics (weight/weight history, BMI)

Nutrition Diagnosis

- Predicted Suboptimal Energy Intake RT changes in taste and appetite and increased nutrient needs due

to chronic catabolic illness AEB scheduled therapy predicted to decrease ability to consume sufficient energy or nutrients, side effects of therapy, and nausea/vomiting.

- Inadequate Protein-Energy Intake RT decreased ability to consume sufficient protein and energy and surgery AEB weight loss of >5% × 1 month, BMI-18, bowel resection, NPO × 3 days, and recent Dx of colon cancer.
- Swallowing difficulty RT pharyngeal/esophageal tumor and esophagitis from radiation therapy AEB dry mucous membranes, pain with swallowing, decreased food intake (eating <50% of meals), and inability to swallow solid foods.

Intervention

- Addition of snacks, nutritional supplements
- Enteral or parenteral recommendations
- Addition of multivitamins and minerals
- Feeding assistance
- Nutrition-related medication management (appetite stimulants, saliva substitute)
- Diet instruction (high kilocalorie and protein, GI symptom management—N&V, dry mouth/throat)
- Communication with Speech Language Pathology, social work, multiple disciplines

Monitoring and Evaluation

- Patient to meet >75% of protein-energy needs by mouth
- Patient intake to meet 100% of estimated needs
- Enteral nutrition to be initiated

- Patient to follow medication recommendations
- BMI to improve
- No further weight loss with goal for weight gain
- Labs to return to normal limits
- Improvement of GI symptoms or problems
- Wound healing

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CHAPTER 8

Cardiovascular Disease

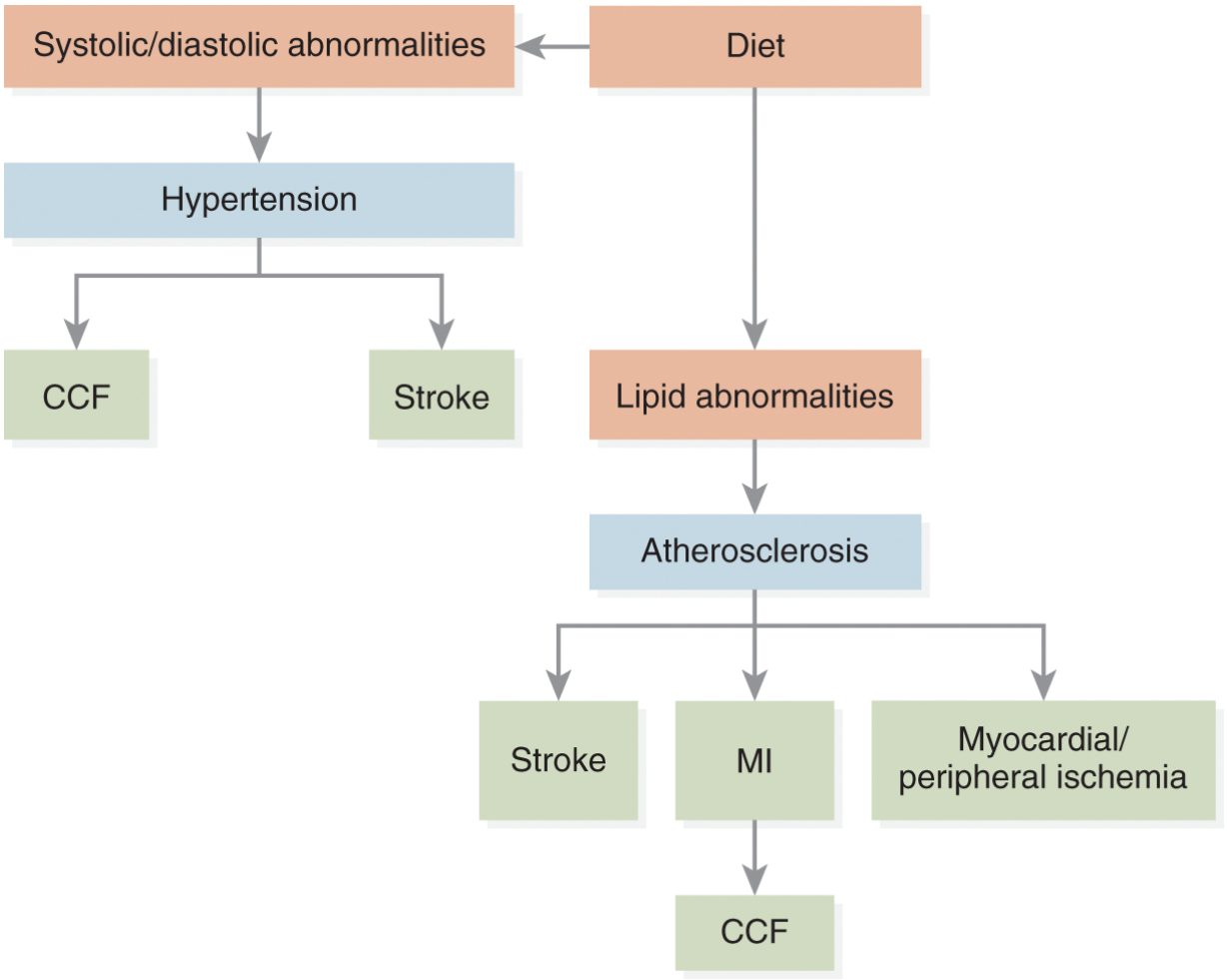
Prof. Dr. Tilakavati Karupaiah, APD, AN

The cardiovascular system includes the heart and blood vessels, which are functionally linked to other systems to facilitate metabolic functions, nutrient and gas transport, temperature and pH stabilization, maintenance of homeostasis, and the initiation of defense mechanisms. Diseases affecting the heart, coronary heart disease (CHD) leading to myocardial infarction (MI), and the circulatory network, cerebrovascular accident (CVA) or stroke, are collectively grouped as cardiovascular disease (CVD). Of note, diseases of the heart remained the top cause of mortality for Americans in 2019. Compared to the previous declining CVD mortality rates reported for 2009–2019, the trend changed with the occurrence of the COVID-19 pandemic, as indicated by a 4.1% increase for CHD and 4.9% for CVA between 2010 and 2020.[1] The data was only partially collected for the 2019–2020 cycle and was combined with 2017–2018 data to produce nationally representative estimates.[1] Both CHD (41.2%) and CVA (17.3%), however, remained major contributors to mortality in Americans[1] with significant racial disparities related to CVD mortality and risk factors (**Box 8.1**). Medical nutrition therapy (MNT) is a crucial component of lifestyle management, requiring specific changes in the diet to target the signs and symptoms of CVD, particularly hyperlipidemia, hypertension and dyslipidemias, as well as potential disease complications (**Figure 8.1**).

Box 8.1 Racial and Gender Disparities for CVD Risk Factors

- Among males, the highest **prevalence of obesity** was in Hispanics (44.6%). Among females, the highest obesity prevalence occurred in Blacks (56.8%).
- Percentage **prevalence of hypertension** was highest among Black males (55.8%) and females (56.9%).
- Although **hypercholesterolemia** rates dropped, its prevalence was the highest among White and Asian females (37.2 and 37.7% respectively), while among males it was the highest for Asians (40.7%).
- **Dyslipidemia** prevalence presenting with hypertriglyceridemia and low HDL-C levels was highest in Hispanic persons.
- **CVD** and **stroke** prevalence at all ages were highest in Blacks who were **more than twice as likely to die** as non–Hispanic-Asian or Pacific-Islander persons.
- **CHD, MI** and **HF** events were highly prevalent in Black females, whereas White males faced the highest prevalence for **CHD** and **MI**.

Data from “Tsao CW, Aday AW, Almarzooq ZI, Anderson CAM, Arora P, Avery CL, Baker-Smith CM, Beaton AZ, Boehme AK, Buxton AE, Commodore-Mensah Y, Elkind MSV, Evenson KR, Eze-Nliam C, Fugar S, Generoso G, Heard DG, Hiremath S, Ho JE, Kalani R, Kazi DS, Ko D, Levine DA, Liu J, Ma J, Magnani JW, Michos ED, Mussolino ME, Navaneethan SD, Parikh NI, Poudel R, Rezk-Hanna M, Roth GA, Shah NS, St-Onge M-P, Thacker EL, Virani SS, Voeks JH, Wang N-Y, Wong ND, Wong SS, Yaffe K, Martin SS; on behalf of the American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2023 update: a report from the American Heart Association. *Circulation*. 2023;147:e93-e621. doi: 10.1161/CIR.0000000000001123



Description

Figure 8.1 CVD and nutrition intervention targets. Blue box denotes disease conditions; green boxes denote endpoints of organ/systemic dysfunction.

Coronary Heart Disease

Disease Process

Coronary heart disease (CHD) begins with atherosclerosis, a chronic inflammatory process in response to arterial wall injuries leading to lesion formation.[2] Endothelial injuries are instigated by one or more factors such as elevated or oxidized low-density lipoprotein (LDL), free radicals, toxins associated with smoking, infectious microorganisms, high blood pressure (BP), high serum cholesterol, insulin resistance, shear stress, homocysteine, diets high in saturated fat, or a sedentary lifestyle.[3] Atherogenesis results in endothelial dysfunction with abnormal vascular smooth muscle growth, decreased anticoagulant and anti-inflammatory properties, and impaired vasomotor control.[4] Notably, endothelial dysfunction causes reduced nitric oxide production, which adversely affects vasodilation.

The core event in endothelial dysfunction is the trapping of LDLs within the subendothelial space, which subsequently undergo progressive oxidation initiated by free radicals from the intimal wall.[2] Oxidized or modified LDLs induce inflammatory responses, causing lesions in the intima, increased recruitment and adherence of monocytes and T lymphocytes to the atheroma, and penetration of monocytes into the tunica intima.[5] Monocytes convert into macrophages and readily take up oxidized LDLs, which subsequently become transformed into foam cells that eventually form the lipid core of the atherosclerotic plaque.[6] Oxidized LDLs add to endothelium injury, leading to the release of platelet-derived growth factor (PDGF), which in turn stimulates the proliferation and migration of smooth muscle cells into intima that take up oxidized LDL to form foam cells.[7]

The high lipid- or cholesterol-enriched atheromatous plaque is prone to rupture, as the lesion is not stable. The ruptured fatty plaque is exposed to blood flow, attracting the deposition of small, dense LDL, which subsequently leads to thrombus formation and platelet activation. The continuous deposition or accumulation of LDL and foam cells will progressively occlude the lumen of the blood vessel and slowly induce thrombosis, a proximate cause for a CVD event that, depending on the site, may be angina pectoris, ischemic heart disease, or MI.[4] Lipoprotein particles vary in size, with small, dense LDL particles proposed to be more atherogenic than larger- or normal-sized LDL particles and are considered to be an independent risk factor for CVD (**Table 8.1**).[8]

TABLE 8.1 Lipoprotein Subclasses and Atherogenic Risk

Lipoprotein Subclasses	Size in Nanometer (nm)	Atherogenicity
Large LDL	21.2–23.0	↓
Small LDL	18.0–21.2	↑
Large HDL	8.8–13.0	↓
Medium HDL	8.2–8.8	↓
Small HDL	7.3–8.2	↑

HDL, high-density lipoprotein; LDL, low-density lipoprotein; ↓, decreases risk; ↑, increases risk.
Data from Samia Mora, et al., "LDL Particle Subclasses, LDL Particle Size, and Carotid Atherosclerosis in the Multi-Ethnic Study of Atherosclerosis (MESA)," *Atherosclerosis* 192, no. 1 (2007): 211-217.

In clinical practice, for all lipoprotein carriers of cholesterol, LDL, very low-density lipoproteins (VLDL), and remnants are considered atherogenic and labeled together as non-high-density lipoproteins (non-HDL). Dyslipidemia, an additional clinical term, characterizes elevated atherogenic lipoproteins (LDL, VLDL, Lp(a), and remnants) and low levels of HDL.

Although elevated LDL is recognized as the major atherogenic lipoprotein for primary target treatment, the 2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol Committee (denoted as the 2018 AHA/ACC/multisociety cholesterol guideline) recommends the intensity of LDL-C lowering therapy should be based on absolute risk to patients, with those at the highest risk receiving the most intensive cholesterol reduction.[9] Accordingly, very high risk is defined as a history of multiple ASCVD events or of one event + multiple high-risk conditions. Specifically, the two goals for LDL-C lowering would be targeting ≥ 50% LDL-C reduction in secondary prevention using high-intensity statins and ≥35% reduction in primary prevention using moderate-intensity statins. Of note, no numerical targets were identified with the exception of an LDL-C threshold goal of 1.8 mmol/L (70 mg/dL) if prescribing PCSK9 inhibitors in very high-risk patients on maximal statin therapy + ezetimibe.

Referral to an RD/RDN is recommended for any patient with ASCVD and/or baseline LDL-C >190 mg/dL, or baseline LDL-C ≥190 mg/dL or for patients with ASCVD and baseline LDL-C ≥190 mg/dL who did not achieve reducing LDL-C ≥50% and LDL-C <70 mg/dL (or non–HDL-C <100 mg/dL) on maximally tolerated statin therapy in combination with nonstatin therapy. The dietitian referral may also be considered for other patients unable to achieve adequate LDL-C reduction.

The year 2013 heralded disengagement with using the Framingham 10-year Risk Score (FRS) algorithm, which was derived from a white sample population, as well as recognition that the FRS scope was limited to only outcomes related to CHD.[10] FRS has been replaced with risk equations for a 10-year estimated ASCVD risk of >7.5% based on a pooled cohort equation (PCE) factoring 5 large population groups in the USA (**Box 8.2**.[11] The ASCVD Risk Estimator is intended for use in patients without ASCVD and with an LDL-C <190 mg/dL in order to establish a baseline reference at initial visit. The 10-year risk estimation is provided for first hard ASCVD events (defined as the first occurrence of nonfatal MI, CHD death, or fatal or nonfatal CVA) as the outcome of interest with required baseline information on age, sex, race, total cholesterol, high-density lipoprotein (HDL) cholesterol, systolic BP, blood pressure-lowering medication use, diabetes status, and smoking status, with follow-up information on change in status for clinical data of the patient. The ASCVD Risk Estimator is importantly viewed as a tool to inform decision-making by the clinician in primary prevention of ASCVD, when lifetime risks for ASCVD are provided for adults 20 through 59 years of age at the initial visit.

Box 8.2 Calculation Steps for 10-Year Risk Estimate for Hard ASCVD

Step 1—The natural log of age, total cholesterol, HDL-C, and systolic BP (treated or untreated value) are first calculated.

Step 2—Interaction terms between the natural log of age are multiplied with values for total cholesterol, HDL-C, and treated or untreated systolic BP.

Step 3—Calculated values are then multiplied by the coefficients from equation parameters (“Coefficient” column) for the specific race—sex group of the individual derived from pooled cohort equations for estimation of the 10-year risk of ASCVD[a].

Step 4—The "Coefficient × Value" calculation provides the results of the multiplication for the risk profile for each parameter.

Step 5—The sum of the "Coefficient × Value" column is then calculated for the individual, shown as "Individual Sum" for each race and sex group.

Step 6—The estimated 10-year risk of a first hard ASCVD event is formally calculated[a] as 1 minus the survival rate at 10 years ("Baseline Survival"), raised to the power of the exponent of the "Coefficient × Value" sum minus the race- and sex-specific overall mean "Coefficient × Value" sum; or, in equation form: $1 - S10.^{(IndX'B - MeanX'B)}$

[a]Web-based ASCVD Risk Calculators are available at http://www.acc.org/tools-and-practice-support/mobile-resources/features/2013-prevention-guidelines-ascvd-risk-estimator and http://my.americanheart.org/cvriskcalculator.
Data from David C. Goff Jr., et al., "2013 ACC/AHA Guideline on the Assessment of Cardiovascular Risk—A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines," *Circulation*, no. 129 (2014): S49-S73.

Treatment and Nutrition Intervention

The 2018 ACC/AHA multisociety cholesterol guidelines prioritize primary prevention of ASCVD over the life span besides targeting the management of blood cholesterol, which constitutes a major ASCVD risk (**Table 8.2**).[9] The Task Group's position is that intensive lifestyle modification is a critical component of health promotion and ASCVD risk reduction, both prior to and in concert with, the use of cholesterol-lowering drug therapies.[9–11] According to the follow-up 2019 ACC/AHA guidelines for primary prevention of ASCVD, lifetime risk estimates for ASCVD should target adults 20 through 59 years of age at the initial visit, with such information (diet and exercise or physical activity) facilitating the clinician-patient discussion regarding risk reduction through lifestyle change.[12] Encouragement of a heart healthy lifestyle requires identifying barriers and facilitators for desired behavior changes to achieve recommendations for diet, physical activity, and maintaining optimal weight. Lifestyle change counseling provided for

ASCVD prevention should therefore adopt a comprehensive patient-centered approach that includes collaborative decision-making between the clinician and patient. Such approaches require incorporating team-based care to enable access to telehealth monitoring, follow-up support aids, and patient education. An additional assessment of social determinants of health is also critical in primary prevention, as the clinician is recommended to tailor advice to a patient's socioeconomic and educational status, as well as cultural, work, neighborhood, and home environments. Low health literacy or disadvantaged educational background levels of clients should be factored into counseling.

TABLE 8.2 LDL-C Targets for Statin Therapy Combined with Lifestyle Modification

Targets	Patient Groups	Statin & Non-Statin Therapy	Intensive Lifestyle Modification
Primary prevention	Children aged 0–19 y should be assessed periodically for lifetime risk[a] and calculated 10-year risk of ASCVD[b].	Children with diagnosis of familial hypercholesterolemia (FH) or abnormal lipid profiles; and fail 3-month lifestyle therapy	Emphasize a heart-healthy lifestyle across the life course for all individuals. Lifestyle therapy is primary prevention for individuals with metabolic syndrome. Lifestyle therapy is primary prevention for individuals with hypercholesterolemia.
Hypercholesterolemia (not very high-risk ASCVD)	Adults 20–39 y	Consider statin if family history, premature ASCVD and LDL-C ≥160 mg/dL (≥4.1 mmol/L)	Encourage lifestyle to reduce ASCVD risk
	Adults 40–75 y and LDL-C ≥70 to <190 mg/dL (≥1.8–<4.9 mmol/L) without diabetes mellitus Calculated 10-y ASCVD risk indicators are: ■ Low- <5% ■ Borderline- 5 to <7.5%[c] ■ Intermediate- ≥7.5–<20%[c] ■ high risk ≥20%[c] Coronary artery calcium (CAC) scores may guide statin use decision if risk is still unclear.	Clinician-patient risk discussion initiated for borderline, intermediate and high risk ASCVD patients before starting statins Topics include: ■ risk enhancers ■ patient's preference ■ drug costs and adverse effects of statins **Moderate-intensity** statins considered for borderline and intermediate-risk patients (30%–49% LDL-C reduction) **High-intensity** statins for high-risk ASCVD patients to achieve LDL-C lowering ≥50% **If high-intensity statin not tolerated, use moderate intensity statin.** If on maximal statin and LDL-C ≥70 mg/dL/1.8 mmol/L, add **ezetimibe**	Emphasize lifestyle for low to borderline ASCVD risk groups. Note—All individuals receiving statins should be counseled on healthy lifestyle habits
	Adults >75 y	Clinical assessment, Risk discussion [as above]	
Hypercholesterolemia (very high-risk ASCVD)	Age 20–75 y with severe hypercholesterolemia (LDL-C ≥190 mg/dL/4.9 mmol/L). Calculation of ASCVD 10-y risk not required	**Initiate** high-intensity or maximal statin therapy. Add **ezetimibe** if LDL-C ≥70 mg/dL (1.8 mmol/L). A proprotein convertase subtilisin/kexin type 9 inhibitor (PCSK9-I) with LDL-C ≥ 220 mg/dL despite maximally tolerated statins and ezetimibe	Review lifestyle habits for diet, physical activity, body weight/BMI, and tobacco use. Endorse healthy lifestyle with counseling and patient education material. Referral to dietitian required. Adherence to LM targets should be assessed.
	Age 20–75 y with severe hypercholesterolemia (LDL-C ≥190 mg/dL/4.9 mmol/L) and triglycerides ≤300 mg/dL/ 3.4 mmol/L.	**Maximally** tolerated statin therapy with **ezetimibe** if LDL-C lowering < 50% or ≥100 mg/dL (2.6 mmol/L) and addition of bile sequestrants.	*Evaluate diet as a secondary cause of hypertriglyceridemia:* ■ Elevated LDL-C—saturated or trans fats, weight gain, anorexia ■ Elevated TG—weight gain, very low-fat diets, high intake of refined carbohydrates, excessive alcohol intake; ethnic-based diets should be assessed for carbohydrate and fat consumption levels *Assess sedentary behaviors.* *Assess less favorable diets.*
	Age 30–75 y with heterozygous FH and LDL-C ≥100 mg/dL (2.6 mmol/L).	**Maximally** tolerated statin therapy with **ezetimibe** and addition of **PCSK9 inhibitor**.	Review lifestyle habits for diet, physical activity, body weight/BMI, and tobacco use.
	Age 40–75 y with baseline LDL-C ≥ 220mg/dL (5.7 mmol/L).	**Maximally** tolerated statin therapy with **ezetimibe** and addition of **PCSK9 inhibitor**.	Review lifestyle habits for diet, physical activity, body weight/BMI, and tobacco use.
With Clinical ASCVD	Individuals ≤75 y with clinical ASCVD not at high risk.	Consider **high-intensity statin** (moderate-intensity statin if high-intensity statin not tolerated). With **maximal statin** therapy and LDL-C ≥70 mg/dL (≥1.8mmol/L) ezetimibe may be added.	Counsel on intensive healthy lifestyle habits: ■ Regularly monitor adherence to lifestyle management. ■ Reinforce healthy lifestyle during follow-ups 3–12 mo.
	Age >75 y with clinical ASCVD not at high risk.	Initiate **moderate- or high-intensity** statin	

With Diabetes	Individuals 40–75 y of age with diabetes regardless of ASCVD risk estimation.	**Moderate-intensity statin**.	Counsel on intensive healthy lifestyle habits: ■ Diet should be adjusted for diabetes. ■ Regularly monitor adherence to lifestyle management. ■ Reinforce healthy lifestyle during follow-ups 3–12 mo.
	Individuals 40–75 y of age with diabetes and with LDL-C 70–189 mg/dL (1.7 to 4.8 mmol/L) with 1st ASCVD event.	Evaluate benefit for **high-intensity statin** based on individual's global risk (age group, race, and gender) and patient preference, lab values.	
	In adults with diabetes with multiple ASCVD risk factors.	**High-intensity statin**	
	Age >75 y with diabetes.	If not on statins, to initiate statin therapy. If on statin therapy, continue use of statins.	
	Adults with diabetes with 10-y ASCVD risk 20% or higher.	**Maximally** tolerated statin therapy with **ezetimibe**.	
	Age 20 to 39 y with long term diabetes and co-morbidities such as albuminuria, eGFR <60ml/min/1.73m^2, retinopathy, neuropathy, ankle-brachial index (ABI) <0.9.	Initiate statin therapy.	

High-intensity statin: lower LDL-C by ≥50%, such as Atorvastatin (40)–80 mg; Rosuvastatin 20 (*40*) mg; for history of hemorrhagic stroke and Asian ancestry.
Moderate-intensity statin: lower LDL-C by 30–49%, such as Atorvastatin 10 (*20*) mg; Rosuvastatin (*5*) 10 mg; Simvastatin 20–40 mg; Pravastatin 40 (*80*) mg; Lovastatin 40 (*80*) mg; Fluvastatin XL 80 mg; Fluvastatin 40 mg BID; Pitavastatin 1–4 mg.
Low-intensity statin: lower LDL-C by <30%, such as simvastatin 10mg, pravastatin 10–20 mg, lovastatin 20 mg, fluvastatin 20–40 mg.
[a]Lifetime risk constitutes cigarette smoking, elevated blood pressure, LDL-C, hemoglobin A1C [*if indicated*].
[b]ASCVD Risk estimate—A spreadsheet enabling estimation of 10-year and lifetime risk for ASCVD and a Web-based calculator are available at http://my.americanheart.org/cvriskcalculator and https://www.acc.org/tools-and-practice-support/mobile-resources/features/2013-prevention-guidelines-ascvd-risk-estimator.
[c]ASCVD Risk enhancers—family history of premature ASCVD, persistent elevated LDL-C ≥160 mg/dL (≥4.1 mmol/L), chronic kidney disease, metabolic syndrome, inflammatory diseases, ethnicity (South Asian ancestry), women-specific conditions (preeclampsia, premature menopause), persistently elevated triglycerides (≥175 mg/mL), elevated biomarkers (hsCRP ≥2.0 mg/L, Lp[a] >50 mg/dL or >125 nmol/L, apoB ≥130 mg/dL, ankle-brachial index (ABI) <0.9.

Data from Grundy, Scott M., et al., "2018 AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines," *Circulation* 139, no. 25 (2019): e1082-e1143, https://www.ahajournals.org/doi/10.1161/CIR.0000000000000625.

Description

The goal of the primary prevention approach is "ideal cardiovascular health," which identifies eight metrics for assessing individual and community health behaviors in individuals aged 40–75 years and children without clinical ASCVD or diabetes and with LDL-C <190 mg/dL (**Box 8.3**).[13] To meet this goal, lifestyle treatments target schools and workplaces in local communities throughout the nation, providing health coaching at the community level for both children and adults in specific diet and lifestyle management (**Box 8.4**).

Box 8.3 Ideal Cardiovascular Health: 7 Metrics to Assess Population Behaviors

- Current smoking status
- BMI
- Physical activity

- Healthy Diet Score
- Total cholesterol
- Blood pressure
- Fasting plasma glucose

"The metrics with the greatest potential for improvement are health behaviors, including diet quality, physical activity, and body weight."

BMI, body mass index.
Data from Robert H. Eckel, et al., "2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines," *Circulation* 129, no. 2 (2013): S75-S99.

Box 8.4 Lifestyle Management Guideline for Diet and Lifestyle: Key Recommendations

Heart Healthy Diet	Physical Activity
Adults and Children	***Children***
In the context of a DASH-type dietary pattern, to achieve at least four of five key components of a healthy diet: 1. Fruits and vegetables: >4.5 cups/d 2. Fish: more than two, 3.5-oz servings/wk (preferably oily fish) 3. Fiber-rich whole grains (>1.1 g of fiber per 10 g of carbohydrates): three 1-oz-equivalent servings/d 4. Sodium: <1,500 mg/d 5. Sugar-sweetened beverages: <450 kcal (36 oz)/wk	*Ideal:* ≥60 minutes of moderate to vigorous physical activity per day *Intermediate:* 1 to 59 minutes of moderate to vigorous physical activity per day *Poor:* No physical activity
Children/Adults	**Adults**
• *Ideal:* Diet Score 4 to 5 • *Intermediate:* Diet Score 2 to 3 • *Poor:* Diet Score 0 to 1	*Ideal:* At least 150 minutes of moderate or 75 minutes of vigorous physical activity each week *Intermediate:* 1 to 149 min/wk moderate or 1 to 74 min/wk

Heart Healthy Diet	Physical Activity
	vigorous activity *Poor:* No physical activity

Data from Robert H. Eckel, et al., "2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines," *Circulation* 129, no. 2 (2013): S75-S99.

For adults aged 40–75 years with or without ASCVD or diabetes and with LDL-C >190 mg/dL, the Task Group developed diet, exercise/physical activity recommendations for preventing or lowering ASCVD risk and provided evidence ratings for each recommendation (**Box 8.5**).[12,14] In addition, the AHA/ACC proposed promoting healthy dietary patterns, such as adopting Mediterranean and DASH diets to individuals and communities to target modifiable CVD risk factors of BP and lipids (**Box 8.6**).[13] Recommending a heart healthy diet to avoid weight gain and address BP and lipids requires engagement with lifestyle counseling which is consistent with racial and ethnic preferences such as Asian and Hispanic/Latino groups who exhibit regional differences in lifestyle preferences. With these ethnic groups undergoing acculturation, the expected challenges will be to avoid increased sodium, sugar, and calories.

Box 8.5 Lifestyle Factor Recommendations for Adults in Prevention of ASCVD

Diet and Exercise or Physical Activity Recommendations	Evidence Rating
1. Emphasizing intake of vegetables, fruits, legumes, nuts, whole grains; and fish to decrease ASCVD risk factors.	*COR: I, LOE: B-R*

Diet and Exercise or Physical Activity Recommendations	Evidence Rating
• Adapt this dietary pattern to appropriate calorie requirements, personal and cultural food preferences, and nutrition therapy for other medical conditions (including diabetes mellitus). • Achieve this pattern by following plans such as the Mediterranean style, DASH, Healthy US-Style and healthy vegetarian diets.	
2. Replacing saturated fat with dietary monounsaturated and polyunsaturated fats benefits reducing ASCVD risk. • Aim for 5 to 6% of calories from saturated fat. • Use nontropical LIQUID oils rather than tropical oils (coconut, palm and palm kernel), and animal fats (butter and lard). • Aim for low-fat or fat-free dairy products instead of full-fat diary products • Choose healthy protein sources (lean cuts of meat and poultry, legumes and nuts, fish, seafood)	*COR: IIa, LOE: B-NR*
3. Avoiding *trans* fat to reduce ASCVD risk. • Reduce percentage of calories from *trans* fat. • Avoid partially hydrogenated fats.	*COR: III, LOE: B-NR*
4. Reducing dietary cholesterol will benefit decreasing ASCVD risk. • Moderate egg intake. • Replace animal protein (red meat with plant protein (legumes and nuts) • Aim for healthy sources of animal protein (fish, seafood).	*COR: IIa, LOE: B-NR*
5. Reducing sodium intake will benefit decreasing ASCVD risk. • Choose minimally processed instead of ultraprocessed foods. • Choose and prepare foods with little or no salt.	*COR: IIa, LOE: B-NR*

Diet and Exercise or Physical Activity Recommendations	Evidence Rating
6. Minimizing the intake of processed meats, refined carbohydrates and sweetened beverages to reduce ASCVD risk. • Choose healthy instead of processed protein sources (lean cuts of meat and poultry, legumes and nuts, fish, seafood). • Choose foods made mostly with wholegrains rather than refined grains. • Reduce added sugar in food and beverage preparation or intake of processed foods and beverages with added sugars (glucose, dextrose, sucrose, corn syrup, honey, maple syrup, concentrated fruit juice).	*COR: IIa, LOE: B-NR*
7. Achieving and maintaining a healthy body weight benefits ASCVD risk reduction. • Engaging in at least 150 minutes per week of accumulated moderate-intensity or 75 minutes per week of vigorous-intensity aerobic physical activity (or an equivalent combination of moderate and vigorous activity) • Adjusting energy intake according to physiological need and age group. • Adoption of healthy dietary pattern concurrent with portion control and energy balance.	*COR: 1, LOE: B-NR*
8. Achieving and maintaining weight loss by adults with overweight and obesity improves ASCVD risk factor profile. • Achieving a clinically meaningful weight loss (≥5% initial weight). • Consuming reduced calorie diet ≥ 500 kcal from baseline intake. • Engaging in at least 200–300 minutes per week of accumulated high levels of physical activity	*COR: 1, LOE: B-R*

Diet and Exercise or Physical Activity Recommendations	Evidence Rating

For adults aged 40 to 75 years with or without ASCVD or diabetes and with LDL-C >190 mg/dL.
NHLBI, The National Heart, Lung, and Blood Institute; USDA, U.S. Department of Agriculture; COR, class of recommendation - I (strong benefit), IIa (moderate benefit), III (harm, strong); LOE, level (quality) of evidence (B, R-randomized, NR- not randomized).
Data from Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA guideline on primary prevention of cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol*. 2019;74:e177-e232; Lichtenstein AH, Appel LJ, Vadiveloo M, et al. 2021 dietary guidance to improve cardiovascular health: a scientific statement from the American Heart Association. *Circulation*. 2021;144:e472-e487.

Box 8.6 Healthy Dietary Patterns to Target Both BP and Lipid Lowering

Type of Diet	Description	Clinical Targets
MED Pattern	• Higher in fruits (particularly fresh), vegetables (emphasizing root and green varieties), whole grains (cereals, breads, rice, or pasta), fatty fish (rich in omega-3 fatty acids). • Lower in red meat (and emphasizing lean meats). • Substitute lower-fat or fat-free dairy products	• May benefit BP management • No consistent effect on plasma LDL-C, HDL-C, and TG

Type of Diet	Description	Clinical Targets
	for higher-fat dairy foods. • Use oils (olive or canola), nuts (walnuts, almonds, or hazelnuts), or margarines blended with grape-seed or flaxseed oils in lieu of butter and other fats. • Overall moderate in total fat (32–35% of total calories), relatively low in saturated fat (9–10% of total calories), high in fiber (27–37 g/d), and high in polyunsaturated fatty acids (particularly omega-3s).	

Type of Diet	Description	Clinical Targets
DASH Dietary Pattern	• High in vegetables, fruits, low-fat dairy products, whole grains, poultry, fish, and nuts. • Low in sweets, sugar-sweetened beverages, and red meats.	• Evidence strong for BP lowering in all and subgroups (men and women, African and non-African groups, hypertensive and non-hypertensive adults) • Evidence strong for lowering of LDL-C and HDL in all and similar effect for all subgroups
	• Low in saturated fat, total fat, and cholesterol. • Rich in potassium, magnesium, and calcium, as well as protein and fiber.	• No effect on TG
DASH Variations/Glycemic Index/Load Dietary Approaches	Two variations of the DASH dietary pattern: replacement of 10% of total daily carbohydrate calories with calories from either protein or unsaturated fat.	• May benefit BP management • May benefit lowering of LDL-C and TG with marginal increase in HDL-C

TG, triglyceride.
Data from Robert H. Eckel, et al., “2013 AHA/ACC Guideline on Lifestyle Management to Reduce Cardiovascular Risk: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines,” *Circulation* 129, no. 2 (2013): S75-S99.

Hypertension

On the basis of NHANES 2017–2020 data, hypertension (HTN) is a significant contributor (1.3%) to total mortality in the U.S. population. An estimated 122.4 million adults ≥20 years of age (46.7%) were affected by HTN, with the highest prevalence in African Americans (55.8% in men and 56.9% in women).[1,15] HTN prevalence in young adults 20 to 44 years of age (28.5%) is also a concern.[1] For adults diagnosed with HTN, only 43.7% are under control, with young adults with the lowest rates of control.[1] Untreated HTN significantly increases the risk of CVA, with 77% of those experiencing a first CVA having BP >140/90 mmHg, while diabetics with BP <120/80 mmHg have half the lifetime CVA risk than those with just HTN.[16] In contrast, eliminating HPT is estimated to reduce CVD mortality by 30.4% among males and 38.0% among females.[17] It is critical to understand that the interpretation of HTN prevalence in the U.S. adult population is now substantially higher (48.6%)[1] as defined by the high blood pressure (BP) ACC/AHA 2017 guideline, which is SBP ≥130 mmHg, DBP ≥80 mmHg compared to when prevalence data (32%) was defined by the previous JNC 7 definition.[18]

Disease Process

Blood is pumped to the body by the left ventricle of the heart, imparting a pressure that is opposed by the resistance of the blood vessels through which it flows. The balance of these two opposing forces produces BP, which rises and falls as the heart contracts and relaxes. The peak, when the heart contracts, is known as the systolic pressure and the minimum, when the heart relaxes, as diastolic pressure. Homeostatic regulation of BP via the kidney and

the sympathetic nervous system (SNS) also has a role in controlling BP peripherally.[19,20] Maintenance of BP within certain limits enables adequate blood flow to the brain and other tissues. Systolic-diastolic abnormalities lead to the development of essential HTN and event endpoints.[21]

The resistance of blood vessels increases when the diameter of arterial vessels narrows with progressive atherosclerosis. Regulatory mechanism failure from a hyperactive SNS or renin–angiotensin system also contributes to vasoconstriction and HTN. In the short term, elevated BP damages the endothelial lining of the arteries, allowing increased entry of LDL-C. In the long term, arterial stiffening and end-stage kidney disease develop with untreated BP. Left ventricular hypertrophy develops over time with uncontrolled HTN leading to heart failure, sometimes referred to as congestive heart failure. Occlusion of blood vessels in the heart can cause MI, retinopathy of the eyes, or in the brain, a CVA.

BP is one of the eight metrics of cardiovascular health[1] and the AHA, by incorporating HTN into its strategic plan, seeks to reduce the death rate from CVD and CVA by 20% by 2020 and beyond.[22] Additionally, the *Million Hearts® project*, spearheaded by the Centers for Disease Control and Prevention (CDC), strategizes *with* 5-year goals to prevent a million heart attacks and strokes at population level through clinical targets, and at the community level by adopting lifestyle management (LM) issues to support actions to improve and achieve control of HTN.[23] LM is recognized as a crucial non-pharmacological approach to HTN management with clear evidence-based goals (all COR:1 and LOE:A) for weight loss, a healthy diet, reduced intake of sodium, enhanced intake of dietary potassium, physical activity, and moderation of alcohol intake.[24]

Because a high percentage of the population has undiagnosed HTN, identification and diagnosis are key steps in achieving control of HTN. The ACC/AHA 2017 guidelines

designated normal BP to be a systolic level <120 mmHg and a diastolic level <80 mmHg and HTN diagnosis based on BP levels at three stages: elevated, stage 1 HTN, and stage 2 HTN (**Box 8.7**).[18]

Box 8.7 ACC/AHA 2017 Classification of Blood Pressure

BP Category	Systolic (mmHg)		Diastolic (mmHg)
Normal	<120	And	<80
Elevated	120–129	Or	<80
Stage 1 HTN	130–139	Or	80–89
Stage 2 HTN	≥140	Or	≥90

Data from Paul K. Whelton, et al., "2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines," *Hypertension* 71, no. 6 (2018): e13-e115, DOI: 10.1161/HYP.0000000000000065

Description

Treatment and Nutrition Intervention

The ACC/AHA 2017 Task Group developed an HTN treatment algorithm as part of a multifactorial approach to improve BP control (**Box 8.8**), which is referenced by a joint AHA and ACC science advisory on effective management of hypertension.[24] LM is strongly recommended in prevention as well as control approaches to HTN (**Box 8.9**).[12,24] For people with Stage 1 HTN (systolic <130 mmHg and diastolic <80 mmHg) and without clinical CVD or 10-year ASCVD risk ≥10%, treatment begins with LM for 6 months only, and upon assessment if BP is > target then BP-lowering medication may be added to treatment. Otherwise patients with clinical CVD and having 10-year ASCVD risk ≥10%, then treatment with BP-lowering medication and non-

pharmacological strategies are considered. A higher limit for BP-lowering medication will be implemented for Stage 2 (>140/90 mmHg) or HTN crisis (>180/120 mmHg).[24] Depending on the HTN state, patient's race, and co-morbid status with regard to the presence of heart failure, stroke, diabetes, and/or chronic kidney disease, the choice between monotherapy or combination drug therapy varies, but all patients are recommended to implement LM. Of the various drugs used to treat HTN, some have nutrition implications. Antihypertensives are grouped according to mechanistic function and may interact with specific foods or nutrients or cause gastrointestinal distress, fluid retention, or dehydration (**Box 8.10**). Team-based care to improve BP control is recommended at the health-system level with multidisciplinary healthcare members, of whom the dietitian is integral in contributing skills and knowledge related to medical nutrition therapy.[18]

Box 8.8 Hypertension Treatment Algorithm

Patient Groups	Antihypertension Therapy[a]		Lifestyle Modification[b]
	Indication	Monitoring	
Normal BP with systolic BP <120 or diastolic <80 mmHg	No	*Reassess annually*	Promote optimal lifestyle habits
Elevated BP with systolic BP 120–129 or diastolic <80 mmHg	No	*Reassess 3 to 6 mo.*	Non-pharmacological therapy[c] is preferred. Adherence and impact should be assessed 3 to 6 mo.
Stage 1 hypertension with systolic BP 130 to 139 mmHg or diastolic BP 80 to 89 mmHg	No	*Reassess after 6 mo.*	Non-pharmacological therapy.[c]
■ With Clinical ASCVD or estimated 10-y CVD risk ≥10%	Yes	*Reassess in 1 mo.*	Non-pharmacological therapy.[c]
Stage 2 hypertension with systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg	Yes	*Reassess in 1 mo:* *If goal not met—assess and optimize adherence to therapy, consider intensified therapy.* *If goal is met—reassess in 3–6 mo.*	Non-pharmacological therapy.[c]

[a]**Suggested drugs** are adjusted as per HTN stage per age, race, and co-morbidities.
Stage 1 HTN patients require initial monotherapy with dosage titration with sequential addition of other agents if required to achieve BP target.
Stage 2 HTN patients are initiated with combination therapy using 2 first-line agents of different classes or in a fixed-dose combination.
African-American patients with/out diabetes but without heart failure/CKD—initial therapy is thiazide diuretic or CCB.
First-line drugs include thiazide diuretic, CCB, ACE-I or ARB.

Stable Ischemic Heart Disease: BB, ACE-I, ARB
Angina Pectoris: CCB, thiazide diuretic
Aortic Disease: BB
Atrial fibrillation: ARB
Heart Failure: ACE-I or ARB, MRA, thiazide diuretic, BB, ARNI
Heart Failure with Preserved Ejection Fraction: ACE-I or ARB, BB, thiazide
Diabetes: ACE-I or ARB, thiazide diuretic, CCB
Chronic Kidney Disease: ACE-I or ARB
Secondary Stroke or TIA: thiazide diuretic, ACE-I, ARB
Abbreviations: ACE-I, angiotensin-converting enzyme inhibitor; ARNI, angiotensin receptor-neprilysin inhibitors; ARB, angiotensin II receptor blocker; BB, β-blocker; BP, blood pressure; CCB, calcium channel blocker; HTN, hypertension; MRA, mineralocorticoid receptor antagonist; TIA, transient ischemic attack

[b]**Social determinants of health** should be considered in lifestyle modification therapy:
- Diet and sedentary lifestyle
- Poverty
- Income inequality
- Food insecurity
- Unhealthy food environment of neighborhoods
- Health literacy

[c]**Non-pharmacological therapy**:
Note- All Recommendations COR:1; LOE:A
[i] Weight loss (aim for ideal body weight or—1kg for overweight status)
[ii] A heart-healthy diet as per dietary patterns (DASH etc.)
[iii] Sodium reduction (optimal is <1,500 mg/d but aim for 1,000 mg/d)
[iv] Potassium supplementation aiming for 3,500–5,000 mg/d (preferable with diet modification)
[v] Increased physical activity:
- Aerobic
  - 90–150 min/wk
  - 65%–75% heart rate reserve
- Dynamic resistance
  - 90–150 min/wk
  - 50%–80% 1 rep maximum
  - 6 exercises, 3 sets/exercise, 10 repetitions/set
- Isometric resistance
  - 4 × 2 min (hand grip), 1 min rest between exercises, 30%–40% maximum voluntary contraction, 3 sessions/wk
  - 8–10 wk

[vi] Moderate alcohol consumption
- women ≤ 1 standard drinks/d
- men ≤ 2 standard drinks/d

Data from (i) Paul K. Whelton, et al., "2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines," *Hypertension* 71, no. 6 (2018): e13-e115, DOI: 10.1161/HYP.0000000000000065 (ii) Carey, Robert M., Wright Jr, Jackson T., Taler. Sandra J., Whelton, Paul K. Guideline-Driven Management of Hypertension- An Evidence-Based Update. Circulation Research. 2021;128:827-846. https://doi.org/10.1161/CIRCRESAHA.121.318083; and (iii) Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA guideline on primary prevention of cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. *J Am Coll Cardiol.* 2019;74:e177-e232.

Description

Box 8.9 Lifestyle Modification Specifics to Benefit BP Lowering in Adults

Target	Recommendations for BP Lowering	Evidence Rating
1. Community action to adopt healthful behaviors: • Health coaching to modify diets. • Health coaching to read food labels. • Understand fresh versus processed food choices. • Identify healthy food retail services in neighborhoods.	Consume a dietary pattern that emphasizes intake of vegetables, fruits, and whole grains; includes low-fat dairy products, poultry, fish, legumes, non-tropical vegetable oils, and nuts; and limits intake of sweets, sugar-sweetened beverages, and red meats: • Adapt this dietary pattern to appropriate calorie requirements, personal and cultural food preferences, and nutrition therapy for other medical conditions (including diabetes mellitus). • Achieve this pattern by following plans such as the DASH dietary pattern, the USDA Food Pattern, or the AHA Diet.	*COR: I, LOE: A*
	Decrease sodium intake.	*COR: I, LOE: A*

Target	Recommendations for BP Lowering	Evidence Rating
1. Individual health management: • Health literacy should be factored in. • Consider use of mobile apps in engagement. • Family inclusion in education. • Encourage self-management goals. • Provide plans for exercise, sodium-reduction meal plans for breakfast, lunch and dinner. • Identify environmental barriers to healthy food shopping.	a. Consume no more than 2,400 mg/d of sodium. b. Further reduction of sodium intake to 1,500 mg/d is desirable since it is associated with an even greater reduction in BP. c. Reduce sodium intake by at least 1,000 mg/d since that will lower BP, even if the desired daily sodium intake is not yet achieved.	*COR: IIa, LOE: B*
	Combine the DASH dietary pattern with lower sodium intake.	*COR: I, LOE: A*
	In general, advise adults to engage in aerobic physical activity to lower BP: 3 to 4 sessions a week, lasting on average 40 minutes per session and involving moderate to vigorous intensity physical activity.	*Grade: B (moderate); COR: IIa, LOE: A*

Abbreviations: NHLBI, The National Heart, Lung, and Blood Institute; USDA, U.S. Department of Agriculture; COR, class of recommendation; LOE, level of evidence.
Data from (i) Paul K. Whelton, et al., “2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for

Target	Recommendations for BP Lowering	Evidence Rating
the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines," *Hypertension* 71, no. 6 (2018): e13-e115, DOI: 10.1161/HYP.0000000000000065, and (ii) Carey, Robert M., Wright Jr, Jackson T., Taler. Sandra J., Whelton, Paul K. Guideline-Driven Management of Hypertension- An Evidence-Based Update. Circulation Research. 2021;128:827-846. https://doi.org/10.1161/CIRCRESAHA.121.318083.		

Box 8.10 Medications Used in HTN Management with Nutritional Implications

Antihypertensive Class	Mechanism of Action	Nutritional Implications
Diuretics	Promote excretion of excess sodium and water, thus helping control BP. Likely use in combination therapies.	May cause hyperkalemia if potassium-sparing. • Monitor mineral salts/supplements. May increase urinary potassium, zinc, calcium, or magnesium excretion (e.g., thiazides). • Weakness, leg cramps, or being tired may result. • Eating potassium-rich foods may offset significant potassium loss.
β-Blockers	Reduce heart rate, the heart's workload, and the heart's	Gastrointestinal side effects, including heartburn, gas,

Antihypertensive Class	Mechanism of Action	Nutritional Implications
	output of blood, which lowers BP.	diarrhea, vomiting, or constipation. • Monitor dehydration.
ACE inhibitors	Angiotensin causes arteries supplying the kidney as well as the rest of the body to become narrow in uncontrolled hypertension. Angiotensin-converting enzyme (ACE) inhibitors help the body produce less angiotensin, which helps blood vessels vasodilate, which, in turn, lowers BP.	May cause increased mineral retention (potassium, calcium, or magnesium). • Monitor mineral salts/supplements.
Angiotensin II receptor blockers (ARBs)	Block the effects of angiotensin. ARBs block the angiotensin receptors so the angiotensin fails to constrict the blood vessel. This means blood vessels stay open and BP is reduced.	
Calcium channel blockers (CCBs)	Prevent calcium from entering the smooth muscle cells of the heart and arteries, thus preventing hardening and reduced contractility of the vessels. CCBs	• Grapefruits and grapefruit juice can affect the action of many CCBs. • Alcohol interferes with the effects of CCBs and increases side effects.

Antihypertensive Class	Mechanism of Action	Nutritional Implications
	relax and open up narrowed blood vessels, reduce heart rate, and lower BP.	Common side effects: edema, constipation, diarrhea, gastroesophageal reflux disease.
α-Blockers	Reduce arteries' resistance, relaxing the muscle tone of the vascular walls.	Side effects: weight gain.
α-2 Receptor agonist	Reduce BP by decreasing the activity of the sympathetic (adrenaline-producing) portion of the involuntary nervous system.	
Combined α- and β-blockers	Combined α- and β-blockers are used as an IV drip for those patients experiencing a hypertensive crisis and at risk for heart failure.	
Central agonists	Decrease blood vessels' ability to tense up or contract via a different nerve pathway than the α- and β-blockers, but accomplish the same goal of BP reduction.	Side effects: dry mouth, constipation.
Peripheral adrenergic inhibitors	Reduce BP by blocking neurotransmitters in the brain, which block the smooth	Side effects: diarrhea or heartburn.

Antihypertensive Class	Mechanism of Action	Nutritional Implications
	muscles from getting the “message” to constrict. Rarely used unless other medications do not help.	
Vasodilators	Cause the muscle in the walls of the blood vessels (especially the arterioles) to relax, allowing the vessel to dilate (widen). This allows blood to flow through better, thus reducing BP.	Side effects: may cause fluid retention (marked weight gain).

The Dietary Approaches to Stop Hypertension (DASH) eating plan has been shown to lower BP when managing patients with HTN (**Table 8.3**).[25] The DASH eating plan can be incorporated into any diet, including those with diabetes. It emphasizes an eating plan lower in sodium content that is also low in saturated fat, cholesterol, and total fat and higher in fruits, vegetables, and low-fat dairy foods compared with other dietary patterns. Higher potassium intake through increased fruit and vegetable intakes can also help lower BP. Sodium chloride or salt reduction is the core of LM, with nutrition coaching required to target several approaches to supporting healthy behaviors (**Box 8.11**).[26,27]

TABLE 8.3 DASH Eating Plan Based on Energy Level

Food Group	Serving Size	Calories per Serving[a]	Servings as per Daily Calorie Plan			
			1,600 kcal	2,000 kcal[b]	2,600 kcal	3,100 kcal
Grains	1 slice bread 1 cup ready-to-eat cereal[c] ½ cup cooked rice, pasta, or cereal	75	6–7	7–8	9–10	11–12
Vegetables	1 cup raw leafy vegetable ½ cup cooked vegetable 6-oz vegetable juice	Estimated as ~25 kcal	3–4	4–5	5–7	6–8
Fruits	1 medium fruit ¼ cup dried fruit ½ cup fresh, frozen, or canned fruit 6-oz fruit juice	60	3–4	4–5	5–7	6–8
Low-fat or fat-free dairy	8-oz milk 1 cup yogurt 1½-oz cheese	125 of low fat or 90 if fat-free	2	2–3	3–4	3–5
Meats, poultry, fish	3-oz cooked lean meat, skinless poultry, or fish	158 if meat/poultry, but 85 if fish	≤2	2 or fewer	3 or fewer	4 or fewer
Nuts, seeds, dry beans, and peas	⅓ cup or 1½-oz nuts 1 Tbsp or ½-oz seeds ½ cup cooked dry beans	45	3–4/wk	4–5/wk	5–7/wk	6–8/wk
Fats and oils[d]	1 Tsp soft margarine 1 Tbsp low-fat mayonnaise 2 Tbsp light salad dressing 1 Tsp vegetable oil	45	2	2–3	3–4	3–5
Sweets	1 Tbsp sugar 1 Tbsp jelly or jam ½-oz jelly beans 8-oz lemonade	60	4/wk	5/wk	7/wk	8/wk
Sodium	*Check food labels for sodium content per serving size*	2,300 mg *1,500 mg for high risk*[e]				

[a]Calories estimated from website: http://glycemic.com/DiabeticExchange/The%20Diabetic%20Exchange%20List.pdf.
[b]Gray area in the recommended DASH eating plan. Servings are calculated for a 2,000 kcal diet. http://www.nhlbi.nih.gov.
[c]Serving sizes vary between ½ cup and 1¼ cups. Check the product's nutrition label.
[d]Fat content changes serving counts for fats and oils: For example, 1 Tbsp of regular salad dressing equals 1 serving, 1 Tbsp of low-fat salad dressing equals ½ serving, and 1 Tbsp of fat-free salad dressing equals 0 servings.
[e]For African Americans at high risk for hypertension. Lower sodium goal effective at lowering BP.

Data from Government of the United States, National Heart, Lung, and Blood Institute, *Your Guide to High Blood Pressure with DASH*, NIH Publication No. 06-4082, (Washington, DC: U.S. Department of Health and Human Services, 2006).

Description

Box 8.11 Education Topics for Achieving Sodium Reduction

Topics	Tips	Why?
But, I *never* use the salt shaker—so I must be	Where does sodium come from? Forty-four percent of sodium consumed comes from 10 types of food: breads and rolls, cold cuts and cured meats such as	Sixty-five percent of sodium intake comes from food and meals purchased at retail stores and ~ 25%

Topics	Tips	Why?
eating a low-sodium diet	deli or packaged ham, or turkey, pizza, fresh and processed poultry, soups, sandwiches such as cheeseburgers, cheese, pasta dishes, meat- mixed dishes such as meat loaf with tomato sauce, and snacks such as chips, pretzels, and popcorn.[26]	consumed at restaurants. Barely 10% comes from salt added during cooking and at the table.[26] Average intake ~ 3,400 mg/d (excludes table and cooking salt).[27]
Label language	**Sodium-Free/Salt-Free** Less than 5 mg sodium per serving. **Very Low Sodium** 35 mg or less per serving. **Low Sodium** 140 mg or less sodium per serving. **Unsalted or No Added Salt** No salt during processing does not necessarily mean “sodium-free”. **Reduced/Less Sodium** At least 25% less sodium per serving than the same food with no sodium reduction. **Light in Sodium/Lightly Salted** At least 50% less sodium. **Low-Sodium Meal** 140 mg or less sodium per 100 g (3½ oz).	Label reading—best way to avoid excess sodium.
Salt substitution	Compare products within food categories:	Label reading—best way to avoid excess sodium by substituting
	Oats, deli foods, tomato products, cereals, sauces, canned goods.	“high” for “low” sodium content products in each food class.

Topics	Tips	Why?
Eating out	**Forming the sodium budget** Plan a sodium allowance for meals eaten outside.	Being aware of the sodium content of food can make a *big* difference in sodium intake.
Meal planning	**Fresh versus processed**	Using whole unprocessed foods in low-sodium meals can save more than *3,130 mg* of sodium.
Cooking skills	**Flavor without salt** **Beef:** dry mustard, nutmeg, onion, sage, pepper, ginger, garlic **Lamb:** garlic, curry, mint, rosemary **Chicken:** paprika, thyme, sage, parsley, curry, ginger, garlic **Fish:** dry mustard, paprika, curry, lemon juice, dill, basil **Eggs:** pepper, dry mustard, paprika, tarragon **Asparagus:** lemon juice, nutmeg, onion **Green beans:** lemon juice, nutmeg, onion **Broccoli:** lemon juice, oregano **Cabbage:** mustard, caraway seed, vinegar **Carrots:** allspice, ginger, cloves **Cauliflower:** nutmeg, celery, seed **Peas:** onion, mint **Potatoes:** parsley, chives **Squash:** ginger, basil, oregano **Tomatoes:** basil, oregano, sage, thyme	Flavors are taste substitutes for low sodium. Flavor enhancers are inexpensive and delicious.

Acute Care for Cardiovascular Events

Myocardial Infarction

Disease Process and Nutrition Intervention

The prevalence of myocardial infarction (MI) in adults ≥20 years of age, as indicated by the NHANES 2017–2020 data, is 3.2%, with rates doubling in males (4.5%) compared to females (2.1%).[1] Most MI patient cases are new, while a third of these may be silent. An MI cuts off blood supply to the heart, causing an oxygen deficit to cells of the heart muscle, leading to necrosis; therefore, the more prolonged the time to treatment to restore blood flow, the greater the damage to the heart muscle.[28] The critical stage for an MI patient is within the first 48 hours, followed by the acute stage that lasts between 3 and 14 days, and convalescence for the next 15 days to 3 months. Post-MI arrhythmias, nausea and vomiting, and fatigue are common symptoms.[29]

MNT in the acute stage focuses on reducing stress on the heart and preventing arrhythmias (**Box 8.12**).[30] As the patient moves to the next stage, a liquid diet is progressed as tolerated to soft, easily digested foods offered in small, frequent meals, with the exclusion of potential stimulants, such as caffeine, to reduce the potential for arrhythmias. In addition, serving foods at body temperature may also help in this regard. Prior to discharge, patient education should include the 2013 ACC/AHA recommendations for dietary modification and encouragement to follow up with the cardiac rehabilitation program and outpatient nutrition counseling.[13] Potential nutrition implications for medication used in the treatment of acute MI and other acute CVD events are shown in **Box 8.13**.

Box 8.12 MNT Strategies in Acute MI

Treatment Phase	MNT Approach	Assess and Monitor
Post-MI/critical stage *Rest to reduce heart strain*	To reduce heart strain: • *Use clear to full liquid diets* • *Small volume feeding*	I/O charts, LDH, CPK, BP, BUN, Pco_2, Po_2, triglycerides, PT • Aspiration • Vomiting
Prevent arrhythmias	To minimize arrhythmias: • *Food must be at body temperature.* • *Exclude stimulants such as coffee.*	• Abdominal distention • Flatulence • Constipation
Acute stage/recovery	• Liquid diet progress to soft easily digested foods. • Small frequent meals. • Exclude gas-forming foods. • Exclude stimulants to minimize arrhythmias.	• Abdominal distention • Constipation
Convalescence/stable stage	• Either Med or DASH diet as per ACC/AHA guidelines.	• Optimize weight by using calorie plan • Sodium control

Box 8.13 Nutritional Implications of Medications Used in Acute CVD Events

Acute Event	Drugs	Nutritional Implications
Myocardial infarction	CCBs	Common side effects: edema, constipation, diarrhea, gastroesophageal reflux disease
	β-Blockers	Gastrointestinal side effects, including heartburn, gas, diarrhea, vomiting, or constipation Monitor dehydration
	Anticoagulant and thrombolytic therapy—Warfarin (Coumadin) or heparin	Excessive intake of vitamin K foods alters PT values Also dong quai, fenugreek, gingko, ginseng Vitamin E and CoQ10 contraindicated
	Mexitil, Rythmol, Procan	Nausea, vomiting, or constipation Bitter taste, nausea, anorexia, or diarrhea with Procan

Acute Event	Drugs	Nutritional Implications
Heart failure	Thiazide diuretics	Deplete urinary potassium, zinc, calcium, or magnesium excretion (e.g., thiazides)
	Furosemide (Lasix)	Weakness, leg cramps, or being tired may result
	KCl	Side effect: hyperkalemia
	Digitalis	Deplete potassium when taken with Lasix Side effect: anorexia or nausea
	Arterial vasodilators	Side effect: nausea and vomiting
	Anticoagulant and thrombolytic therapy—Warfarin (Coumadin) for bedridden patients	As above
	ACE inhibitors	Side effect: nausea and vomiting, abdominal pain, hyperkalemia
Cerebrovascular accident	Anticoagulant and thrombolytic therapy—Warfarin (Coumadin) or heparin	
	Reserpine	Cramping and diarrhea

ACE, angiotensin-converting enzyme; CCBs, calcium channel blockers.

ADIME 8.1 ADIME At-A-Glance: Coronary Heart Disease and Hypertension

Assessment

- BMI ≥25, recent wt gain, waist circumference >40 in men and >35 in women
- Sedentary lifestyle
- Excess dietary intake of energy, trans and saturated fatty acids, proinflammatory foods, salt/sodium, alcohol.
- Inadequate dietary intake of fruits and vegetables—anti-inflammatory foods and antioxidants.
- BP levels: TG, LDL-C, HDL-C, inflammatory markers
- Diabetes, hx of previous CVD event
- Food and nutrition knowledge deficit, beliefs/attitudes, or practices about food, nutrition.

Nutrition Diagnosis

- Overweight RT sedentary lifestyle AEB BMI of 27 and recent wt gain of 10% × 1 year.
- Class 1 obesity RT excessive energy intake AEB BMI of 31 and diet hx of 2,600 kcal/d.
- Excessive SFA/TFA RT knowledge deficit AEB diet hx and elevated LDL-C of 190 mg/dL.
- Intake of PUFA inconsistent with needs RT undesirable food choices AEB LDL-C of 190 mg/dL.
- Excessive sodium intake RT knowledge food choices AEB diet hx of low fruit/veg intake.
- Limited adherence to recommendations RT low interest in change AEB diet hx of low compliance.

Intervention

- Decrease energy (1,800 kcal) and increase physical activity (20 minutes, 3 × /wk).
- Decrease SFA/TFA to <6% total energy and increase PUFA/MUFA to 15% total energy.
- Decrease sodium intake to 1,500 mg/d.
- Increase potassium intake to 3 to 5 g/d by following DASH diet pattern.
- Referral to outpatient nutrition counseling for decreased SFA/energy and increased PUFA diet.

Monitoring and Evaluation

- Body weight
- Dietary intake
- BP, blood lipids, inflammatory markers (if available)

Heart Failure

Disease Process and Nutrition Intervention

Heart failure (HF) or congestive heart failure (CHF) occurs when the heart muscle is unable to pump blood efficiently in the lower ventricles. This failure commonly arises from the narrowing of arteries resulting from coronary artery disease, HTN, chronic pulmonary disease, or heart damage from a previous MI, gradually leaving the heart too weak or stiff to fill and pump efficiently. Right-sided HF causes pitting edema in all extremities, whereas left-sided HF results in pulmonary edema, rales, and dyspnea. Decreased renal blood flow causes elevated blood urea nitrogen (BUN), and cardiac cachexia presents in advanced-stage HF.

ADIME 8.2 ADIME At-A-Glance: Acute Myocardial Infarction[a]

Assessment

- Oral intake
- Changes in digestion, absorption, or elimination (check for constipation, flatulence, abdominal distention)
- Impaired or difficult movement of food and liquid from oral cavity to stomach (check for swallowing ability, vomiting, choking/gagging, aspiration)

Nutrition Diagnosis

- Altered GI function RT medications AEB abdominal distention and constipation
- Swallowing difficulty RT impaired movement of food/liquid AEB vomiting and aspiration

Intervention

- Liquid diet, progress as tolerated from clear to full liquid to soft food
- Small volume, frequent feedings
- Exclude gas-forming foods
- Referral to outpatient nutrition counseling

Monitoring and Evaluation

- I&Os
- PO intake (food, fluid)
- Swallowing function, abdominal distention

[a]See CHD and HTN for post-MI/discharge ADIME.

Based on NHANES 2017–2020 data, an estimated ~6.7 million Americans ≥20 years of age had HF, and prevalence is projected to increase by 46% by 2030, affecting more than 8 million people ≥18 years of age with the disease.[1] The lifetime risk for a 45-year-old individual in the community to experience HF ranges from 20% to 46%, but the risk increases with higher BP and BMI.[2] Antecedent HTN is present in 75% of patients with chronic HF, but 11.2% within the community without antihypertensive therapy at the start develop HF over 10 year.[18] The recent 2022 AHA/ACC/HFSA *Guideline for the Management of Heart Failure* therefore recommends therapeutic interventions be tailored according to the development and progression of disease.[31] The ACC/AHA stages of HF accordingly aim to modify risk factors (stage A), treat risk and structural heart disease to prevent HF (stage B), and reduce symptoms, morbidity, and mortality (stages C and D).

Treatment for a patient with HF focuses on improving the related signs and symptoms, which increases the life span. Short-term management focuses on reducing edema and preventing cachexia, whereas long-term management emphasizes lifestyle changes, including dietary salt restriction, weight loss if indicated, and physical activity (**Box 8.14**).[31–34]

Box 8.14 MNT Strategies for HF

Treatment Phase	MNT Approach	Assess and Monitor
Restoration of hemodynamic stability	NPO with IV fluids for first 24 hours	• Monitor: I/O charts, electrolytes, BP, BUN, oliguria, Pco_2, Po_2,

Treatment Phase	MNT Approach	Assess and Monitor
		triglycerides, PT, albumin, prealbumin • Avoid overhydration
Elimination or reduction of edema	Sodium restriction—<1,000 mg if severe or 2,000 to 3,000 mg if less severe Fluid restriction—500 to 1,000 cc depending on severity of HF	• Daily I/O charting • Daily weight monitoring • Fluid plan—75% for meals, 25% for medications and in between sipping
Distention and elevation of diaphragm	Small frequent meals. Rest before and after meals. High calorie diet—low-volume supplements needed. Indications for tube feeding—small bowel feeding ideal.	• Poor appetite • Excessive refeeding • Aspiration • Gastric ileus
Prevent cardiac cachexia	Small frequent meals. Rest before and after meals. High-calorie protein—low-volume supplements needed. Indications for tube feeding (small bowel feeding) or even parenteral nutrition.	• Poor appetite • Fatigue • Muscle depletion • Aspiration
Prevent pressure ulcers	Calorie and protein optimization to promote wound healing	• Improving mobility in the bedbound patient • Improving circulation • Weight optimization
Long-term goals	Assess functional status.	• Calorie and protein optimization essential

Treatment Phase	MNT Approach	Assess and Monitor
	Attain ideal body weight to decrease O_2 needs. Replete lean body mass. Limit cardiac stimulants. Fluid and sodium goals. Moderate aerobic activity.	• Weight gain or loss should be monitored • Adequate fiber from food or supplementation is necessary to prevent constipation

Cerebrovascular Accident (Stroke)

Disease Process and Nutrition Intervention

Stroke is the fifth leading cause of death in the United States. Annually, an estimated 795,000 Americans experience a new or recurrent stroke, with about 610,000 first attacks and 185,000 recurrent attacks.[1] According to the NHANES 2017–2020 data, 9.4 million Americans >20 years of age had self-reported experiencing a stroke, while an overall prevalence of 3.3% was reported for this period. It is projected that by 2030, an additional 3.4 million US adults ≥18 years of age will have had a stroke. High-risk segments of the population for stroke include the older age groups, African Americans, individuals of lower socioeconomic status, and people living in the Southeastern United States.

ADIME 8.3 ADIME At-A-Glance: Heart Failure

Assessment

- BMI, UWL, decrease in appetite and food intake
- Muscle wasting, loss of adipose tissue
- BP, SOB, pitting edema, oliguria
- Excessive dietary sodium intake

Nutrition Diagnosis

- Inadequate oral intake RT decreased appetite, AEB recent UWL of 5% × 3 weeks, poor PO intake of 40%
- Unintended weight loss RT inadequate oral intake AEB UWL of 5% × 3 weeks, poor PO intake of 40%
- Chronic disease-related malnutrition RT HF AEB UWL of 5% × 3 weeks, poor PO intake of 40%, muscle wasting
- Excessive sodium intake RT knowledge deficit AEB exacerbation of edema, diet hx

Intervention

- Small, frequent feedings
- Provision of commercial beverage product
- Insert enteral feeding tube
- Feeding assistance
- Referral to Home Delivered Meals Program

Monitoring and Evaluation

- Body weight
- PO intake (food, fluid)
- Hydration status, BUN, edema

CVA types include ischemic stroke (IS), which accounts for 87%; intracranial hemorrhage (ICH) at 10%; and subarachnoid hemorrhage (SAH) contributing 3%. BP is a

powerful risk determinant for both IS and ICH. Transient ischemic attacks (TIAs), which block blood flow to the brain for no more than 5 minutes, may serve as an early warning for severe CVA.

Depending on the site of the CVA and the extent of brain damage, unconsciousness or paralysis may occur. Left CVA affects sight and hearing; whereas right CVA, bilateral or brain stem CVA, causes significant feeding and swallowing food problems with associated speech problems.[35] Neurogenic deficits are motor-related, causing muscle weakness of the tongue and lips, and sensory and cognitive deficits. Patients with the right CVA are typically unable to coordinate food from plate to mouth, taste, chew, or swallow food, causing dysphagia.

If dysphagia is present, which may be temporary or permanent, the food matrix or texture becomes critical.[35,36] Liquids may be aspirated or may cause drooling, and chewing difficulties with normal-texture foods may lead to choking. Patients may require enteral nutrition consisting of nasogastric feeding for the short term and percutaneous endoscopic gastrostomy feeding in the long term if the speech and swallowing reflexes are not rehabilitated. MNT focuses on the specific stage of CVA and feeding problems related to the type of stroke and area of the brain affected in order to prevent dehydration and malnutrition (**Box 8.15**).[37–40]

Box 8.15 MNT Strategies for CVA

Treatment Phase	MNT Approach	Assess and Monitor
Initial	NPO with IV fluids for first 24 hours	• Monitor: I/O charts, electrolytes, BP, BUN, triglycerides, PT • Avoid overhydration

Treatment Phase	MNT Approach	Assess and Monitor
If patient is comatose	Transit from NPO to liquids. Tube feeding required (nasogastric, gastrostomy, or jejunostomy).	• Elevation of bed-head to prevent aspiration • Monitoring of gastric residual volumes
If patient is not comatose, dysphagia assessment required	Sip feeding or tube feeding may be required depending on short- or long-term needs.	• Aspiration prevention • Hydration adequacy • Pressure sores increase nutritional requirement
Those with lower category of dysphagia assess saliva secretion	For those not requiring liquids, transition to thick pureed liquids or to mechanical soft diet.	• Eating slowly. • Easy-to-chew foods necessary. • Identify foods that trigger choking (dry) or coughing (tart) or drooling (sweet).
Stable long-term goals • Assess functional status	If dysphagia is permanent, patient may be dependent totally or partially on enteral supplements or modified textured foods: • Use thickeners to make semisolids of liquid foods (soups, beverages, juices, and shakes). • Syringe or training cups useful. For first stroke patients with mild disabilities and return to good quality of life, a preventive approach to	• Hospital readmission for aspiration-related pneumonia. • Six to 8 cups of fluid to prevent dehydration. • Calorie and protein optimization essential. • Adequate fiber from food or supplementation is necessary to prevent constipation. Consider prune juice as stool softener (add to tube feeding). • Weight gain or loss should be monitored.

Treatment Phase	MNT Approach	Assess and Monitor
	the second event is critical: • Lifestyle modification emphasizing adherence to the DASH dietary pattern and adoption of the 1,500 mg sodium limit are important.	

ADIME 8.4 ADIME At-A-Glance: Cerebrovascular Accident

Assessment

- BMI, UWL, decrease in appetite and food intake, dehydration
- Swallowing difficulty
- Self-feeding ability
- Ability to prepare foods/meals

Nutrition Diagnosis

- Swallowing difficulty RT CVA AEB coughing and choking, poor PO intake of 60%
- Inadequate fluid intake RT CVA AEB high BUN, high serum osmolality, high Na, thirst
- Inadequate protein-energy intake RT CVA and decreased appetite AEB UWL of 6% × 3 weeks, poor PO intake of 60%

- Self-feeding difficulty RT CVA AEB dropping of utensils and decreased PO intake of 60%
- Unintended weight loss RT CVA, swallowing difficulty, and poor PO intake of 6% × 3 weeks AEBUWL of 6% × 3 weeks
- Impaired ability to prepare foods/meals RT recent CVA AEB reduced mobility

Intervention

- Provision of commercial beverage product
- Increased fluid diet
- Dysphagia diet/liquid consistency—nectar thick liquids
- Feeding assistance
- Referral to Home Delivered Meals Program

Monitoring and Evaluation

- Body weight, BUN, serum osmolality
- PO intake (food, fluid)
- Swallowing function, self-feeding ability

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CHAPTER 9

Diabetes

Diabetes mellitus is a disease known since ancient times, cited by both the Greeks and Egyptians as early as 1500 BC.[1] In its various forms, it affects over 37 million Americans every year, and another 96 million people have prediabetes.[2] It is the seventh leading cause of death and a major cause of complications, such as heart disease, kidney disease, blindness, and amputations.[3]

Diabetes: Classification, Screening, and Diagnosis

Classification of Diabetes

To provide comprehensive and effective medical and nutritional care for patients with diabetes, we must first understand the definitions and classifications of diabetes, which are issued by the American Diabetes Association (ADA). **Table 9.1** includes the four clinical classes of diabetes. It should be noted that the traditional standards of type 1 diabetes occurring only in children and type 2 diabetes only in adults are no longer accurate, as both diseases occur in both cohorts.[4] Assigning a type of diabetes to an individual often depends on the circumstances present at the time of diagnosis, with individuals not always fitting clearly into a single category.

TABLE 9.1 Classification of Diabetes Mellitus

Category	Characteristics
Type 1 diabetes	Due to autoimmune destruction of pancreatic β-cells, usually leading to absolute insulin deficiency, including latent autoimmune diabetes of adulthood. Accounts for 5–10% of diagnosed diabetes in the United States.
Type 2 diabetes	Due to a non-autoimmune progressive loss of adequate β-cell insulin secretion frequently on the background of insulin resistance and metabolic syndrome. Accounts for 90–95% of diagnosed diabetes in the United States.
Other specific types of diabetes	Specific types of diabetes as a result of other causes: monogenic diabetes syndromes (such as neonatal diabetes and maturity-onset diabetes of the young [MODY]); diseases of the exocrine pancreas (such as cystic fibrosis

Category	Characteristics
	and pancreatitis); genetic defects of the β-cell; genetic defects in insulin action; endocrinopathies; drug- or chemical-induced diabetes (such as with glucocorticoid use, in the treatment of HIV/SIDS, or after organ transplantation).
Gestational diabetes mellitus (GDM)	Diabetes diagnosed in the second or third trimester of pregnancy that was not clearly overt diabetes prior to gestation.

Data from American Diabetes Association. 2. Classification and diagnosis of diabetes: standards of care in diabetes–2023. Diabetes Care. 2023;46(Supp 1):S19-S40. Accessed March 9, 2023. https://doi.org/10.2337/dc23-S002.

Screening for Diabetes

Adults

Dietitians and other healthcare providers should encourage regular screening and testing for both prediabetes and type 2 diabetes, especially for individuals in high-risk groups. There is currently a lack of clinically validated screening programs for type 1 diabetes in a clinical setting.[4] Screening for prediabetes and type 2 diabetes with an informal assessment of risk factors or validated tools (such as the ADA Diabetes Risk Test) is recommended for all asymptomatic adults, regardless of age, to guide providers on whether performing diagnostic tests is appropriate. Testing criteria for prediabetes and type 2 diabetes are listed in **Box 9.1**.

Box 9.1 Criteria for Screening for Diabetes or Prediabetes in Asymptomatic Adults

1. Testing should be considered in adults with overweight or obesity (BMI ≥25 kg/m^2 or ≥23 kg/m^2 in Asian American individuals) who have one or more of the following risk factors:

- First-degree relative with diabetes
- High-risk race/ethnicity (African American, Asian American, Latino, Native American, Pacific Islander)
- History of cardiovascular disease
- Hypertension (≥130/80 mm Hg or on therapy for hypertension)
- HDL cholesterol level <35 mg/dL and/or triglyceride level >250 mg/dL
- Individuals with polycystic ovary syndrome
- Physical inactivity
- Other clinical conditions associated with insulin resistance (severe obesity, acanthosis nigricans)

2. People with prediabetes (A1C ≥ 5.7%, IGT, IFG) should be tested yearly.
3. People who were diagnosed with GDM should have lifelong testing at least every 3 years.
4. For all other people, testing should begin at age 35 years.
5. If results are normal, testing should be repeated at a minimum of 3-year intervals, with consideration of more frequent testing depending on initial results and risk status.
6. People with human immunodeficiency virus (HIV)

IFG, impaired fasting glucose; IGT, impaired glucose tolerance.
Data from American Diabetes Association. 2. Classification and diagnosis of diabetes: standards of care in diabetes–2023. Diabetes Care. 2023;46(Supp 1):S19-S40. Accessed March 9, 2023. https://doi.org/10.2337/dc23-S002.

Children

Because of the dramatic increase of type 2 diabetes in children and adolescents over the last decade, the ADA recommends screening children at increased risk for type 2 diabetes and prediabetes in this group.[4] **Box 9.2** shows the screening criteria for type 2 diabetes and prediabetes in asymptomatic children and adolescents.

Box 9.2 Risk-Based Screening for Type 2 Diabetes or Prediabetes in Asymptomatic Children and Adolescents (persons aged <18 Years)

Screening should be considered in youth (after 10 years of age or after onset of puberty, whichever is earlier) who have overweight (>85th percentile) or

obesity (>95th percentile) and who have *one or more additional risk factors based on the strength of their association with diabetes as indicated by evidence grades (**A** to **D**, with **A** the strongest)*:

- Maternal history of diabetes or GDM during the child's gestation: **A**
- Family history of type 2 diabetes in first- or second-degree relative: **A**
- Race/ethnicity (African American, Asian American, Latino, Native American, Pacific Islander): **A**
- Signs of insulin resistance or conditions associated with insulin resistance (acanthosis nigricans, dyslipidemia, hypertension, polycystic ovary syndrome, or small-for-gestational-age birth weight): **B**

GDM, gestational diabetes mellitus.
Data from American Diabetes Association. 2. Classification and diagnosis of diabetes: standards of care in diabetes–2023. Diabetes Care. 2023;46(Supp 1):S19-S40. Accessed March 9, 2023. https://doi.org/10.2337/dc23-S002.

Pregnancy

The best available evidence reveals that many cases of GDM represent preexisting hyperglycemia that is detected by routine screening in pregnancy since routine screening is not widely performed in nonpregnant individuals of reproductive age.[4] Ideally, screening should take place for individuals who are at risk for diabetes and are planning pregnancy. If individuals are not screened before pregnancy, the ADA recommends testing for undiagnosed prediabetes and diabetes before 15 weeks of gestation at the first prenatal visit in individuals with risk factors using standardized diagnostic criteria. Those found to have diabetes by the standard diagnostic criteria used outside of pregnancy should be classified as having diabetes complicating pregnancy (most often type 2 diabetes, rarely type 1 or monogenic diabetes) and should be managed accordingly.[4] Those who meet the lower glycemic criteria for GDM should be diagnosed with that condition and managed accordingly. Other pregnant individuals without risk factors should be screened for GDM between 24 and 28 weeks of gestation. The current recommendations for screening for GDM in pregnancy come from the International Association of Diabetes and Pregnancy Study Groups (IADPSG). **Box 9.3**

lists the risk factors that increase an individual's risk of developing GDM. Further information on diabetes in pregnancy, including the detection and diagnosis of GDM, can be found in the Pregnancy chapter of this publication.

Box 9.3 Screening for Gestational Diabetes Mellitus

Recommendation: Screen for GDM in all asymptomatic pregnant individuals after 24 weeks of gestation

Factors that increase the risk of developing GDM:

- Overweight or obesity
- Increased maternal age (>25 years)
- History of GDM
- Given birth to a baby weighing over 9 pounds
- Family history of type 2 diabetes
- Polycystic ovary syndrome (PCOS)
- Race/ethnicity (African American, Alaska Native, Hispanic or Latino, Native American, Native Hawaiian, Pacific Islander)

Data from Centers for Disease Control and Prevention. Diabetes Risk Factors: Gestational Diabetes. Updated April 5, 2022. Accessed March 9, 2023.

Diagnosis of Diabetes

Children and Nonpregnant Adults

Diabetes may be diagnosed using fasting plasma glucose (FPG), a 2-hour plasma glucose during an oral glucose tolerance test (OGTT), or hemoglobin A1C (A1C). A1C was not previously recommended for the diagnosis of diabetes in part because of a lack of standardized assays. However, assays are now highly standardized, and the ADA supports the use of A1C as a diagnostic test for diabetes using a method that is certified by the National Glycohemoglobin Standardization Program (NGSP) and traceable to the Diabetes Control and Complications Trial (DCCT) reference assay.[4] A1C has several advantages to the FPG and OGTT,

including greater convenience (no fasting required), greater preanalytical stability, and fewer day-to-day perturbations during stress.[4] These advantages must be balanced by greater cost, limited availability of A1C testing in certain regions of the developing world, the lower sensitivity of A1C at the designated cut point, and the incomplete correlation between A1C and average glucose in certain individuals.[4] When using A1C to diagnose diabetes, clinicians should consider that this test is an indirect measure of average blood glucose levels and that other factors may impact hemoglobin glycation independently of glycemia, including hemodialysis, pregnancy, HIV treatment, age, race/ethnicity, genetic background, and anemia/hemoglobinopathies.[4] **Box 9.4** lists the laboratory testing methods and criteria currently recommended for the diagnosis of diabetes.

Box 9.4 Criteria for the Diagnosis of Diabetes[a]

1. **FPG ≥126 mg/dL**
   - Fasting is defined as no caloric intake for at least 8 hours.

Or

2. **2-hour plasma glucose ≥200 mg/dL during an OGTT**
   - The test should be performed as described by the World Health Organization (WHO), using a glucose load containing the equivalent of 75-g anhydrous glucose dissolved in water.

Or

3. **A1C ≥6.5%**
   - The test should be performed in a laboratory using a method that is NGSP-certified and standardized to the DCCT assay.

Or

4. **In a patient with classic symptoms of hyperglycemia or hyperglycemic crisis, a random plasma glucose ≥200 mg/dL**
   - Random is defined as any time of day without regard to time since last meal.

[a]In the absence of unequivocal hyperglycemia, diagnosis requires two abnormal test results from the same sample or in two separate test samples. A1C, hemoglobin A1C; DCCT, diabetes control and complications trial; FPG, fasting plasma glucose; NGSP, National Glycohemoglobin Standardization Program; OGTT, oral glucose tolerance test.
Data from American Diabetes Association. 2. Classification and diagnosis of diabetes: standards of care in diabetes–2023. Diabetes Care. 2023;46(Supp 1):S19-S40. Accessed March 10, 2023. https://doi.org/10.2337/dc23-S002.

Prediabetes

An intermediate group of subjects has been recognized whose glucose levels do not meet the criteria for diabetes but are too high to be considered normal.[4] Individuals who fall into this category have impaired fasting glucose (IFG) and/or impaired glucose tolerance (IGT) and are referred to as having "prediabetes." These individuals are considered at risk for the future development of diabetes and cardiovascular disease. Prediabetes is associated with obesity (especially abdominal or visceral obesity), dyslipidemia with high triglycerides and/or low high-density lipoprotein (HDL) cholesterol, and hypertension. The same tests used to screen and diagnose diabetes may be used to detect individuals with prediabetes (see **Table 9.2**).

TABLE 9.2 Diagnostic Criteria for Diabetes and Prediabetes[a]

	Fasting Plasma Glucose (mg/dL)	A1C (%)	2-Hour Plasma Glucose in the OGTT (mg/dL)
Normal	<100	≤5.6	≤139
Prediabetes	100–125	5.7–6.4	140–199
Diabetes	≥126	≥6.5	≥200

[a]Each criterion must be confirmed by repeat testing unless unequivocal symptoms of hyperglycemia are present.

Data from American Diabetes Association. 2. Classification and diagnosis of diabetes: standards of care in diabetes–2023. Diabetes Care. 2023;46(Supp 1):S19-S40. Accessed March 10, 2023. https://doi.org/10.2337/dc23-S002.

Description

Gestational Diabetes

Screening for GDM can be accomplished with either of two strategies: the “one-step” strategy is supported by the IADPSG, an international consensus group with representatives from multiple obstetrical and diabetes organizations, including the ADA; and the “two-step” strategy is a recommendation from the National Institutes of Health (NIH) consensus panel of experts in obstetrics/gynecology, maternal–fetal medicine, pediatrics, diabetes research, and biostatistics. The strategies are summarized in the Pregnancy chapter of this book.

Medical Nutrition Therapy

Medical nutrition therapy (MNT) is an integral component of diabetes prevention, management, and self-management. The following recommendations by the ADA for MNT in diabetes are based in part on the Dietary Guidelines and the Recommended Dietary Allowances (RDAs) from the Institute of Medicine of the National Academy of Sciences.[5]

- Provide an individualized MNT program as needed to achieve treatment goals, provided by a registered dietitian, preferably one who has comprehensive knowledge and experience in diabetes care.
- For individuals with overweight or obesity, behavioral modification to achieve and maintain a minimum weight loss of 5% is recommended.
- There is no ideal macronutrient pattern for people with diabetes. Meal plans should be individualized while keeping nutrient quality, total calorie, and metabolic goals in mind.
- A variety of eating patterns can be considered for the management of type 2 diabetes and to prevent diabetes in individuals with prediabetes.
- Reducing overall carbohydrate intake has demonstrated the most evidence for improving glycemia and may be applied to a variety of eating patterns that meet individual needs and preferences.
- Carbohydrate intake should emphasize nutrient-dense carbohydrate sources that are high in fiber (at least 14 g fiber per 1,000 kcal) and minimally processed. Eating plans should emphasize non starchy vegetables, fruits, legumes, and whole grains, as well as dairy products, with minimal added sugars.

- In individuals with type 2 diabetes, ingested protein appears to increase insulin response without increasing plasma glucose concentrations. Therefore, carbohydrate sources high in protein should be avoided when trying to treat or prevent hypoglycemia.
- An eating plan emphasizing elements of a Mediterranean eating pattern rich in monounsaturated and polyunsaturated fats may be considered to improve glucose metabolism and lower cardiovascular disease risk.
- Adults with diabetes who drink alcohol should do so in moderation (no more than one drink per day for adult women and no more than two drinks per day for adult men).
- Sodium consumption should be limited to <2,300 mg/day.

ADIME 9.1 ADIME At-A-Glance: Type 1 Diabetes

Assessment

- Ht, wt, BMI
- Labs: blood pressure, fasting plasma glucose, A1C (quarterly), lipid profile, serum creatinine, BUN, GFT, microalbuminuria (annually)
- Blood glucose self-monitoring log, food history/logs, vitamin/mineral/supplement use

Nutrition Diagnosis

- Altered nutrition-related laboratory values for blood glucose RT insufficient or inappropriate insulin administration AEB A1C of 7.5% and consistent

preprandial hyperglycemia per blood glucose logs despite good eating habits.

Intervention

- Review insulin regimen and timing of food intake with patient.
- Recalculate insulin-to-carbohydrate ratio and sensitivity factor.
- Consult with physician regarding adequacy of current insulin prescription.

Monitoring and Evaluation

- Improvements in blood glucose levels; have patient submit blood glucose log results for 1 week.
- Recheck A1C in 3 months; goal level <7%.

Carbohydrate Counting

Carbohydrate counting is a meal planning approach for people with diabetes that focuses on balancing carbohydrate food choices throughout the day. It places emphasis on the total amount of carbohydrate consumed rather than on the source or type of carbohydrate consumed and is based on the fact that carbohydrate, whether from sugars or starches, has the greatest impact on postprandial blood glucose levels compared with protein and fat. Two levels of carbohydrate counting have been defined: basic and advanced.

Basic Carbohydrate Counting

The basic approach aims to increase carbohydrate awareness by promoting consistency in the timing and amount of carbohydrate intake. People with diabetes should

learn about the relationship among food, physical activity, diabetes medications, and blood glucose levels. In addition, education on how to identify carbohydrate foods, appropriate portion or serving sizes, weighing and measuring foods, and how to read food labels should be included.[6]

Carbohydrate counting is based on the principle that one carbohydrate serving (or carb choice) is equal to 15 g of carbohydrate and can be a starch, fruit, milk, or sweet/dessert. Patients can use basic carbohydrate counting with type 1 or type 2 diabetes although it is more likely to be used by people with type 2 diabetes. Although the type of carbohydrate consumed is not as important as the amount and timing in this method, carbohydrate intake from vegetables, fruits, whole grains, legumes, and dairy products should be emphasized over intake from other carbohydrate sources, especially those that contain added fats, sugars, or sodium.

Advanced Carbohydrate Counting

This approach is for those patients who have mastered basic carb counting and wish to have more freedom in their carbohydrate choices and/or tighter blood glucose control, such as those with type 1 diabetes who use a basal-bolus insulin regimen or an insulin pump. For patients using this method, the following skills are recommended:[7-9]

- Understanding target blood glucose levels
- Ability to quantify food intake and use basic math skills
- Understanding the action of insulin and the basal-bolus insulin concept
- Ability to carry out pattern management and keep adequate records
- Ability to calculate a bolus insulin dose using the insulin-to-carbohydrate ratio (ICR)

- Willingness to check blood glucose before and after meals
- Ability to correct blood glucose using insulin sensitivity factor (SF), which can also be referred to as insulin correction factor.

Advanced carbohydrate counting involves calculating an individual's ICR and insulin SF. ICR is the number of grams of carbohydrate counteracted by 1 unit (U) of rapid-acting or short-acting (bolus) insulin. The method used to calculate the ICR is the 450/500 Rule (**Box 9.5**). The ICR allows flexibility in food choices because any number of carbohydrates can be covered with a matching dose of insulin.

Box 9.5 The 450/500 Rule for Calculating the ICR

- If using rapid-acting insulin (i.e., Humalog or Novolog), use the 500 Rule. If short-acting (regular) insulin is used (i.e., Humulin R or Novolin R), use 450.
- Divide 450 or 500 by the total daily dose (TDD) of insulin.
  Example: A person uses Humalog insulin at 50 U per day. Their ICR would be 1:10 based on the equation:

  500/50 U (TDD) of insulin = 10 g of carbohydrate covered by 1 U of insulin, or a **1:10 ICR**
- The TDD includes all basal plus bolus insulin.
- It should be noted that the 450/500 Rule works best for patients with type 1 diabetes with *no* insulin production, using the basal/bolus approach to insulin therapy. People with type 2 diabetes who require insulin injections often produce some insulin naturally, so TDD cannot be calculated reliably.
- ICR can vary from person to person, and for the same person during different times of the day, based on their physical activity, insulin needs, body weight, hormonal changes, and other variables. As always, self-monitoring of blood glucose is imperative.

Data from (i) American Diabetes Association. Insulin. Accessed July 5, 2023. https://diabetes.org/healthy-living/medication-treatments/insulin-other-injectables, (ii) Weinstock RS. Management of blood glucose in adults with type

1 diabetes. UpToDate.com. Updated January 10, 2023. Accessed July 18, 2023. https://www.uptodate.com/contents/management-of-blood-glucose-in-adults-with-type-1-diabetes-mellitus, and (iii) Cornelius BW. Patients with type 2 diabetes: Anesthetic management in the ambulatory setting, Part 2, pharmacology and guidelines for perioperative management. *Anesth Prog*. 2017;64(1):39-44. Accessed July 18, 2023. https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5278535/.

When a patient's blood glucose level goes unexpectedly high, a correction bolus of insulin can be given to lower the level back to baseline. This correction value is referred to as the insulin sensitivity factor (can also be referred to as correction factor). The 1,500/1,800 Rule is a commonly accepted formula for estimating the drop in a person's blood glucose per unit of short-acting or rapid-acting insulin (**Box 9.6**).[7-9] Knowing their SF can help a person with type 1 diabetes determine the correct dose of bolus insulin to correct an elevated blood glucose level.

Box 9.6 Calculating Insulin Sensitivity Using the 1,500/1,800 Rule for Blood Glucose Correction

- Use the 1,500 Rule for patients using short-acting (regular) insulin (i.e., Humulin R or Novolin R). Use the 1,800 Rule for those using rapid-acting insulin (i.e., Humalog or NovoLog).
- Determine the total daily dose (TDD) of all basal and bolus insulin.
- Divide 1,500 or 1,800 by the TDD to find the SF.
- *Example:* 1,800 divided by 50 U (TDD) of insulin = 36. So, it would be estimated that 1 U of rapid-acting insulin would lower the blood glucose by 36 mg/dL.
- This rule works best when basal insulin makes up about 50% of TDD.
- SF can vary from person to person, and for the same person during different times of the day, based on their physical activity, insulin needs, body weight, hormonal changes, and other variables. As always, self-monitoring of blood glucose is imperative.

Data from (i) American Diabetes Association. Insulin. Accessed July 8, 2023. https://diabetes.org/healthy-living/medication-treatments/insulin-other-injectables, (ii) Weinstock RS. Management of blood glucose in adults with type

1 diabetes. UpToDate.com. Updated January 10, 2023. Accessed July 18, 2023. https://www.uptodate.com/contents/management-of-blood-glucose-in-adults-with-type-1-diabetes-mellitus, (iii) Cornelius BW. Patients with type 2 diabetes: Anesthetic management in the ambulatory setting, Part 2, pharmacology and guidelines for perioperative management. *Anesth Prog*. 2017;64(1):39-44. Accessed July 18, 2023.

ADIME 9.2 ADIME At-A-Glance: Type 2 Diabetes

Assessment

- Ht, wt, BMI, waist circumference
- Labs: blood pressure, fasting plasma glucose, A1C (quarterly), lipid profile, serum creatinine, BUN, GFR, microalbuminuria (annually)
- Blood glucose self-monitoring log, food history/logs, vitamin/mineral/supplement use

Nutrition Diagnosis

- Inconsistent carbohydrate intake RT lack of knowledge and confusion regarding carb counting and portion sizes AEB food records with excessive carb choices at meals/snacks and postprandial glucose levels above desired targets

Intervention

- Nutrition prescription: 18 carb choices with 3–4 choices per meal and 2–3 choices per snack.
- Review carb-containing foods and appropriate portion sizes, particularly when dining out.
- Review label reading and meal planning.

Monitoring and Evaluation

- Improvements in blood glucose levels; have patient submit blood glucose log results for 1 week.
- Food intake logs to show more consistent carb intake at meals and snack; have patient submit logs for 1 week.

The calculations of ICR and SF should be monitored and adjusted frequently based on the patient's blood glucose levels, because the values can change based on may factors such as hormonal changes, physical activity, body weight, and others.

Exchange Lists

The Exchange Lists for Meal Planning (**Table 9.3**) were one of the first meal planning systems used to help patients with diabetes control their blood glucose levels. This system separates foods into six categories (or lists) based on their macronutrient content. Depending on the energy level, an exchange pattern consists of a set number of exchanges from each group. Within each list, foods can be exchanged, but the serving size may vary.

TABLE 9.3 Exchange Lists for Meal Planning

Food Groups	Kilocalories	Carbohydrate	Protein	Fat
Carbohydrates				
Starch	80	15	3	0–1
Milk—skim and low-fat				
2%	120	12	8	5
Whole	150	12	8	8
Fruit	60	15	—	—
Vegetables	25	5	2	—
Meat and Meat Substitutes				
Very lean	35	—	7	1
Lean	55	—	7	2–3
Medium-fat	75	—	7	5
High-fat	100	—	7	8
Fat	45	—	—	5

Description

Although most health professionals prefer to use carbohydrate counting as the method of choice for managing blood glucose because of its ease of use, many patients still rely on the Exchange system to help keep their diabetes under control. In addition, because some patients with type 2 diabetes would benefit from even modest weight loss, the Exchange lists can be a useful tool for the registered dietitian in planning a weight-loss regimen. This system can be used to plan a more Mediterranean-style eating plan, which has been shown to not only aid with weight loss but also help patients with diabetes decrease their cardiovascular disease risk by improving their lipid profiles.[10] The Mediterranean-style diet has also been shown to reduce the incidence of type 2 diabetes, which can be helpful in the prediabetes population.

Pharmacologic Management of Diabetes

Oral Hypoglycemic Medications

See **Table 9.4** for a listing of oral medications for diabetes.

TABLE 9.4 Oral Medications for Type 2 Diabetes

Mode of Action	Class Brand Name (Generic Name)	When to Take?
Stimulate insulin secretion from the pancreas	**Second-generation sulfonylureas**	
	Glucotrol (glipizide)	30 min before meal
	Glucotrol XL (glipizide)	Take with first meal of day
	Micronase/DiaBeta (glyburide)	Take with first meal of day
	Glynase (micronized glyburide)	Take with first meal of day
	Amaryl (glimepiride)	Take with first meal of day
	Meglitinide–Benzoic acid derivative	
	Prandin (repaglinide)	within 30 min before meal

Mode of Action	Class Brand Name (Generic Name)	When to Take?
	Meglitinide–d-phenylalanine derivative	
	Starlix (nateglinide)	1–30 min before meals
Improve insulin sensitivity via the liver and peripheral tissue	**Biguanides**	
	Glucophage (metformin HCL)	Take with meals
	Glucophage XR, Fortamet, Glumetza (metformin HCL extended-release)	Once daily with evening meal
	Riomet (metformin HCL liquid)	Take with meals
	Thiazolidinediones	
	Actos (pioglitazone)	Take without regard to meals
Delay glucose absorption in GI	**α-Glucosidase inhibitors**	
	Precose (acarbose)	Take with first bite of main meals
	Glyset (miglitol)	Take with first bite of main meals
Increase insulin secretion and decrease glucagon secretion	**DPP-4 inhibitors**	Once daily without regard to meals
	Januvia (sitagliptin)	

Mode of Action	Class Brand Name (Generic Name)	When to Take?
	Onglyza (saxagliptin)	
	Tradjenta (linagliptin)	
	Nesina (alogliptin)	
Dopamine-2 agonist	**Dopamine-2 agonists**	
	Cycloset (bromocriptine)	Once daily within 2 hours of waking, with food
Blocks glucose reabsorption in the kidney	**SGLT-2 inhibitors**	
	Invokana (canagliflozin)	Once daily before first meal
	Farxiga (dapagliflozin)	Once daily, in morning, with or without food
	Jardiance (empagliflozin)	Once daily, in morning, with or without food
	Steglatro (ertugliflozin)	Once daily, in the morning
Mechanism of action in diabetes is unknown	**Bile acid sequestrants**	
	Welchol (colesevelam HCL)	Six tablets once daily or three tablets twice daily, with a meal and liquid

Mode of Action	Class Brand Name (Generic Name)	When to Take?

DPP, dipeptidyl peptidase; SGLT, sodium glucose transporter.
Data from (i) American Diabetes Association. 9. Pharmacologic approaches to glycemic treatment: Standards of medical care in diabetes-2023, (ii) RxList.com, accessed July 19, 2023, http://www.rxlist.com.

Insulin

See **Table 9.5** for a listing of insulin and insulin analogs and their action.

TABLE 9.5 Insulin and Insulin Analogs

Type of Insulin: Brand Name (Generic Name)	Onset	Peak	Duration (h)	When to Administer
Rapid-acting Humalog, Admelog, Lyumjev (insulin lispro) NovoLog, Fiasp (insulin aspart) Apidra (insulin glulisine)	5–15 min	30–90 min	3–5	0–15 min before meals
Rapid-acting by inhalation Afrezza (technosphere insulin)	12–15 min	60 min	2–3	
Short-acting Humulin R, Novolin R (Regular human [R])	30–60 min	2–4 h	5–8	30–45 min before meals
Intermediate-acting Humulin N, Novolin N (NPH human [N])	1–3 h	6–10 h	12–16	Before a.m. and p.m. meal or before a.m. meal and at bedtime
Long-acting				
Basaglar, Lantus, Toujeo (insulin glargine)	1 h	No peak	Up to 24	In a.m. *or* at bedtime
Semglee (insulin glargine-yfgn)	1 h	No peak	Up to 24	In a.m. *or* at bedtime
Levemir (insulin detemir)	1 h	No peak	Up to 24	Once or twice daily
Tresiba (insulin degludec)			Up to 42	Once daily
Combinations/Mixtures				
Humulin 70/30, Novolin 7/30 (NPH/Regular)	30–60 min	2–12	14–24	Before breakfast and dinner
Humalog Mix 50/50 (protamine/lispro)	25–30 min	.5–3 h	14–24	
Humalog Mix 75/25 (protamine/lispro)	15–30 min	.5–2.5 h	14–24	Before breakfast and dinner
NovoLog Mix 70/30 (protamine/aspart)	5–15 min	1–4	18–24	Before breakfast and dinner

Data from (i) American Diabetes Association. 9. Pharmacologic approaches to glycemic treatment: Standards of medical care in diabetes-2023, (ii) Donnor T, Sarkar S. Insulin-pharmacology, therapeutic regimens and principles of intensive insulin therapy. *Endotext*. National Library of Medicine. Updated February 15, 2023. Accessed July 18, 2023. https://www.ncbi.nlm.nih.gov/books/NBK278938, and (iii) RxList.com, accessed July 19, 2023, http://www.rxlist.com.

Description

Other Injectable Medications for Diabetes

Glucagon-Like Peptide-1 Agonists

Incretin mimetics are a class of medication for the treatment of type 2 diabetes. In individuals with type 2 diabetes, the naturally occurring incretin hormones (such as glucagon-like

peptide-1 [GLP-1]) are blunted. An incretin mimetic works to mimic the antidiabetic or glucose-lowering actions of incretins by enhancing glucose-dependent insulin secretion and several other glucoregulatory actions. These medications suppress inappropriately elevated glucagon levels, promote satiety, reduce food intake, and slow the rate of gastric emptying.

Amylin Analogs

Amylin analogs can mimic the actions of amylin, a hormone cosecreted with insulin that helps to slow gastric emptying and reduces elevated glucagon levels postprandially. Amylin, like insulin, is absent or deficient in patients with diabetes. When used with insulin, amylin analogs can help patients achieve improved glycemic control. See **Table 9.6** for a listing of noninsulin injectable medications for the control of diabetes.

TABLE 9.6 Injectable (Noninsulin) Antihyperglycemic Medications

Type of Medication Brand Name (Generic Name)	When to Inject	Indications for Use
GLP-1 Agonists:		

Type of Medication Brand Name (Generic Name)	When to Inject	Indications for Use
Adlyxin (lixisenatide)	Once daily, within 1 hour before first meal of the day	• Indicated for use as an adjunct to diet and exercise to improve glycemic control in adults with type 2 diabetes mellitus • Not for use in patients with type 1 diabetes or for the treatment of diabetic ketoacidosis • Not a substitute for insulin, in insulin-requiring individuals • Not recommended as first-line therapy for patients who have inadequate glycemic control on diet and exercise
Byetta (exenatide)	1–6 min *prior* to the main morning and evening meals (at least 6 h apart). Do not take after meals.	
Bydureon (exenatide extended-release)	Once weekly, any time of day without regard to meals	
Ozempic (semaglutide)	Once weekly, without regard to meals	
Trulicity (dulaglutide)	Once weekly, any time of day, without regard to meals	
Victoza (liraglutide)	Once daily, without regard to meals	
GLP-1/GIP Receptor Agonist		
Mounjaro (tirzepatide)	Once weekly, at any time of the day	
Amylin Analogs:		
Symlin (pramlintide) Used only in type 1 or type 2 patients	Immediately prior to each major meal[a]	Indicated for use in type 1 or type 2 diabetes, as an adjunct treatment in patients who use mealtime insulin therapy who

Type of Medication Brand Name (Generic Name)	When to Inject	Indications for Use
treated with insulin		have failed to achieve desired glucose control despite optimal insulin therapy.

[a]Major meal is defined as containing at least 250 kcal or at least 30 g carbohydrate.
Data from (i) American Diabetes Association. 9. Pharmacologic approaches to glycemic treatment: Standards of medical care in diabetes-2023, (ii) RxList.com, accessed July 19, 2023, http://www.rxlist.com, and (iii) Cleveland Clinic. Health Library. Accesses July 18, 2023. https://my.clevelandclinic.org/health.

Glycemic Control, Blood Pressure and Lipid Goals for Adults with Diabetes

Glycemic Control

Glycemic control is assessed by the A1C test, blood glucose monitoring (BGM), and a continuous glucose monitor (CGM) using time in range (TIR) and/or glucose management indicator (GMI). Both BGM and CGM for daily glucose monitoring allow patients to evaluate the individual responses to therapy and assess whether they are meeting their glycemic targets. With new technology advances, CGM has evolved rapidly in both accuracy and affordability, and many patients are using these monitors to enhance their diabetes self-management and improve their glycemic control.[11]

The A1C test measures a patient's average glycemia over the preceding 2 to 3 months and can help health providers determine whether a patient's metabolic control has been reached and maintained within a target range.[11] A1C should be performed routinely in all people with diabetes at initial assessment and as part of continuing care. The ADA recommends the use of the term *estimated average glucose* (eAG) in reference to A1C levels.[12] Healthcare providers can report A1C results to patients using the same units (mg/dL or mml/L) that patients see routinely in blood glucose measurements. **Table 9.7** shows the correlation between A1C and eAG levels. The ADA recommends the following for A1C testing:[11]

TABLE 9.7 Correlation Between A1C Level and eAG

A1C (%)	eAG (mg/dL)
6	126
6.5	140
7	154
7.5	169
8	183
8.5	197
9	212
9.5	226
10	240
11	269
12	298

The relationship between A1C and eAG is described by the formula

$$28.7 \times \text{A1C} - 46.7 = \text{eAG}$$

A calculator for converting A1C results into eAG is available at http://professional.diabetes.org/diapro/glucose_calc.
Data from (i) American Diabetes Association. 6. Glycemic targets: standards of care in diabetes–2023. Diabetes Care. 2023;46(Supp 1):S97–S110. Accessed July 18, 2023. https://doi.org/10.2337/dc23-S006, and (ii) American Diabetes Association. eAG/A1C Conversion Calculator. Accessed July 16, 2023. http://professional.diabetes.org/diapro/glucose_calc.

- Assess glycemic status (perform the A1C test or other glycemic measurement such as TIR or GMI) *at least two times* per year in patients who are meeting treatment goals and who have stable glycemic control.
- Assess glycemic status at least quarterly and as needed in patients whose therapy has recently changed and/or

who are not meeting glycemic goals.

- The A1C goal for *patients in general* is <7%.
- On the basis of health care professional judgement and patient preference, a more stringent A1C goal may be acceptable and even beneficial if this can be achieved without significant hypoglycemia or other adverse effects of treatment.

Pre- and postprandial plasma glucose testing is an important part of diabetes management. **Table 9.8** lists current recommendations for glycemic control.

TABLE 9.8 Recommendations for Glycemic Control for Nonpregnant Adults with Diabetes

Glycemic Control	ADA Goals	AACE[a] Goals
A1C	<7%	≤6.5%[b] 7%–8%[c]
Preprandial capillary plasma glucose	80–130 mg/dL	<110 mg/dL
Peak (2-hr) postprandial capillary plasma glucose	<180 mg/dL	<140 mg/dL

More or less stringent targets may be appropriate for individual patients if achieved without significant hypoglycemia or adverse events. Individualize targets based on:

- Age/life expectancy
- Comorbid conditions
- Diabetes duration
- Hypoglycemia status
- Individual patient considerations
- Known cardiovascular disease/advanced microvascular complications

Glycemic Control	ADA Goals	AACE[a] Goals

[a]American Association of Clinical Endocrinology.
[b]Recommended for most nonpregnant adults without concurrent serious illness and at low hypoglycemic risk, if it can be achieved safely.
[c]Recommended in persons with a history of severe hypoglycemia, hypoglycemia awareness, limited life expectancy, advanced renal disease, extensive comorbid conditions, or long-standing diabetes mellitus in which the A1C goal has been difficult to attain despite intensive efforts.
Data from (i) American Diabetes Association. 6. Glycemic targets: standards of care in diabetes–2023. Diabetes Care. 2023;46(Supp 1):S97-S110. Accessed July 18, 2023. https://doi.org/10.2337/dc23-S006, and (ii) Blonde L, Umpierrez GE, Reddy SS, et al. American Association of Clinical Endocrinology clinical practice guideline: Developing a diabetes mellitus comprehensive care plan-2022 update. Endocr Pract. 2022;28:923-1049. https://doi.org/10.1016/j.eprac.2022.08.002.

Blood Pressure and Lipids

Hypertension and dyslipidemia are common comorbid conditions for patients with diabetes, and evidence indicates that the control of these coexisting conditions is essential in the treatment of diabetes. The American Association for Clinical Endocrinology (AACE) recommendations for blood pressure and lipids for patients with type 2 diabetes are included in **Table 9.9**.

TABLE 9.9 AACE Lipid and Blood Pressure Targets for Patients with Type 2 Diabetes

Risk Levels	High DM but no Other Major Risk and ⩽40 YOA	Very High DM + Major ASCVD Risks[a]	Extreme DM + Established Clinical CVD
LDL cholesterol (mg/dL)	<100	<70	<55
Non-HDL cholesterol (mg/dL)	<130	<100	<80
Triglycerides (mg/dL)	<150	<150	<150
Apo B (mg/dL)	<90	<80	<70
Blood Pressure	<130/80 mm Hg	<130/80 mm Hg	<120/70 mm Hg

[a]Hypertension, family history of ASCVD (atherosclerotic cardiovascular disease), low HDL-C, smoking, CKD3,4.

YOA, years of age; CVD, cardiovascular disease; LDL, low-density lipoprotein; HDL, high-density lipoprotein; Apo B, apolipoprotein B.

Data from Blonde L, Umpierrez GE, Reddy SS, et al. American Association of Clinical Endocrinology clinical practice guideline: Developing a diabetes mellitus comprehensive care plan-2022 update. Endocr Pract. 2022;28:923-1049. https://doi.org/10.1016/j.eprac.2022.08.002.

Description

Sick Day Management

Educating patients on sick day management is important. Illnesses such as viral colds or flu, infections, injuries, fever, vomiting, and diarrhea all increase the need for insulin. Emotional stress or crises, physical injury, and surgery can also affect blood glucose levels.

General Guidelines for Sick Days

- Test blood glucose every 4 hours and record in logbook.
- Test for urine ketones every 4 hours and record in logbook (type 1 diabetes).
- Continue taking insulin and diabetes medications.
- Supplemental doses of rapid- or short-acting insulin may be needed because of elevated blood glucose levels or the presence of large or persistent ketones.
- Adjustments need to be individualized and based on tested blood glucose levels.
- Drink 8 oz of fluid every hour. If blood glucose is high, drink water or other calorie-, caffeine-free fluids. If blood glucose is low, drink sugar-based liquids.
- Eat at least 15 g of carbohydrate every hour or 45 to 50 g every 3 to 4 hours (**Box 9.7**).
- Soft foods or liquids may be easier to consume.

Box 9.7 Foods for Sick Day Management (~15 g Carbohydrate Each)

½ cup (4 oz) fruit juice	1 cup (8 oz) milk (nonfat)[a]

1 cup (8 oz) Gatorade or sports drink	½ cup (4 oz) regular, nondiet soft drink
½ cup unsweetened applesauce	3 squares graham cracker
1 slice toast	1 cup soup
½ cup regular gelatin	6 saltine crackers
½ cup oatmeal	1 popsicle (single)
½ cup rice or pasta	½ cup ice cream
¼ cup sherbet	½ cup frozen yogurt
3 glucose tablets	8 Lifesavers
2 tablespoons raisins	

[a]Avoid food high in fat content as it may slow gastric emptying and absorption of carbohydrate.

When to Call the Doctor

- Persistent hyperglycemia (blood glucose >240 mg/dL) even after extra insulin
- Persistent vomiting and/or diarrhea (for more than 6 hours)
- Sick or have fever for more than 2 days and are not getting better
- Symptoms that might signal ketoacidosis of dehydration (chest pains, difficulty breathing, breath smells fruity, lips or tongue dry and cracked)
- Moderate to large amounts of ketones in urine

Acute Complications of Diabetes

Hypoglycemia

Hypoglycemia, or low blood glucose, is a major limiting factor in the glycemic management of type 1 and type 2 diabetes.[11] Hypoglycemia can also be fatal, particularly as a complication of type 1 diabetes.[13] **Box 9.8** gives details on the definition, signs & symptoms, causes and treatment of hypoglycemia.

Box 9.8 Hypoglycemia

Levels of Hypoglycemia	Level 1: Plasma glucose <70 mg/dL and ≥54 mg/dL (an alert to take action). Level 2: Plasma glucose <54 md/dL (requires immediate action). Level 3: A severe event characterized by altered mental and/or physical status requiring assistance from another person for recovery.
Signs and Symptoms	Hunger, sweating, shakiness, irritability, dizziness, lightheadedness, difficulty speaking, sleepiness, confusion and disorientation, anxiety, tachycardia, weakness, unconsciousness
Causes	• Excessive insulin or oral medications • Inappropriate timing of insulin in relation to food intake • Inadequate food intake (not eating enough or skipping meals/snacks) • Prolonged duration or increased intensity of exercise • Alcohol intake without food
Treatment	**Level 1 hypoglycemia: The 15/15 rule** 1. Check blood glucose (BG). • If BG is 55–69 mg/dL, take **15 g** carbohydrate.[a]

	2. Wait **15 minutes** and recheck BG. • If BG <70, repeat step 1. • If BG >70, monitor for signs/symptoms of low BG. • Eat the next meal or snack within 1 hour. **Level 2 or 3 hypoglycemia:** 1. Glucagon injection or dry nasal spray is the best way to treat severely low BG. Caregivers, family members, friends, school personnel, and others should be made aware of hypoglycemia signs/symptoms. If available, it should be used whether the person is alert or unconscious. 2. If glucagon is not available and the person is alert and able to swallow without risk of aspiration, offer a fast-acting source of sugar such as fruit juice, regular soft drink, glucose gel, honey, syrup, or jelly. 3. If unconscious and no glucagon is available, call 911.
Special note	People who take acarbose (Precose) or miglitol (Glyset) should know that only pure glucose (dextrose), available in tablet or gel form, will raise the blood glucose level during a hypoglycemic episode. Other quick-fix food and drinks will not raise the level quickly enough because these medications slow the digestion of other forms of carbohydrate.

[a]Pure glucose is the preferred treatment, but any form of carbohydrate that contains glucose will raise blood glucose. Examples of 15 g carbohydrate include 4 oz fruit juice, 2 Tbsp raisins, 3 glucose or 4 dextrose tablets, 5–6 oz regular soda, 7–8 lifesavers, 1 Tbsp sugar, honey, corn syrup, or jelly.

Data from (i) American Diabetes Association. 6. Glycemic targets: standards of care in diabetes–2023. *Diabetes Care*. 2023;46(Supp 1):S97-S110. Accessed July 18, 2023. https://doi.org/10.2337/dc23-S006, (ii) Agiostratidou G, Anhalt H, Ball D, et al. Standardizing clinically meaningful outcome measures beyond HbA1C for type 1 diabetes: A consensus report of the America Association of Clinical Endocrinology, the American Association of Diabetes Educators, the American Diabetes Association, the Endocrine Society, JDRF International, The Leona M. and Harry B. Helmsley Charitable Trust, the Pediatric Endocrine Society, and the T1D Exchange. *Diabetes Care*. 2017;40(12):1622-1630. doi: 10.2337/dc17-1624, and (iii) Hypoglycemia. National Institute of Diabetes and Digestive and Kidney Diseases. Accessed July 19, 2023, https://www.niddk.nih.gov/health-information/diabetes/overview/preventing-problems/low-blood-glucose-hypoglycemia.

Diabetic Ketoacidosis and Hyperosmolar Hyperglycemic State

Hyperglycemia can lead to diabetic ketoacidosis (DKA) or hyperosmolar hyperglycemic state (HHS), both of which are life-threatening conditions. DKA is a state of severe metabolic decompensation manifested by the overproduction of ketone bodies and keto acids, resulting in metabolic acidosis.[14] DKA is characterized by severe disturbances in carbohydrate, protein, and fat metabolism and is most frequently seen in those with type 1 diabetes. HHS is a metabolic derangement most frequently seen in type 2 diabetes and is usually precipitated by illness or infection that leads to profound dehydration due to fluid loss or inadequate oral hydration. HHS is similar to DKA except that insulin deficiency is not as prevalent. **Table 9.10** gives a comparison of DKA and HHS.

TABLE 9.10 Comparison of DKA and HHS

	DKA	HHS
Age	Under 40 years of age	Over 60 years of age
Hallmark features	• More common in patients with type 1 diabetes • Can occur in type 2 diabetes under conditions of extreme stress (infection, trauma, cardiovascular or other emergencies) • Hyperglycemia, ketosis, ketonuria, metabolic acidosis, dehydration	• Most commonly occurs in patients with type 2 diabetes but can occur in type 1 in conjunction with DKA. • Markedly elevated blood glucose, hyperosmolality, profound dehydration, no significant ketosis
Signs and symptoms	Malaise, increase in polyuria, polydipsia, dehydration, fruity	Lethargy, nausea, vomiting, weakness,

	DKA	HHS
	breath odor of ketones, fatigue, blurred vision, weakness, abdominal pain, nausea, vomiting, disorientation, confusion	blurred vision, dry mouth, muscle cramps, mental status changes, coma
Causes	Inadequate insulin therapy (whether omitted or insufficient insulin regimen), illness, infection, and new onset of diabetes	Illness, infection, noncompliance, and undiagnosed diabetes are common precipitating factors
	DKA	**HHS**
Plasma glucose	>250 mg/dL	600 mg/dL or greater
Ketones	Blood: >3.0 mmol/L (<0.6 is normal) Urine: moderate to large amounts	Blood: Normal or mildly elevated Urine: trace amounts
Arterial pH	Low (<7.3)	Normal
Serum osmolality	<320 mOsm/kg	>320 mOsm/kg
Bicarbonate concentration	Low	Normal or mildly reduced
Treatment	Insulin administration, fluid resuscitation, correction of electrolyte imbalance, monitoring for complications of treatment	Fluid and electrolyte replacement and insulin administration

Data from (i) Gosmanov AR, Gosmanova EO, Kitabchi AE. Hyperglycemic crises: Diabetic ketoacidosis and hyperglycemic hyperosmolar state. Endotext. Updated May 9, 2021. Accessed July 19, 2023. https://www.ncbi.nlm.nih.gov/books/NBK279052/, (ii) Avichal D, Blocher NC. Medscape: Hyperosmolar Hyperglycemic State. Updated May 26, 2023. Accessed July 19, 2023, http://emedicine.medscape.com/article/1914705-overview, and (iii) Hamdy O. Medscape: Diabetic Ketoacidosis (DKA). Updated

	DKA	HHS

January 19, 2021. Accessed July 19, 2023. http://emedicine.medscape.com/article/118361-overview.

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CHAPTER 10

Gastrointestinal Disease

Considering the importance of the gastrointestional (GI) tract's key roles in maintaining life and health, it becomes clear how profoundly GI diseases can affect nutritional status, and ultimately overall health. Nutritional problems can arise from both upper gastrointestinal tract (UGI) and lower gastrointestinal tract (LGI) diseases. These problems can be caused by the underlying disease process, disease symptoms, interventions, and/or self-imposed dietary restrictions. For these reasons, a comprehensive nutritional assessment is essential, with particular focus on a detailed diet history, as are highly individualized interventions.

Diseases of the Upper Gastrointestinal Tract

Gastroesophageal Reflux Disease

Disease Process

Gastroesophageal reflux is the process in which acid from the stomach refluxes up through the lower esophageal sphincter (LES) into the esophagus, with inflammation of the sensitive tissue, resulting in esophagitis. If the acid rises to the pharynx or larynx, it is termed laryngopharyngeal reflux, often called silent reflux. Some patients experience both types of reflux, while other patients have only one type. When reflux is chronic into the esophagus or the larynx/pharynx, it is termed *gastroesophageal reflux disease* (GERD) or laryngopharyngeal reflux (LPR), respectively, and increases risk for Barrett esophagus, a precancerous condition, and esophageal adenocarcinoma (EAC).[1] Acute esophagitis can be caused by ingesting a caustic compound, often a medication, such as nonsteroidal anti-inflammatory drugs (NSAIDs), and it can arise from repeated vomiting, especially when self-induced as in anorexia and bulimia. More commonly, the initiating step is inappropriate relaxation of the LES and can be caused by many factors, some of which are related to diet and lifestyle (**Box 10.1**). Medications and substances that slow gastric emptying can also cause or exacerbate reflux (**Box 10.2**).

Box 10.1 Factors Affecting LES Pressure

Reduce Pressure (Open)

Alcoholic beverages
Caffeine

Chocolate
Cigarettes
Dietary fat
Mint oils

High pressure on stomach

- Overeating, drinking

Hormone level

- Progesterone (pregnancy, late phase of menstrual cycle)

Medications

- Anticholinergics: Atropine, Bentyl, Robinul, and Scopolamine
- Bronchodilators: Albuterol (Proventil, Ventolin), metaproterenol (Alupent), montelukast (Singulair), terbutaline (Brethine), theophylline (Aerolatte, Slo-Bid, Slo-Phyllin, Theo-24, Theo-Dur, Theolair, Uniphyl), and zafirlukast (Accolate)

Increase Pressure (Close)

Dietary protein

Medications

- Bethanechol
- Metoclopramide

Box 10.2 Medications and Substances That Slow Gastric Emptying

Medications

Calcium Channel Blockers

Adalat, Calan, Cardene, Cardizem, DynaCirc, Isoptin, Nimotop, Norvasc, Plendil, Procardia, Sular, Vascor, and Verelan

Opiates/Opioids

Alfenta, Buprenex, codeine, Dalgan, Darvon, Demerol, Dilaudid, Dolophine, Levo-dromoran, Nubain, Roxicodone, Sublimaze, Sufenta, Talwin, and Ultiva

Tricyclic Antidepressants

Anafranil, Desipramine/Norpramin, Imipramine/Tofranil, Nortriptyline/Pamelor, and Protriptyline/Vivactil

Other Substances

Alcohol
Marijuana
Tobacco

A common cause of GERD is hiatal hernia, particularly type 1 (sliding), which accounts for 95% of all hiatal hernias, with studies reporting that over half of GERD diagnoses were associated with hiatal hernias.[2] Another potential cause of GERD is eosinophilic esophagitis, esophageal inflammation resulting from allergy, most often food allergy, with up to 20% of patients experiencing GERD.[3] In achalasia, a neurologic disorder of the lower esophagus in which the LES fails to relax, causing dysphagia (see Chapter 4), the disease process itself and treatments can also result in GERD.[4]

Treatment and Nutrition Intervention

Treatment for GERD and LPR includes diet and lifestyle changes, medications, and, failing these, surgery. Medications to reduce gastric acidity include antacids, H_2 receptor antagonists, and proton pump inhibitors (PPIs). Reducing acidity of the gastric contents renders the reflux fluid less caustic. Chronic and long-term use of acid-reducing medications may cause malabsorption or interference with several nutrients, especially PPIs, including folic acid, vitamin B_{12}, iron, calcium, zinc, and magnesium.[5,6] Dietary considerations focus on prevention of reflux and reduction of gastric acidity, which involves manipulating dietary constituents that affect LES pressure, reducing gastric acid secretion, and avoiding tart or acidic foods and

irritating spices in a disease exacerbation, or flare[7] (**Box 10.3**). These changes, in conjunction with a Mediterranean diet supplemented with alkaline water (pH >8.0) resulted in better improvement than a PPI regimen in patients with laryngopharyngeal reflux.[8] Smoking exerts a wide range of GI effects, most of which worsen GI problems, including reduced LES pressure and increased acid secretion.[9]

Box 10.3 Dietary and Other Recommendations for GERD

Diet to Prevent Reflux	Other Treatment Aspects	Diet in Acute Esophagitis
Eat small, frequent meals, avoid large meals.	• Do not lie down after eating (wait 3 h).	• Avoid acidic foods (citrus fruits, tomatoes).
Avoid single high-fat meals.	• Elevate the head of the bed, if needed.	• Avoid spicy foods (red and black peppers).
Eat low-fat, higher protein meals.	• Limit or avoid smoking.	• Follow a bland, soft diet.
Limit alcohol.	• Use antacids to lower gastric acidity. • Lose weight, if indicated. • Wear loose-fitting clothing.	• Eat small, frequent meals.
Avoid foods that lower LES[a] pressure: chocolate, coffee, mints, garlic, onions, and cinnamon.		

Diet to Prevent Reflux	Other Treatment Aspects	Diet in Acute Esophagitis
Avoid drinking liquids with meals; drink between meals.		

[a] Based on individual experience.

Peptic Ulcer Disease

Disease Process

Peptic ulcer disease (PUD) is the collective term for ulcers, eroded areas of tissue in either that stomach or the duodenum. The erosion arises from the stomach's own acid and pepsin, most commonly instigated by *Helicobacter pylori* (*H. pylori*), which secretes proteins and toxins that attract macrophages, resulting in irritation and inflammation in gastric epithelial cells.[10] Another major cause of PUD is the chronic use of medications that irritate the mucosa, most commonly NSAIDs and corticosteroids, which are associated with a relative risk of 4.7 for bleeding and perforation.[11,12] The stress of severe illness or trauma can produce ulcers, termed *stress ulcers*, which is the reason for prophylactic use of acid-reducing drugs in some acute care patients.

Of nutritional concern, PUD often results in self-imposed dietary restriction and concomitant reduction in essential nutrients. In addition, pain and other GI symptoms may affect food intake and compromise nutritional status. PUD can lead to life-threatening complications including perforation, bleeding, and obstruction.

ADIME 10.1 ADIME At-A-Glance: GERD

Assessment

- High body mass index (BMI)/high waist circumference (WC)
- Epigastric pain, erosions/ulceration, UGI bleeding
- Diet Hx; meal pattern, high fat intake, and alcohol intake
- Laboratory: hgb, hct; if long-term use PPIs, B_{12}, folate, Ca, and Mg

Nutrition Diagnosis

- Food–medication interaction of PPI use × 3 years and B_{12} RT GERD Dx, AEB low serum B_{12} level
- Class 1 Obesity RT xs energy intake and sedentary lifestyle, AEB BMI 32, diet Hx

Intervention

- Small, frequent meals; avoid large meals, especially high-fat meal
- Fat 30% or less, higher protein (especially if GI bleed)
- Limit alcohol, smoking, foods that lower LES pressure (caffeine, chocolate, and min oils)
- Modest weight loss, if indicated
- Supplementation with cited nutrients if PPI use

Monitoring and Evaluation

- Body weight, WC
- Laboratory values

- Control of GI symptoms

Treatment and Nutrition Intervention

The history of PUD treatment reflects various regimens that were not evidence based, therefore ineffective, and often nutrient deficient.[13] Treatment for PUD involves determining the cause of the disease and, when it exists, eradicating *H. pylori*. The foods and substances to avoid are those that may irritate the mucosa or increase acid secretion (**Box 10.4**).[7] Avoiding smoking is another important aspect of lifestyle change for treating PUD.[9]

Box 10.4 Goals and Nutrition Intervention for PUD

Goals

1. Optimize nutritional intake to correct any deficiencies (iron, B_{12} most common) and to meet nutritional needs.
2. Adopt dietary and lifestyle factors to minimize symptoms and pain and promote healing.

Nutrition Intervention

1. Avoid foods that increase gastric acid secretion or irritate gastric mucosa: alcohol, pepper, caffeine-containing products (energy drinks, soft drinks), tea and coffee (including decaffeinated), and chocolate.
2. Avoid eating at least 2 h before bedtime.
3. Individualize the diet for any foods not tolerated.
4. Avoid large meals (distend the stomach); small, frequent meals may be helpful.
5. Ensure adequate intake of protein and vitamin C to promote healing.

Other Recommendations

1. Avoid cigarette smoking.
2. Avoid NSAIDs.

Other aspects of treatment include a variety of medications to accomplish the objectives of eradicating *H. pylori* if present, reducing acid secretion and gastric acidity, and promoting ulcer healing (**Box 10.5**). Gastric surgery is sometimes needed because of complications such as perforation, obstruction, or cancer. After surgery, nutritional problems can arise, such as nutrient malabsorption, poor food intake, and weight loss, depending on the type and extent of the surgery (**Box 10.6**). The most common complication of gastric surgery is dumping syndrome, which often leads to many of these nutritional problems.

Box 10.5 Common Medications in Peptic Ulcer Disease

Antacids
Antibiotics (if *H. pylori* presents)
Ulcer drugs

- H_2 antagonists (cimetidine/Tagamet, famotidine/Pepcid, Nizatidine/Axid)
- Omeprazole/Prilosec, lansoprazole/Prevacid
- Sucralfate/Carafate
- Misoprostol/Cytotec

Box 10.6 Surgery for Peptic Ulcer Disease

Truncal Vagotomy and Pyloroplasty

The truncal vagotomy cuts the main trunks of the vagus nerve on each side of the distal esophagus, it eliminates nerve-induced secretion of acid, it also reduces contractions and delays gastric emptying. To offset these effects, the pyloroplasty is also done. In the pyloroplasty, the pylorus is surgically altered so that it can act as a barrier to contents of the stomach as it empties. The result is that liquids empty more quickly, but solids take longer.

Truncal Vagotomy and Antrectomy

This procedure is more aggressive because the antrectomy connects the antrum and pylorus. When the antrum is altered, the portion of the stomach that secretes gastrin (hormone that stimulates all gastric secretions) is

removed. The two surgical procedures for attaching the remaining stomach to the intestine are Billroth I and Billroth II.

Highly Selective Vagotomy (Proximal Gastric Vagotomy)

This procedure reduces gastric acid secretion but does not interfere with motility (i.e., the stomach's movement). This prevents problems related to gastric emptying (i.e., dumping syndrome).

ADIME 10.2 ADIME At-A-Glance: Peptic Ulcer Disease

Assessment

- Low BMI, UWL, anorexia/decrease in appetite and food intake
- Abdominal pain, fatigue, nausea, and vomiting
- Epigastric pain, UGI bleeding
- Laboratory: hgb, hct; if long-term use PPIs, B_{12}, folate, Ca, and Mg

Nutrition Diagnosis

- Inadequate oral intake RT decreased appetite from abdominal pain and nausea with PUD Dx, AEB recent UWL of 6% × 2 months, and PO intake of <50%

Intervention

- Protein 1.0 g to 1.2 g/kg for repletion
- NGS, individualize per tolerance
- Avoid eating 2 hours before bedtime
- Avoid large meals
- Supplementation with cited nutrients if PPI used

Monitoring and Evaluation

- Body weight
- Laboratory values for specific nutrients
- Control of GI symptoms

Dumping Syndrome

Disease Process

The major nutritional complication, dumping syndrome, is a group of symptoms that result from rapid emptying of undigested food into the jejunum from the stomach and proceeds through distinct phases of early, intermediate, and late.[14] Some patients experience only early-phase dumping syndrome, whereas others have both early and late. The rapid movement of the hyperosmolar load into the small intestine pulls fluid from the vascular compartment into the intestine, producing gastrointestinal and vasomotor symptoms of pain, nausea, dizziness, sweating, and tachycardia. These symptoms occur within 10–20 minutes of eating, the undigested food moves to the colon and symptoms of bloating, cramping, gas, and diarrhea ensue, termed *the intermediate phase*.

A patient who experiences the late phase of dumping syndrome will experience symptoms within 1–3 hours of eating. Late phase is caused by reactive hypoglycemia, which arises from an excess release of insulin in response to a high intestinal concentration of carbohydrate and rapid absorption of glucose into the blood. This produces symptoms of sweating, shakiness, weakness, and confusion.

Treatment and Nutrition Intervention

Nutrition intervention is a mainstay of treatment, the postgastrectomy diet or antidumping diet (**Box 10.7**).[7] In addition, functional lactose intolerance tends to be a common problem after surgery. Treatment may also include

drugs to slow gastric emptying; somatostatin and its synthetic analog orectide; and acarbose, an α-glycoside hydrolase inhibitor, which interferes with carbohydrate absorption.

Box 10.7 Postgastrectomy Dietary Recommendations

1. Eat small, frequent meals.
2. Avoid large amounts of liquids with meals; consume fluids 1–2 h before or after meals.
3. Avoid extremes in food temperature.
4. Avoid activity and lie down for 1 h after meals.
5. Eat 50–60% of calories from complex carbohydrates, less than 15% of kilocalories from simple sugars.
6. Eat 20% of calories from protein with a protein-rich source at all meals.
7. Eat about 30% from fat;
8. Consume diet moderate in sodium, 3 g of sodium or less daily.
9. Avoid concentrated sweets.
10. Avoid foods containing lactose.

ADIME 10.3 ADIME At-A-Glance: Dumping Syndrome

Assessment

- Low BMI, UWL, anorexia/decrease in appetite and food intake
- Abdominal pain, nausea and vomiting, steatorrhea
- Laboratory: hgb, hct; B_{12}

Nutrition Diagnosis

- Unintended weight loss RT dumping syndrome post partial gastrectomy and subsequent malabsorption,

AEB recent UWL of 7% × 1 month, steatorrhea

Intervention

- Protein 1.0–1.2 g/kg
- Restrict to <15% of kcals from simple sugars
- 30% fat
- Small, frequent meals
- Avoid lactose
- Supplement of B_{12}, Fe, other nutrients as needed

Monitoring and Evaluation

- Body weight
- Laboratory values for specific nutrients
- Control of GI symptoms

Diseases of the Lower Gastrointestinal Tract

Diverticular Disease: Diverticulosis and Diverticulitis

Disease Process

Diverticular disease includes diverticulosis and diverticulitis, of which the latter is considered a complication of the former. Diverticulosis is the asymptomatic presence of small herniations of the colon, most often the sigmoid colon, which form pouches, and diverticulitis refers to inflammation and/or infection of these pouches. This can occur throughout the intestine but is most common in the sigmoid colon.[15,16] Potentially life-threatening complications can occur in diverticulitis and include intestinal obstruction, bleeding, abscess, fistula, and perforation.

Because of the significant differences in disease prevalence among more economically developed and less developed countries, the Western diet has long been implicated in the etiology, particularly a low-fiber diet leading to constipation and straining during defecation.[17] However, a large population-based study compared patients with irritable bowel syndrome, a disease that significantly increases risk for diverticular disease and presents in several forms, of which the two major forms are diarrhea predominant and constipation predominant.[18] They reported that diarrhea-predominant irritable bowel syndrome was the strongest predictor of diverticular disease. In addition, a meta-analysis showed that lowering dietary fiber intake reduced constipation.[19] A more definitive study using colonoscopy data reported that both a high- fiber diet and

increased bowel movement frequency were associated with a higher prevalence of diverticulosis.[20]

Treatment and Nutrition Intervention

If a person has had diverticulitis in the past, future flares, if not too severe, can often be treated at home by means of rest, fasting at first and then a clear liquid diet, and antibiotics and/or anti-inflammatory medications, if needed. As the symptoms subside, usually within a few days, nutrition therapy consists of a soft, low-fiber diet, and gradual return to a normal level of fiber.[7] Although the evidence does not support a protective effect of a high-fiber diet in the prevention of diverticulosis, limited evidence suggests that it may reduce the risk of diverticulitis and subsequent complications.[16] Although clinicians had prescribed the antibiotic rifaximin and probiotics (20), the American Gastroenterology Association, in the Clinical Practice Update on Medical Management of Colonic Diverticulitis, has stated that there is insufficient evidence for their use.[16]

In hospitalized patients, severe diverticulitis warrants complete bowel rest and only small sips of water or ice chips (**Box 10.8**).[7] When appropriate, the diet is progressed to clear liquids, and oral nutrition supplementation may be indicated depending on the need for extended nothing by mouth (NPO) or clear liquid diet status in conjunction with poor nutritional status upon admission. For most patients, antibiotics and bowel rest alleviate symptoms within 2–5 days. After a clear liquid, patients progress to a bland or GI soft diet, with no nuts, seeds, or fibrous vegetables. The diet is low fiber for up to 1 month, after which the patient should consume a diet with the recommended level of fiber: Dietary Reference Intake (DRI) of 28 g (women) and 35 g (men). An increase beyond the DRI is not warranted[7] (**Table 10.1**).

Box 10.8 Dietary Recommendations for Diverticular Disease

During Acute Diverticulitis Requiring Hospitalization

1. NPO or clear liquid diet, if bowel rest is indicated depending on severity

Diverticulosis (After Diverticulitis Has Subsided)

1. Gradual increase in daily fiber intake to DRI level of 28 g (women) and 35 g (men)
2. Ensure adequate daily intake of fluid, concomitant with increase in dietary fiber

TABLE 10.1 Fiber Content of Foods

Food	Amount	Fiber (g)
Bread: whole wheat	1 slice	2
Cereal, bran	½ cup	10
Cereal, cooked oatmeal	½ cup	2
Fruit: apple, banana, kiwi, and pear	1 med.	2
Legumes: cooked baked beans, kidney beans, and navy beans	½ cup	8
Legumes: garbanzo, lima beans, lentils, and split peas	½ cup	5
Vegetables: cooked broccoli, green beans, corn, winter squash, raw carrots, and peppers	½ cup	3

ADIME 10.4 ADIME At-A-Glance: Diverticular Disease

Assessment

- Decrease in appetite and food intake
- Abdominal pain, nausea and vomiting, and fever
- Diet Hx (fiber intake)
- Laboratory: hgb, hct

Nutrition Diagnosis

- Food and nutrition knowledge deficit RT to new Dx, AEB diet Hx

Intervention

- Advance from NPO to low fiber
- After discharge, gradual fiber increases to DRI
- Adequate fluid intake of fluid

Monitoring and Evaluation

- Fiber intake
- Laboratory values for specific nutrients
- Control of GI symptoms

Irritable Bowel Syndrome

Disease Process

Irritable bowel syndrome (IBS) is one of the most common functional gastrointestinal disorders, affecting from 10 to 20% of the international population.[21] The cause of IBS is

unknown, but theories suggest that people with IBS have an exaggerated response to specific stimuli, which include intolerance or hypersensitivity to specific substances in foods, infection, and gut hormones.[22] Research also suggests potential participation in the pathophysiology of mucosal immune activation and inflammation, and alterations in intestinal permeability, serotonin metabolism, and the intestinal microbiome.[23]

Symptoms vary and can include abdominal cramping and pain, bloating, flatulence, and alternating bouts of both constipation and diarrhea or only one of these, with Rome IV subgroups consisting of IBS with predominant constipation (IBS-C), IBS with predominant diarrhea (IBS-D), IBS with mixed bowel habits (IBS-M), and IBS unclassified (IBS-U).[24] Because the symptoms are somewhat nonspecific and variable, it is important to rule out other LGI diseases. The Rome IV criteria for IBS diagnosis specify that patients must have had recurrent abdominal pain a minimum of 1 day per week for the previous 3 months accompanied by 2 or more of the following:

1. Related to defecation (may be increased or unchanged by defecation)
2. Associated with a change in stool frequency
3. Associated with a change in stool form or appearance

The guidelines also include other symptoms that may facilitate diagnosis: altered stool frequency; altered stool form; altered passage of stool (straining or urgency); mucorrhea; patient’s perception of abdominal bloating or distention.

Treatment and Nutrition Intervention

Treatment emphasizes diet and lifestyle changes and, when needed, medications. Lifestyle changes emphasize establishing regular eating patterns, regular bowel habits,

possible dietary changes, and stress reduction and management (**Box 10.9**).[7] A significant number of IBS patients are lactose intolerant, and other common problematic foods include caffeine, alcohol, gas-forming vegetables, and wheat or yeast.[25] To determine whether specific foods are provoking flares, an elimination and reintroduction approach can be helpful for some patients,[7] as can adherence to a diet in low-fermentable oligo-, di-, and monosaccharides and polyols (FODMAPs), particularly in the presence of small intestinal bacterial overgrowth (SIBO) (**Box 10.10**).[26,27] However, the contribution of SIBO to IBS and the efficacy of the low-FODMAP diet is somewhat controversial. The American College of Gastroenterology (ACG) has made a "conditional recommendation" for a limited trial of the low-FODMAP diet, citing "very low quality of evidence" and patient difficulty in adherence to the diet[28] (**Table 10.2**). However, the American Gastroenterological Association (AGA) referred to the diet as "currently the most evidence-based diet intervention for IBS" in their Clinical Practice Update, which included best practice advice.[29] Another potential treatment is the use of probiotics, with Sabaté reporting significant improvement in patients using species *Bifidobacterium longum* 35624.[30] However, the ACG recommendations "suggest against probiotics," issuing a conditional recommendation citing "very low quality of evidence." The ACG also suggests the use of peppermint, although citing "low quality of evidence."[28]

Box 10.9 Recommendations for Irritable Bowel Syndrome

1. Normalize eating patterns; eat at a relaxed pace at consistent times, with small, frequent meals; and ensure adequacy of all nutrients.
2. Adjust diet for food allergies and intolerances; common problem foods include wheat, yeast, and eggs.
3. Reduced lactose (if lactose intolerant).

4. Low-FODMAP diet (avoid foods high in fructose, sorbitol, xylitol and mannitol, and gas-forming foods).
5. Use of prebiotics and probiotics.
6. Progress slowly to a diet adequate in fiber (25–35 g/d).
7. Ensure adequate fluid intake of 2–3 quarts of water daily.
8. Foods to avoid: alcohol, black pepper, caffeine, chili powder, cocoa/chocolate, coffee, colas, garlic, red pepper, spicy foods, and sugars.
9. Avoid high-fat intake.
10. Supplement with B-complex vitamins, calcium, vitamin D, and riboflavin (if lactose is not tolerated).
11. Supplement of 1 Tbsp daily of a bulking agent, such as Metamucil, may be helpful; avoid bran supplements, as they may be irritating.

Box 10.10 Low FODMAP Diet Recommendations

Foods High in FODMAPs: Limit or Avoid				
Fructans	**Fructose**	**Galactans**	**Lactose**	**Polyols**
Fruits: persimmon and watermelon *Grains:* rye and wheat products in high amounts *Vegetables:* artichokes, asparagus, beets, broccoli, Brussels sprouts, cabbage, eggplant, fennel, garlic, leek, okra, onions, and shallots *Other:* pistachios and products containing chicory, inulin	*Fruits:* apple, mango, pear, and watermelon *Sweeteners:* agave, corn syrup, fructose, high-fructose corn syrup, and honey	*Legumes:* baked beans, chickpeas, kidney beans, lentils, soybeans	*Cheeses:* cottage cheese, cream cheese, ricotta, and other soft, unripened cheeses *Milk:* cow's, goat's, and sheep's milk, and products from milk such as ice cream and yogurt	*Fruits:* apple, apricot, avocado, blackberries, cherries, nectarine, peach, pear, plum, and watermelon *Sweeteners:* isomalt, maltitol, mannitol, sorbitol, xylitol *Vegetables:* cauliflower, corn, green bell pepper, and mushrooms
Foods Low in FODMAPs: Appropriate for Diet				
Fruits	**Dairy**	**Grains**	**Vegetables**	**Miscellaneous**
Banana, blueberries, boysenberries, cantaloupe, cranberries, grapes, grapefruit, honeydew, kiwi, lemon, lime, orange, passion fruit, raspberries, rhubarb, strawberries, and tangerines	*Cheese:* brie, camembert, and hard cheeses *Ice cream:* gelato and sorbet *Milk:* lactose-free milk, oat, rice, and soy milk (and tofu) *Yogurt:* lactose-free products	Arrowroot, corn products (polenta), gluten-free products, millet, oats, psyllium, quinoa, rice, spelt, and tapioca	Alfalfa and bean sprouts, bamboo shoots, bok choy, carrot, celery, endive, ginger, green beans, lettuce, olives, parsnip, potato, pumpkin, red bell pepper, spinach, squash, sweet potato, tomato, turnip, yam, and zucchini	*Sweeteners:* any artificial sweetener not ending in "ol," glucose, sucrose (table sugar)

Data from Alexander C. Ford, et al., "American College of Gastroenterology Monograph on Management of Irritable Bowel Syndrome," *American Journal of Gastroenterology* 113, supp 2 (2018): 1-18.

Description

TABLE 10.2 American College of Gastroenterology Clinical Guideline: Management of Irritable Bowel Syndrome

Diet-Related Recommendations

Intervention	Quality of Evidence	Recommendation
Low FODMAP diet	Very low	Limited trial of a low FODMAP diet in patients with IBS to improve global IBS symptoms. Conditional recommendation.
Fiber	Moderate	Soluble, but not insoluble, fiber be used to treat global IBS symptoms. Strong recommendation.
Probiotics	Very low	Suggest against probiotics for the treatment of global IBS symptoms. Conditional recommendation.
Peppermint oil	Low	Use of peppermint to provide relief of global IBS symptoms. Conditional recommendation.

Data from Lacy BE, Pimentel M, Brenner DM, et al. ACG Clinical Guideline: management of irritable bowel syndrome. *Am J Gastroenterol*. 2021;116(1):17-44. doi:10.14309/ajg.0000000000001036

Patients with IBS exhibit a wide range of symptoms and responses to various dietary treatments, so it may be helpful to the individual IBS patient to follow these recommendations, as well as traditional dietary advice, such as eating small, frequent meals at regular and consistent times, ensuring adequate fluid intake, and avoiding any foods that appear to cause problems.[7] In addition to an elimination diet to find problem foods, a person with IBS can

keep a food diary to record food and beverage intake and see if flares are associated with certain foods.

Medications typically focus on the Rome IV subgroup and specific symptoms, such as antidiarrheal, anticholinergic, antispasmodics (although the ACG recommends the use of antispasmodics to treat global IBS symptoms[28]), bulk-forming laxatives, and prokinetic drugs. In addition, medications may include antidepressants (tricyclic antidepressants or serotonin receptor antagonists), chloride channel activators, and guanylate cyclase C (GC-C) agonists.[31] The antibiotic rifaximin (Xifaxan) is effective in treating SIBO, with the consequent symptom of gas in IBS-D.

ADIME 10.5 ADIME At-A-Glance: Irritable Bowel Syndrome

Assessment

- Abdominal pain, nausea, diarrhea, constipation, abnormal stooling, and xs flatulence
- Diet Hx (food intolerance, sensitivities), chronic laxative use, and avoidance of specific foods
- Laboratory: specific nutrients, if diarrhea predominant IBS and/or avoidance of specific foods

Nutrition Diagnosis

- Altered GI function FT IBS Dx, AEB alternating diarrhea/constipation with subsequent avoidance of specific foods

Intervention

- Small, frequent meals; ensure adequacy of all nutrients

- Adjust diet for food allergies/intolerances; avoid lactose (if lactose intolerant [LI])
- Low-FODMAP diet
- Gradual increase to adequate fiber, with adequate fluid
- Avoid high fat intake
- Supplement with B-complex vitamins, calcium, vitamin D, and riboflavin, if LI
- Possible supplement of 1 Tbsp daily of a bulking agent

Monitoring and Evaluation

- Meal pattern; changes in stools (frequency and consistency)
- Fiber and fluid intake
- Laboratory values for specific nutrients
- Control of GI symptoms

Inflammatory Bowel Disease

Disease Process

Inflammatory bowel disease (IBD) consists of two major types of chronic diseases: Crohn disease (CD) and ulcerative colitis (UC), which both produce inflammation of the intestine. Both diseases tend to cause nutrient malabsorption, leading to malnutrition, and both can cause life-threatening complications[7] (**Table 10.3**). They both increase the risk for chronic diseases including cardiovascular disease, respiratory disease, cancer, arthritis, kidney disease, and liver disease.[32] The diseases appear to represent heterogeneous disorders of potentially multiple etiologies with genetic and environmental interactions, which cause the immunologic response that undergirds the

disease processes.[33] Several aspects of the diseases are similar, but each also has its own pattern of attack and specific nutrient problems (**Box 10.11**). One significant difference between the two diseases is that surgery, which is often a necessity, can cure UC by removing the entire colon. In contrast, some patients with CD may require subsequent surgeries after the initial surgery, and the disease has no cure.[34,35]

TABLE 10.3 Differentiation Between Crohn Disease and Ulcerative Colitis

Parameter	UC	Crohn's
Typical age of onset	15–30	Same
Organ involved	Colon only	GIT anywhere
Tissues involved	Surface only	All layers
Distribution	Continuous	Segments
Cancer risk	Yes (10 years)	Probably
Rectal bleeding	Common	Occasional
Steatorrhea	No	Common
Diarrhea	Frequent	Frequent
Vomiting	Yes	Yes
Smoking	Lowers risk	Increases risk
Oral contraceptives	No effect on risk	Increases risk
Hormone replacement therapy	Increases risk	No effect on risk

Box 10.11 Nutrition Problems in Inflammatory Bowel Disease

Nutrient	UC	CD
Protein	Yes, because of inflammation, diarrhea, intake	Same
Fat	No	Yes, because of malabsorption
B_{12}	No	Yes, because of malabsorption, if ileal involvement
Vitamins A, D, E, and K	Yes, because of medications	Yes, because of malabsorption and medications
Fe	Yes, because of bleeding	Yes, because of bleeding and malabsorption, if duodenal involvement
Cu, Zn, Se	Yes, because of diarrhea	Yes, because of malabsorption and diarrhea

Crohn Disease

CD is also known as regional enteritis, granulomatous ileitis, and ileocolitis. It can strike anywhere in the GI tract; however, it demonstrates a predilection for the terminal ileum. In contrast to UC, in which only the mucosa and submucosa are affected, all intestinal tissue layers may be affected.[36] Complications include obstruction, fissures, and fistulas, which increase the risk for mortality. The nutritional problems in CD are potentially more severe compared with UC, because the small intestine is usually involved (Box 10.11).

Another nutritional concern is the potential for severe diarrhea, often steatorrhea from fat malabsorption, in cases of ileal involvement. In addition to direct nutrient malabsorption, people with CD experience pain and other GI symptoms that adversely affect appetite and worsen after eating, the latter of which may cause a fear of eating, further reducing nutrient and intake and worsening malnutrition.

Treatment and Nutrition Intervention

Treatment for CD depends on the phase of the disease and, if during a flare, its severity. Medications used in CD target specific problems; for example, during a flare, drugs target the inflammation, pain, and diarrhea, and may cause nutrition-related problems (**Box 10.12**).[7,37] In disease exacerbation that is not severe, patients can be treated on an outpatient basis, but if severe, hospitalization is necessary along with parenteral nutrition (PN), although small enteral feedings may be tolerated (**Box 10.13**). Exclusive enteral nutrition (EEN) may be effective in some patients for induction of remission in the following cases: strictures, abscesses, fistulas, perioperative setting to avoid the need for surgery, and postsurgical reduction of complications.[7] However, EEN can present a compliance challenge for some patients. To address the EEN compliance problem but recognizing the 80% remission rate, researchers developed a whole-foods diet, CD-TREAT, consisting of common solid foods in a group of 25 adults and five children.[38]

Box 10.12 Nutrition Implications of Medications Used in Inflammatory Bowel Disease

Drug Class	Drugs	Potential Nutrition Problems
5-Aminosalicylic acid derivative agents	Mesalamine rectal, mesalamine, sulfasalazine, and balsalazide	Nausea, vomiting, acid reflux, diarrhea, and folate depletion
Corticosteroids	Prednisone, methylprednisolone, budesonide, hydrocortisone, and prednisolone	Fluid retention, increase in appetite, increase in blood glucose, nausea, weight gain; may need to reduce sodium; increase protein, potassium, and calcium
Immunosuppressive agents	Mercaptopurine, methotrexate, and tacrolimus	Anorexia, nausea, vomiting, and mouth sores
Monoclonal antibodies	Infliximab, adalimumab, and certolizumab pegol	Abdominal pain, nausea, vomiting, and irritation of mouth or tongue
Alpha 4 integrin inhibitors	Natalizumab and vedolizumab	Diarrhea and stomach pain
Antibiotics	Metronidazole and ciprofloxacin	Anorexia, nausea, and vomiting
Antidiarrheal agents	Loperamide, diphenoxylate-atropine	Anorexia, bloating, constipation, stomach pain with nausea and vomiting
Bile acid sequestrants	Cholestyramine and colestipol	Acid reflux, constipation, nausea, vomiting, and stomach pain

Drug Class	Drugs	Potential Nutrition Problems
Anticholinergic agents	Dicyclomine, hyoscyamine, and propantheline	Dry mouth and nausea

Box 10.13 Dietary Recommendations for Inflammatory Bowel Disease

In Acute Exacerbation, Depending on Severity:

1. Enteral feedings or parenteral nutrition, if severe.
2. Progress to low-fat, low-fiber, high-protein, high-kilocalorie (if weight loss), small, and frequent meals as tolerated.
3. Avoid nuts, seeds, legumes, and coarse grains.
4. Avoid lactose, wheat, and gluten, if not tolerated.
5. Vitamin/mineral supplements: vitamins D and B_{12}, folate; minerals iron, zinc, calcium, and magnesium.
6. If taking corticosteroids, limit sodium intake.
7. If fat malabsorption (common with Crohn's), consider use of medium-chain triglycerides.
8. Maintain good hydration, consider oral rehydration solutions (ORS) for severe diarrhea.
9. Consider Exclusive EN (EEN) in CD patients with active inflammatory strictures, enterocutaneous fistulas, or prior to surgery. For induction of remission, 6–8 weeks of EEN should be considered.

In Remission:

1. Eat a nutrient-dense diet, with adequate protein (1.0 g/kg body weight) and energy.
2. Gradually increase dietary fiber to recommended level; fruits and vegetables may be protective.
3. Avoid foods high in oxalate, if kidney stone occurs (Box 10.16).
4. Avoid lactose, wheat, and gluten, if not tolerated.
5. Consider use of probiotics in collaboration with physician; recommend Visbiome for patients with pouchitis.

6. Consider six small meals rather than three large meals.
7. Recommend a trial of low FODMAP diet.
8. Recommend increased fluid intake via ORS in patients with severe diarrhea, and those with high jejunoscopy or ileostomy output.
9. Recommend EN in cases of insufficient intake via oral diet and insufficient intake oral feeding plus oral nutritional supplementation.
10. Recommend 1–2 mg folic acid daily for patients on sulfasalazine and methotrexate.
11. Recommend calcium supplementation of 1,000–1,500 mg/day in patients avoiding dairy products.

The CD-TREAT diet included limited foods such as *full-fat milk, rice cereal, apple and pineapple juices, white bread, cheddar cheese, lettuce, mashed potatoes, chicken, salmon, and rice pudding.*

Subjects followed the CD-TREAT diet for 7 days, then a 14-day wash-out period, and crossover to an exclusive enteral nutrition diet (EEN). The CD-TREAT diet was better tolerated and demonstrated similar effects on inflammation reduction, gut microbiome, and clinical response. The authors concluded that like EEN and antibiotic therapy, the CD-TREAT diet reduced nutrients (primarily fiber) that promote microbial growth, thus changing the gut environment to suppress inflammatory microbes and reduce the gut-associated immune response (Box 10.13).[7]

Patients should be encouraged to consume a nutrient-dense diet, with particular attention to specific nutrients in periods of remission in order to prevent nutrient deficiencies when a disease exacerbation occurs with the nutrient malabsorption that may attend it. Nutrients of concern include protein; fat-soluble vitamins; vitamins C, B_6, and B_{12} and folate; and the minerals iron, zinc, copper, calcium, potassium, and magnesium.

Ulcerative Colitis

Disease Process

Although symptoms are similar to CD, UC typically strikes the sigmoid segment of the colon, with extensive involvement of the rectum.[34] It often spreads further up throughout the colon so that the entire organ may be affected, but it never involves the small intestine. Complications in UC are as severe as those for CD, including toxic colitis in which the organ begins to dilate. The dilation can happen in hours or days, and when severe, it is termed *toxic megacolon*, which can lead to perforation. Rectal bleeding is a common problem that often causes iron-deficiency anemia. Other nutritionally relevant problems include protein losses, electrolyte disturbances, dehydration, anorexia, and weight loss. In contrast to CD, most of the nutritional problems are not directly caused by malabsorption (Box 10.11).[7] The diagnosis can be confirmed with sigmoidoscopy, which can also indicate disease severity.[39]

Treatment and Nutrition Intervention

Treatment for UC is similar to that for CD, with the goals of countering the inflammation, alleviating symptoms, and correcting dehydration and nutrient deficiencies.[7] As with CD, when the disease is in remission, a nutrient-dense diet with adequate protein and energy is the best approach to stave off nutritional problems between flares (Box 10.13). Several medications can counter the inflammation, for which the approach is stepwise and based on severity, and they can be combined. These medications include aminosalicylates, corticosteroids, immunomodulators, and some can be delivered orally, via enema, or topical application.[40] Sulfasalazine, an aminosalicylate that has been available for decades, interferes with folic acid absorption, so patients using this drug should take a folate supplement[37] (Box 10.12).

ADIME 10.6 ADIME At-A-Glance: Inflammatory Bowel Disease

Assessment

- UWL, decreased appetite, and intake
- Abdominal pain, nausea, diarrhea (UC), steatorrhea (Crohn's)
- Diet Hx, avoidance of specific foods
- Laboratory: hgb/hct, serum proteins, and specific nutrients

Nutrition Diagnosis

- Inadequate oral intake RT Abdominal pain from UC, AEB UWL of 7% × 2 months and diet Hx
- Impaired nutrient utilization RT malabsorption (Crohn's), AEB steatorrhea, 7% UWL × 2

Intervention

Acute:

- Enteral feedings or parenteral nutrition, if severe
- Progress to low-fat, low-fiber, high-protein, high-kilocalorie (if weight loss), small, frequent meals AT
- Avoid nuts, seeds, legumes, coarse grains; lactose, wheat, and gluten, if not tolerated
- Supplement of vitamins D and B_{12}, folate, Fe, Zn, Ca, and Mg
- Limit sodium, if corticosteroids

Monitoring and Evaluation

- Weight

- Diarrhea and steatorrhea
- PO intake (especially protein)
- Laboratory values for specific nutrients
- Control of GI symptoms

Celiac Disease

Disease Process

Celiac disease, also known as nontropical sprue, gluten-induced sprue, gluten-induced enteropathy, and gluten-sensitive enteropathy, is caused by an inappropriate T-cell-mediated immune response to various compounds of specific grain proteins in genetically sensitive individuals, which causes inflammation and damage to mucosal cells in the proximal small intestine.[41] In wheat, the compound is gliadin, one of two protein components of the main protein gluten, with similar compounds in other grains including hordein in barley and secalin in rye.

Celiac affects 0.67% of Europeans, with a higher prevalence in Ireland and Italy, 0.4% in some parts of the United States,[41] although other prevalence estimates indicate 1% of most populations.[42] In addition to genetics, risk factors include Down syndrome, type 1 diabetes, chronic arthritis in childhood, and other autoimmune diseases. Patients' disease presentation varies widely particularly at different ages with signs and symptoms from gastrointestinal, neurologic, hormonal, and skin disorders. Gastrointestinal symptoms can include abdominal bloating, pain, diarrhea, constipation,[43] and children with IBS are four times more likely to have celiac disease.[44] Because it affects the small intestine, the disease is characterized by malabsorption of nutrients with numerous nutritional problems including weight loss, anemia, bone disease, and growth deficits in children.

Intestinal biopsy via esophagogastroduodenoscopy (EGD) provides confirmation of the diagnosis, although initial screening can include CD-specific serum antibodies, such as tissue transglutaminase, and nonbiopsy testing may be warranted for some children.[42]

Treatment and Nutrition Intervention

The treatment for celiac disease is the lifelong elimination of gliadin/gluten from the diet, which must be done as soon as possible to prevent extensive damage to the intestine.[42] Some patients with refractory celiac disease, either a more severe or long-standing and unresponsive to diet, corticosteroids become necessary.[45] As with IBD, damage of the intestinal tissue may necessitate the removal of the affected sections of the small intestine. And as with CD, this poses nutritional concerns because of the small intestine's role in nutrient digestion and absorption. In contrast to CD, however, removal of the toxin from the diet precludes further need for surgery. If a sufficient amount of small intestine is removed, this can cause short bowel syndrome (SBS).

Because of potentially long-standing nutrient malabsorption, it is important to identify and correct any nutrient deficiencies (**Box 10.14**), with special focus on vitamin D, folate, iron, and calcium.[7] Additionally, protein should be increased to 1.0–2.0 g/kg body weight, with additional energy in cases of weight loss and growth deficits in children. If fat malabsorption is present, medium-chain triglycerides are also helpful. Lactose intolerance is likely to be a problem because of inflammation of mucosal tissue upon diagnosis, and it is important to avoid lactose long term, if intolerance persists after recovery.

Box 10.14 Dietary Recommendations in Celiac Disease

Initial Diagnosis

1. Correct any nutritional deficiencies (special attention to folate, vitamin D, calcium, and iron).
2. Protein intake should be 1–2 g/kg body weight for adults.
3. Energy intake should be increased in cases of weight loss or in growth deficits in children.
4. Reduce dietary fiber initially and increase gradually as tolerated.
5. Avoid lactose and determine whether intolerance persists.

Patient Education: Foods to Avoid

Breads, Cereals, and Grains

- Gluten, wheat, whole wheat flour, enriched flour, soft wheat flour, high-gluten flour, high-protein flour; all flour containing wheat, rye, or barley, bran, graham, wheat germ, malt, kasha, bulgur, spelt, kamut, triticale, couscous, farina, seitan, semolina, durum, durum flour, groats, millet, whole wheat berries, and wheat starch
- Commercially prepared mixes for biscuits, cornbread, muffins, pancakes, and waffles
- Pasta, regular noodles, spaghetti, macaroni, and most packaged rice dishes
- Bread crumbs, cracker meal, pretzels, and matzo
- Gelatinized starch, which may contain wheat protein

Milk/Dairy

- Malted milk, Ovaltine; chocolate milk with cereal additive
- Some sour cream, some yogurt, and some nondairy creamers
- Products containing lactose, if lactose tolerance is present

Meat/Fish/Poultry

- Meats prepared with wheat, rye, or barley (bologna, chili, hot dogs, luncheon meats, and sandwich spreads)
- Creamed meats, breaded or bread-containing products (i.e., croquettes, Swiss steak, and meat loaf)
- Meats injected with hydrolyzed vegetable protein
- Tuna in vegetable broth, meat, or meat alternatives containing gluten stabilizers
- Eggs in sauces with gluten

Fruits and Vegetables

- Breaded or creamed vegetables or vegetables in sauce
- Some canned, baked beans
- Some commercially prepared vegetables and salads
- Thickened or prepared fruits, some pie fillings

Fats/Oils and Sweets

- Some commercially prepared salad dressings
- Some commercial candies, chocolate-coated nuts
- Commercial cakes, cookies, pies, and doughnuts made with wheat, rye, or barley
- Prepared dessert mixes including cookies and cakes
- Puddings thickened with wheat flour
- Ice cream or sherbet with gluten stabilizers; ice cream containing cookies, crumbs, or cheesecake; ice cream cones

Alcohol

- Ale, beer, gin, whiskies, vodka distilled from grain

Miscellaneous

- Herbal teas with malted barley or other grains with gluten (see Bread/Cereal/Grain list)
- Most canned soups, cream soups and soup mixes, and bouillon
- Some curry powder, dry seasoning mixes, gravy extracts, meat sauces, catsup, mustard, horseradish, soy sauce, chip dips, chewing gum, distilled white vinegar, cereal extract, cereal beverages (Postum), root beer, yeast extract, malt syrup, malt vinegar, and commercial infant dinners with flour thickeners
- Caramel color and MSG may not be tolerated
- Herbal and nutritional supplements
- Prescription and over-the-counter medicines
- Vitamin and mineral supplements
- Children’s modeling dough, such as Play-Doh
- Cosmetics: lipstick, lip gloss, and lip balm
- Skin and hair products
- Toothpaste and mouthwash
- Communion wafers

The patient must avoid all sources of gluten in the diet, with the most obvious being foods that contain wheat, rye, and barley. At one time, oats were also eliminated from the diet, and while this is no longer the case, commercial oat products may be contaminated with wheat in processing. For this reason, it may be useful after the initial diagnosis to avoid oats and gradually introduce into the diet observing for signs of intolerance. In addition to obvious foods containing wheat, rye, or barley, the patient must also learn to read food labels for ingredients made from parts of one of the grains including cereal, starch, flour, thickening agents, emulsifiers, stabilizers, hydrolyzed vegetable proteins, caramel coloring, and monosodium glutamate (Box 10.14).[7] Hidden sources of gluten include dietary supplements, prescription and over-the-counter medications, cosmetics, hygiene products, and communion wafers.[46]

ADIME 10.7 ADIME At-A-Glance: Celiac Disease

Assessment

- UWL, decreased appetite and intake
- Abdominal pain, nausea, diarrhea, and steatorrhea
- Diet Hx, avoidance of specific foods
- Laboratory: anti-TTG antibodies, IgA, IgG, serum proteins, hgb/hct, and specific nutrients

Nutrition Diagnosis

- Impaired nutrient utilization RT malabsorption (Celiac, AEB steatorrhea, 7% UWL × 2 months)

Intervention

- Protein 1.0–2.0 g/kg; energy for repletion/regain of lost weight
- Correct deficiencies (especially folate, vitamin D, Ca, and Fe)
- Gradually increase fiber AT after initial reduction
- Avoid lactose, if LI
- Instruction on gluten-free diet

Monitoring and Evaluation

- Weight
- Diarrhea and steatorrhea
- PO intake (especially protein)
- Laboratory values for specific nutrients
- Control of GI symptoms

Intestinal Surgery: Ostomy, Short Bowel Syndrome, Intestinal Transplant

Disease Process

Several gastrointestinal diseases may necessitate intestinal surgery that involves removal of sections of intestine. In the case of the colon, the entire organ may be removed with minimal long-term effects on nutrient absorption. However, many nutritional problems may arise depending on how much small intestine is lost: Loss of 50% or more of the small intestine produces nutrient malabsorption and places a patient at risk for SBS, which is defined as in adults is defined as less than 180–200 cm of remaining small bowel (normal length 275–850 cm),[47] and more than 75% of the small intestine is associated with a high mortality rate.[48]

When the body's nutritional demands cannot be met by the gastrointestinal absorptive surface, intestinal failure (IF)

results. The remaining bowel can adapt to recover intestinal function, and determinants of intestinal adaptability include type and location of the resection, patient age and nutritional status, and content of the diet.

After intestinal surgery to remove sections of the intestine, it may be necessary to create a stoma for waste elimination. In a colostomy, part of the colon, the rectum, and anus are removed, and the remaining segment of colon is brought out through the abdominal wall to form the stoma. An ileostomy involves a colectomy, removal of the entire colon, the rectum, and anus, and brining the ileum through the opening. Ileostomy causes more problems because of the digestive and absorptive role of this portion of the small intestine.[7] In addition to nutritional problems, diet can influence various aspects of bowel regularity and stool consistency.

Ileostomy: Treatment and Nutrition Recommendations

An ileostomy produces a watery stool, making postsurgical adjustment more of a challenge than with colostomy. Also, ileostomy often causes fat malabsorption because bile is not reabsorbed, resulting in malabsorption of fat-soluble vitamins, which necessitates supplementation (**Box 10.15**).[7] Deficiency of vitamin B_{12} is also common, as absorption of the vitamin occurs in the terminal ileum. The risk for both kidney and gallstones increases with steatorrhea, and so it is important to monitor for and counter the development of these conditions and be prudent to avoid foods high in oxalate (**Box 10.16**). Ileostomy also causes excessive fluid losses, along with electrolytes, making dehydration a special concern. Obstruction is a possible complication, so thorough mastication of foods is important.

Box 10.15 Dietary Recommendations for Ileostomy

1. Clear liquid diet following surgery.
2. Progress to a bland, low-fiber diet with extra kilocalories and protein (1.5 g/kg/d).
3. Continue with low-fiber diet for about 4 weeks; include sources of pectin such as oatmeal and applesauce.
4. Supplement with vitamins C and B_{12}, folate, calcium, magnesium, and iron.
5. Maintain good hydration status and supplement with sodium and potassium, as needed, especially in hot weather.
6. Avoid eating before bedtime.
7. If gassiness is a problem, avoid cruciferous vegetables, legumes, and other potential gas-forming fruits and vegetables and FODMAPs (Box 10.10).
8. Follow additional recommendations for specific foods, as needed, for colostomy (Box 10.17).

Box 10.16 Foods High in Oxalate

Beets
Celery
Chocolate/cocoa
Nuts
Peanut butter
Rhubarb
Soy
Spinach
Strawberries
Sweet potatoes
Tea
Vitamin C supplements
Whole wheat

ADIME 10.8 ADIME At-A-Glance: Ileostomy

Assessment

- UWL, decreased appetite and intake
- Abdominal pain, nausea, diarrhea, and steatorrhea
- Diet Hx, avoidance of specific foods
- Laboratory: serum proteins, hgb/hct, specific nutrients (especially B_{12})

Nutrition Diagnosis

- Altered GI function RT ileostomy, AEB diarrhea
- Impaired nutrient utilization (B_{12}) RT malabsorption from ileostomy, AEB low serum B_{12} level

Intervention

- Clear liquid diet post-surgery
- Progress to a bland, low-fiber diet, adequate energy, protein, fluid, and electrolytes individualized for the patient
- Begin with smaller, more frequent meals
- Low-oxalate diet
- Supplement of vitamins C and B_{12}, folate, Ca, Mg, and Fe
- Adequate fluids, possible Na and K supplement PRN, especially in hot weather
- Avoid eating before bedtime
- Low-FODMAP diet, if flatulence problem
- Monitor for lactose intolerance
- Monitor for fat malabsorption

Monitoring and Evaluation

- Weight
- Diarrhea and steatorrhea

- PO intake (especially protein)
- Laboratory values for specific nutrients
- Control of GI symptoms

Colostomy: Treatment and Nutrition Recommendations

With a colostomy, the stool consistency is close to normal or normal, depending on how much of the colon was removed. If the colostomy is on the right side, the stool will be mushy, whereas a left-sided colostomy produces a firm stool. Odor is a significant problem for the person with a colostomy, and attention to specific foods that cause more of a problem is important. Because potential odor-causing foods are nutrient dense, it is better to focus on other approaches, such as deodorizers. Many of the same diet recommendations for ileostomy apply to colostomy, although the possible nutritional problems are more likely with ileostomy (**Box 10.17**).[7]

Box 10.17 Dietary Recommendations for Colostomy

1. A clear liquid diet follows surgery and an oral diet follows for most patients within 2 days.
2. Progress to a low-fiber diet with extra kilocalories and protein (1.5 g/kg/d) and adequate vitamins and minerals.
3. After approximately 2 weeks, gradually increase fiber intake.
4. The following foods should be avoided only if they consistently cause undesirable effects, *as they may cause obstructions:*
   bamboo shoots, bean sprouts, celery, citrus fruit membranes, coconut, coleslaw, corn, fruits with skins and seeds, green beans, lettuce, mushrooms, nuts, peas/pea pods, popcorn, potato skins, raw carrots, raw/dried fruit, relishes, seeds, spinach, tough meats, and vegetables.

May cause odor or gas
Antibiotics, asparagus, beer, broccoli, Brussels sprouts, cabbage family vegetables, carbonated beverages, cauliflower, corn, cucumbers, deep fried

foods, dried beans/peas, eggs, fish, melons, milk, mustard, nuts, onions, pastries, pickles, radishes, some vitamin/mineral supplements, spicy foods, strong-flavored cheeses, and turnips.

May contribute to diarrhea

Beans, beer/wine, broccoli, coffee, fresh fruits/vegetables and juices, green leafy vegetables (especially spinach), highly spiced foods, licorice, and prune juice:

1. Foods such as applesauce, bananas, boiled milk, cheese, milk, peanut butter, rice, and tapioca may reduce diarrhea (reducing fluid intake does not reduce diarrhea, and it may cause dehydration).
2. Consume 1–2 quarts of fluid daily, between meals, with adequate or increased sodium.

Dietary/Behavior Modifications

- Establish regular meal times.
- Eat slowly and chew thoroughly with mouth closed.
- Avoid chewing gum or drinking from straws to prevent excess gas.
- If a food has been eliminated from the diet because of diarrhea, constipation, odor, or gas, retest tolerance after 2–3 weeks.

Short Bowel Syndrome

Disease Process

With removal of 50% or more of the small intestine in a resection, SBS is a prime concern, especially if the terminal ileum and ileocecal valve were removed. Although intestinal length varies widely as does ability to adapt, a length of 200 cm or less[49] meets the criteria for SBS, while the Center for Medicare and Medicaid Services uses <153 cm as the criteria.[48] Since the intestine is capable of adaptation, more important than the length of the section removed is the length and location of the remaining bowel, specifically the jejunum or ileum. For example, bile salt and vitamin B_{12} absorption occur in the ileum, which is also the site of active sodium chloride transport, which facilitates fluid reabsorption. Additionally, the ileum has a higher capacity for adaptation relative to nutrient absorption.[48]

The most significant nutritional problems associated with SBS arise from malabsorption (**Box 10.18**). Although the survival rate declines as more of the intestine is removed, if the ileum and ileocecal valve remain intact, even an 80% resection can prove to be tolerated.[50] Resections of the jejunum initially cause significant malabsorption, because most nutrients are digested and absorbed within the first 40 inches of the small bowel. However, after the adaptation period, the ileum takes over this function, as the remaining intestine grows in length, diameter, and thickness.

Box 10.18 Nutritionally Relevant Problems in Short Bowel Syndrome

Deficit or Problem	Result
Bile acid	Fat malabsorption, bacterial overgrowth
Absorptive surface	Malabsorption or nutrients
Fluid reabsorption	Dehydration
Increased motility, short GIT	Dumping syndrome and diarrhea
Loss of ileocecal valve	Bacterial overgrowth (B_{12} deficiency)
Oversecretion of gastric acid	Damage to duodenum, pancreatic enzyme activity altered
Pancreatic enzyme activity	Maldigestion and malabsorption

Deficit or Problem	Result
Fat malabsorption	Steatorrhea, increased risk for kidney stones, gallstones, fat-soluble vitamin deficiency, and bone disease

Because of its importance in long-term survival, and the fact that it is the only controllable variable, enhancing the intestine's ability to adapt is the focus of treatment. Nutrition is the key to recovery and enhancing the intestine's adaptive ability. The reason is that many of the typical symptoms a patient with SBS will experience are nutritionally relevant: diarrhea, steatorrhea, weight loss, muscle wasting, bone disease, and malabsorption of several nutrients. SIBO is another nutritionally relevant problem that appears to be common in SBS, particularly when the ileocecal valve has been removed and part of the colon remains.[48]

The occurrence of SIBO presents several deleterious nutritional consequences. The bacterial overgrowth can cause inflammation and atrophic changes that produce nutrient malabsorption. In addition, the excess microbes deconjugate bile acids, causing fat malabsorption, and compete for B_{12}, exacerbating the potential for deficiency of the vitamin. Other gastrointestinal symptoms resulting from SIBO also aggravate diarrhea, which can adversely affect food intake, and may increase the risk of intestinal failure-associated liver disease (IFALD), a serious complication that can cause cholestasis, steatosis, and fibrosis, leading to cirrhosis.[51]

Treatment and Nutrition Intervention

Treatment and nutrition intervention focuses on managing symptoms, enhancing intestinal adaptation, and

maintaining adequate nutritional and hydration status (**Box 10.19**).[7] A major problem for most SBS patients is voluminous diarrhea, which, if not controlled, can adversely affect nutritional status and bowel recovery. Although diet alone cannot control the diarrhea, careful food selection and fluid intake can help reduce fecal output and optimize nutrient absorption, in conjunction with medications.[52]

Box 10.19 Dietary Recommendations for Short Bowel Syndrome

Phase 1: Immediately Following Intestinal Resection Surgery

1. Parental nutrition (PN) is required. The extent of the resection and overall health of the subject will determine the duration of PN.
2. Enteral nutrition (EN) may be useful; consider only in stable patients who have begun oral intake, which is insufficient.

Phase 2: Initiation of Oral Feedings

1. Fat: if colon is present, diet should be <20%; without colon, 30–40% of calories.
2. Carbohydrate: if colon is present, diet should be 60%; without colon, 40–50% of calories; focus is on complex carbohydrate, both must restrict simple sugars.
3. Protein: 20–30% of calories, emphasis on high biologic value sources at each meal and snack.
4. As oral diet tolerance improves:
   - 5–10 g/d soluble fiber, if stool output is >3 L/d.
   - Avoid high-oxalate foods (Box 10.16), only in patients with colon in continuity and who have had a kidney stone and with the presence of hyperoxaluria.
   - Vitamin/mineral supplements that include calcium, magnesium, zinc, iron, manganese, vitamin C, selenium, potassium, folic acid, B_{12} and B-complex vitamins, and water miscible forms of vitamins A, D, E, and K.
   - Salt: increase in patients with end jejunostomy/ileostomy.
   - Fluids: ORS may be needed; total fluid may need to be restricted.
   - Modified FODMAP restriction, avoid fructose, high-fructose corn syrup, sugar alcohols such as sorbitol, and limit lactose only if intolerant (Box 10.10).

Diet and Behavior Modifications

- Smaller, more frequent meals: 4–6 meals/d.
- Consume fluids between meals in small amounts.

Medications are important in managing the symptoms of diarrhea, steatorrhea, and SIBO, such as those that slow gastric emptying and intestinal motility and reduce gastric secretion, which can help to increase nutrient digestion and absorption, which can help to increase nutrient digestion and absorption and reduce stool output.[52] For optimal efficacy, patients should take their antimotility medications consistently and 30–60 minutes prior to meals. However, it is important to note that reduced absorptive surface area and accelerated transit time typically cause drug malabsorption.

Feeding progresses in a stepwise process consisting of three phases that will vary with the patient relative to the bowel's adaptive capacity, the extent of resection, and resulting remnant anatomy (Box 10.19).[53] For these reasons, the dietitian must individualize the nutrition therapy to the patient, particularly based on the patient's remnant anatomy and the corresponding specific roles of the affected GI tract sections. Although the main aspects of the nutrition approach are similar, the presence of the colon may necessitate additional modifications, especially the need to restrict oxalate with some colon in continuity in select patients (see Box 10.16). However, a low oxalate diet is difficult for patient adherence and restrictive of nutritious foods, so the dietitian should recommend it only in patients who have a history of a kidney stone and hyperoxaluria confirmed by urinalysis.

The first phase is within 1–3 months after surgery, characterized by voluminous diarrhea, fluid losses, and significantly limited absorption. According to the American Gastroenterological Association (AGA) Clinical Practice Update on Management of Short Bowel Syndrome Clinical

Guidelines, almost all patients will require PN support in this first phase following resection, and most will not be able to discontinue the PN prior to discharge.[49] After 5 years, only half of patients can be weaned from PN, and if it cannot be done within 2 years more than 94% will remain PN-dependent. In patients with an end-jejunostomy, which is a resection or exclusion of the ileum and colon, intestinal adaptation is significantly lower making it more likely to require permanent parenteral support.[48] Although EN had been proposed as stimulating adaptation of the bowel remnant, the AGA has stated it is rarely and suggests consideration of EN in stable patients in used combination with oral feeding if intake is inadequate after the use of appetite stimulating drugs.[49]

In the second phase, 4–12 months, nutrient absorption begins to improve, and the patient begins to regain weight. During this time as oral intake is reestablished, many patients need to continue PN. Maintenance of adequate hydration status is important, and an oral rehydration solution (ORS) is useful to improve absorption, reduce secretions via the sodium-glucose-coupled transport system. The use of an ORS is particularly important for patients with an end-jejunostomy and those with some colon continuity.[48]

In the final phase, which lasts 13–24 months, bowel adaptation results in a return to more normalized nutrient absorption. The timing of the different dietary changes is variable but may take months or even years before a patient can resume a regular diet, and some patients cannot be weaned off PN. Malabsorption in SBS causes adaptive hyperphagia or excessive hunger in order to compensate.[49] The AGA has stated that nutrition therapy “should focus on maintaining compensatory hyperphagia, rather than on excessive dietary restrictions.” They recommend a “hyperphagic diet” that provides at least 50% higher energy over a patient’s estimated needs. The recommendation to

eat small, frequent meals (5–6) throughout the day facilitates the attainment of the high energy requirements and is better tolerated.[7]

In addition to small, frequent meals, other key dietary points include avoiding all forms of simple sugars, and paying careful attention in chewing foods extremely well (Box 10.19). Although widely recommended and used, studies on the efficacy of compounds such as glutamine, somatropin (a recombinant human growth hormone), and glucagons, in promoting intestinal adaptation are limited. Teduglutide, an injectable analog of naturally occurring glucagon-like peptide-2 (GLP-2), binds to GLP-2 receptors activating the release of intestinal mediators to increase intestinal absorption of both fluid and nutrients.[47] It is recommended in patients 1 year or older who are PN-dependent, but side effects include nausea, vomiting, abdominal pain and distension, fluid overload, stoma blockage, and injection site reactions. More serious complications include the potential to accelerate the growth of colonic polyps and neoplasms, biliary disease, and pancreatitis.[52] However, its long-term use has demonstrated sustained efficacy in reducing PN volume.

Intestinal Transplantation

—Thomas Pietrowsky, MS, RD, and **Maria Segovia**, MD

Intestinal transplantation is an option for patients with intestinal failure who suffer severe complications because of total parenteral nutrition (TPN) dependence. Intestinal failure is defined as the loss of absorptive capacity of the small bowel as a result of severe GI disease or surgically induced short gut syndrome.[54] The total number of intestine transplants performed annually in the United States reached a peak of 198 in 2007 and decreased to 82 in 2022.[55] There has been no significant change in graft survival after

intestine transplant across the period from 2010 through 2021. Patient survival at 1 year after intestine transplant is 83.8% as of 2021 and survival after 5 years is 61.2%.[56]

ADIME 10.9 ADIME At-A-Glance: Short Bowel Syndrome (and Intestinal Transplant)

Assessment

- Current PO intake or nutrition support regimen
- Length and location of remaining functional bowel
- GI symptoms including vomiting, diarrhea
- Ostomy output and hydration status
- Lab values
- Baseline vitamin levels (vitamins A, D, E, B_1, B_2, B_6, B_{12}, and C, and folic acid) and mineral levels (chromium, copper, manganese, selenium, and zinc)

Nutrition Diagnosis

- TPN dependent because of malabsorption R/T short gut syndrome or intestinal dysmotility
- Complications with TPN R/T liver cholestasis, central vein thrombosis, line sepsis, or severe dehydration

Intervention

- Short term:
  - POD 1 to 2: Initiate PN.
  - POD 3 to 7: Initiate enteral feedings and taper PN as tube feedings increase.
  - 2 weeks: Begin clear liquids and advance as tolerated to regular diet, taper enteral feedings as oral intake increases.
- Long term:
  - Small, frequent meals with supplements and ORS.

- Low fiber with complex carbohydrates.
- Limit lactose and concentrated sweets, low oxalate.
- Limit fat with chylous ascites.

Monitoring and Evaluation

- PO intake and tolerance using food journals
- Weight, protein status
- I & Os (ostomy output, hydration)
- Electrolyte balance
- Labs:
  - Monthly: zinc
  - Every 6 months: D, B_{12}, and B_6, iron studies, and lipid panel
- Every year: vitamins A and C, selenium, copper, manganese, and chromium. Academy of Nutrition and Dietetics. Nutrition Care Manual. Accessed March 18, 2023. Available for subscription at: https://www.nutritioncaremanual.org

Indications for and Types of Intestinal Transplant

Patients with intestinal failure are eligible for intestine transplant if they meet any of the following criteria according to the Centers for Medicare and Medicaid Services (CMS)[54]:

- Imminent or evident liver failure of TPN-induced liver injury
- Thrombosis of at least two central veins
- Two or more episodes of line sepsis per year that require hospitalization
- Single episode of line-related fungemia, septic shock, or acute respiratory distress
- Recurrent episodes of severe dehydration despite intravenous fluid supplementation

Types of intestinal transplants are as follows:

- Isolated intestine: intestinal failure without liver disease.
- Combined liver/intestine: intestinal failure with TPN-associated liver failure.
- Multivisceral: includes the stomach, pancreas, and intestine with or without the liver. Indicated in patients with massive gastrointestinal polyposis, traumatic loss of abdominal viscera, extensive abdominal desmoid tumors, locally aggressive non-metastasizing neoplasms, generalized hollow visceral myopathy/neuropathy, and complete thrombosis of the splanchnic arterial or portal venous systems.[57]

Nutrition Before Intestine Transplant

When evaluating patients for intestine transplant, a thorough nutritional assessment should be performed. The evaluation should assess medical history and current diagnoses, diet history including oral intake and nutritional support, amount and location of remaining functional bowel, GI symptoms, anthropometrics including weight history, laboratory values, ostomy output, and appropriateness of the current diet order or TPN regimen. Collaboration with home infusion providers is important when assessing labs and changes with home TPN prescriptions. Living environment should also be considered, including persons in the household and who will perform shopping and meal preparation.

Once a patient is deemed a candidate for intestine transplant, there are several nutritional goals to consider. Maintaining adequate hydration is vital to help preserve renal function, and additional intravenous hydration may be needed to replace fluid losses. Preserving kidney function prior to transplant is especially important since some post-transplant medications, including tacrolimus, can have potentially nephrotoxic effects.[58] Hepatic function should be

preserved by adjusting parenteral lipids and dextrose as needed to help avoid hepatic steatosis. Since patients can experience significant weight loss of 20% or more of their pretransplant weight in the postoperative period, optimization of nutritional status is vital for improving outcomes after transplant.[59] Measuring pertinent serum vitamin and trace element levels and correcting deficiencies are also recommended in the pretransplant phase.[58]

Post-Transplant Nutrition—Short Term

Nutritional goals in the early post-transplant period are similar to other major surgeries (Box 10.19). Adequate nutrition is critical to help promote weight gain, decrease risk of infection, and improve wound healing. The ultimate nutritional goal post-transplant is to reach nutritional autonomy in which the patient can maintain weight, electrolyte balance, and adequate hydration.

The transition to an oral diet can occur rather quickly after transplant.[53] PN can usually be started 24–48 hours after surgery. Calorie intake should be increased as tolerated to 30–35 kcal/kg. Recommended protein requirements are 1.5–2.0 g/kg. PN can be weaned as enteral nutrition is initiated and increased, and some centers will place feeding tubes at the time of transplant. Surgical complications, episodes of rejection, or infections may delay PN weaning.

Enteral nutrition should be initiated as soon as possible after surgery to help maintain gut integrity. Enteral feedings can generally be initiated 3–7 days post-transplant once allograft motility is established.[58] Routes of administration can vary among transplant centers. Feedings should be started at full strength and advanced slowly (5–10 mL/h) every 12–24 hours to goal rate. Polymeric formulas are usually tolerated well. Formulas that are lower in potassium but maintain a high protein content may be beneficial because of the risk of hyperkalemia from some

immunosuppression medications, and lower fat formulas may be used if chylous ascites develops.

Although the rate of diet advancement is individualized, an oral diet can usually be initiated within 2 weeks after transplant. A clear liquid diet should be started and advanced as tolerated to a regular diet. Patients should begin a low-fiber diet consisting of small, frequent meals. Lactose should also be limited in patients who show signs of lactose intolerance. Concentrated sweets should be restricted if patients experience diarrhea or hyperglycemia. Fats can contribute a significant source of calories and generally should not be restricted unless there is a medical indication, such as chylous ascites. Oral rehydration solutions should also be added once diet is initiated to help avoid dehydration.

Post-Transplant Nutrition—Long Term

Tolerance to diet can vary, and some patients may have difficulty tolerating lactose and concentrated sweets long term. Food journals are useful for assessing oral intake and any GI symptoms with eating. Nutrition therapy should emphasize maintaining weight and protein status, adequate hydration, and electrolyte balance. Suggested nutritional labs for monitoring long term include the following[60]:

- Monthly: zinc
- Every 6 months: vitamin D, vitamin B_{12}, vitamin B_6, iron studies, and lipid panel
- Every 12 months: vitamin A, vitamin C, selenium, copper, manganese, and chromium

As oral intake improves over time, intestine transplant patients can be at a higher risk of becoming overweight or obese. Therefore, education and reinforcement of healthy eating guidelines are prudent. Conversely, many patients can have eating issues because of anxiety from GI

symptoms they experienced before transplant. Psychologists can be valuable members of the transplant team, especially in helping intestine transplant patients return to a regular diet.

Developing a foodborne illness is a potential risk for posttransplant patients since immunosuppression medications weaken the immune system. The following are tips for preventing foodborne illnesses[61]:

1. Always wash hands before eating and preparing meals.
2. Wash fruits and vegetables thoroughly. Avoid raw sprouts due to increased risk of contamination.
3. Water quality/safety: if there is a concern about water quality, boil water for 1 minute to help kill impurities. Patients should be encouraged to contact the EPA Safe Water Hotline at 1-800-426-4791 or local community for water safety information.
4. Cross-contamination occurs when bacteria are spread from one food product to another (especially when handling raw meat, poultry, seafood, and eggs). To prevent cross-contamination:
   - Separate raw meat from other food items when shopping, handling, and preparing meals.
   - Consider using more than one cutting board in food preparation (for example, raw foods on a “red” colored board, cooked foods on a “green” cutting board).
5. Cooking temperatures: never consume raw or undercooked meat. Check the internal temperature by using a meat thermometer. Keep hot foods hot and cold foods cold.
   - Ground meat: 160°F (71.1°C)
   - Steak and roast: 145°F (62.8°C)
   - Chicken: 165°F (73.9°C)
   - Seafood: 145°F (62.8°C)

- Precooked seafood: 165°F (73.9°C)
- Leftovers and lunch meat: reheat to165°F (73.9°C)

6. Refrigerand freeze effectively.
    - Maintain a consistent refrigerator temperature of 40°F or below and a freezer temperature of 0°F or below.
    - Refrigerate or freeze meat, poultry, eggs, and seafood within 2 hours of cooking or purchasing.
    - Never thaw food at room temperature. Thaw foods in refrigerator, cold water, or microwave. If thawed in water or microwave, cook immediately.
7. Avoid unpasteurized foods such as unpasteurized milk, cheese made with unpasteurized milk, honey, and apple cider.
8. Avoid undercooked or raw eggs (runny egg yolks).
9. Never consume food past its expiration date.

Although intestine transplant is still a relatively new medical procedure, in concert with intensive nutritional therapy, many patients with intestinal failure can discontinue TPN and experience an improved quality of life.

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CHAPTER 11

Hepatobiliary Disease

The liver, pancreas, and gallbladder have been referred to as the “accessory organs” because of their participation in digestion. Given the crucial role that the liver, in particular, plays in nutrient absorption, metabolism, synthesis, and storage, diseases affecting this organ can profoundly and adversely affect nutritional status. Chronic liver disease, to which any liver disease or injury can progress, affects 4.5 million Americans accounting for 56,585 deaths as the ninth leading cause of death.[1] The pancreas, a dual organ of both endocrine and exocrine function, critically participates in digestion and also in blood glucose regulation. Problems with this organ affect both of these functions. The gallbladder’s role is limited, with minimal potential effect on nutritional status. However, the presence of gallstones, which affects 10–20% of Americans, 33% of whom will develop acute cholecystitis, may necessitate nutrition intervention and surgery.[2]

Liver Disease

Several conditions can cause inflammation or damage to the liver and adversely affect organ function, including exposure to toxins or hepatotoxic drugs and dietary supplements, infection, physical injury, genetic abnormality resulting in accumulation of minerals, or an autoimmune response. Because of the organ's role in metabolism of most nutrients, liver diseases have significant potential to negatively affect nutritional status, and these include hepatitis, hepatic steatosis (fatty liver), and cirrhosis, with excessive alcohol intake being a common trigger for all. In advanced liver disease, malnutrition is the most common complication occurring in 5–92% of patients, depending on the patient population and screening process, and it is even more common in alcohol-induced liver disease.[3]

The pathophysiologic processes underlying the various forms of liver disease, such as inflammation and lipid infiltration, can lead to irreversible diffuse fibrosis of hepatic tissue known as cirrhosis, or end-stage liver disease (ESLD). Prior to this advanced stage, the liver has extensive regenerative ability and reserve capacity, posing a challenge for diagnosis, as significant function must be lost before disease is clinically evident.

Liver function tests (LFTs) provide information on the presence and extent of liver disease (**Box 11.1**).[4] LFTs indicate the functional status of the liver, as with the synthesis of proteins such as serum albumin and prothrombin; and indication of liver injury, as with various enzymes. These enzymes include aspartate aminotransferase (AST) and alanine aminotransferase (ALT). Although not specific for liver disease, other enzymes that indicate biliary tract obstruction, whether in the liver or in

the bile channels outside of the liver, include alkaline phosphatase (ALP) and gamma-glutamyl transpeptidase (GGT).[5]

Box 11.1 Liver Function Abnormalities in Liver Disease

Function	Test	Derangement
Bile synthesis	Bilirubin (serum, urine, fecal); direct (conjugated) and indirect (unconjugated)	Bilirubin not excreted in feces (clay-colored); indirect bilirubin high in liver disease and direct bilirubin high in biliary tract disease
Carbohydrate metabolism	Oral glucose tolerance test, blood glucose	Normal until advanced disease; low in acute disease; high in chronic disease
Carbohydrate metabolism	Oral glucose tolerance test, blood glucose	Normal until advanced disease; low in acute disease; high in chronic disease
Lipid metabolism	Triglyceride, lipoproteins/cholesterol, ketones	All low in severe disease
Protein metabolism	**Urea:** BUN, NH_3	BUN low, NH_3 high in advanced disease
	Plasma proteins: albumin, transferrin, amino acid ratios (BCAA vs. AAA), A1AT	Low protein levels BCAA: AAA ratio skewed toward higher AAA
	Prothrombin time (PT)	PT increased

Function	Test	Derangement
	Enzymes: ALT, AST, GGT, ALP	High

Hepatitis

Disease Process

Hepatitis is inflammation of the liver induced by the presence of a toxin, such as alcohol, numerous medications and some dietary supplements (**Box 11.2**),[6] environmental toxins, or viral infection.[7] The viral forms of hepatitis include viruses A, B, C, D, and E, with D only arising in patients with hepatitis B virus (HBV), as it requires a helper function of HBV for replication.[8] Both hepatitis A virus (HAV) and hepatitis E virus (HEV) are spread by contact with infected persons or consumption of infected foods, generally from the fecal–oral route. The other forms are transmitted parenterally through contaminated blood and body fluids. Patients with HAV generally recover within 3–6 months, and rarely develop chronic liver disease. In contrast, chronic liver disease develops in up to 6% of adults and 90% of infants infected with HBV and 85% of hepatitis C virus (HCV) patients.[9]

Box 11.2 Dietary Supplements Associated with Hepatotoxic Effects

Aloe Vera
Black cohosh
Comfrey
Chaparral
Fenugreek
Germander
Ginkgo

Green tea extract
Horse chestnut
Jin Bu Huan
Kava
Ma Huang
Saw Palmetto
Senna
Skullcap
Yohimbe
Bodybuilding and weight loss supplements of all types are the most likely to cause liver damage (most are proprietary formulations).

Data from "LiverTox," National Institute of Diabetes and Digestive and Kidney Disorders, US National Institutes of Health, Updated March 2, 2023. https://livertox.nih.gov/drugliverinjury.html.

Acute hepatitis proceeds through three stages: prodromal, icteric, and posticteric.[8] The prodromal phase is generally 2 weeks in duration after exposure and ends with the onset of jaundice (icteric). If infection is the cause, the patient can transmit the virus during this time. Symptoms may include nausea, anorexia, low-grade fever, and hepatomegaly. In the icteric phase, which can last up to 1 month, up to 85% of patients will be jaundiced, although it is less likely to occur in children and even less likely in infants.[10] In this stage, symptoms include clay-colored stools, dark urine, and abdominal pain in 40% of patients.

Patients with HBV and HCV are often asymptomatic, and in the case of type C, symptoms arise only after extensive hepatic damage. In the recovery phase, after jaundice, the liver is still enlarged, but function begins to return. Patients with HBV and HCV may develop chronic liver disease after recovery, and although uncommon in HAV, relapsing hepatitis A may occur and is more common in the elderly.[10]

Treatment and Nutrition Intervention

In acute hepatitis, the goals are to promote liver regeneration and prevent further injury, if a toxin is the cause. The nutritional therapy components include a diet adequate in energy, protein, and micronutrients without the

need for food restrictions (**Box 11.3**).[11] If anorexia and gastrointestinal symptoms have caused weight loss, repletion of energy reserves will be necessary. Small, frequent meals facilitate the provision of adequate energy and nutrient intake in patients with weight loss and gastrointestinal symptoms. If disease progresses to chronic liver disease and cirrhosis, more problems with eating and nutrient restrictions can make consuming adequate food and fluids more difficult.

Box 11.3 Nutrition Intervention for Hepatitis

Adequate energy, protein, carbohydrate, and micronutrients
Small, frequent feedings may be necessary for patients with anorexia and gastrointestinal symptoms.
For patients with weight loss, repletion may be necessary.

ADIME 11.1 ADIME At-A-Glance: Hepatitis

Assessment

- Low Body mass index (BMI), Unintended weight loss (UWL), anorexia/early satiety, decrease in appetite and food intake
- Abdominal pain, fatigue, nausea, and vomiting
- Ascites, edema, jaundice
- Computed tomography (CT) scans, abdominal ultrasound, Endoscopic Retrograde Cholangiopancreatography (ERCP), cholangiogram
- Viral antibodies, Liver function tests (LFTs), electrolytes, glucose, hepatic proteins

Nutrition Diagnosis

- Inadequate oral intake Related to (RT) decreased appetite from abdominal pain, AEB recent UWL of 5% × 2 weeks, and PO intake of <50%

Intervention

- Provision of higher energy/protein diet (30–35 kcal/kg, 1.0–1.2 g/kg).
- Progress as tolerated to small, frequent feedings.
- Adequate fluids.

Monitoring and Evaluation

- Body weight, dry weight
- PO intake (food, fluid)
- Laboratory values
- Control of GI symptoms

Nonalcoholic Fatty Liver Disease and Nonalcoholic Steatohepatitis

Disease Process

Fatty liver consists of an accumulation of lipids in hepatic tissue because of an imbalance between endogenous synthesis, reduced oxidation, or delivery to the organ and removal via very-low-density lipoprotein (VLDL).[12] It can arise as an early and reversible stage of liver disease from alcohol abuse, but when this is not the cause, it is nonalcoholic fatty liver disease (NAFLD). In up to one-third of patients with NAFLD, inflammation and liver cell death lead to nonalcoholic steatohepatitis (NASH).

Although the condition is reversible, it may progress to chronic and irreversible damage. NAFLD is also an independent risk factor for the development of type 2

diabetes. Several toxins and conditions can cause NAFLD, including drugs; environmental toxins; specific diseases such as diabetes, obesity, insulin resistance (IR), and dyslipidemia; surgical procedures such as jejunal ileal bypass; and long-term parenteral nutrition.[13]

Treatment and Nutrition Intervention

If related to toxins, eliminating the underlying cause is the most important treatment component. Nutrition intervention includes weight loss, if appropriate, limiting high-glycemic index foods particularly sweetened beverages and those containing fructose, following dietary fat recommendations of the American Heart Association, and limiting alcohol intake.[14] Coffee, tea, and caffeine appear to be protective against the development of fibrosis, although the intake threshold for hepatoprotective effects is not yet clear.[15]

Supplementation with α-lipoic acid may improve disease markers in NAFLD, based on animal studies,[16] and clinical trials are underway investigating its efficacy.[17] Vitamin E supplementation significantly reduced hepatic oxidative stress in NAFLD patients;[18] however, it is contraindicated in patients with diabetes.[12] A study of patients with type 2 diabetes and BMI over 25 years old, 95% of whom had NAFLD biomarkers, showed that a monitored ketogenic diet for 1 year significantly improved the biomarkers and advanced fibrosis.[19]

Cirrhosis

Disease Process

Cirrhosis is a chronic degenerative disease, in which hepatic tissue becomes fibrous, with impairment of liver function and ultimately leading to liver failure. It is the final stage of many forms of chronic liver disease. With progression to ESLD, the fibrosis becomes increasingly extensive, with fewer remaining functional hepatocytes. In the United

States, the most common causes are viral hepatitis (HCV, HBV), with alcoholic liver disease being the next leading cause.[20] Potential complications of cirrhosis include malnutrition, esophageal varices, ascites, edema, fat malabsorption, portal hypertension, IR and diabetes, and hepatic encephalopathy (HE).

ADIME 11.2 ADIME At-A-Glance: Nonalcoholic Fatty Liver Disease

Assessment

- Low BMI, UWL, decrease in appetite and food intake, or high BMI/waist circumference (diabetes, insulin resistance, dyslipidemia)
- Hepatotoxic drugs or alcohol, diabetes, obesity, insulin resistance, dyslipidemia, jejunal ileal bypass, long-term TPN
- Abdominal pain, fatigue, nausea, and vomiting
- Ascites, edema, and jaundice
- CT scans, abdominal ultrasound, ERCP, and cholangiogram
- LFTs, electrolytes, glucose, hepatic proteins, and vitamins/minerals

Nutrition Diagnosis

- Inadequate oral intake R/T decreased appetite from abdominal pain, AEB recent UWL of 5% × 2 weeks, and PO intake of <50%

Intervention

- Provision of higher energy/protein diet (30–35 kcal/kg, 1.0–1.2 g/kg); restricted energy, if high BMI.

- Progress as tolerated to small, frequent feedings.
- Adequate fluids.
- If diabetes/insulin resistant, CHO management for glycemic control.
- Possible benefit of supplementation: vitamin E (not in patients with diabetes) and α-lipoic acid.
- Coffee, tea, and caffeine may help prevent fibrosis.

Monitoring and Evaluation

- Body weight, dry weight
- PO intake (food, fluid)
- Laboratory values
- Control of GI symptoms

After cirrhosis is diagnosed, it is important to ascertain whether the patient has compensated or decompensated cirrhosis, as prognosis and survival vary significantly.[21] Compensated cirrhosis often does not produce symptoms, although clinicians may detect portal hypertension and varices, both esophageal and gastric. Patients with decompensated cirrhosis experience symptoms caused by hepatic insufficiency including jaundice, hemorrhagic varices, ascites, and HE. Scoring systems for predicting prognosis, transplant consideration, and mortality, include the Model for End-Stage Liver Disease (MELD) and the Child-Turcotte-Pugh (CTP) score. The MELD score is based on serum levels of bilirubin, creatinine, and international normalized ratio.[21] Scoring ranges from 4 to 60 points, with 30 points correlating to a 3-month survival rate of 65%.

Diabetes and IR, common in cirrhosis, in turn increase the risk of further cirrhosis complications, including liver carcinogenesis.[22] Up to 60% of patients with cirrhosis develop EV, and if acute variceal hemorrhage occurs, risk

for mortality is up to 20%.[23] One of the most serious complications is HE, which is clinically evident in 33% of patients and may affect 66% of patients in a more mild form.[24] HE is a spectrum of neuropsychiatric abnormalities, which occurs in five stages, leading to coma (hepatic coma), and is a major concern, as the stage four mortality rate of 80%.[25] A preliminary stage "0" subclinical, which had been termed *subclinical HE*, is now recognized as minimal hepatic encephalopathy (MHE). In MHE, no changes in behavior or personality can be identified, but there is impaired psychomotor testing, with minimal changes in memory, concentration, intellectual function, and coordination.

Classification of HE is based on the underlying disease, and when HE arises from cirrhosis, common precipitating factors include hyperammonemia, specific medications, systemic inflammation, infection, gut dysbiosis and gut microflora metabolites such as indoles, oxindoles and endotoxin.[26] Somatic protein depletion has been shown to increase the risk for HE.[27] Malnourished patients have a significantly higher mortality rate,[28] making early nutritional assessment and intervention in cirrhosis patients imperative.

Treatment and Nutrition Intervention

Aspects of both treatment and nutrition intervention focus on the goals of maintenance of adequate nutrition, prevention tissue catabolism, and the control of complications (**Box 11.4**).[11] Dietary restrictions in the control of complications may need to be liberalized to promote optimal intake and prevent or reverse malnutrition, which develops in as many as 92% of patients and is more prevalent in patients with alcoholic cirrhosis.[3] Sodium restriction of 2,000 mg may be needed for controlling edema and ascites, and fluid restriction if hyponatremia is present.[11] Since cirrhosis adversely affects immunity, patients with cirrhosis should avoid raw shellfish, which can

contain *Vibrio vulnificus*, a virulent bacterium that causes serious infection. In addition, infection can increase the rate of protein catabolism.[29]

Box 11.4 Nutrition Intervention for Cirrhosis and Hepatic Encephalopathy

Cirrhosis

Energy
High kilocalorie diet of 35 kcal/kg actual body weight for patients within normal range of BMI; To facilitate high energy intake, include energy-dense foods and small frequent meals, especially a carbohydrate and protein-containing bedtime snack minimize periods of fasting
Macronutrients
Protein: 1.2–1.5 g/kg (70–100 g), as tolerated and depending on nutritional status
Fat: Reduce fat and possible use of medium chain triglyceride in patients with fat malabsorption
Carbohydrate: consistent carbohydrate intake to prevent fasting state and provide glucose for energy metabolism; if diabetes or IR is present, careful management of BGL may help worsening of cirrhosis
Sodium and Fluid
Avoid unnecessary sodium restriction in order to facilitate high energy intake, unless patient develops ascites, in which restrict to 2,000 mg/day
Restrict fluid if serum sodium is less than 125 mg/dL
Vitamin Supplementation
Water Soluble: B-complex, folate, vitamin C
Fat-soluble: A, D, E
Mineral Supplementation
Selenium
Copper
Zinc
No alcohol
Avoid raw shellfish

Chronic/Intolerant Hepatic Encephalopathy

Recommendations as above, in addition:
Intake of 0.25 g/kg/day of oral BCAA can be recommended to meet total protein requirements
Possible benefit of providing percentage of plant-based and dairy-based protein sources to reduce ammonia production
Enteral and Parenteral Nutrition
Immediate enteral nutrition for patients with malnutrition who cannot achieve energy intake goals orally; PEG tube is normally contraindicated for patients

due to risk for ascites and bleeding
Parenteral nutrition for patients in whom oral or enteral nutrition cannot meet nutrient needs; PPN is typically contraindicated due to potential fluid limitation; when conjugated bilirubin is > 2 mg/dL, reduce or eliminate copper and manganese from solution

The diet should include adequate energy of up to 75% higher than calculated needs, and energy determinations must be based on dry weight. In the presence of diabetes, IR, and hyperglycemia, management of carbohydrate intake is also important to prevent worsening of cirrhosis and further complications.[22,30] Fat malabsorption with steatorrhea is common, and medium-chain triglyceride (MCT) may be useful, along with dividing meals into small, frequent feedings. However, MCT can cause diarrhea and acidosis, so use should be carefully monitored.[31] The diet should include high-quality protein of 1.0 g to 1.5 g/kg body weight, and dietary sources of BCAA (dairy, eggs, and vegetable) rather than aromatic amino acids (AAA) (meats) may be helpful. Oral BCAA supplementation may also be useful in improving protein status and preventing malnutrition, as well as improving abnormal glucose tolerance.[11] A systematic review reported that oral BCAA supplementation, but not intravenous, significantly improved manifestations of HE.[32]

Vitamin and mineral supplementation is important, consisting of vitamin C; folate; B-complex; water-soluble forms of vitamins A, D, E, and K; and calcium, potassium, magnesium, and zinc. Supplementation of vitamins A and D, however, must not be excessive so as to avoid hepatotoxicity. As with preventing progression in reversible forms of liver disease, coffee intake also appears to have the same effect in cirrhosis, with one study reporting significant benefits at four or more cups daily leading its authors to state, *“Are we ready to write a prescription for coffee? Most likely, ‘yes.’ There is sufficient evidence to provide biological plausibility for coffee as an antifibrotic*.”[33]

Although a recent systematic review concluded that the intake threshold for hepatoprotective effects has not been demonstrated.[15]

Treatment of the onset of HE includes administration of lactulose, a synthetic disaccharide that reduces ammonia in the gut, and antibiotics such as rifaximin, to reduce bacterial production of ammonia and toxins.[32] In addition, L-ornithine and L-aspartate (LOLA) preparation is useful in reducing ammonia by increasing the use of ammonia in the urea cycle.[34] Microbial alterations in the gut arise in tandem with decompensated cirrhosis, which is characterized by the disease complications including HE.[35] For this reason, prebiotics and probiotics could be potentially useful, and lactulose is sometimes classified as a prebiotic, however, more studies are needed to demonstrate their effectiveness.[22] To obtain any possible benefits, patients can be encouraged to consume a diet with the recommended level of fiber, as well as probiotic foods.

Nutrition intervention depends on the stage of HE, with refractory HE necessitating a reduction of protein intake to 0.8 g/kg/day for short duration.[11] Protein restriction may also be needed for an acute flare-up but must be of short duration, as these patients have a high catabolic rate and wasting.[34] Vegetable protein (higher in BCAA) may be better tolerated than animal protein (higher in AAA), relative to ammonia production, and oral supplementation of 0.25 g/kg/day of BCAA as part of total protein intake is useful.[11] If a patient cannot resume oral intake, enteral nutrition is warranted and should be initiated as soon as possible.[25] Prevention of malnutrition is critical, and adequate protein intake is key in this regard, with most NHE and chronic HE patients being able to tolerate a daily intake of more than 60–80 g of protein per day.[25]

ADIME 11.3 ADIME At-A-Glance: Cirrhosis and Hepatic Encephalopathy

Assessment

- Low BMI, UWL, anorexia/early satiety, decrease in appetite and food intake
- Abdominal pain, fatigue, nausea and vomiting, and steatorrhea
- Ascites, edema, jaundice, and GI bleeding (especially esophageal varices)
- Alcohol, hepatotoxic drugs, diabetes, obesity, insulin resistance, dyslipidemia, jejunal ileal bypass, and long-term TPN
- CT scans, abdominal ultrasound, ERCP, and cholangiogram
- LFTs (especially ammonia), electrolytes, glucose, hepatic proteins, hgb/hct, chol, and vitamins/minerals

Nutrition Diagnosis

- Inadequate oral intake RT decreased appetite from abdominal pain, AEB recent UWL of 5% × 2 weeks, and PO intake of <50%

Intervention

- Provision of higher-protein diet (1.2–1.5 g/kg, as tolerated); BCAA protein sources versus AAA
- Oral BCAA supplementation, if protein not tolerated
- If steatorrhea, MCT
- CHO to prevent hypoglycemia; if diabetes, CHO management
- Small, frequent feedings

- Restrict Na 2 to 3 g, if edema, ascites; possible fluid restricted if ascites (1.0–1.5 L/d), depending on use of diuretic
- If esophageal varices, soft, and low fiber
- Provision of supplement of B-complex, fat-soluble vitamins
- Avoidance of raw shellfish, alcohol
- Adequate fluids

Monitoring and Evaluation

- Body weight, dry weight
- PO intake (food, fluid)
- Laboratory values
- Control of GI symptoms and other symptoms
- Prevention of neurologic symptoms (HE)

Gallbladder Disease

Disease Process

The presence of gallstones is cholelithiasis, and problems with the organ generally stem from this common condition, affecting over 33 million in the United States and 20% of the population over age 65.[36] Relatively static conditions, such as in pregnancy and total parenteral support (TPN), give rise to biliary sludge, which promotes crystals to form and later become gall stones. Constituents of the stones include bilirubin, cholesterol, calcium salts, lipid, protein, and other materials. Of the various types of gallstones, cholesterol stones are the most common accounting for 85%. Although 80% of people with gallstones are asymptomatic, serious complications include cholecystitis (inflammation of the organ), pancreatitis, and cancers of the gallbladder and liver. In addition, gallbladder disease significantly increases the risk for developing type 2 diabetes.[37]

Risk factors for the development of gallstones include being female, obese, Mexican American, Native American, the use of estrogen and oral contraceptives, and long-term TPN.[38] In addition, several diseases and conditions increase the risk, including diabetes, metabolic syndrome, hypothyroidism,[39] intestinal diseases causing ileal dysfunction such as Crohn's disease, cystic fibrosis, sickle cell disease, bariatric surgery, rapid weight loss, and pregnancy due to higher levels of progesterone reducing contractility. Genetic susceptibility confers an approximately 25% higher risk with several identified gene candidates.[40] More recently, researchers have shown that the gut microbiota influences stone formation via effects on bile acid and cholesterol metabolism.[41] Epidemiologic studies have implicated low-fiber diets high in energy and refined

carbohydrates. In contrast, higher intakes of vitamin C, coffee, and calcium appear to be protective.[42,43]

The disease becomes symptomatic with either stone impaction in the cystic duct or inflammation. The former causes either epigastric or right upper quadrant (RUQ) pain of varying intensity with nausea and vomiting, often after a high-fat meal.[38] Inflammation causes similar but more severe symptoms, sometimes accompanied by fever and leukocytosis, often requiring hospitalization.

Treatment and Nutrition Intervention

Laparoscopic cholecystectomy is generally recommended for patients with symptomatic cholelithiasis who are suitable candidates.[40] Some patients may be unwilling to undergo surgery, and in these patients, nutrition recommendations include following a diet low in fat (<30%) with adequate fiber and vitamin C, in small frequent feedings, and reduced energy, if weight loss is indicated.[11,44] In acute cholecystitis requiring hospitalization, nutrition intervention includes NPO status with progression to a low-fat diet. Post-surgical cholecystectomypatients should follow a clear liquid diet immediately after surgery, and gradually progress to a low-fat diet in smaller more frequent meals. Most patients can resume a regular diet, but some patients (5–40%) experience bloating, abdominal pain and diarrhea due to the laxative effect of an increased amount of bile in the large intestine and may require a slower progression to their regular diet.[46]

Pancreatic Disease

Diseases of the pancreas can significantly impact nutritional status, given the dual role of the organ, that is, exocrine and endocrine. Dysfunction related to the former role will affect digestion and absorption of nutrients, potentially resulting in malnutrition, whereas dysfunctions of the latter cause diabetes mellitus. Because of the direct malabsorption that occurs with failure of the exocrine pancreas, nutritional assessment and intervention are critical in preventing malnutrition.

Acute Pancreatitis

Disease Process

In the United States, the incidence of acute pancreatitis (AP), a sudden inflammation of the pancreas, is up to 50 events per 100,000 adults.[47] AP can lead to chronic pancreatitis (CP) and is also raises the risk for pancreatic cancer.[48] The higher risk for pancreatic cancer is probably due to the acute inflammation itself, and the risk increases concomitantly with recurrent episodes. In addition, CP also increases the risk for pancreatic cancer and the risk is additive with that of AP. Symptoms are similar for AP and CP and include pain of varying intensity in the upper abdomen radiating to the back, nausea, vomiting, and steatorrhea. In AP, serum levels of pancreatic enzymes, lipase and amylase, are elevated; however, in CP, they may be close to normal because of atrophy of the pancreatic parenchyma, leading to fibrosis in advanced disease.[49]

The most common cause of AP is the presence of gallstones in the common bile duct, followed by alcohol abuse, which together account for 80% of cases.[50] Although

most cases of AP in patients abusing alcohol occur in those who have been chronic users for 5–10 years, AP can sometimes occur after weekend binge drinking and from one excessive dose that causes an initial attack.[47] Additional causes of AP include genetics, obstruction of the pancreatic duct, which can arise from narrowing of the duct or pancreatic cancer, hypercalcemia, hypertriglyceridemia (>1000 mg/dL), abdominal trauma or surgery, various medications and infections (especially in children) (**Box 11.5**),[47,49] and cigarette smoking.[51] About 5% of cases are caused by endoscopic retrograde cholangiopancreatography (ERCP).[47] More recently, cannabis use has been linked to an increased risk for AP.[52]

Box 11.5 Medications and Microbes Causing Acute Pancreatitis

Medications	Infections
5-aminosalicylic acid compounds	*Ascaris*
6-Mercaptopurine	Candida
Azathioprine Corticosteroids Didanosine Dideoxyinosine	Coxsackie B virus
Estrogens	Cytomegalovirus (CMV)
Furosemide	Escherichia coli
Methyldopa Octreotide	*Epstein–Barr virus*
Pentamidine Sulfasalazine Sulfonamides	HIV

Medications	Infections
Sulindac	*Legionella*
Tetracycline	*Leptospirosis*
Thiazides	Mumps
Trimethoprim–sulfamethoxazole	*Salmonella*
Valproic acid	*Shigella*

Treatment and Nutrition Intervention

Medical nutrition therapy for AP depends on the severity of the patient's condition (**Box 11.6**).[11] In mild to moderate cases, a low-fat oral diet can begin immediately in the absence of nausea and vomiting and once pain has subsided. In severe cases, nasogastric or nasojejunal delivery of enteral is the preferred nutrition therapy, which will help prevent complications of infections. If enteral feeding is not possible, or if nutrient needs cannot be achieved by this method, parenteral nutrition may be necessary. If inflammation is extensive, protein and energy needs are high because of catabolism. Patients with fat malabsorption should receive fat-soluble vitamin supplementation. Alcohol should be avoided by all patients, and in those with a history of alcohol abuse, daily supplemental thiamin (100 mg), folate (1 mg), and general multivitamin are indicated.

Box 11.6 Dietary Recommendations for Acute Pancreatitis

Feeding Progression

Severe AP: Early enteral nutrition within 24–48 hours of admission is preferable mild to moderate AP: Early oral feeding within 24–48 hours as tolerated (when patient is not in pain and not experiencing nausea or vomiting)

General

Protein: 1.2–1.5 g/kg of body weight per day; if severe AP: 2.0 g/kg of body weight
Fat: Low-fat only in AP patients experiencing GI symptoms
Multivitamin mineral supplement pancreatic replacement therapy (PERT): Evaluate need every 6 months after AP for at least 18 months, and more frequently in cases of significant malabsorption
Abstinence from alcohol

ADIME 11.4 ADIME At-A-Glance: Acute Pancreatitis

Assessment

- Low BMI, UWL, decrease in appetite and food intake
- Abdominal pain, nausea and vomiting, and steatorrhea
- Hypovolemia
- Alcohol abuse
- Laboratory: amylase, lipase, CRP, glucose, electrolytes, and lipids

Nutrition Diagnosis

- Inadequate oral intake RT decreased appetite from abdominal pain, AEB recent UWL of 5% × 2 weeks, and PO intake of <50%

Intervention

- Progress if mild, small, frequent, oral, low-fat feedings; PERT with each meal

- Mild to moderate: NGT or nasojejunal formula containing peptides and MCT
- If unable to feed enterally: parenteral nutrition
- In extensive inflammation, protein, and energy needs higher
- If alcohol abuse, supplement thiamin (100 mg), folate (1 mg), general multivitamin
- Abstinence from alcohol, caffeine (GI stimulant), smoking

Monitoring and Evaluation

- Body weight, dry weight
- PO intake (food, fluid)
- Laboratory values
- Control of GI symptoms and other symptoms

Chronic Pancreatitis

Disease Process

CP involves inflammation, which, after resolution, results in irreversible fibrosis and calcification of pancreatic tissue.[53] Some CP patients will have episodes of AP, and as the disease progresses, acinar cells are destroyed and nutrient malabsorption occurs, causing steatorrhea, which can lead to malnutrition. Islet cells are destroyed with disease progression, producing diabetes in up to 70% of patients, and classified as pancreatogenic diabetes or type 3c diabetes mellitus.[54] Symptoms are similar to those of AP with severe abdominal pain, which subsides over time as the disease worsens; however, some patients have no pain.[53]

The most common cause of CP is heavy alcohol ingestion over several years, which accounts for 50% of cases.[53] As with alcohol, other causes and risk factors are similar to those of AP and include genetic pancreatic diseases (cystic fibrosis is the most common), gallbladder disease, hypercalcemia, hypertriglyceridemia, hyperparathyroidism, organ trauma, cigarette smoking, certain medications, and autoimmune diseases. CP can also occur after an episode of AP in which the pancreatic duct is damaged, causing inflammation of the organ and the development of scar tissue.

Treatment and Nutrition Intervention

Abstinence from alcohol and tobacco is an important aspect of intervention to remove the underlying disease cause, in the case of alcoholic CP, and to prevent further damage to the pancreas, as both appear to have both direct and indirect toxic effects on the organ.[55] In CP, eating may exacerbate the chronic abdominal pain, which can reduce food intake and leading to further weight loss, which mainly arises from nutrient malabsorption, particularly fat. For all of these reasons, nutrient deficiencies and protein energy malnutrition are common, particularly in alcoholic CP. Goals for nutritional intervention include repletion of nutritional status and reduction of malabsorption. Pancreatic replacement enzymes are important therapeutic agents to achieve these goals (**Box 11.7**).

Box 11.7 Dietary Recommendations for Chronic Pancreatitis

Protein: 1.0 g/kg body weight, up to 2.0 g/kg for repletion
Small, frequent meals (4–8)
PERT with each meal
Fat variable, based on tolerance; MCT, if steatorrhea severe
Adequate diet or supplement: calcium, magnesium, fat-soluble vitamins, B-complex vitamins, vitamin C, zinc

Abstinence from alcohol and tobacco

Fat restriction is not usually necessary, except in cases of refractory steatorrhea.[11] In addition, MCT may be helpful if steatorrhea cannot be controlled.[53] For most patients, protein should be 1.0–1.5 g/kg body weight. However, protein needs may be as high as 2.0 g/kg in a severely compromised patient who has had significant weight loss. Small, frequent meals (four to eight) are better tolerated and may promote higher absorption, particularly of dietary fat. Supplementation with fat-soluble vitamins and vitamin B_{12} and possibly other micronutrients is often warranted because of chronic nutrient malabsorption in general and fat in particular. Some patients may require enteral nutrition, if they are unable to consume adequate energy through dietary intake. This may include nocturnal feedings, depending on the patient's nutritional status and tolerance.

ADIME 11.5 ADIME At-A-Glance: Chronic Pancreatitis

Assessment

- Low BMI, UWL, decrease in appetite and food intake
- Abdominal pain, nausea and vomiting, and steatorrhea
- Hypovolemia
- Alcohol abuse
- Laboratory: amylase, lipase, CRP, glucose, electrolytes, and lipids

Nutrition Diagnosis

- Impaired nutrient utilization RT to Dx and nonadherence to PER with meals, AEB steatorrhea and

recent of 5% × 2 weeks

Intervention

- Small, frequent meals (4–8)
- PERT with each meal
- Fat variable, based on tolerance; MCT, if steatorrhea severe
- Adequate diet or supplement: calcium, magnesium, fat-soluble vitamins, B-complex vitamins, vitamin C, and zinc
- Abstinence from alcohol, smoking

Monitoring and Evaluation

- Body weight
- PO intake
- Laboratory values
- Control of GI symptoms and other symptoms

Pancreaticoduodenectomy (Whipple Procedure)

Procedure and Nutrition Intervention

Pancreatic cancer is the fourth leading cause of cancer in the United States.[56] It accounts for 3% of all cancer cases and 7% of cancer mortality, and the incidence and mortality are increasing. The disease may necessitate a pancreaticoduodenectomy, or Whipple procedure (WP), which involves removal of the head of the pancreas, distal bile duct, gallbladder, duodenum, first few centimeters of jejunum, and in some cases, up to half of the distal stomach, and pylorus.[57] Numerous nutrition problems typically arise, including gastroparesis, dumping syndrome, diabetes, malabsorption, lactose intolerance, weight loss,

and malnutrition.[11] Specific nutrient deficiencies that can develop include vitamins A, D, E, K, and B_{12}; and minerals: iron, calcium, zinc, copper, and selenium.

Nutrition intervention targets all of the potential nutrition problems. The specific dietary measures include small, frequent meals (five to six), limited fluid at meals (up to 5 oz) and drinking fluids up to 40 minutes after a meal, avoidance of simple sugars and sugar alcohols, inclusion of protein at every meal, and limiting fat to less than 30% of total calories. In addition, if lactose intolerance occurs, the patient should be instructed to avoid lactose and use specialized products in which lactose has been prehydrolyzed. The patient should also be encouraged to eat slowly and chew all foods thoroughly. A vitamin and mineral supplement is also an important part of nutrition intervention with close monitoring for weight loss and possible malnutrition.

Exocrine Pancreatic Insufficiency

Disease Process

Exocrine pancreatic insufficiency (EPI) occurs when the pancreatic enzymes (amylase, protease, and lipase) are deficient, giving rise to maldigestion.[58] The underlying pathological cause may be due to the pancreas, most commonly CP, pancreatic malignancies, and especially resulting in the WP, diabetes (both types), cystic fibrosis, or outside the pancreas including celiac disease, IBD, autoimmune pancreatitis, and Zollinger-Ellison syndrome.

Symptoms may be overt, such as steatorrhea and weight loss caused by fat malabsorption, or generalized gastrointestinal problems similar to other gastrointestinal diseases, such as diarrhea, gas and bloating, and abdominal pain. Up to 90% of patients with CP will have EPI,[59] however, 33% of CP patients are affected by SIBO[60] without EPI, which

may cause symptoms similar to EPI. Severe EPI is associated with higher mortality in patients with chronic pancreatitis, but even less severe cases can adversely affect nutritional status and lead to malnutrition.

Treatment and Nutrition Intervention

The potential for EPI to adversely affect nutrition status, as with CP and the WP, makes a thorough nutrition assessment and monitoring crucial. The high occurrence of EPI in CP patients necessitates careful monitoring of nutrition assessment parameters and possible nutritional deficiencies.[59] The mainstay of treatment is the use of PERT, with similar nutritional interventions as with CP and WP. These nutrition interventions include moderate fat intake, vitamin supplementation, particularly fat-soluble vitamins A, D, E, K, as well as avoidance of alcohol and smoking.[11,61]

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CHAPTER 12

Kidney Disease

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The majority of kidney cases requiring nutrition intervention include patients hospitalized with acute kidney injury, those living with progressive chronic kidney disease (CKD), and those with end-stage kidney disease (ESKD). The United States Renal Data System (USRDS 2022) reports on kidney disease epidemiology.[1] Their data spanning 2017 up to March 2020 due to the COVID-19 pandemic indicated 14.0% of adults in the United States had low eGFR (<60 mL/min/1.73 m^2) or albuminuria (urinary ACR ≥30 mg/g) or both using the Kidney Disease: Improving Global Outcomes (KDIGO) criteria,[2] which approximates 31.2 million individuals with CKD and a 19.1% increase since 2005–2008.[1] However, in this survey period, a higher incidence of COVID-19 and higher mortality after diagnosis of COVID-19 among patients with CKD and ESKD meant reduced prevalence rates of diagnosis in 2020.

CKD prevalence during the last pre-pandemic year (2019 to March 2020) was highest amongst Black individuals (6437 per million population [pmp]) which was approximately twice that of Hispanic individuals (3474 pmp), three times that of Asian individuals (2361 pmp), and greater than four times that of White individuals (1504 pmp).[1] The overall prevalence of CKD was also higher among individuals with lower income (16.5% below the poverty line vs. 14.0% above) and among those with lower education (20.4% with less than a high school education vs. 12.0% with at least some college).[1]

Acute Kidney Injury

Acute kidney injury (AKI) represents an abrupt reduction in glomerular filtration rate (GFR), resulting in the reduced ability of the kidney to excrete metabolic wastes with dysregulation of fluid, electrolyte, and acid-base homeostasis.[3] It often results from an illness, surgery, severe trauma, or rapidly progressive, intrinsic renal disease.[4] But the highly infectious COVID-19 disease became a significant cause of AKI by implicating multiorgan disruption. Although the overall rate of hospitalization with AKI decreased in 2020, arising from reduced hospital admissions due to the COVID pandemic, AKI events worsened as indicated by a 16% increase in the need for dialysis among those with AKI in 2020 compared to 2019.[1] Mortality outcomes after AKI requiring dialysis (AKI-D) during a COVID-19 hospitalization in 2020–2021 were substantially worse than outcomes after AKI-D without COVID-19 (74.1% vs. 35.2%, respectively).[1]

Although AKI is reversible in many cases if diagnosed and treated early, it is increasingly recognized as a risk factor for the development of CKD. There is a significantly increased long-term risk of CKD and ESKD for many AKI survivors, even after initial recovery of kidney function. Of hospital survivors of AKI without pre-existing CKD post-discharge, CKD was diagnosed in 11.6% within 3 months, which progressively increased to 20.7% by 24 months.[1] This patient group also faced a high mortality rate, with 36.7% having died by 24 months. Severe AKI occurs in >5% of critically ill patients and is associated with 40–70% mortality rates.[3]

Disease Process

AKI is suspected when there is a rapid rise in serum creatinine (SCr) associated with oliguria.[5] The causes of AKI are varied and can be divided into three general categories:

- As an adaptive response to severe volume depletion and hypotension, with the kidney's structure and function intact (prerenal)
- In response to inflammatory, ischemic, or cytotoxic insults, which affect both kidney structure and function (intrinsic or intrarenal)
- When the passage of urine is blocked (postrenal)

The majority of AKI cases arise from prerenal causes and are multifactorial, particularly in the critically ill setting.[5,6] A listing of the causes of AKI in each category can be found in **Table 12.1**.

TABLE 12.1 Causes of Acute Kidney Injury

AKI Category	Causes
Prerenal (decreased blood flow)	• Hypotension from any cause • Congestive heart failure • Systemic vasodilation (e.g., sepsis and neurogenic shock) • Volume depletion • Renal loss from diuretic overuse, osmotic diuresis (e.g., diabetic ketoacidosis) • Extrarenal loss from vomiting, diarrhea, burns, excessive sweating, blood loss • Hypercalcemia
Intrinsic renal or intrarenal	• Ischemia • Sepsis or direct infections • Atheroembolism or thromboembolism • Thrombotic microangiopathy • Systemic necrotizing vasculitides

AKI Category	Causes
	• Hypoperfusion from systemic hypotension • Toxins • Endogenous (e.g., hemolysis, rhabdomyolysis, tumor lysis syndrome, and myeloma) • Exogenous (drugs, radiographic contrast agents, bacteria, fungi, and viruses) • Acute glomerular or tubulointerstitial nephritis • Tumor • Stones • Scar tissue formation • Tissue inflammation
Postrenal (obstruction)	• Stones • Tumor • Prostate hypertrophy • Neurogenic bladder • Bladder, prostate, or cervical cancer

Data from (i) Gilbert S, Weiner DE, Bomback AS, Perazella MA, Rifkin DE. *National Kidney Foundation Primer on Kidney Diseases*. 8th ed. 688pp. Philadelphia, PA: Elsevier, 2022, and (ii) Moore PK, Hsu RK, Liu KD. Management of acute kidney injury: core curriculum 2018. *Am J Kidney Dis*. 2018;72(1):136-148.

The clinical diagnosis of AKI is based on changes in SCr and/or urine output as surrogates for changes in GFR. A system for the classification of AKI has been established by the International Work Group of the KDIGO initiative in order to define short- and long-term changes to the excretory outputs of the kidney (see **Table 12.2**).[3,7,8] The three stages categorize the magnitude of the increase in SCr and/or the duration of oliguria, in increasing severity. However, the definition of AKI should be within the clinical context of the individual patient, as there are still limitations to using the SCr and urine output.[6,8]

TABLE 12.2 Classification of Acute Kidney Injury Severity

Stage	Serum Creatinine Criteria[a]	Urine Output Criteria
I	Increased 1.5–1.9 times baseline OR > 0.3 mg/dL increase within 48 h	<0.5 mL/kg/h for >6 h
II	Increased 2.0–2.9 times baseline	<0.5 mL/kg/h for >12 h
III	Increased >3.0 times baseline OR a value of ≥4 mg/dL OR need for KRT > 4 weeks	<0.3 mL/kg/h for >12 h or anuria for >12 h

[a]Increase in serum creatinine should be both abrupt (within 1–7 d) and sustained (>24 h).
Data from (i) Moore PK, Hsu RK, Liu KD. Management of acute kidney injury: core curriculum 2018. *Am J Kidney Dis*. 2018;72(1):136-148, and (ii) Marlies O, Joannidis M. Acute Kidney Injury 2016: Diagnosis Workup. *Crit Care*. 2016;20(1):299.

Treatment and Nutrition Intervention

The AKI patient typically presents with metabolic derangements resulting from both the uremic burden and inflammatory processes.[6] Therefore, metabolic acidosis, hyperkalemia, and protein catabolism need to be assessed. Stress hyperglycemia with peripheral insulin resistance is a distinctive clinical feature of critical illness. Short-term dialysis may be necessary to treat AKI, with the goal of preventing renal damage and returning the kidneys to baseline function. Medications may include the following:

- Intravenous (IV) sodium chloride to correct dehydration.
- Diuretics, such as furosemide, to correct fluid overload.
- Bicarbonate to correct severe acidosis.

- Potassium-exchange resins to bind potassium in the gut to correct hyperkalemia.
- Insulin to correct hyperglycemia.

Specific nutritional goals are based on whether or not dialysis is planned to reduce the uremic burden, and the presence of multiple organ complications. Careful daily monitoring of the metabolic status of the AKI patient is necessary, and nutritional goals should be determined based on these findings as outlined in **Table 12.3**.[9,10]

TABLE 12.3 Basis for Setting Nutritional Goals in Acute Kidney Injury

Nutritional Specifics	Requirements	Basis
Energy	• Calculation should be based on usual or ideal body weight. • Assess calories from enteral or parenteral feeding and choice of dialysis modality.	• Actual body weight is affected by water balance. • Energy expenditure rarely exceeds 1.3 times basal energy expenditure. • 27 kcal/kg/d is optimal in critically ill patients with AKI. • Refeeding syndrome risk with overfeeding.
Protein	• At least 1.5 g of protein per kg BW/d[11] required to achieve a less negative or nearly positive nitrogen balance.	• Protein catabolic rate values are 1.4–1.8 g/kg/d in patients with AKI on dialysis. • Amino acid losses increased during dialysis.

Nutritional Specifics	Requirements	Basis
		• Excessively higher nitrogen intakes increase urea production.
Trace elements	• Daily supplementation with standard doses of multitrace element (copper, zinc, selenium, magnesium, chromium, and/or iron) preparations recommended.	• Serum levels low due to acute phase of illness. • Losses also occur with dialysis.
Water-soluble vitamins	• Vitamin C requirement is 50–100 mg/d but increased (up to 150–200 mg/d) with continuous modalities of dialysis. • Thiamine supplementation is also required if the patient is on standard parenteral feeding.	• Serum levels low due to acute phase of illness. • Losses also occur with dialysis.
Fluid	• Restriction during oliguric or anuric phases of AKI.	• Hypervolemia during acute phase of AKI
Delivery	• Enteral route is the preferred choice. • Concentrated enteral formula required (1.5–2.0 kcal/mL). • Parenteral nutrition may supplement enteral feeding.	• Volume restriction requires use of concentrated formula. • Achieving prespecified nutritional targets may justify combining enteral and parenteral feeding.

Data from (i) Fiaccadori E, Maggiore U, Cabassi A, Morabito S, Castellano G, Regolisti G. Nutritional evaluation and management of AKI patients. *J Ren Nutr*. 2013;23(3):255-258, and (ii) Meyer D, Mohan A, Subev E, Sarav M,

Nutritional Specifics	Requirements	Basis

Sturgill D. Acute kidney injury incidence in hospitalized patients and implications for nutrition support. *Nutr Clin Prac*. 2020;35(6):987-1000.

Table 12.4 lists the nutritional recommendations of both ASPEN and KDIGO, and monitoring parameters for patients with AKI.

TABLE 12.4 Nutrition Intervention and Monitoring in Acute Kidney Injury

	Recommended Amounts		
Nutrient Rationale	**ASPEN**	**KDIGO**	**Monitor**
Protein ■ Avoid net negative nitrogen balance associated with hypercatabolism ■ Sufficient to reduce serum urea and improve urinary output[12] ■ Sufficient to maintain somatic and visceral protein status ■ Replace protein losses from KRT ■ Correct hyperkalemia	1.2–2.0 g/kg/d On hemodialysis or CRRT: additional 0.2 g/kg/d up to 2.5 g/kg/d	Not on dialysis: 0.8–1.0 g/kg/d On HD or PD: 1.0–1.5 g/kg/d On CRRT: up to 1.7 g/kg/d	BUN Serum creatinine Serum chloride Urine output GI tolerance
Energy ■ Sufficient to spare protein for synthesis ■ Sufficient to prevent tissue protein catabolism ■ Titrate for stress-induced hyperglycemia	25–30 kcal/kg/d (use usual body weight for normal weight patients; use ideal body weight for obese or critically ill) Indirect calorimetry is preferred	20–30 kcal/kg/d in patients with any stage of AKI Indirect calorimetry is preferred	Blood glucose Insulin dose
Electrolytes ■ Sodium—restriction during oliguric phase ■ Potassium—hyperkalemia from tissue catabolism; control to prevent cardiac arrhythmias	Sodium—20–40 mEq/d (2–3 g) during oliguric phase Potassium—30–50 mEq/d during oliguric phase Phosphorus—limit as needed Restriction not required on dialysis	Not stated	Blood levels of sodium, potassium, and phosphorus
Fluid ■ Control in early oliguric phase ■ Net intake must balance output ■ Maintain normal blood pressure	500 mL + previous days output Restriction not required on dialysis	Not stated	I/O charts Weight change Blood pressure IVs Parenteral or enteral fluid volumes

ASPEN, American Society for Parenteral and Enteral Nutrition; BUN, blood urea nitrogen; CRRT, continuous renal replacement therapy; GI, gastrointestinal; I/O, in-out charting; IV, intravenous.

Data from (i) Kellum JA, Lameire N, Aspelin P, et al. Kidney disease: improving global outcomes (KDIGO) acute kidney injury work group. KDIGO clinical practice guideline for acute kidney injury. *Kidney Intl Suppl.* 2012;2(1):1-138, (ii) Palevsky PM, Liu KD, Brophy PD, et al. KDOQI US commentary on the 2012 KDIGO clinical practice guideline for acute kidney injury. *Am J Kidney Dis.* 2013;61(5):649-672, and (iii) McClave SA, et al. Guidelines for the provision and assessment of nutrition support therapy in the adult critically ill patient: Society of Critical Care Medicine and American Society for Parenteral and Enteral Nutrition. *JPEN J Parenter Enteral Nutr.* 2016;40(2):159-211.

Description

ADIME 12.1 ADIME At-A-Glance: Acute Kidney Injury

Assessment

- Ht, wt, BMI, wt changes, dry wt, SBW
- Labs: blood pressure, glycemia, serum creatinine, BUN, GFR, albumin, electrolytes, calcium, phosphorus, inflammatory markers (CRP), acid–base balance (pH, CO_2, chloride), I&Os
- Food history/logs, vitamin/mineral/supplement use, appetite status
- NFPE: presence of edema, muscle wasting, subcutaneous fat loss

Nutrition Diagnosis

- Inadequate protein energy intake RT AKI (stress hypermetabolism/catabolism), initiation of CRRT, and decreased ability to consume sufficient protein/energy AEB unintentional weight loss of 1.8% in 1 week, intake <25% × 3 days, and new NPO status

Intervention

- Initiate enteral nutrition Nepro at 20 mL/h via NG tube and increase by 10 mL/h every 8 hours to goal rate of 45 mL/h to provide 1,944 kcal, 87 g protein, 785 mL free water
- Request metabolic cart study

Monitoring and Evaluation

- Goal rate for EN reached by day 2.

- Reassess energy needs based on metabolic cart study.

Chronic Kidney Disease

CKD represents a gradual and progressive loss of kidney function, which is irreversible. The GFR, as a measure of kidney function, decreases proportionately from stages 1–5 CKD in tandem with acidosis, anemia, bone disorders, and uremic symptoms (see **Box 12.1**).[5] The progression in GFR decline (termed *renal insufficiency*) is inversely proportional to SCr levels.[5] Initially, as renal tissue loses function, there are few signs or symptoms because the remaining tissue increases its performance (renal adaptation). With a GFR falling to 50% of normal, up to 75% of renal tissue function may already be lost.[13] The principal causes leading to CKD are diabetic nephropathy and hypertension, which together account for up to two-thirds of all cases.[1] It is also noted that in the United States, approximately 11.6% of post-AKI discharges develop CKD within 3 months, with the overall rate doubling by the end of 2 years.[1]

Box 12.1 Definition and Classification of Chronic Kidney Disease

Definition of CKD Based on the NKF-KDOQI Guidelines:

1. Kidney damage for ≥3 months, with or without decreased kidney function. Kidney damage is defined as pathologic abnormalities or markers of damage, including abnormalities in blood, in urine test, or on diagnostic imaging studies.
2. Decreased kidney function measured by GFR <60 mL/min/1.73 m^2 for ≥3 months, with or without kidney damage.

Stage	Description	GFR[b]	Symptoms	Action
Pre-CKD	At increased risk	>90 with CKD risk factors		■ Screening for CKD ■ CKD risk reduction
1	Kidney damage with normal or increased GFR	≥90	Albuminuria, proteinuria, hematuria	■ Diagnosis and treatment ■ Treatment of comorbid conditions ■ Slowing progression ■ CVD risk reduction
2	Kidney damage with mild decrease in GFR	60–89	Albuminuria, proteinuria, hematuria	■ Estimating progression
3[a]	Moderate decrease in GFR	30–59	Early to chronic renal insufficiency	■ Evaluating and treating complications ■ Start nutrition intervention
4	Severe decrease in GFR	15–29	Chronic renal insufficiency, late renal insufficiency, pre-ESKD	■ Preparation for RRT ■ Nutrition counseling
5	Kidney failure	<15	Renal failure, uremia, ESKD	■ RRT if uremia is present

CVD, cardiovascular disease; RRT, renal replacement therapy.

[a]KDIGO guidelines[2] established in 2012 further divide stage 3 CKD into the following categories, with concurrence from NKF-KDOQI:

3a (GFR 45–59): mild to moderate decrease in GFR or early renal insufficiency.

3b (GFR 30–44): moderate to severe decrease in GFR or chronic renal insufficiency.

[b]Glomerular filtration rate (mL/min/1.72 m^2).

Data from Inker LA, Astor BC, Fox CH, et al. KDOQI U.S. commentary on the 2012 KDIGO clinical practice guidelines for the evaluation and management of CKD. *Am J Kidney Dis.* 2014;63(5):P713-735.

Description

The staging of CKD differentiates clinical treatment goals and priorities[2,14] and also provides the rationale for medical nutrition therapy, which differs with progression status and kidney replacement therapy (KRT) options provided to survive ESKD.

Individuals at stage 3 CKD comprise the largest percentage of CKD patients in the United States.[1] The demographics of CKD progression for a cohort at stage 3 CKD in 2015 followed up to 2020 (see **Box 12.2**) therefore

affords an understanding on the priorities for dietitian intervention.[1]

Box 12.2 Demographics of Chronic Kidney Disease Progression from Stage 3 for an USRDS 2015 Cohort

- Only 31.6% remained at stage 3 while 10.4% progressed to stage 4, 2.5% progressed to stage 5, 42.9% died before ever reaching ESKD, and only 2.5% progressed to ESKD.
- Individuals with stage 4 CKD were over 6 times as likely to progress to ESKD (16.1%).
- Those at stage 5 CKD were over 8 times as likely (20.9%) to progress to ESKD, as those with stage 3 CKD (2.5%).
- At stage 4 CKD, younger individuals (66–69 years) were more likely to progress to ESKD than those who were older (66–74 years).
- Although Black individuals had a higher incidence of CKD progression and advancement to ESKD for all stages of CKD, they were less likely to die over the ensuing year than White individuals, regardless of CKD stage.

CKD: Nondialysis

Disease Process

At the nondialysis (ND) stage of CKD, also referred to as CKD ND, preservation of kidney function for as long as possible is a priority.[15] Early renal insufficiency is characterized by increasing blood urea nitrogen (BUN) and SCr concentrations and decreasing creatinine clearance.[5] As the GFR declines, the progressive loss of functioning nephrons impairs a range of physiologic functions in the CKD patient relative to acid–base balance, potassium and phosphate excretion, vitamin D metabolism, increased renin production, impaired erythropoietin production, and retention of protein metabolites.[5]

The development of the uremic syndrome quantitatively and qualitatively characterizes the deterioration of cardiovascular, neurologic, hematologic, immunologic, and

other systems.[5] Increased catabolism leads to malnutrition and acidosis, which further increases the uremic burden. By stages 4 and 5 of CKD, full-blown uremia is a complex clinical picture comprising fatigue, anorexia, weight loss, itching, muscle cramps, pericarditis, and sensory and cognitive disturbances.[5,16]

Treatment and Nutrition Intervention

Laboratory values should be monitored at least monthly and reviewed with the patient to determine any necessary therapeutic interventions (see **Table 12.5**). Some values may be monitored more frequently, such as hemoglobin, hematocrit, calcium, and phosphorus. These lab values can be affected by medications or acute medical conditions, and in both cases, more frequent monitoring will significantly improve patient outcomes. Some biochemical parameters, such as lipid profile, are tested less frequently, generally on a quarterly basis.

TABLE 12.5 Commonly Monitored Laboratory Values in Kidney Disease

Test	Normal Chronic Kidney Disease Range
Albumin	WNL for laboratory >4.0 g/dL is ideal
Blood urea nitrogen	<20 mg/dL; 60–80 mg/dL (dialysis)
Calcium–phosphorus product	<55 mg/dL
Corrected calcium[a]	8.5–10.2 mg/dL
Chloride	WNL

Test	Normal Chronic Kidney Disease Range
Bicarbonate (CO_2), total	≥ 22 mEq/L
Cholesterol	WNL
Creatinine	0.8–1.4 mg/dL; 2–15 mg/dL
Ferritin	≥100 ng/mL for PD/CKD 200–500 ng/mL for HD
Glucose	WNL ≤200 non-fasting
Hematocrit	33–36% (dialysis)
Hemoglobin	•9–11 g/dL
Phosphorus, inorganic (serum)	3.5–5.5 mg/dL (dialysis); 2.7–4.6 mg/dL (nondialysis/stages 3–4)
Potassium (serum)	3.5–5.0 mEq/L
Parathyroid hormone, intact	150–300 pg/mL (stage 5); 70–110 (stage 4); 35–70 (stage 3)
Sodium	WNL

[a]*Corrected* Ca (mg/dL) = serum calcium (mg/dL) + 0.8 (4.0 − serum albumin (g/dL)).
WNL, within normal limits.
Data from (i) National Kidney Disease Education Program (NKDEP). Making Sense of CKD- A concise guide for managing chronic kidney disease in the primary care setting. July 2014, NIH Publication No.14-7989, and (ii) McCann L. *Pocket Guide to Nutrition Assessment of the Patient with Kidney Disease*. 6th ed. CRN-NKF, National Kidney Foundation Inc., New York, NY, 2021.

Primary goals in the treatment of CKD include the preservation of kidney function and maintaining adequate nutritional status, thus delaying the need for dialysis as long

as possible. The NKF has issued national guidelines, KDOQI, which provide evidence-based clinical practice guidelines for all stages of CKD.[2] These guidelines strongly recommend nutrition evaluation and monitoring as early as stage 2 or 3. To prevent further decline of kidney function, the cornerstone of treatment is intensive control of diabetes and hypertension, if present. Nutritional intervention benefit control of uremic symptoms such as anorexia, diarrhea, and vomiting that frequently accompany the decline in GFR (see **Table 12.6**). Preventing malnutrition in the predialysis patient is of critical importance to overall patient outcome.[5] In planning diets for patients, the rationale for multiple nutrient recommendations must be considered and adjusted to patient needs accordingly:

TABLE 12.6 KDOQI Nutrition Guidelines for CKD ND (Stages 3–5 CKD)

Nutrients	Stages 3–5 CKD
Protein (g/kg BW/d)	0.55–0.60 as calculated for a normal diet. OR 0.28–0.43 with a very low protein diet supplemented with keto acids/amino acid analogs to meet 0.55–0.60 goal. For a patient with diabetes: 0.6–0.8 as calculated for a normal diet.
Energy (kcal/kg/d)	25–35 based on age, sex, activity level, body composition, weight status goals, and concurrent illness.
Phosphorus (mg/d)	Based on laboratory values. Adjust dietary phosphate intake when serum phosphorus levels are elevated above target range of the CKD stage. Consider bioavailability of phosphorus (animal, vegetable, or additives) when assessing the patient's diet.
Sodium (mg/d)	<2,300 based on labs and blood pressure

Nutrients	Stages 3–5 CKD
Potassium (g/d)	Adjust per laboratory values
Calcium (mg/d)	800–1,000; maintain serum calcium WNL
Fluid (mL/d)	Unrestricted with normal urine output May need restriction with CHF

WNL, within normal limits; CHF, congestive heart failure.
Data from Ikizler TA, Burrowes JD, Byham-Gray LD, et al. KDOQI clinical practice guideline for nutrition in CKD: update 2020. *Am J Kidney Dis*. 2020;76(3):S1-S107.

- **Energy and protein** calculations are typically based on standard body weight (SBW) or actual body weight and provide for maintenance only. Calorie provision should be adequate to prevent protein catabolism for gluconeogenesis. Activity level and stress factors should be taken into account when planning for caloric and protein requirements. High biologic value (HBV) protein should provide at least 50% of the total protein consumed.[17]
- **Potassium** may be liberalized if potassium-depleting diuretics are in use. Restrictions are implemented only with hyperkalemia. Once a potassium restriction is needed, diabetic patients should use caution in selecting interventions for the treatment of hypoglycemic reactions (see **Table 12.7**).

TABLE 12.7 Low Potassium Hypoglycemic Interventions (15 g CHO)

Food Item	Portion Size
Regular gelatin	½ cup
Honey or sugar	1 Tbsp
Regular soda or apple juice	½ cup
Glucose gel	1 tube
Glucose tablets	4

Data from McCann L. *Pocket Guide to Nutrition Assessment of the Patient with Kidney Disease*. 6th ed. CRN-NKF, National Kidney Foundation Inc., New York, NY, 2021.

- **Very-low-sodium** diets are generally not palatable enough to sustain adequate calorie and protein intake for most patients. Initiation of dialysis should be considered when the low palatability of diets with multiple food restrictions prevents adequate nutritional intake.[17]
- Preventing mineral bone disease requires lowering high serum **phosphorus** and maintaining serum **calcium** from CKD stage 3 onwards. Limiting dietary phosphorus alone or in combination with phosphate binders is critical to managing hyperphosphatemia. Educating the patient to read food labels to identify phosphorus-containing additives is also vital. Phosphate-binders, taken with each meal and snack, bind phosphorus in the gastrointestinal (GI) tract and reduce the amount of phosphorus absorbed into the blood. Controlling phosphorus intake will automatically limit calcium intake, since many high-phosphorus foods are also rich in calcium. Early control of phosphorus intake will also help control parathyroid hormone (PTH) levels.
- Elemental **calcium** intake from both the diet and medications should be limited to 2,000 mg daily. Patients

should read labels to avoid products that are fortified with calcium.

- **Fluid** needs to be limited in predialysis patients only when congestive heart failure appears imminent (stages 4–5). Fluid restrictions for patients on diuretics may cause the progression of renal failure because of volume depletion.

ADIME 12.2 ADIME At-A-Glance: Chronic Kidney Disease

Assessment

- Ht, wt, BMI, wt changes, dry wt, SBW.
- Labs: blood pressure, serum creatinine, BUN, GFR, electrolytes, calcium, phosphorus microalbuminuria, inflammatory markers (CRP), Hgb, Hct, iron.
- Food history/logs, vitamin/mineral/supplement use, appetite status.
- NFPE: presence of edema, muscle wasting, subcutaneous fat loss.

Nutrition Diagnosis

- Excessive sodium intake (RT intake) of foods high in sodium × 7 days AEB elevated blood pressure, reported intake of sodium of >4 g/d, and 3+ pitting edema in hands and feet.

Intervention

- Nutrition prescription: add 2 g sodium restriction.
- Review sodium-containing foods.
- Review label reading and meal planning.

Monitoring and Evaluation

- Improvements in blood pressure and edema
- Food intake logs to show decreased sodium intake and appropriate food choices have patient submit logs for 1 week

CKD: Dialysis

Initiation of dialysis as a kidney replacement therapy (KRT) option starts at stage 5 CKD when GFR falls to <15 mL/min/1.72 m^2, although initiation at higher levels of kidney function may be necessary for some patients.[2] The KDOQI refers to dialysis-dependent CKD as CKD 5D.

The two main KRT options are hemodialysis (HD) and peritoneal dialysis (PD). In HD, blood from an artery circulates through a mechanical dialyzer, where it is filtered and returned to the body via the parallel vein. HD is an intermittent therapy that takes place 3 times per week, with sessions lasting 3–5 hours. HD is typically performed at a dialysis center; however, at-home dialysis treatments are becoming more popular. In PD, the patient's own peritoneum serves as the filtration membrane. The dialysis solution (dialysate) is placed into the peritoneal cavity, periodically drained, and replaced with a fresh solution via a catheter (the process is called an exchange). To allow for removal of waste products from the blood, a hyperosmolar dextrose dialysate is used, which results in some dextrose being absorbed by the patient during the process. The daily amount of dextrose absorbed depends on the concentration of dextrose used for each exchange and on the number of exchanges (see **Box 12.3**). Currently, three concentrations of PD dialysate are available: 1.5%, 2.5%, and 4.25%. Dextrose and kilocalorie absorption should be taken into

consideration when developing a diet plan for peritoneal patients. It is important to monitor patients with diabetes closely for glucose control. In both HD and PD, the diet is more liberalized compared with that in the predialysis state.

Box 12.3 Simple Estimation of Dextrose Kilocalories Absorbed from Peritoneal Dialysis

- Continuous cycling peritoneal dialysis (CCPD)[a] allows **40%** dextrose absorption.
- Continuous ambulatory peritoneal dialysis (CAPD) allows **60%** dextrose absorption.
- Each gram of dextrose = 3.4 kcal.
- Patient's weight gain or loss should also be factored into assessing caloric intake.

Dialysate Dextrose Concentration	Grams of Dextrose/L	kcal/L from Dextrose	kcal/L with CCPD (40%)	kcal/L with CAPD (60%)
1.5%	15 g	51 kcal	20–26 kcal	31–36 kcal
2.5%	25 g	85 kcal	34–43 kcal	51–60 kcal
4.25%	42.5 g	145 kcal	58–73 kcal	87–102 kcal

Example: A CAPD patient uses 4 L of 1.5% dialysate and 4 L of 4.25% dialysate daily.
4 L of 1.5% = 124 kcal (31 kcal/L × 4 L)
4 L of 4.25% = 348 kcal (87 kcal/L × 4 L)
Total kcal absorbed from dextrose = **472**

Data from McCann L. *Pocket Guide to Nutrition Assessment of the Patient with Kidney Disease.* 6th ed. CRN-NKF, National Kidney Foundation Inc., New York, NY, 2021.

Description

[a]CCPD is an automated form of peritoneal dialysis and may also be referred to as APD.

Treatment and Nutrition Intervention

Laboratory values monitored in CKD can be found in Table 12.5. A listing of nutrient needs for dialyzed patients can be

found in **Table 12.8**, with additional considerations for specific nutrients as follows:

- **Energy and protein** needs should be based on SBW or adjusted edema-free body weight (aBW_{ef}).[17]

$$aBW_{ef} = BW_{ef} + [(SBW - BW_{ef}) \times 0.25],$$

where BW_{ef} is actual edema-free body weight and SBW is the standard body weight (NHANES II data).

TABLE 12.8 Daily Nutrient Needs for Dialyzed Patients

	Hemodialysis	Peritoneal Dialysis
Protein[a] (g/kg)	1.0–1.2	
Energy[a] (kcal/kg)	25–35	25–35 Include dextrose kcals absorbed from PD
Potassium (g/d)	Adjust per serum levels	Adjust per serum levels; typically unrestricted
Sodium (mg/d)	<2,300	<2,300
Phosphorus (mg/d)	Based on laboratory values. Adjust dietary phosphate intake when serum phosphorus levels are elevated above target range of the CKD stage. Consider bioavailability of phosphorus (animal, vegetable, or additives) when assessing the patient's diet.	
Calcium (mg/d)	To maintain serum calcium WNL. Adjust according to intake (diet, dialysate, binders, and supplements) and vit D analog/calcimimetics use.	
Fluid (mL/d)	750–1,000 plus urine output in 24 h	1,000–2,000 depending on urine output, cardiac status, blood

	Hemodialysis	Peritoneal Dialysis
		pressure; adjust to maintain fluid balance

[a]For metabolically stable patients
WNL, within normal limits.
Data from (i) Ikizler TA, Burrowes JD, Byham-Gray LD, et al. KDOQI clinical practice guideline for nutrition in CKD: update 2020. *Am J Kidney Dis*. 2020;76(3):S1-S107, and (ii) Kidney Disease: Improving Global Outcomes (KDIGO) CKD Work Group. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. *Kidney Intl Suppl*. 2013;3(1):1-150, and (iii) Inker LA, Astor BC, Fox CH, et al. KDOQI U.S. commentary on the 2012 KDIGO clinical practice guidelines for the evaluation and management of CKD. *Am J Kidney Dis*. 2014;63(5):713-735.

Goals should be adjusted for stress, illness, and infections as needed. Protein recommendations for dialysis are higher because of losses through the artificial kidney membrane and blood loss in HD, and through the peritoneal membrane in PD. PD patients in particular face a high risk of protein malnutrition, partly because of greater losses of amino acids and proteins (8–10 g/d) in the dialysate through the daily exchanges. Appetite suppression, as a direct effect of dialysate dextrose calories as well as from uremia, may contribute to suboptimal food intake. HBV protein could account for at least 50% of the protein allowance. Total calories for PD patients should be adjusted for the calories absorbed from the dialysate to control potential obesity, hyperlipidemia, and hyperglycemia (in patients with diabetes).

- **Potassium** restriction is only necessary if the patient is hyperkalemic. Potassium allowance is liberalized with PD treatment because of frequent dialysis exchanges that consistently remove potassium; potassium supplementation may be needed at times to maintain serum levels in the normal range. HD patients may have

increased potassium needs when potassium-depleting diuretics are used or increased losses are related to the etiology of renal failure, as in polycystic kidney disease or high residual kidney function. For diabetic patients requiring the standard potassium restriction, care should be taken in selecting interventions for hypoglycemic reactions (Table 12.7).

- **Sodium** intake is limited to help control volume expansion, thirst, and edema. High dietary intake and inadequate sodium removal during HD result in excess fluid intake and hypertension. Chronic extracellular volume overload is associated with higher cardiovascular morbidity and mortality. The PD diet is more liberal in sodium because of greater losses to the dialysate.
- **Phosphorus/phosphate** intake is limited to maintain serum phosphorus levels in goal range and reduce stimulation of PTH secretion (Table 12.8). Strategies in patient education to limit dietary phosphorus intake while ensuring adequate protein intake requires teaching about (i) limiting the amount of phosphorus/phosphate for every 1 g of protein intake (phosphorus/protein) in food with a favorable ratio <12 mg/g, (ii) demineralization, which requires lengthy boiling (30 min to 1 h) of protein-rich foods (meat, peas, beans, tubers, etc.) before discarding the water, and (iii) identifying phosphorus/phosphate sources in the diet (i.e., animal, vegetable, and additives), and on food labels (additives).[16,18] The phosphorus level is also controlled with the inclusion of phosphate-binding medications. These medications, taken with meals and snacks, bind phosphorus in the GI tract and reduce the amount of phosphorus absorbed into the blood.
- Elemental **calcium** intake should be regulated according to serum levels and consider sources from oral intake and medication. Calcium-based phosphate binders and

calcium-fortified foods that are widespread in the food supply must be given close attention in meeting this dietary limitation.

- **Fluid** limitations are dictated by the amount of urine output and interdialytic weight gains. Goal fluid gains between HD treatments should not exceed 5% of the estimated dry weight. Patients should be free of shortness of breath, edema (peripheral, facial, and ascites), and significant elevation in blood pressure prior to treatment. Fluid intake is more liberal in PD.
- **Vitamin** requirements could follow the recommended dietary allowance (RDA). Nutrition supplements designed specifically for dialysis patients can be useful because these products offer the best nutrient profile with limited fluid for a dialysis patient; however, many patients can also tolerate the standard or basic supplements. Intake of vitamins and minerals may be decreased because of the restriction of foods containing potassium, which limits fruit and vegetable intake. Water-soluble vitamins are also lost through dialysis. Peripheral neuropathy and hyperoxalemia, particularly in HD, are associated with high-dose pyridoxine and vitamin C supplementation. Vitamin A supplementation leads to retention and toxicity. Megavitamin therapy should be discouraged.

Renal osteodystrophy is common in both dialysis groups and typically in the form of *osteomalacia* (bone demineralization); *osteitis fibrosa cystica*, which is caused by elevated PTH; and *metastatic calcification* of joints and soft tissue. Limiting calcium and phosphorus, and treatment with calcitriol and phosphate binders plays a large role in improving these skeletal disorders. Monitoring of serum phosphorous and calcium levels should be more frequent when patients are on calcitriol therapy. Calcitriol should be discontinued in the presence of elevated calcium-

phosphorous product, especially with severe hyperparathyroidism.

Malnutrition and Dialysis

Malnutrition, common in dialysis patients, is either protein only (in PD) or protein energy malnutrition (in HD). The presence of malnutrition aggravates comorbidities, leading to poor prognosis. Causative factors are reduced dietary intake, poor appetite, increased uremia, metabolic acidosis, increased catabolism, and the dialysis procedure itself. Adherence to low- or very-low-protein diets from the non-dialysis ESKD period inadvertently sets up the suboptimal nutrient milieu contributive to malnutrition in the KRT period.[19] Inflammation also common to dialysis patients, may lead to cachexia and muscle wasting.

This severe form of malnutrition is termed as protein energy wasting (PEW) and is persistent as the combining of inflammation, uremia, and hypercatabolism leads to depletion of muscle and fat stores.[20] PEW is associated with frailty, poor physical activity, poor quality of life, and higher risk of death. The International Society of Renal Nutrition and Metabolism (ISRNM) has provided guidelines[20] for PEW diagnosis (see **Box 12.4**).

Box 12.4 The ISRNM Criteria for PEW Diagnosis

1. Serum chemistry: low serum levels of albumin, transthyretin, or total cholesterol
2. Body mass: unintentional weight loss over time, decreased BMI, or total body fat percentage
3. Muscle mass: decreased muscle mass over time, mid-arm muscle circumference, or creatinine appearance
4. Dietary intake: unintentional decreased protein (DPI) or decreased energy intake (DEI)

Note: Diagnosis of PEW requires three of four main components.
BMI, body mass index.
Data from Fouque D, et al. A proposed nomenclature and diagnostic criteria for protein–energy wasting in acute and chronic kidney disease. *Kidney Intl*. 2008; 73(4):391-398.

The following corrective interventions may be implemented when providing medical nutrition therapy (MNT) for a malnourished dialysis patient:

- The average protein consumption in ESKD patients is low (less than 1 g/kg/d in •50% of subjects) along with an equivalent caloric deficit. Thus nutritional intervention should start early for patients whose spontaneous intake is <30 kcal/kg/d and <1.0 g/kg/d of protein.[21]
- In patients reporting poor appetite, consider diet liberalization and food preferences in meal planning. Intake of foods higher in potassium, sodium, and phosphorus in limited amounts can help the patient increase total calories and protein. Intake of protein-rich food or oral supplements (snacks or light meals) is effective in mitigating the catabolism associated with HD and to increase total protein intake.[22]
- If intake remains inadequate, use of appetite stimulants, liquid supplements, protein, and calorie modular, as well as more aggressive interventions (tube feeding or parenteral nutrition), should be considered.
- Oral nutrition supplementation (ONS) is recommended as the first step of nutritional support for ESKD patients with suboptimal dietary intake.[21,23] ONS can add up to 10 kcal/kg and 0.3–0.4 g of protein/kg daily to spontaneous intake, helping the patient to achieve nutritional targets. Nutrition supplements designed specifically for kidney disease patients can be useful. These supplements have nutrient profiles that fit either low or high protein requirements, are calorically dense, and can help meet fluid, phosphorus, potassium, and sodium limitations.

Many protein modular products in the form of powders, bars, and cookies are available as options for improving protein deficits.

- Intradialytic parenteral nutrition (IDPN) is now proposed as an intensive treatment option to address PEW by the ISRNM.[21] IDPN is the administration of nutrients (usually a mixture of amino acids, dextrose, and lipid emulsions) during each dialysis session, through the extracorporeal circulation lines. IDPN is recommended only if the patient has a spontaneous intake of at least 20 kcal/kg/d and 0.8–0.9 g/kg/d of proteins.[21,24] Safe IDPN administration in each dialysis session involves the delivery of not more than 1 L of fluids, 1,000 kcal, and 50 g of amino acids.[24]

Pregnancy in Nondialysis and Dialysis

Increasing cases of pregnancy in women at early stages of CKD are being encountered although there is higher risk for adverse outcomes relating to hypertension, preeclampsia, intrauterine growth restriction, and preterm birth.[25] In contrast pregnancy in women with ESKD is rare and carries very high rates of neonatal complications, including miscarriage, stillbirths, prematurity, and small-for-gestational-age (SGA) births.[5]

Unlike the metabolic and physiological changes faced by women without CKD, women with CKD face increased risk for nutritional deficiencies and wider metabolic issues relating to the uremic burden.[25] Related evidence regarding metabolic and nutritional adaptations during pregnancy in women with CKD is scarce although inferences for nutrition care practice are being drawn from prevailing clinical guidelines for a healthy pregnancy and majorly from case-based management.

Maternal weight gain, lower incidence of SGA and low birth weight (LBW), and less frequent neonatal intensive care unit hospitalization are measurable clinical outcomes in

monitoring the pregnant CKD patient that should also be adopted in the provision of nutrition care.[25]

Pregnancy in Nondialysis

At CKD stages 1 and 2, energy and protein intake recommendations are similar to requirements for a healthy pregnancy.[25] However, the nutritional prescription for an expectant CKD mother at advanced stages 3 and 4 needs to accommodate the physiological cost of the trimesters along with the metabolic issues of the uremic burden. Additionally, beyond the •50% expansion of plasma volume with normal pregnancy, anemia is a prevailing comorbidity in CKD related to a higher risk of prematurity, LBW, and adverse perinatal outcomes.[25]

The nutritional management at stages 3–5 therefore should be on an empirical basis with the following considerations:[25]

- Energy and protein recommendations are based on the pregestational weight of the mother. If it is not available, then the current dry weight is an option for calorie/protein calculation.
- Energy predictive equations can be used with the pregestational weight of the CKD mother, with an additional 85 kcal, 285 kcal, and 475 kcal/d for the first, second, and third trimesters respectively.
- Moderate-protein-restricted diets (0.6–0.8 g/kg/d of protein) supplemented with amino acids such as keto-analogs (1 tablet/8–10 kg) have been shown to benefit a lower frequency of early preterm delivery, very low birth weight (LBW) (<1,500 g), and SGA infants. Plant-based diets have been successfully explored in some case studies.
- Anemia is diagnosed with hemoglobin levels <12.0 g/dL and characterized as hyperproliferative, normochromic,

and normocytic due to the insufficient production of erythropoietin. Oral iron supplementation (120 mg/d) is necessary in severe anemia (<8 g/dL) and if not tolerated then IV iron supplementation is considered.

- Low serum 25-OH-D levels prevail during pregnancy, which call for periodic monitoring to plan supplementation if necessary.
- Sodium and potassium recommendations are individualized based on serum concentrations, blood pressure, medications, hydration status, and kidney function.

Pregnancy in Dialysis

Many nephrologists prescribe long and frequent HD for pregnant women with ESKD, which is necessary to maintain fluid balance, control blood pressure, and reduce uremic toxins of both the mother and the growing fetus.[5] However, the current evidence base is too weak to determine or recommend an optimal session duration.[26] HD sessions offered 6 times per week improve pregnancy-related outcomes. A Canadian dialysis cohort study reported live birth rates of 48% in women dialyzed ≤20 h/wk, 75% in women dialyzed for 30 h/wk, and 85% in women dialyzed for >36 h/wk.[27] Using this Canadian study data, an ungraded statement was made that live birth rates increase with increasing time per HD session.[26]

The nutritional management of the pregnant patient should be on an empirical basis with the following considerations:[25,26]

- Pregnant patients are likely to be dialyzed daily (BUN is maintained below a range of 50–60), allowing for more diet liberalization in all areas.
- For pregnancy undergoing HD an intake of 30–35 kcal/kg pregestational weight plus an additional 300–400 kcal/d

from the second trimester supports physiological needs.

- Daily protein intake should increase by an additional 20 g above standard dialysis calculations.
- Trace elements should be monitored to determine whether supplementation is needed.
- Monitoring of maternal and infant weight gain is important, and post-dialysis weight should be used for reference.
- Close monitoring of interdialytic weight gain will determine signs and symptoms of fluid overload, and if excessive, fluid removal is necessary.
- Worsening anemia in the pregnant patient requires synthetic erythropoietin, iron, and folate supplementation to support increased blood volume.

Medications in CKD: Nondialysis and Dialysis

The CKD medications are functionally diverse, targeting the symptoms of kidney functions that are declining in stages 3–4 CKD patients, and are absent in dialysis patients (see **Table 12.9**). Phosphate binders, intravenous iron, and PTH-suppressing medications will have dose titration based on the laboratory results.[14] Dose titration is generally addressed monthly in a chronic dialysis patient. Aside from these CKD-specific medications, comorbidity presence of diabetes, hyperlipidemia, and/or hypertension will also add to the medication list of CKD patients. All patients should be monitored for common side effects of medications that may contribute to GI intolerance as well as drug–nutrient interactions.[16]

TABLE 12.9 Common Medications in CKD

Category	Medication	Comments
Phosphate binders		• All binders must be taken with each meal and snack
• Calcium-based	• Phoslo, Calphron, Eliphos (calcium acetate); Tums, Os-cal, Caltrate (calcium carbonate)	• Monitor for hypercalcemia
• Calcium-free, aluminum-free	• Renvela, Renagel (sevelamer hydrochloride), Fosrenol (lanthanum carbonate)	• Dose is titrated based on serum phosphorous level and diet intake • Renagel may exacerbate metabolic acidosis and should be monitored during use
• Iron-based	• Auryxia (ferric citrate) • Velphoro (sucroferric oxyhydroxide)	• May cause dark stools
• Aluminum-based	• Aluminum hydroxide	• Use of aluminum should be limited to <14 days because of risk of aluminum toxicity
Potassium binder	• Veltassa	• Binds many oral medications, reducing their effectiveness; other oral meds must be administered at least 6 h before or 6 h after Veltassa • Binds to magnesium, so monitor for hypomagnesemia
Vitamin supplements		

Category	Medication	Comments
Renal-specific formulas	• Dialyvite, Nephrocaps, Nephro-Vite, Rena-Vite • NephPlex, RenaPlex	• Taken daily, usually in the evening to accommodate dialysis treatment days when fluid is removed • Use to replace water-soluble vitamin losses
Iron supplements		
• Oral iron	• Ferrous sulfate, ferrous gluconate, ferrous fumarate	• Oral iron should not be taken with binders
• IV iron	• Venofer (iron sucrose), Ferrlecit (sodium ferric gluconate complex in sucrose), Infed (iron dextran)	• IV iron is administered during hemodialysis treatments
Vitamin D		
• Oral vitamin D	• Rocaltrol (calcitriol)[a]	• Oral agents are to be taken consistently as ordered
• Oral and IV vitamin D	• Hectorol (doxercalciferol)	• IV forms are administered during hemodialysis • Calcium levels should be monitored closely with the use of Rocaltrol and Sensipar
• IV vitamin D	• Calcijex (calcitriol)[a]	
• Oral calcimimetic agent	• Sensipar (cinacalcet)[a]	

Category	Medication	Comments

[a]All agents used to manage PTH levels.
Data from (i) Inker LA, Astor BC, Fox CH, et al. KDOQI U.S. commentary on the 2012 KDIGO clinical practice guidelines for the evaluation and management of CKD. *Am J Kidney Dis*. 2014;63(5):713-735. *American Journal of Kidney Diseases* 2014, 63(5):713-735, (ii) Treatment of Hyperkalemia: Veltassa® accessed April 15, 2023, https://www.viforpharma.com/us/products/nephrology-products, (iii) The Internet Drug List. RxList, accessed April 15, 2023, http://www.rxlist.com, and (iv) Lederer E, Hyperphosphatemia Medication: Phosphate Binders. Medscape, Updated Aug 19, 2022, Accessed April 15, 2023, http://emedicine.medscape.com/article/241185-medication#3.

Posttransplant Considerations

Kidney transplantation is the preferred KRT option of CKD patients. KDOQI guidelines refer to non–dialysis-dependent CKD of any stage (1–5), with a kidney transplant as CKD–T.[26]

In the immediate postoperative period, protein needs are increased to offset postsurgical hypercatabolism and to enhance wound healing.[16] In this period, fluid restriction may be needed in a patient with significant fluid overload, but in long-term maintenance, fluid is generally unrestricted. In the stable chronic posttransplant period, most patients should follow the general nutrition guidelines for any healthy individual. **Table 12.10** lists the nutrient recommendations for posttransplant patients. Immunosuppressive treatment to prevent kidney allograft rejection is a lifelong strategy and the cornerstone of successful transplantation; however, associated comorbidities such as hyperlipidemia, diabetes mellitus, hypertension, obesity, and osteoporosis may necessitate the need for nutrition intervention.[5]

TABLE 12.10 Daily Nutrient Needs for Kidney

Transplant Patients

Nutrients	Acute Posttransplant	Long-Term Maintenance
Protein (g/kg)	1.2–2.0	0.6–0.8 without diabetes 0.8–0.9 with diabetes
Energy (kcal/kg)	30–35	25–35; maintain healthy body weight
Fat	30% of total calories ≤10% saturated fat	30% of total calories 7–10% saturated fat
Potassium (g)	2–4; maintain serum levels WNL	Individualize based on serum level, graft function, and immunosuppressant therapy
Sodium (g)	2–4; based on blood pressure and fluid balance	2–3; based on blood pressure, immunosuppressant effect on fluid balance
Phosphorus	Individualize based on serum levels	Individualize based on serum levels
Fluid	Limit if edema or volume overload is present	Unrestricted, unless overloaded

WNL, within normal limits.
Data from McCann L. *Pocket Guide to Nutrition Assessment of the Patient with Kidney Disease*. 6th ed. CRN-NKF, National Kidney Foundation Inc., New York, NY, 2021.

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CHAPTER 13

Pulmonary Disease

Chronic lower respiratory disease is the sixth leading cause of death in the United States, preceded by heart disease, cancer, COVID-19, unintentional injuries, and stroke,[1] and the third leading cause of death worldwide.[2] Currently, more than 34 million Americans are living with chronic lung diseases such as asthma, emphysema, or chronic bronchitis.[3] Pulmonary diseases have a significant impact on nutritional status, and the clinician's role is crucial in nutrition assessment and patient education to prevent or correct malnutrition.

Chronic Obstructive Pulmonary Disease

Disease Process

Chronic obstructive pulmonary disease (COPD) is a common, progressive respiratory condition characterized by persistent airflow limitation and respiratory symptoms, most commonly dyspnea, cough, and sputum production.[4,5] COPD is caused by interactions between genes and environmental exposures, including tobacco and toxic particles and gases from household and outdoor air pollution, occurring over the lifetime of the individual.[5] In the United States, COPD includes two main conditions; chronic bronchitis (clinically defined) and emphysema (pathologically or radiologically defined).[6] COPD patients typically have chronic bronchitis or emphysema, or a mixture of both.[6,7] The diagnosis of COPD requires evidence of airflow limitation which is defined as an abnormally reduced ability to exhale efficiently and is measured by spirometry.[4] Common causes of COPD include cigarette smoking, pipe smoking, and cigar smoking; passive exposure to cigarette smoking; occupational dust and chemicals; air pollution; and genetic factors.

Chronic Bronchitis

Chronic bronchitis is defined as a chronic cough with sputum production occurring on most days for a minimum of 3 months per year for 2 consecutive years.[5,7] Chronic bronchitis characteristics may include little to no weight loss (patients may have overweight or obesity), wheezing, use of accessory muscles of respiration, and signs of right heart failure including cyanosis and edema in the extremities.[8]

Chronic bronchitis becomes chronic obstructive bronchitis if spirometric evidence of airflow obstruction develops.[4,7]

Emphysema

Emphysema involves the enlargement of the airspaces distal to the terminal bronchioles that causes destruction of lung parenchyma, leading to loss of elastic recoil[4,7] (lung hyperinflation, airflow limitation, and air trapping follow).[7] Characteristics of emphysema may include wheezing, shortness of breath, weight loss, a barrel chest, and a reddish complexion due to the tendency to breathe with pursed lips, and distant heart sounds. Patients with emphysema typically have little or no cough or expectoration.[8] Nutritional depletion is significantly greater in patients with emphysema than in those who have chronic bronchitis.

Treatment and Nutrition Intervention

COPD patients are at risk of weight loss and nutritional deficiencies because of a 15–25% increase in resting energy expenditure (REE) owing to the extra effort to breathe, a higher energy cost of daily activities, reduced caloric intake relative to need because of dyspnea, and the catabolic effect of inflammatory cytokines.[7] Additional factors that may influence energy requirements include chronic infections, chest physical therapy, and pulmonary rehabilitation exercise programs.[9] Medical nutrition therapy (MNT) should focus on prevention and treatment of weight loss and other comorbidities.

Energy, Macronutrients, and Fluid

- Indirect calorimetry (IC), when available, is the preferred method of determining energy expenditure in COPD patients, especially during an exacerbation.[9] When using

predictive equations to assess energy needs, increases for the presence of inflammation or physiologic stress and the level of physical activity must be included.

- There is no data specifically indicating protein requirements in adults with stable COPD. The dietary reference intakes (DRIs) should be used, and the dietitian should individualize protein needs for patients with malnutrition or catabolic stress.[9]
- A high-kilocalorie, high-protein diet is necessary to correct malnutrition in patients with COPD. Start with 30–35 kcal/kg and 1.2–1.7 g protein/kg.[9] Body mass index (BMI) and weight change should be used to evaluate weight status.
- Patients with stable COPD generally have normal fluid requirements. Fluid intake should be 1 mL/kcal in the absence of any mitigating circumstances such as vomiting, diarrhea, infection, fever, or edema.
- When utilizing nutrition support, overfeeding (providing more than 35 kcal/kg unless measured by IC) should be avoided since it may result in dyspnea, hypercapnia, hypoxia, and respiratory acidosis. To avoid these issues, nutritional supplementation should aim to reduce metabolic CO_2 production, lower respiratory quotient, and improve lung function.[10]

Patient Education

- Gas-forming foods may cause discomfort for some patients because they bloat the abdomen and make breathing more difficult. Foods to avoid/use in moderation include onions, cauliflower, broccoli, melons, peas, corn, cucumbers, cabbage, Brussels sprouts, turnips, raw apples, and beans—except green beans. Fried and greasy foods can also cause gas or bloating.

- Medications that make breathing easier and/or clear airways should be used about 1 hour before eating.
- If oxygen is prescribed, make sure it is worn during food procurement, preparation, and eating.
- Eat small, frequent meals and snacks to help compensate for shortness of breath and possible limited oxygen supply to the gastrointestinal (GI) tract. Drink beverages at the end of the meal to avoid early satiety.
- Consider food choices that are easy to chew, swallow, and digest, with nutrients easily absorbed.
- Provide strategies to increase the nutrient density of foods, such as adding the following to foods to increase caloric and/or protein content: butter, margarine, whipped cream, half and half, cream cheese, sour cream, salad dressings, mayonnaise, honey, jam, sugar, granola, dried fruits, cottage or ricotta cheese, whole milk, powdered milk, ice cream, yogurt, eggs, nuts, seeds, wheat germ, and peanut butter.
- Avoid excess sodium, as it may increase edema and make breathing more difficult. Provide the patient with a list of high-sodium foods.
- Encourage consumption of foods high in omega-3 fatty acids (salmon, haddock, mackerel, tuna, and other fish sources).[9]
- Encourage consumption of a diet that meets the recommended dietary allowance (RDA) for antioxidant vitamins A, C, and E.[9]

ADIME 13.1 ADIME At-A-Glance: COPD

Assessment

- Ht, wt, BMI, unintentional wt loss

- Labs: Hgb, Hct, electrolytes, serum iron, arterial blood gases
- Appetite/interest in eating, fatigue, diminished ability to shop for food and prepare meals, shortness of breath (SOB), food intake
- Nutrition-focused physical exam (NFPE): barrel chest, digital clubbing, cyanosis, loss of fat/muscle, edema

Nutrition Diagnosis

- Unintentional weight loss RT generalized fatigue and decreased ability to walk and stand while preparing food AEB 6.5% weight loss in 30 days
- Inadequate mineral intake (iron) RT decreased intake AEB low serum iron, Hgb, and Hct levels

Intervention

- Small, frequent mini-meals and snacks using easy-to-prepare, nutrient-dense food choices
- Add oral supplements (Ensure) to increase caloric and nutrient intake
- Add iron supplement to daily multivitamin
- Consult physician regarding possible need for oxygen therapy during food preparation and eating
- Refer to social work for possible at-home meal delivery service

Monitoring and Evaluation

- Improvements in caloric intake
- Monitor weight changes

Cystic Fibrosis

Disease Process

Cystic fibrosis (CF) is an autosomal recessive genetic disease of the mucus and sweat glands. CF is characterized by the secretion of thick, tenacious mucus that obstructs the ducts and glands of the respiratory tract, sweat and salivary glands, intestine, pancreas, liver, and reproductive tract. Complications include bronchitis, pneumonia, glucose intolerance, and malabsorption caused by decreased availability of digestive enzymes, decreased bicarbonate secretion, decreased bile acid reabsorption, and decreased nutrient absorption by intestinal microvilli.[11,12]

CF is caused by a defect in the cystic fibrosis transmembrane conductance regulator (CFTR) gene, which codes for a chloride transporter found on the surface of the epithelial cells that line the lungs and other organs. There are over 1,700 known mutations of the CFTR gene categorized into five classes depending on where in the pathway the CFTR protein becomes ineffective.[13] There are channels in these lining cells through which sodium and chloride ions can pass. Normally, the movement of ions brings water to the surface of the airway and keeps the mucus moist. The defective gene acts to block the chloride channels, which causes the mucus to dry out. It is difficult for a person to shift the thick mucus, which then becomes prone to infection by bacteria. This defect also accounts for the high levels of sodium and chloride that are present in the saliva, tears, and sweat of patients with CF.[14] In the digestive tract, the mucus blocks the movement of pancreatic digestive enzymes into the duodenum, and if left untreated can lead to malnutrition. Individuals with CF also

have difficulties absorbing the fat-soluble vitamins A, D, E, and K.[11]

Treatment and Nutrition Intervention

The risk for malnutrition in CF patients is high because of increased nutrient needs, decreased intake, and malabsorption.[11-13] The goals of nutritional care include controlling malabsorption with pancreatic enzyme replacement; providing adequate energy, protein, and other nutrients; and preventing nutritional deficiencies. A full nutrition assessment should be conducted by a dietitian[13,15]

- at diagnosis
- when indicated by nutrition screening
- up to monthly for the first 6 months of life; up to every other month until 1 year of age; and up to quarterly until 2 years of age
- annually for those >2 years of age; and
- when disease or treatment course changes

CFTR Modulator Therapy

CF is incurable and medical therapies have long relied on interventions that treat the symptoms and effects of the disease. In recent years, research has focused on the study of CFTR modulator medications that target the underlying gene defect as opposed to targeting the effects of the disease. CFTR modulators are a class of drugs that act by improving production, intracellular processing, and/or function of the defective CFTR protein and are a major revolution in the treatment and management of CF.[16] Since the introduction of CFTR modulators, the number of individuals with CF who qualify for this therapy has increased dramatically, and the goal of the CF Foundation is

to provide some type of CFTR modulation therapy for all people with CF.[13]

Energy

Energy requirements for patients with CF vary widely, and many factors such as gender, age, basal metabolic rate (BMR), physical activity, severity of lung disease, severity of malabsorption, and the use of CFTR modulator therapy must all be considered when determining appropriate energy levels. It has been well established in the literature that energy needs for individuals with CF are higher and can range from 110 to 200% of recommended dietary allowances (RDA) at various times.[13,17]

- For patients with CF <20 years of age who are growing normally without malabsorption, the dietitian may estimate energy needs using the RDA (see Appendix D) or the Institute of Medicine (IOM) active lifestyles formulas with a CF stress factor of 1.1–2.0 (energy needs should be individualized based on the factors mentioned in the paragraph above).
- In adults >20 years of age with CF, the dietitian may estimate energy needs annually or with unintentional weight changes using standard energy expenditure equations × 1.25, since estimated energy requirements for the general population may underestimate needs in adults with CF (energy needs should be individualized based on the factors mentioned in the paragraph above).
- Needs can increase to 120–150% of the estimated energy requirements with increased activity and severity of disease (with some teenagers needing 3,000–4,000 kcal/d).
- Needs can be as high as 200% of estimated energy requirements during episodes of acute respiratory tract infections or disease exacerbations.

- Nocturnal tube feedings may be used to prevent or treat growth failure or malnutrition (usually providing 30–50% of established nutritional needs).

Protein and Fluid

For individuals with CF who are not at risk for malnutrition, it is recommended that they consume macronutrients (protein, carbohydrates, and fat) in the same percent distribution recommended for individuals of the same age without CF.[13] Historically, a high-fat diet was recommended due to malabsorption and a high risk of malnutrition, but with improved treatment options and CFTR modulator therapy, there has been a decline in the number of individuals with CF that are undernourished. As with energy needs, protein needs may be higher during times of stress or disease exacerbation. Individuals with CF typically have normal fluid requirements unless an underlying disease process requires a restriction such as heart or kidney failure.

Patient Education

- Vitamin/mineral supplementation should include a daily CF-specific vitamin, with dosing depending on age and laboratory fat-soluble vitamin levels (A, D, E, and K). Vitamin K should also be given to patients who are taking antibiotics secondary to depletion of beneficial GI flora.[15]
- Additional micronutrient supplementation may vary depending on laboratory values and dietary intake but can include additional vitamin D, iron, and calcium.[15]
- Pancreatic enzyme capsules are taken by mouth with all meals and snacks that contain fat and/or protein. Enzymes work best for approximately 30–45 minutes. For infants and young children, capsules may be opened and the microspheres mixed with a soft, acidic food, such as applesauce. Avoid mixing enzymes with milk-based foods such as yogurt or pudding, or anything with pH >6.0 (the

alkaline pH will destroy the enteric coating and inactivate the enzymes by exposing them to stomach acid).

- The sodium requirement for individuals with CF is higher than the general population due to elevated loss of sodium chloride in sweat[17]:
  - Infants: 500–1,000 mg/day
  - Children: 1,000–4,000 mg/day
  - Adolescents and adults: 6,000 mg/day
  - The sodium requirement for a person with CF may change over time so the adequacy of sodium intake should be reviewed on a regular basis, particularly during these time periods
    - Seasonal variations (requirements are likely to be higher in warmer months)
    - Changes in levels of physical activity and exercise
    - Change in work environment (if required to work outdoors in hot climates)
    - Major changes to nutrient sources that may impact dietary sodium intake (such as reliance on enteral nutrition or oral supplements)

Cystic Fibrosis-Related Diabetes

Cystic fibrosis-related diabetes (CFRD) is a unique type of diabetes that is prevalent in the CF population. CFRD is unique because it has features of both type 1 and type 2 diabetes, including insulin insufficiency from pancreatic scarring and insulin resistance.[17]

CF patients should be screened annually for CFRD starting at age 10 with a 2-hour oral glucose tolerance test. Insulin is currently the only medication that has proven effective in CFRD; oral agents are not indicated. Nutrition therapy incorporates carbohydrate counting so insulin can be adjusted at mealtimes, but dietary restrictions are kept to a

minimum to avoid weight loss. Fat intake should be unrestricted to adequately meet energy targets, and protein intake should be maintained at normal levels (15–20% of total energy intake). Patients should be encouraged to balance regular meals and snacks, and insulin should be dosed accordingly.[17]

Respiratory Failure

Disease Process

Respiratory failure (RF) is a life-threatening impairment of the gas-exchange functions of the lungs, either oxygenation or carbon dioxide elimination, or both. RF is defined as either type 1 or type 2.[18] Type 1 or hypoxemic RF occurs when the respiratory system cannot adequately provide oxygen to the body leading to a drop in blood oxygen levels and is characterized by an arterial oxygen tension (PaO_2) of <60 mm Hg, with a normal or low carbon dioxide tension ($PaCO_2$).[19] Type 1 is the most common form of RF, and some examples include cardiogenic or noncardiogenic pulmonary edema, pneumonia, and pulmonary hemorrhage.[18] Type 2 or hypercapnic RF occurs when carbon dioxide cannot be adequately removed causing a rise in arterial carbon dioxide levels and is characterized by a $PaCO_2$ of >50 mm Hg.[19] Type 2 RF patients who are breathing room air often develop hypoxemia as well. The two most common causes of type 2 RF include severe acute exacerbation of asthma (status asthmaticus) and exacerbation of COPD.[20] RF due to COPD is termed acute-on-chronic RF (ACRF). Other causes include drug overdose, neuromuscular disease, and chest wall abnormalities.

ADIME 13.2 ADIME At-A-Glance: Cystic Fibrosis

Assessment

- Ht (length in children), wt, BMI, head circumference, mid-arm circumference, triceps skinfold, and growth

velocity
- Labs: serum vitamin A, D, E levels, PT/PTT, fasting plasma glucose; A1C (quarterly); fecal elastase levels (newly diagnosed patients), Hgb, Hct, zinc levels
- Appetite/oral intake, steatorrhea, abdominal pain, and edema
- Eating behaviors in children
- Physical activity level (exercise)
- Pancreatic enzyme replacement therapy

Nutrition Diagnosis

- Inadequate oral intake RT increased nutrient needs owing to chronic lung disease and shortened meal times AEB decline in growth velocity and shortened meal time owing to typical childhood behaviors

Intervention

- Increase caloric intake by adding two snacks per day and additional calories to prepared foods with butter, milk, gravy, etc.
- Provide at least two servings per day of oral supplements of choice
- Educate family on importance of increasing calories and nutrients to support lung function and reverse decline in growth velocity
- Refer to psychologist for help with mealtime behaviors to maximize intake

Monitoring and Evaluation

- Improvements in growth

- Consider tube feeding if weight goals are not achieved

RF can be either acute or chronic. Acute hypercapnic RF develops quickly, over minutes to hours, and usually results in a pH <7.3. Chronic RF develops over several days or longer, allowing time for renal compensation and an increase in bicarbonate concentration. Therefore, the pH is usually only slightly decreased.[19]

Arterial Blood Gases

The confirmation of the diagnosis of RF is based on arterial blood gas (ABG) analysis. ABG measurement is a blood test that is performed to determine the oxygen, carbon dioxide, and bicarbonate content, as well as the pH (acidity), of the blood. Its main use is in pulmonology, as many lung diseases feature poor gas exchange, but it is also used in nephrology and electrolyte disturbances. In pulmonology, the test is used to evaluate respiratory diseases and conditions that affect the lungs, to measure the effectiveness of ventilation and oxygen therapy, and to evaluate overall acid–base balance.[21,22] See **Table 13.1** for ABG components.

TABLE 13.1 Arterial Blood Gases

Analyte	Normal Range	Interpretation
pH	7.35–7.45	<7.35 = acidosis >7.45 = alkalosis <6.8 or >7.8 is usually fatal
$PaCO_2$	35–45 mm Hg	Partial pressure of carbon dioxide in arterial blood (reflects CO_2 concentration). A high $PaCO_2$ (respiratory acidosis) indicates hypoventilation;

Analyte	Normal Range	Interpretation
		a low $PaCO_2$ (respiratory alkalosis) indicates hyperventilation.
PaO_2	70–100 mm Hg	Partial pressure of oxygen in arterial blood. Values below 60 may require immediate action and possibly mechanical ventilation.
SaO_2	94–100%	Saturation of hemoglobin available for transporting oxygen in the arteries
HCO_3	22–26 mEq/L	Bicarbonate-metabolic indicator of kidney's role in maintaining normal pH values. A low HCO_3 indicates metabolic acidosis; a high HCO_3 indicates metabolic alkalosis.
CO_2	19–24 mEq/L	Dissolved carbon dioxide in the blood.
Base excess	–2 to 2 mEq/L	Represents the amount of buffering anions in the blood, with HCO_3 being the largest. A negative-base excess (deficit) indicates metabolic acidosis. A positive-base excess indicates metabolic alkalosis or compensation to prolonged respiratory acidosis.

Data from (i) Grogono AW. Acid–Base Balance Tutorial. Tulane University Department of Anesthesiology. Accessed July 27, 2023. www.acid-base.com/index.php, and (ii) Pagana KD, Pagana TJ, Pagana TN. *Mosby's Manual of Diagnostic and Laboratory Tests*, 7th ed. Elsevier-Mosby, 2022.

Acid–Base Balance

When the pulmonary system is compromised by diseases such as RF or COPD, the ability of the lungs to regulate acid–base balance can be affected. Acid–base balance within the body is required to provide an optimal environment for enzymatic and cellular activity. When normal physiology is altered, acid–base imbalances may result. These can be defined as either acidosis or alkalosis. When the acid–base

disturbance results from a primary change in HCO_3, it is a metabolic disorder; when the primary disturbance alters blood $PaCo_2$, it is a respiratory disorder. Compensation for these disturbances can be respiratory or metabolic in nature and is intended to minimize further pH changes.[21] **Box 13.1** lists acid–base disturbances and the body's physiologic response.[21,22]

Box 13.1 Acid–Base Disturbances

Respiratory Acidosis

- **Common causes:** asphyxia, respiratory depression (drugs and central nervous system trauma), pulmonary disease (pneumonia, COPD, and respiratory underventilation).
- **Mechanism of compensation:** kidneys will retain HCO_3 and excrete H^+ to increase pH.

	Uncompensated	Compensated
pH	<7.35	Normal
$PaCO_2$	↑	↑
HCO_3	Normal	↑

Respiratory Alkalosis

- **Common causes:** hyperventilation (anxiety and pain respiratory overventilation) and pulmonary emboli.
- **Mechanism of compensation:** kidneys will excrete HCO_3 and retain H^+ to decrease pH.

	Uncompensated	Compensated
pH	>7.45	Normal
$PaCO_2$	↓	↓

	Uncompensated	Compensated
HCO_3	Normal	↓

Metabolic Acidosis

- **Common causes:** diabetic ketoacidosis, shock, renal failure, intestinal fistula, diarrhea, and starvation.
- **Mechanism of compensation:** Respiratory rate increases, and lungs "blow off" excess CO_2 to increase pH.

	Uncompensated	Compensated
pH	<7.35	Normal
$PaCO_2$	Normal	↓
HCO_3	↓	↓

Metabolic Alkalosis

- **Common causes:** excessive vomiting, diuretics, hypercalcemia, antacid overdose.
- **Mechanism of compensation:** Respiratory rate decreases to retain CO_2 and decrease pH.

	Uncompensated	Compensated
pH	>7.35	Normal
$PaCO_2$	Normal	↑
HCO_3	↑	↑

Treatment and Nutrition Intervention

Mechanical ventilation is used when a patient's spontaneous ventilation is inadequate to maintain life. In addition, it is indicated as a measure to control ventilation in critically ill patients and as prophylaxis for the impending collapse of other physiologic functions. Physiologic indications include respiratory or mechanical insufficiency and ineffective gas exchange.[23]

It is important for the dietitian working in intensive care to be familiar with ventilator modes and settings. This information is useful when conducting nutrition assessments and calculating nutritional requirements for RF patients on mechanical ventilation. **Table 13.2** describes common ventilator modes.

TABLE 13.2 Ventilator Modes

Mode	Name	Description
ACV	Assist-control ventilation	Triggered by patient breaths, but if patient fails to trigger the threshold, a mechanically controlled breath is delivered.
CMV	Continuous mandatory ventilation	Ventilator delivers breaths at a set rate, and volume or pressure, regardless of patient effort.
CPAP	Continuous positive airway pressure	Positive pressure applied during spontaneous breathing and maintained throughout the entire respiratory cycle, without ventilator assistance.
IMV	Intermittent mandatory ventilation	Combination of spontaneous and CMV—patient can breathe spontaneously between ventilator breaths that are delivered at a set rate and volume or pressure.
MMV	Mandatory minute ventilation	Patient breathes spontaneously, yet a minimum level of minute ventilation is ensured.

Mode	Name	Description
PEEP	Positive end-expiratory pressure	Positive pressure applied during machine breathing and maintained at end-expiration.
PSV	Pressure support ventilation	Provides a preset level of positive pressure during each inspiratory effort by the patient.
SIMV	Synchronized intermittent mandatory ventilation	Combines spontaneous and IMV. Intermittent ventilator breaths are synchronized to spontaneous breaths to reduce competition between the ventilator and the patient. If no inspiratory effort is sensed, the ventilator delivers a breath.

Energy and Protein

The use of predictive equations to estimate energy expenditure is a regular part of the nutrition assessment completed by a dietitian. However, in a critically ill patient with pulmonary disease, normal predictive equations may be of little value. The measurement of resting energy expenditure (REE) through IC has been shown to be more accurate than published formulas used to predict REE.[24] IC is the measurement of gas exchange used to indicate a patient's cellular metabolic activity. IC measures oxygen consumption and carbon dioxide production to calculate REE and the respiratory quotient. This information can be used to prevent over- and underfeeding of patients. Therefore, IC is the preferred method of determining energy expenditure in critically ill patients.

- If IC is not available, start with 20–25 kcal/kg to avoid overfeeding (25–30 kcal/kg in ambulatory patients).
- Once initiated, monitor pulmonary status, body weight, and fluid balance closely to avoid overfeeding.

- Two predictive equations have been shown to be useful in the critical care setting for patients on mechanical ventilation.[25] The Penn State (2003b) and the Modified Penn State (2010) equations can be used and are found in **Box 13.2**.
- Protein should be provided at 1.2–1.5 g/kg/d.

Box 13.2 Predictive Equations for Patients on Mechanical Ventilation

Penn State (2003b) equation: use for nonobese or obese patients

$$RMR = Mifflin\ (0.96 + V_E\ (31) + T_{max}\ (167) - 6{,}212$$

Modified Penn State (2010) equation: use for obese patients

$$RMR = Mifflin\ (0.71) + V_E\ (64) + T_{max}\ (85) - 3{,}085$$

Both equations above use the Mifflin–St. Jeor equation

$$\textit{Men:}\ RMR = (9.99 \times W) + (6.25 \times H) - (4.92 \times A) + 5$$

$$\textit{Women:}\ RMR = (9.99 \times W) + (6.25 \times H) - (4.92 \times A) - 161$$

RMR, resting metabolic rate; V_E, expired minute ventilation; T_{max}, maximum body temperature in previous 24 hour in degree Celsius; W, weight in kg using actual BW; H, height in cm; A, age.

Data from Academy of Nutrition and Dietetics. Evidence Analysis Library: Critical Illness Guideline (2012) Determining Resting Energy Expenditure. Accessed July 26, 2023. https://www.andeal.org.

Respiratory Quotient

The respiratory quotient (RQ) measures the ratio of the volume of carbon dioxide (VCO_2) produced to the volume of oxygen consumed (VO). This is represented by the following equation:

$$RQ = VCO_2/VO_2$$

The RQ was once thought to be useful as a means to determine the source of substrate metabolism (fat, carbohydrate, or protein), although this assumption was

never verified by research.[24] Stored CO_2 in the body can be mobilized with hyperventilation and would thus reflect an increase in CO_2 excretion but not necessarily production, which would have an erroneous effect on the measured RQ. A clinical benefit of the measured RQ is to validate an IC study. Measured values of RQ that fall outside of the physiologic range of 0.67–1.3 can infer serious problems with test validity and almost always represent human or machine calibration errors. In addition, RQ may be used as a measure of tolerance to feeding. A significant rise in measured RQ, especially above 1.0 in response to enteral or parenteral feeding, may indicate mild respiratory compromise and the need for alteration in the nutrition support regimen. However, the application of the RQ as a marker of substrate use has limited clinical usefulness.[26]

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Appendix A

Laboratory Assessment

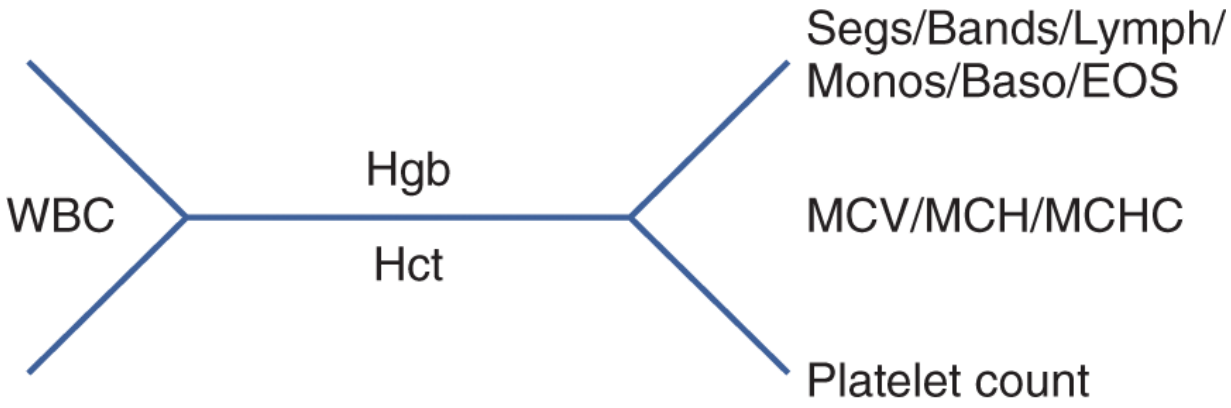
Kelly Sanna-Gouin, RD, CNSC

Laboratory and other diagnostic tests are tools used by clinicians to gain valuable and objective information about their patients. When used in conjunction with additional patient information, such as anthropometric data, a thorough history, and a physical examination, laboratory tests can provide valuable information about a patient's nutritional status and his or her response to medical nutrition therapy.

This appendix contains an alphabetical list of common laboratory (lab) measurements that are relevant to nutrition assessment. This list is not meant to be comprehensive; rather it is a quick reference to the lab tests most commonly used by dietitians in the clinical setting.

Normal values are listed, but it must be noted that normal ranges of lab test results vary significantly, depending on the lab and their methods of testing. It is important to always check the normal values at the facility where the test is performed. This information is almost always given directly adjacent to the patient-specific lab result. In this book, Reference Ranges for blood tests are reported in the conventional U.S. system first, then in the SI system (International System of United or Système Internationale

d’Unités), if available. Critical values are also listed, if applicable. All values given are for adults. Conditions that may cause test results to be increased or decreased are listed under the heading “Clinical Implications.”



Description

Figure A.1 Complete Blood Count.

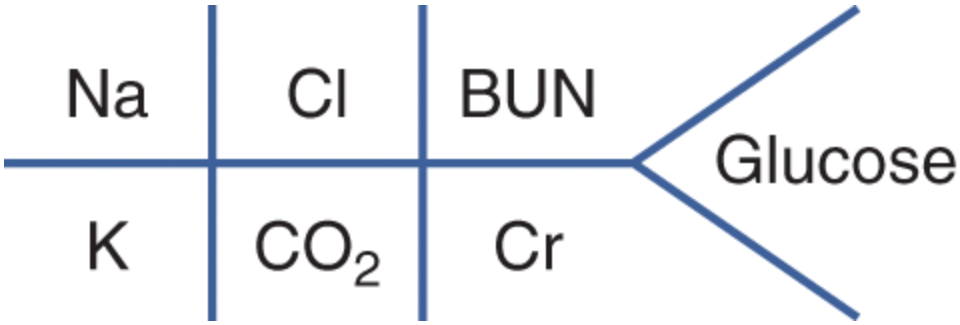


Figure A.2 Electrolytes.

Description

Laboratory Values for Assessing Nutritional Status

Alanine Transaminase (ALT)

Also referred to as Alanine aminotransferase (ALT), Serum Glutamic-Pyruvic Transaminase (SGPT), GPT

Reference Range

4 to 36 U/L

Clinical Implications

Increased Levels

Cirrhosis; hepatic necrosis; hepatitis; hemochromatosis; hepatic steatosis; liver ischemia; hepatic cancer; use of hepatotoxic drugs; mononucleosis; pancreatitis

Decreased Levels

Vitamin B_6 deficiency; chronic kidney disease

Albumin

Reference Range

3.5–5.5 g/dL or 35–50 g/L

Clinical Implications

Increased Levels

Dehydration; shock; hemoconcentration

Decreased Levels

Decreased hepatic synthesis (malnutrition; cirrhosis, liver disease, alcoholism; malabsorption); increased losses (nephrotic syndrome, renal disease; burns; trauma; hemorrhage with fluid replacement, fistulas, protein-losing enteropathies, such as Crohn disease; acute or chronic glomerulonephritis), hemodilution (CHF, pregnancy;

overhydration); acute and chronic inflammation and infections; certain medications such as steroids, insulin, hormones; thyroid disease.

Alkaline Phosphatase

Reference Range

44–147 IU/L or 0.73–2.45 µkat/L

Clinical Implications

Increased Levels

Slight increase in pregnant women and children with growth spurts.
Biliary obstruction; bone disease; eating a fatty meal if blood type O or B; healing fracture; hepatitis; hyperparathyroidism; hyperthyroidism; leukemia; liver disease; lymphoma; osteoblastic bone tumors; osteomalacia; Paget disease of bone; sarcoidosis

Decreased Levels

Hypophosphatasia, hypothyroidism, malnutrition, pernicious anemia, protein deficiency, Wilson disease, zinc deficiency

Amylase

Reference Range

30–220 U/L

Clinical Implications

Increased Levels

Slight increase in pregnancy, the elderly.
Peptic ulcers; intestinal obstruction; pancreatic duct obstruction; cancer; gallbladder attacks; mesenteric thrombosis; post operative abdominal surgery; mumps; macroamylasemia; tubal pregnancy

Decreased Levels

Liver damage; cystic fibrosis; pancreatic cancer; toxemia of pregnancy

Arterial Blood Gases (ABGs; Blood Gases)

Reference Range

pH: 7.35–7.45 (critical values: <7.25 or >7.55)
Pco_2: 35–45 mmHg (critical values: <20 or >60 mmHg)
HCO_3: 21–28 mEq/L (critical values: <15 or >40)
Po_2: 80–100 mmHg (critical values: <40)
O_2 saturation: 94%–99% (critical values: 75% or lower)

Clinical Implications: pH

Increased Levels (Alkalosis)

Metabolic: hypokalemia; hypochloremia; chronic vomiting; aldosteronism; chronic and high-volume gastric suctioning; sodium bicarbonate administration.
Respiratory: hypoxemic states (e.g., congestive heart failure [CHF], cystic fibrosis [CF], carbon monoxide poisoning, pulmonary emboli, shock, acute pulmonary diseases); anxiety, neuroses, psychoses; pain; pregnancy; liver disease; early salicylate poisoning.

Decreased Levels (Acidosis)

Metabolic: ketoacidosis (diabetes, alcohol, starvation); lactic acidosis; severe diarrhea; renal failure; strenuous exercise; increased loss of alkaline body fluids (diarrhea, fistulas, aspiration of gastrointestinal contents, biliary drainage).
Respiratory: respiratory failure; neuromuscular depression; pulmonary edema.

Clinical Implications: Pco_2

Increased Levels

Decreased alveolar ventilation (COPD; bronchitis, emphysema); neuromuscular diseases (such as myasthenia gravis); oversedation; head trauma; other causes of hypoventilation (e.g., Pickwickian syndrome).

Decreased Levels

Hypoxemia; pulmonary emboli; anxiety; pain; pregnancy; other causes of hyperventilation; early salicylate poisoning.

Clinical Implications: HCO_3

Increased Levels

Chronic vomiting or high-volume gastric suction; aldosteronism; COPD; diuretics (thiazide, furosemide).

Decreased Levels

Chronic or severe diarrhea; chronic use of loop diuretics; starvation; acute renal failure; diabetic ketoacidosis.

Clinical Implications: Po_2

Increased Levels

Polycythemia; increased inspired O_2; hyperventilation.

Decreased Levels

Ventilation/perfusion mismatching (asthma, COPD, atelectasis, pulmonary embolism, interstitial lung disease, airway obstruction by foreign body, shock). Alveolar hypoventilation (kyphoscoliosis, neuromuscular disease, head injury, stroke). Right to left shunt (congenital heart disease). Drugs (barbituates, opioids).

Aspartate Aminotransferase (AST)

Also referred to as serum glutamic-oxaloacetic transaminase, SGOT, aspartate transaminase (AST)

Reference Range

0–35 U/L or 0–0.58 µKat/L

Clinical Implications

Increased Levels

Very high AST levels (more than 10 times the highest normal value): Liver damage (acute viral hepatitis, toxins/drugs including acetaminophen overdose, acute fulminant hepatitis); tumor necrosis.
Moderately high AST levels: Chronic liver disease; alcohol abuse; cholestasis; myocardial infarction; heart failure; kidney damage; muscular dystrophy; dermatomyositis; trauma; hemolysis; heatstroke (level dependent on extent of tissue damage); high consumption of vitamin A; pulmonary embolism; carbon tetrachloride (an industrial chemical used to make refrigerants and aerosols).
Slightly high levels of AST: Hepatic steatosis; alcohol abuse; cirrhosis; mononucleosis; drugs (statins, aspirin, barbiturates, HIV medications, herbs).

Decreased Levels

Uremia, vitamin B_6 deficiency, medications, including metronidazole (Flagyl) and trifluoperazine (Stelazine)

Bilirubin

Reference Range

Direct Bilirubin (also referred to as conjugated bilirubin): 0.1–0.3 mg/dL or 1.7–5.1 µmol/L (SI units)
Indirect Bilirubin (also referred to as unconjugated bilirubin): 0.2–0.8 mg/dL or 3.4–12.0 µmol/L (SI units)
Total bilirubin (the total of direct and indirect combined): 0.3–1.0 mg/dL or 5.1–17 µmol/L (SI units)

Clinical Implications

Increased Levels

Anemia; jaundice; cirrhosis; reaction to a blood transfusion; Gilbert syndrome; viral hepatitis; alcohol abuse; gallstones; strenuous prolonged exercise; gallstones; medications (allopurinol, anabolic steroids, some antibiotics, antimalarials, azathioprine, chlorpropamide, cholinergics, codeine, diuretics, epinephrine, meperidine, methotrexate, methyldopa, MAO inhibitors, morphine, nicotinic acid, oral contraceptives, phenothiazines, quinidine, rifampin, salicylates, steroids, sulfonamides, and theophylline)

Decreased Levels

Autoimmune diseases (rheumatoid arthritis, lupus, polymyositis, and Sjogren’s syndrome); cardiovascular disease; caffeine; medications (salicylates, barbiturates)

Blood Urea Nitrogen (BUN)

Reference Range

8–23 mg/dL or 3.6–7.1 mmol/L
(Critical values: >100 mg/dL indicates serious impairment of renal function)

Clinical Implications

Increased Levels

Prerenal (hypovolemia, shock, burns, dehydration, CHF, myocardial infarction [MI], gastrointestinal [GI] bleeding, excessive protein ingestion, catabolism, starvation, or sepsis); renal (renal disease or failure, nephrotoxic drugs); postrenal (urethral obstruction from stones; tumors/congenital anomalies, bladder outlet obstruction from prostatic hypertrophy, cancer, or congenital anomalies).

Decreased Levels

Liver failure; acromegaly; malnutrition; overhydration; negative nitrogen balance; syndrome of inappropriate secretion of antidiuretic hormone (SIADH); pregnancy; nephrotic syndrome; anabolic steroid use; impaired absorption (celiac disease).

C Reactive Protein (CRP)

Reference Range

Normal: 0–0.3 mg/dL
Normal or Minor Elevation: 0.3–1.0 mg/dL
Moderate Elevation: 1.0–10.0 mg/dL
Marked Elevation: >10.0 mg/dL
Severe Elevation: 50.0 mg/dL

Clinical Implications

Increased Levels

Normal or minor elevation: obesity; pregnancy; depression; diabetes; common cold; gingivitis; periodontitis; sedentary lifestyle; cigarette smoking; and genetic polymorphisms.
Moderate elevation: systemic inflammation such as RA, SLE, or other autoimmune diseases; malignancies; myocardial infarction; pancreatitis; bronchitis.
Marked elevation: acute bacterial infections; viral infections; systemic vasculitis; major trauma
Severe elevation: acute bacterial infections.

Decreased Levels

NSAIDS, statins, and magnesium supplementation can falsely decrease levels.

Calcium (Ca)—Total and Ionized Calcium

Reference Range

Total Ca: 9.0–10.5 mg/dL or 2.25–2.75 mmol/L
(Critical values: <6 or > 13 mg/dL or <1.5 or >3.25 mmol/L)
Ionized Ca: 4.5–5.6 mg/dL or 1.05–1.30 mmol/L
(Critical values: <2.2 or >7 mg/dL or <0.78 or >1.58 mmol/L)

Clinical Implications

Increased Levels (Hypercalcemia)

Hyperparathyroidism; cancer with parathyroid hormone (PTH)-producing tumors (metastatic bone cancers, Hodgkin lymphoma, leukemia, and non-Hodgkin lymphoma); Paget disease of the bone; prolonged immobilization; milk–alkali

syndrome; excessive intake of vitamin D, milk, antacids; vitamin A intoxication; Addison disease; granulomatous infections (e.g., sarcoidosis, tuberculosis); drugs (chronic diuretic use – thiazides, lithium).

Decreased Levels (Hypocalcemia)

Pseudohypocalcemia caused by low albumin levels[a]; hypoparathyroidism & pseudohypoparathyroidsim; renal failure; hyperphosphatemia secondary to renal failure; rickets; vitamin D deficiency; osteomalacia; malabsorption; pancreatitis; malnutrition; alkalosis; magnesium deficiency; massive transfusion.

Chloride (Cl)

Reference Range

98–106 mEq/L or 98–106 mmol/L
(Critical values: <80 or >115 mEq/L)

Clinical Implications

Increased Levels (Hyperchloremia)

Dehydration; hyperparathyroidism; renal failure, nephrotic syndrome, renal tubular acidosis; metabolic acidosis; diabetes insipidus; hyperadrenocorticism; eclampsia; hyperventilation, which causes respiratory alkalosis; overtreatment with saline; drugs—acetazolamide, androgens, hydrochlorothiazide, salicylate intoxication.

Decreased Levels

Overhydration; prolonged vomiting or gastric suctioning; chronic diarrhea or high-output GI fistula; ulcerative colitis; excessive sweating; CHF; renal failure with salt deprivation; salt-losing nephritis; metabolic alkalosis; burns; Addison

disease; SIADH; fever; diabetic ketoacidosis; water intoxication; acute intermittent porphyria; drugs—chronic laxative or bicarbonate ingestion, corticosteroids, diuretics.

Cholesterol[b]

Reference Range

Desirable: <200 mg/dL or <5.2 mmol/L
Borderline high: 200–239 mg/dL or 5.18–6.21 mmol/L
High: >240 mg/dL or >6.22 mmol/L

Clinical Implications

Increased Levels (Hypercholesterolemia)

Familial hypercholesterolemia and/or hyperlipidemia; hypothyroidism; poorly controlled diabetes mellitus (DM); nephrotic syndrome; cholestasis; pregnancy (third trimester); obesity; high dietary intake; anorexia nervosa; Cushing syndrome; Werner syndrome; acute MI; atherosclerosis; biliary cirrhosis; hepatocellular carcinoma; pancreatectomy; acute intermittent porphyria; drugs—corticosteroids.

Decreased Levels

Malabsorption; familial (Gaucher disease, Tangier disease); intestinal lymphangiectasia; malnutrition; advanced cancer; hyperparathyroidism; chronic anemias; severe burns; sepsis/stress; severe liver disease (acute hepatitis, cirrhosis, malignancy).

Creatinine (Serum Creatinine)

Reference Range

Female: 0.4–1.1 mg/dL or 44–97 µmol/L
Male: 0.5–1.2 mg/dL or 53–106 µmol/L
(Critical values for female and male: >4 mg/dL)[c]

Clinical Implications

Increased Levels

Impaired renal function (e.g., glomerulonephritis, pyelonephritis, acute tubular necrosis, urinary tract obstruction); muscle disease (gigantism, acromegaly); rhabdomyolysis; shock (prolonged); diabetic nephropathy; CHF; nephrotoxic drugs.

Decreased Levels

Debilitation; decreased muscle mass (e.g., muscular dystrophy, myasthenia gravis); advanced and severe liver disease, with age proportional to decrease in muscle mass.

Erythropoietin (EPO)

Reference Range

4.1–19.5 mU/mL

Clinical Implications

Increased Levels

Anemias associated with bone marrow hyporesponsiveness (iron deficiency, aplastic); hemolytic anemia; secondary polycythemia (high altitude hypoxia, COPD, pulmonary fibrosis); EPO-producing tumors (cerebellar hemangioblastomas, pheochromocytomas, renal tumors); kidney transplant rejection; polycystic kidney disease; treatment with recombinant human EPO; myelodysplasia;

chemotherapy; renal cell carcinoma; adrenal carcinoma; pregnancy.

Decreased Levels

Anemia of chronic disease; renal failure; inflammatory states; primary polycythemia (polycythemia vera); HIV infection with AZT treatment.

Folic Acid (Folate)

Reference Range

2–20 ng/mL, 2–20 µg/L, or 4.5–45.3 nmol/L

Clinical Implications

Increased Levels

Pernicious anemia, vitamin B_{12} deficiency; vegetarianism; recent massive blood transfusion; blind loop syndrome.

Decreased Levels

Inadequate intake (malnutrition, chronic disease, alcoholism, anorexia, diet devoid of fresh vegetables); malabsorption (e.g., small bowel disease); pregnancy; megaloblastic anemia; hemolytic anemia; malignancy; chronic renal disease; drugs that are folic antagonists (phenytoin, aminopterin, methotrexate, antimalarials, alcohol, oral contraceptives).

Glucose (Blood Sugar, Fasting Blood Sugar [FBS])

Reference Range

Fasting defined as no caloric intake for at least 8 hours
70–100 mg/dL or <5.5 mmol/L
(Critical values: <50 and >400 mg/dL)

Clinical Implications

Increased Levels (Hyperglycemia)

DM; Cushing syndrome; acute stress response (MI, cerebrovascular accident [CVA], burns, infection, surgery); pheochromocytoma, acromegaly, gigantism; chronic renal failure; glucagonoma; acute pancreatitis; pregnancy; corticosteroid therapy.

Decreased Levels (Hypoglycemia)

Pancreatic islet cell carcinoma; Addison disease; hypothyroidism; hypopituitarism; liver disease; starvation; insulin overdose.

Glucose, Postprandial (2-Hour Postprandial Glucose [2-Hour PPG])[d]

Reference Range

0–50 years: <140 mg/dL or <7.8 mmol/L
50–60 years: <150 mg/dL
>60 years: <160 mg/dL

Clinical Implications

Increased Levels

DM; gestational diabetes mellitus (GDM); malnutrition; hyperthyroidism; acute stress response (MI, CVA, burns, infection, surgery); Cushing syndrome; pheochromocytoma; chronic renal failure; glucagonoma; diuretic therapy; corticosteroid therapy; liver disease.

Decreased Levels

Insulinoma; hypothyroidism; hypopituitarism; insulin overdose; Addison disease.

Glucose Tolerance (GT; Oral Glucose Tolerance Test [OGTT])

Reference Range

Fasting: <110 mg/dL or <6.1 mmol/L
30 minutes: <200 mg/dL or <11.1 mmol/L
1 hour: <200 mg/dL or <11.1 mmol/L
2 hours: <140 mg/dL or <7.8 mmol/L
3 hours: 70–115 mg/dL or <6.4 mmol/L

Clinical Implications

Increased Levels

DM; acute stress response (MI, CVA, burns, infection, surgery); Cushing syndrome; pheochromocytoma; chronic renal failure; glucagonoma; diuretic therapy; corticosteroid therapy; liver disease; acute pancreatitis; myxedema; Somogyi response to hypoglycemia.

Glycosylated Hemoglobin (GHB; Glycohemoglobin [GHb], Hemoglobin A1C [HbA1C] or [A1C])

Reference Range

Normal: 5.7% or below
Prediabetes: 5.7–6.4%
Diabetes: 6.5% or higher

Clinical Implications

Increased Levels

Newly diagnosed DM; pregnancy; nondiabetic hyperglycemia (acute stress response, Cushing syndrome, pheochromocytoma, glucagonoma, corticosteroid therapy).

Decreased Levels

Hemolytic anemia or other diseases with shortened red blood cell life span such as sickle cell disease and glucose-6-dehydrogenase deficiency; chronic blood loss; chronic renal failure.

Hematocrit (Hct; Packed Cell Volume [PCV])

Reference Range[e]

Male: 40%–54% or 0.40–0.54 volume fraction
Female: 36%–46% or 0.36–0.46 volume fraction

Clinical Implications

Increased Levels

Erythrocytosis; congenital heart disease; polycythemia vera; severe dehydration; severe COPD; diabetic acidosis; transient cerebral ischemia (TIA); trauma; burns.

Decreased Levels

Macrocytic anemia (liver disease, hypothyroidism, vitamin B_{12} deficiency, myelodysplasia); normocytic anemia (early iron deficiency, anemia of chronic disease, hemolytic anemia, acute hemorrhage, bone marrow infiltrates); microcytic anemia (iron deficiency, thalassemia);

hemoglobinopathy; hemodilution; renal disease; pregnancy; peptic ulcer disease; RA.

Hemoglobin (Hgb, Hb)

Reference Range[f]

Male: 14–18 g/dL or 8.7–11.2 mmol/L
Female: 12–16 g/dL or 7.4–9.9 mmol/L
(Critical values: < 7 g/dL or > 20 g/dL)

Clinical Implications

Increased Levels

Congenital heart disease; polycythemia (erythrocytosis); severe dehydration; severe COPD; high altitudes; severe burns.

Decreased Levels

Macrocytic anemia (liver disease, hypothyroidism, vitamin B_{12} deficiency, folate deficiency, myelodysplasia); normocytic anemia (early iron deficiency, anemia of chronic disease, hemolytic anemia, acute hemorrhage, bone marrow infiltrates); microcytic anemia (iron deficiency, thalassemia); hemodilution; hemoglobinopathy; renal disease; pregnancy.

Homocysteine (HCY)

Reference Range

Males 4–12 µmol/L
Females 4–10 µmol/L

Clinical Implications

Increased Levels

homoscystinuria due to defects in cystathionine β-synthase, methionine synthase or intracellular cobalamin metabolism; MTHFR C677T mutation; deficiency of folic acid or B vitamins (e.g., B_{12}, B_6); cigarette smoking; chronic alcohol ingestion; renal failure, systemic lupus erythematosus; hypothyroidism; diabetes mellitus; certain medications (e.g., methotrexate, nicotinic acid, theophylline, L-dopa); and advanced age.

Decreased Levels

Down syndrome; hyperthyroidism.

Iron Level (Fe)

Reference Range

50–175 µg/dL (9–31 mcmol/L)

Clinical Implications

Increased Levels

Hemosiderosis (e.g., multiple transfusions, excess iron administration); acute Fe poisoning (children); hemolytic anemia; pernicious anemia; aplastic or hypoplastic anemia; viral hepatitis; lead poisoning; thalassemia; hemochromatosis; certain medications (estrogens, ethanol, oral contraceptives).

Decreased Levels

Iron-deficiency; nephrotic syndrome; chronic renal failure; many infections; active hematopoiesis; remission of pernicious anemia; hypothyroidism; malignancy (carcinoma); postoperative state; kwashiorkor.

Lipase

Reference Range

0–160 U/L or 0–160 U/L

Clinical Implications

Increased Levels

Acute pancreatitis; perforated or penetrating peptic ulcer, particularly involving the pancreas; obstruction of pancreatic duct by stone; drug-induced spasm of sphincter of Oddi (codeine, morphine, methacholine, cholinergics); chronic pancreatitis; pancreatic pseudocyst; pancreatic malignancy; gastric malignancy or perforation; acute cholecystitis; small bowel obstruction; intestinal infarction; cystic fibrosis; inflammatory bowel disease; acute and chronic renal failure; organ transplantation, particularly associated with a complication (organ rejection, cyclosporine toxicity, cytomegalovirus infection); alcohol abuse; diabetic ketoacidosis; intracranial hemorrhage; lymphoma; chronic liver disease; use of certain drugs: Methodological interference (pancreozymin [contains lipase], deoxycholate, glycocholate, taurocholate [prevents inactivation of enzyme], bilirubin [turbidimetric method]); after endoscopic retrograde cholangiopancreatography (ERCP)

Decreased Levels

In association with methodological interference (presence of hemoglobin, quinine, heavy metals, calcium ions)

Magnesium (Mg)

Reference Range

1.8–3.0 mg/dL (0.75–1.25 mmol/L)
Panic values: <0.5 or > 4.5 mg/dL (<0.2 or >1.85 mmol/L)

Clinical Implications

Increased Levels

Dehydration; tissue trauma; renal failure; hypoadrenocorticism; hypothyroidism; certain medications (aspirin – prolonged use, lithium, magnesium salts, progesterone, triamterene).

Decreased Levels

Chronic diarrhea; enteric fistula; starvation; chronic alcoholism; TPN with inadequate replacement; hypoparathyroidism (especially post thyroid surgery); acute pancreatitis; chronic glomerulonephritis; hyperaldosteronism; diabetic ketoacidosis; CHF; critical illness; Gitelman syndrome (familial hypokalemia-hypomagnesemia-hypocalciuria), hereditary isolated magnesium wasting, induced hypothermia, certain medications (albuterol, amphotericin B, calcium salts, cisplatin, citrates (blood transfusion), cyclosporine, diuretics, ethacrynic acid).

Osmolality (Serum Osmolality)

Reference Range

285–293 mOsm/kg H_2O
Panic values: <240 mOsm/kg or >320 mOsm/kg

Clinical Implications

Increased Levels

Diabetic ketoacidosis; nonketotic hyperosmolar hyperglycemic coma; hypernatremia secondary to dehydration (diarrhea, severe burns, vomiting, fever, hyperventilation, inadequate water intake, central or nephrogenic diabetes insipidus, or osmotic diuresis); hypernatremia with normal hydration (hypothalamic disorders, defective osmostat); hypernatremia with overhydration (iatrogenic or accidental excessive NaCl or $NaHCO_3$ intake); alcohol or other ingestion; hypercalcemia; tube feedings; certain medications (corticosteroids, mannitol, glycerin).

Decreased Levels

Pregnancy (third trimester); hyponatremia with hypovolemia (adrenal insufficiency, renal losses, diarrhea, vomiting, severe burns, peritonitis, pancreatitis); hyponatremia with normovolemia (SIADH); hyponatremia with hypervolemia (CHF, cirrhosis, nephrotic syndrome, postoperative state); certain medications (chlorthalidone, cyclophosphamide, thiazides).

Phosphate (PO_4), Phosphorus (P)

Reference Range

2.5–4.5 mg/dL (0.8–1.45 mmol/L)
Panic values: <1 mg/dL (<0.32 mmol)

Clinical Implications

Increased Levels

Renal failure; calcific uremic arteriolopathy (calciphylaxis): tumor lysis syndrome; massive blood transfusion; hypoparathyroidism; sarcoidosis; neoplasms; adrenal insufficiency; acromegaly; hypervitaminosis D; osteolytic

metastases to bone; leukemia; milk-alkali syndrome; healing bone fractures; pseudohypoparathyroidism; diabetes mellitus with ketosis; malignant hyperpyrexia; cirrhosis; lactic acidosis; respiratory; certain drugs (phosphate infusions or enemas, anabolic steroids, ergocalciferol, furosemide, hydrochlorothiazide, clonidine, verapamil, potassium supplements, and others). Thrombocytosis may cause spurious elevation of serum phosphate, but plasma phosphate levels are normal.

Decreased Levels

Hyperparathyroidism; hypovitaminosis D (rickets, osteomalacia); malabsorption (steatorrhea); malnutrition, starvation or cachexia; refeeding syndrome; bone marrow transplantation; renal phosphate wasting due to autosomal dominant hypophosphatemic rickets; GH deficiency; chronic alcoholism; severe diarrhea; vomiting; nasogastric suction; acute pancreatitis; severe hypercalcemia (any cause); acute gout; osteoblastic metastases to bone; severe burns (diuretic phase); respirator alkalosis; hyperalimentation with inadequate phosphate repletion; carbohydrate administration (e.g., IV D50W glucose bolus); renal tubular acidosis and other renal tubular defects; diabetic ketoacidosis (during recovery); acid-base disturbances; hypokalemia; pregnancy; hypothyroidism; hemodialysis; certain drugs (acetazolamide, β-adrenergic agonists, catecholamines, estrogens, isoniazid, oral contraceptives, prolonged use of thiazides, glucose infusion, insulin therapy, salicylates—toxicity).

Potassium (K)

Reference Range

3.5–5.0 mEq/L

(Critical values: <3.0 or >6.0 mEq/L)

Clinical Implications

Increased Levels (Hyperkalemia)

Excessive dietary or IV intake; acute or chronic renal failure; rhadomyolysis; exercise (transient); renal tubular acidosis type IV (hyporeninemic) hypoaldosteronism; (hyperkalemic) familial periodid paralysis; Addison disease; hypoaldosteronism; aldosterone-inhibiting diuretics (spironolactone, triamterene); crush- or cell-damaging injuries (accidents, burns, surgery, chemotherapy); hemolysis; acidosis; dehydration; certain drugs (potassium salts, potassium-sparing diuretics such as spironolactone, triamterene, eplerenone, nonsteroidal anti-inflammatory drugs, β-blockers, ACE inhibitors, ACE-receptor blockers, high-dose trimethoprimsulfamethoxazole).

Decreased Levels (Hypokalemia)

Deficient dietary or IV intake; burns/trauma/surgery; diarrhea/vomiting/sweating; diuretics; hyperaldosteronism; Cushing syndrome; licorice ingestion; renal tubular acidosis type I and II; alkalosis; glucose administration; osmotic diuresis; (hypokalemic) familial periodic paralysis; subarachnoid hemorrhage; genetic hypokalemic salt-losing tabulopathies such as Gitelman syndrome (familial hypokalemia-hypocalciuria-hypomagnesemia); CF; certain drugs (adrenergic agents, diuretics).

Prealbumin (PAB; Thyroxine-Binding Prealbumin [TBPA], Thyretin, Transthyretin)

Reference Range

15–36 mg/dL or 150–306 mg/L

Clinical Implications

Increased Levels

Hodgkin lymphoma; pregnancy; kidney failure.

Decreased Levels

Malnutrition; liver damage; trauma or burn injury; inflammation; serious infection; hyperthyroidism; certain digestive diseases; serious or long-term illness such as cancer.

Prothrombin Time (Pro-Time, International Normalized Ratio [INR])

Reference Range

11.0–15.0 seconds
INR of 0.8–1.1
Therapeutic range for patients on warfarin 2.0–3.0

Clinical Implications

Increased Levels (Prolonged PT)

Liver disease (hepatitis, cirrhosis); Warfarin; hereditary factor deficiency (factors II, V, VII, X); fibrinogen abnormality (e.g., hypofibrinogenemia, afibrinogenemia, dysfibrinogenemia); circulating anticoagulant affecting the PT system (rarely lupus anticoagulant); vitamin K deficiency; disseminated intravascular coagulopathy (DIC); bile duct obstruction; coumarin ingestion; massive blood transfusion; salicylate intoxication.

Decreased Levels

Thrombophlebitis, MI, pulmonary embolism; recombinant factor FVII (Novoseven) treatment.

Red Blood Cell Count (RBC Count; Erythrocyte Count)

Reference Range

RBC × 10^6/µL or RBC × 10^{12}/L
Adult male: 4.3–6.0 × 10^6/mcL
Adult female: 3.5–5.5 × 10^6/mcL

Clinical Implications

Increased Levels

Erythrocytosis; congenital heart disease; severe COPD; polycythemia vera; severe dehydration; hemoglobinopathies; high altitude; spurious increase with increased white blood cells.

Decreased Levels

Anemia; cirrhosis; hemorrhage; Addison disease; renal disease; bone marrow failure; pregnancy; rheumatoid/collagen vascular diseases (RA, systemic lupus erythematosus [SLE], sarcoidosis); lymphoma/leukemia/Hodgkin lymphoma; chronic infections; excessive IV fluids; spurious decrease with autoagglutination (e.g., cold agglutinins).

Sodium (Na)

Reference Range

136–145 mEq/L or 136–145 mmol/L

(Critical values: <125 or >155 mEq/L)

Clinical Implications

Increased Levels (Hypernatremia)

Increased sodium intake (dietary or IV); decreased sodium loss (Cushing syndrome, hyperaldosteronism); excessive free body water loss (GI, excessive sweating, vomiting, diarrhea; extensive burns, diabetes insipidus, osmotic diuresis, polyuria due to diabetes mellitus or diabetes insipidus); inadequate water intake (coma, hypothalamic disease); certain drugs (steroids, licorice, oral contraceptives).

Decreased Levels (Hyponatremia)

Decreased sodium intake (deficient dietary or IV sodium); increased sodium loss (Addison disease, diarrhea/vomiting, intraluminal bowel loss, diuretic chronic renal insufficiency, gastric suctioning); increased free body water (excessive oral or IV water intake, hyperglycemia, CHF, peripheral edema, pleural effusion, SIADH); certain drugs (thiazides, diuretics, ACE inhibitors, chlorpropamide, carbamazepine, antidepressants—selective serotonin reuptake inhibitors, antipsychotics).

Total Iron-Binding Capacity (TIBC)

Reference Range

255–460 µg/dL or 45–82 µmol/L

Clinical Implications

Increased Levels

Estrogen therapy; polycythemia vera; pregnancy (late); infancy; iron-deficiency anemia; acute and chronic blood loss; acute hepatitis, certain drugs (oral contraceptives).

Decreased Levels

Hypoproteinemia (nephrotic syndrome, starvation, malnutrition, cancer); hemochromatosis; thalassemia; hyperthyroidism; chronic infections; inflammatory diseases; cirrhosis; hemolytic, pernicious, and sickle cell anemias; chronic disease.

Transferrin

Reference Range

200–400 mg/dL

Clinical Implications

Increased Levels

Estrogen therapy; polycythemia vera; pregnancy (late); iron-deficiency anemia.

Decreased Levels

Hypoproteinemia (malnutrition or burns); disorders associated with inflammation or necrosis, chronic inflammation or malignancy; cirrhosis; hemolytic, pernicious, and sickle cell anemias; atransferrinemia (rare); renal disease; nephrotic syndrome; iron overload states; acute liver disease.

Vitamin B_{12} (Cyanocobalamin)

Reference Range

170–820 pg/mL or 121–600 pmol/L

Clinical Implications

Increased Levels

Leukemia (acute myelocytic, chronic myelocytic, chronic lymphocytic, monocytic); polycythemia vera; myeloproliferative disease. (Increased B_{12} levels are not diagnostically useful.)

Decreased Levels

Pernicious anemia; pregnancy; malabsorption syndromes (sprue, celiac disease, steatorrhea, regional enteritis, fistulas, bowel resection, ileal disease, *Diphyllobothrium latum* [fish tapeworm] infestation, small bowel bacterial overgrowth); HIV infection (with or without malabsorption); chronic high-flux hemodialysis, Alzheimer disease; Zollinger–Ellison syndrome; dietary deficiency; folic acid deficiency; vitamin C deficiency; achlorhydria; large proximal gastrectomy; gastric carcinoma; certain drugs (omeprazole, metformin, carbamazepine).

White Blood Count and Differential Count (WBC with Differential)

Reference Range

Total WBCs: 4,500–11,000/mm^3 or 4.5–11 (10^9/L)
(Critical values: <2,500 or >30,000/mm^3)
Lymphocytes: 1,000–4,000/mm^3 (comprise 20%–40% of the total WBC; and in malnutrition, lymphocyte count is reduced)

Clinical Implications

Increased WBC Count (leukocytosis)

Acute infection; leukemic neoplasia or other myeloproliferative disorders; solid tumor (paraneoplastic reaction); G-CSF stimulation; trauma/stress/hemorrhage; tissue necrosis; inflammation; thyroid storm; steroid use; allergies and hypersensitivity reactions; stress; smoking.

Decreased WBC Count (leukopenia)

Infections; constitutional and acquired myeloid hypoplasia, myelosuppression (e.g., chemotherapy, radiation, various drugs); myelodysplasia; collagen vascular diseases; hypersplenism; cyclic neutropenia; autoimmune neutropenia; alcoholism.

Laboratory Test Panels

Laboratory tests are often ordered as panels that are disease or organ specific. Next are some common test panels that have significance to the nutrition assessment. Note that panels may be modified or expanded at different clinical facilities.

Anemia Panel

Complete blood cell (CBC); RBC indices; reticulocyte count
Microcytic: erythrocyte sedimentation rate (ESR); iron panel
Normocytic: ESR; hemolysis profile
Macrocytic: vitamin B_{12}; folate; thyroid-stimulating hormone (TSH)

Basic Metabolic Panel (7 Channel/Chem 7/SMA-7)

Carbon dioxide; chloride, creatinine, glucose, potassium, sodium, urea nitrogen, (BUN).

Complete Blood Cell Count with Differential (Diff)

RBC; Hgb; Ht; red blood cell indices (mean corpuscular volume [MCV], mean corpuscular hemoglobin [MCH], mean corpuscular hemoglobin concentration [MCHC], red blood cell distribution width [RDW]); WBC and Diff count (neutrophils, lymphocytes, monocytes, eosinophils, basophils); blood smear; platelet count; mean platelet volume (MPV).

Comprehensive Metabolic Panel (12 Channel/Chem 12)

Albumin; alkaline phosphatase; alanine aminotransferase (ALT) (serum glutamic-pyruvic transaminase [SGPT]), aspartate aminotransferase (AST) (serum glutamic oxaloacetic transaminase [SGOT]); bilirubin-total; BUN; calcium; carbon dioxide; chloride; creatinine; estimated glomerular filtration rate (eGFR), glucose; potassium; protein-total; sodium; total protein.

Diabetes Mellitus Management

Anion gap; basic metabolic panel; hemoglobin A1C; lipid profile.

Hepatic Function

ALT; albumin; alkaline phosphatase; AST; bilirubin-direct; bilirubin-total; GGT; protein-total; prothrombin time (PT).

Lipid Panel

Cholesterol-total; high-density lipoprotein (HDL); triglyceride; low-density lipoprotein (LDL)
May also contain very-low-density lipoprotein (VLDL); total cholesterol to HDL ratio, LDL-to-HDL ratio.

Pancreatic Panel

Amylase; calcium; glucose; lipase; triglyceride.

Renal Panel

Albumin; basic metabolic panel; calcium; CBC; magnesium; phosphorus. May also contain estimated GFR; protein-total; protein-urine; protein-24-hour urine.

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[a]Because about one-half of blood calcium is bound to albumin, when albumin levels are low, the serum calcium will also be low. Calcium levels can be adjusted with the following equation when serum albumin is low: Corrected calcium = total calcium mg/dL + 0.8 [4 – serum albumin g/dL].

[b]Note: See Cardiovascular Disease chapter for more information on lipoprotein values.

[c]Note: Reduced muscle mass in elderly and young patients may cause decreased values, which may mask renal disease in these age groups.

[d]Note: See Chapter 2 for detailed information on glucose screening for gestational diabetes.

[e]Note: Values may be slightly decreased in the elderly.

[f]Note: Values may be slightly decreased in the elderly.

Appendix B

Food–Drug Interactions

Kelly Sanna-Gouin, RD, CNSC

The following table of medications is not meant to be comprehensive but instead lists common medications that have significant nutritional implications. The list is alphabetical by generic name and cross-referenced by brand or trade name.

Medication	Class and Action	Side Effects	Nutritional Implications
Adalat	See *nifedipine*		
Aldactone	See *spironolactone*		
Apresoline	See *hydralazine*		
Atenolol Tenormin	β-Blocker, antiadrenergic, antiarrhythmic	Diarrhea, constipation, nausea, and vomiting. Possible hypoglycemia. Signs of sympathetic response to hypoglycemia may be masked. May have a decreased insulin release in response to hyperglycemia. Concurrent use of β-adrenergic blockers and St. John's Wort, Ma-huang (ephedra), or yohimbine may result in a decrease in β-adrenergic blocker effectiveness. Concurrent use of β-adrenergic blockers and Fumitory, Lily of the Valley, or Night-blooming Cereus may result in increased effect of β-adrenergic blocker. Consumption of large amounts of orange juice may decrease the effectiveness of atenolol.	Adherence necessary for those following diabetic diet. Monitor blood glucose levels. Be cautious of nonsympathetic signs of hypoglycemia. Consider fluid and electrolyte replacement for diarrhea and vomiting.
Atorvastatin Lipitor	Inhibitor of HMG-CoA reductase	Constipation, diarrhea, gas, upset stomach, and upper right stomach pain. Coenzyme Q10 may be significantly reduced. Concurrent use of atorvastatin and: ■ Niacin may result in an increased risk of myopathy or rhabdomyolysis. ■ St. John's wort may result in reduced effectiveness of atorvastatin. ■ Black cohosh may result in elevated liver enzymes. ■ Echinacea may result in increased hepatotoxic effects. ■ Grapefruit juice or grapefruit-containing foods may cause increased bioavailability of atorvastatin, resulting in an increased risk of myopathy or rhabdomyolysis. ■ Concurrent use of Red Yeast Rice may enhance the adverse/toxic effect of HMG-CoA Reductase Inhibitors.	Avoid intake of large quantities of grapefruit juice or grapefruit-containing foods, for it increases the absorption of statin (>1 qt/d). Eat a low-cholesterol, low-fat diet for best results. Gastrointestinal (GI) problems are generally transient.

Description

Azithromycin Zithromax	Antibiotic Bacteriostatic or bacteriocidal	Occasional nausea, vomiting, diarrhea, abdominal pain, anorexia, stomatitis, bad taste in mouth. Increased serum potassium (1–2%), decreased serum bicarbonate (adults: ≥1%), decreased serum glucose (adults: >1%)	Take with food to avoid GI disturbances. Azithromycin oral suspension should be taken 1 hr before or 2 hr after meals. Eat small, frequent meals to avoid anorexia. Consider fluid and electrolyte replacement for diarrhea. Avoid alcohol.
Bactrim, Bactrim DS	See *sulfamethoxazole*		
Benazepril Lotensin	Antihypertensive ACE inhibitor	Nausea, vomiting, pancreatitis. May increase serum potassium. May decrease serum sodium. Concurrent use of angiotensin-converting enzyme inhibitors and yohimbine or ma huang may result in reduced effectiveness of angiotensin-converting enzyme inhibitors.	Caution with foods high in potassium or potassium supplements. Avoid salt substitutes. Maintain adequate hydration.
Betaloc	See *metoprolol*		
Betamethasone	See corticosteroid		
Biaxin	See *clarithromycin*		
Bumetanide Bumex	Diuretic Loop diuretic	May cause nausea and diarrhea. Ginkgo biloba may cause increased blood pressure. Licorice, bitter melon, can increase the risk of hypokalemia. Goldenrod, forskolin, fo-ti root, birch, cornsilk, maitake, increases effect of bumetanide. Juniper may potentiate or interfere with diuretic therapy. Bumetanide may increase the toxicity of lily of the valley. May increase blood glucose, uric acid, cholesterol, LDL, calcium, and triglycerides. May decrease urinary excretion of calcium and increase excretion of magnesium, sodium, and potassium levels.	Consume foods high in potassium and magnesium. Avoid consumption of natural licorice. Monitor electrolyte levels and consider supplementation. Caution with calcium supplements.

Description

Bupropion Wellbutrin, Zyban	Antidepressant Serotonin and norepinephrine reuptake inhibitor	Dry mouth, upset stomach, vomiting, dysphagia, weight loss, and constipation. Alcohol may increase side effects. Caffeine increases risk of bupropion toxicity. Ethanol and bupropion increase the toxicity of each other.	Monitor weight. Use ice chips or chew gum for dry mouth. Avoid alcohol.
Bumex	See *bumetanide*		
Calan	See *verapamil*		
Calcijex	See *calcitriol*		
Calcium salts PhosLo, Caltrate, Dicarbosil, OsCal, Titralac, Tums, Citracal, Calcitrate	Calcium-based phosphorus binder	Hypercalcemia, stomach pains, flatulence, nausea, vomiting, constipation, dry mouth, thirst, and frequent urination. Concurrent use of calcium and oxalic acid foods (spinach, rhubarb, beet greens, asparagus, etc.) may result in decreased calcium exposure. Concurrent use of calcium and phytic acid foods (beans, seeds, nuts, whole grains, etc.) may result in decreased calcium effectiveness.	Maintain adequate hydration, serum magnesium, phosphate, potassium levels, and urine calcium levels.
Calcitrate	See *calcium salts*		
Calcitriol Calcijex, Rocaltrol	Vitamin D	Nausea, vomiting, dehydration, dry mouth, constipation, metallic taste in mouth, increased thirst, decreased appetite, weight loss, and fatty stools. May increase aluminum concentration, hypercalcemia, serum cholesterol, serum phosphorous, and magnesium concentration.	Avoid use of antacids. Only works in conjunction with appropriate intake of calcium. Consider low-phosphate diet if on dialysis. Can cause growth suppression.
Caltrate	See *calcium salts*		
Camzyos	See mavacamten		
Capoten	See *captopril*		
Capozide	See *captopril*		

Description

Captopril	Antihypertensive ACE inhibitor	Nausea, salty or metallic taste, mouth sores. May increase serum potassium. May decrease serum sodium. Concurrent use of angiotensin-converting inhibitors and yohimbine, ma huang, calcium carbonate may result in reduced effectiveness of angiotensin-converting enzyme inhibitors.	Caution with foods high in potassium or potassium supplements. Avoid salt substitutes. Maintain adequate hydration.
Catapres	See *clonidine*		
Celexa	See *citalopram*		
Cholestyramine Questran, Prevalite	Bile acid sequestrant	Constipation, nausea, vomiting, abdominal pain, indigestion. Occasional diarrhea. May decrease serum potassium and calcium. Binds fat-soluble vitamins A, D, E, and K; folic acid; and β-carotene.	Mix with 3–6 oz of liquid, such as juice, milk, or water for powdered form. Irritating to GI tract. Take before meals. Take fat-soluble vitamins in a water-miscible form or take a supplement prior to initial daily dose of drug. Monitor nutrient levels if long-term use of drug is indicated. Consider high-fiber diet for constipation.
Cinacalcet Sensipar	Calcimimetic	High fat intake may increase cinacalcet concentration of plasma. Anorexia, nausea, vomiting, and diarrhea. Concurrent use of cinacalcet and DHEA, food may result in increased bioavailability of cinacalcet. May cause serum hypocalcemia. Concurrent use of cinacalcet and eucalyptus, grapefruit juice, or grapefruit-containing foods may increase the effects of cinacalcet.	Avoid taking with grapefruit juice or grapefruit-containing foods.

Description

Ciprofloxacin	Antibacterial	Upset stomach, vomiting, stomach pain, indigestion, altered taste, hiccups. Inflammation of the stomach leading to possible diarrhea. Aluminum, magnesium, calcium, ferrous sulfate, and zinc are thought to form chelation complexes, preventing the drugs from being absorbed. May decrease the level or effectiveness of vitamin K1. Concurrent use of fluoroquinolones and dandelion may result in decreased fluoroquinolone effectiveness. Concurrent use of ciprofloxacin and: ■ Iron may result in decreased ciprofloxacin effectiveness. ■ Fennel may result in decreased bioavailability of ciprofloxacin and possible antibiotic treatment failure. ■ Zinc may result in decreased ciprofloxacin effectiveness. ■ Caffeine may result in increased caffeine concentrations and enhanced central nervous system stimulation.	Take with 8 oz of water. Ensure adequate fluid intake. Consider fluid and electrolyte replacement for vomiting and diarrhea. Should not be taken with dairy products or calcium-containing fluids. Do not need to avoid foods containing these products.
Cisplatin Platinol, Platinol-AQ	Alkylating agent	Loss of appetite, weight loss, diarrhea, nausea, vomiting, hiccups, stomatitis, pancreatitis, and altered taste.	Drink plenty of fluids for drug; irritates the kidney. Vomiting is severe. May consider antiemetic therapy. Monitor weight. Encourage food intake when patient feels best (e.g., morning).
Citalopram Celexa	Antidepressant Selective serotonin reuptake inhibitor (SSRI)	Nausea, diarrhea, vomiting, anorexia, dry mouth, and dyspepsia. May decrease serum sodium. Ethanol and celexa increase toxicity of the other. Fish oil triglycerides will increase the level or effect of citalopram by anticoagulation. Concurrent use of SSRIs and St. John's wort, Ginkgo biloba may result in an increased risk of serotonin syndrome (hypertension, hyperthermia, myoclonus, mental status changes).	Consider fluid and electrolyte replacement for diarrhea and vomiting. Use ice chips or chew gum for dry mouth. Avoid alcohol.
Citracal	See *calcium salts*		

Description

Clarithromycin Biaxin	Antibiotic Bacteriostatic or bacteriocidal	Occasional nausea, vomiting, glossitis, diarrhea, abdominal pain, anorexia, stomatitis, bad taste in mouth, hypoglycemia, pancreatitis. Calrithromycine will increase the level or effect of eucalyptus. Grapefruit, DHEA will increase the level or effect of clarithromycin. St. John's wort will decrease the level or effect of clarithromycin.	Take with food to avoid GI disturbances. Eat small, frequent meals to avoid anorexia. Consider fluid and electrolyte replacement for diarrhea. Avoid alcohol.
Clonidine Catapres, Duraclon	α-Antiadrenergic	Constipation, nausea, vomiting, dry mouth, and drowsiness. Ethanol and clonidine increase the effects of each other. Clonidine may also mask hypoglycemia. Concurrent use of clonidine and yohimbine may result in reduced clonidine effectiveness. Use of Cornsilk, maitake, reishi, increases effects of clonidine. Concurrent use of clonidine and ma huang, shepherd's purse, may result in increased blood pressure.	Avoid alcohol intake, for it can exacerbate drowsiness. Use ice chips or chew gum for dry mouth.
Clopidogrel Plavix	Antiplatelet	Upset stomach, stomach pain, diarrhea, and constipation. Ginger may increase the possibility of bleeding. Concurrent use of clopidogrel and Ginkgo biloba, chaparral (also known as creosote bush), bladderwrack (type of seaweed), vitamin A, kava, licorice, clove oil, garlic, anise, boldo, motherwort, cat's claw, bilberry, bogbean, curcumin, celery, evening primrose, black currant, borage, feverfew, dandelion, angelica, meadowsweet, astraglus, tan-shen (red sage), skullcap, guggul, hawthorn, arnica, green tea, horse chestnut seed, or ginger may result in an increased risk of bleeding. Concurrent use of clopidogrel and grapefruit juice or grapefruit-containing foods may result in reduced exposure of the active clopidogrel metabolite.	Consider small, frequent meals for anorexia. Consider fluid and electrolyte replacement for diarrhea. Consistency in dietary and supplemental intake must be consistent to achieve steady level of anticoagulation. Those on long-term use should be monitored for bone density. Avoid taking with grapefruit juice or grapefruit-containing foods.

Description

Corticosteroids *Dexamethasone* (Decadron, Hexadrol) *Hydrocortisone/ cortisol* (Cortef) *Methylprednisolone* (Depo-Medrol, Medrol, Solu-Medrol) *Prednisolone* *Prednisone* (Deltasone) *Triamcinolone* (Aristocort, Kenalog)	Anti-inflammatory, immunosuppressant, hormone	May cause GI symptoms such as esophagitis, nausea, vomiting. May increase risk of GI bleeding. May cause edema, bruising, seizure, elevated blood pressure, acne, rash, slow wound healing, insomnia, psychological disturbances, masking of infection, weakness, headache. Long term use can cause osteoporosis, pancreatitis, cataracts, Cushing's syndrome, decreased growth velocity in children. Avoid alcohol. Serum albumin < 3 g/dL can result in increased drug effects. Can increase serum glucose levels. Other common effects on serum-based labs: elevated Na, low K, low Ca, low Zn, low uric acid, low vit C and vit A, elevated TG, elevated cholesterol, decreased T3 & T4, decreased TSH, increased hgb, increased RBC	Reduced sodium diet indicated. Increases protein needs if not otherwise contraindicated. May also increase needs for potassium, Vitamins A, C, and D, folic acid, calcium, and phosphorus. Increased appetite and wt gain (except with triamcinolone which can cause anorexia).
Cotrimoxazole/ trimethoprim/ Sulfamethoxazole Bactrim, Bactrim DS	Bactericidal	May cause anorexia, nausea, vomiting, diarrhea, abdominal pain, or stomatitis. May hinder folate metabolism. Trimethoprim may increase serum potassium. Willow bark increases levels of sulfamethoxazole.	Take with 8 oz of water on an empty stomach. Eat small, frequent meals to avoid anorexia. Take a folate supplement. Consider fluid and electrolyte replacement for diarrhea.
Coumadin	See *warfarin*		
Covera-HS	See *verapamil*		
Cyclophosphamide Cytoxan	Alkylating agent, nitrogen mustard	Nausea, vomiting, loss of appetite, and weight loss. Concurrent use of cyclophosphamide and St. John's wort may result in reduced cyclophosphamide effectiveness.	Drink plenty of fluids for drug; irritates bladder and kidneys. Encourage food intake when patient feels best (e.g., morning).
Cytoxan	See *cyclophosphamide*		
Dexamethasone	See corticosteroid		

Description

Dialyvite Diatx, Nephrocaps, Nephrovite	B-complex with vitamin C and biotin	Abdominal pain, cramps, dyspepsia, and nausea.	Taken when vitamins are inadequate, usually after dialysis when fluid is removed. Used to replace water-soluble vitamin losses.
Diatx	See *Dialyvite*		
Dicarbosil	See *calcium salts*		
Digoxin Lanoxin	Antiarrhythmic, cardiac glycoside	Occasional diarrhea, loss of appetite, lower stomach pain, nausea, and/or vomiting. May reduce potassium levels and increase urinary excretion of magnesium. Concurrent use of digoxin and St. John's wort, khella, charcoal, and kaolin may result in reduced digoxin efficacy. Concurrent use of digoxin and calcium may result in a serious risk of arrhythmia and cardiovascular collapse. Concurrent use of digoxin and oleander, sea cucumber, carob, licorice, lily of the valley, Pheasant's eye, Chan-Su, cascara sagrada (buckthorn), aloe, squill, hawthorn, or senna may result in increased risk of digoxin toxicity. The use of Siberian ginseng, tan-shen, or ashwagandha may result in falsely elevated digoxin levels.	Hypomagnesemia, hypokalemia, and hypercalcemia elevate drug toxicity. Ensure adequate consumption if potassium and magnesium. Care should be taken with calcium supplements and antacids.
Doxercalciferol Hectorol	See *calcitriol*		
Duraclon	See *clonidine*		
Dyrenium	See *triamterene*		
Effexor, Effexor XR	See *venlafaxine*		

Description

Enalapril Vastotec	Antihypertensive ACE inhibitor	Nausea, salty or metallic taste, mouth sores. May increase serum potassium. May decrease serum sodium. Concurrent use of angiotensin-converting enzyme inhibitors and yohimbine, ma huang, calcium carbonate may result in reduced effectiveness of angiotensin-converting enzyme inhibitors. Concurrent use of rose hips may cause hypotension.	Caution with foods high in potassium or potassium supplements. Avoid salt substitutes. Maintain adequate hydration.
Ery-Tab	See *erythromycin*		
Erythromycin Ery-Tab	Antibiotic Bacteriostatic or bacteriocidal	Occasional nausea, vomiting, diarrhea, abdominal pain, anorexia, stomatitis, bad taste in mouth. Concurrent use of erythromycin and eucalyptus or autumn crocus (colchicine) may result in increased plasma levels of eucalyptus or autumn crocus and increased risk of toxicity. Concurrent use of erythromycin and grapefruit juice or grapefruit-containing foods, or calcium carbonate may result in increased erythromycin bioavailability.	Take with food to avoid GI disturbances. Eat small, frequent meals to avoid anorexia. Consider fluid and electrolyte replacement for diarrhea. Avoid alcohol. Avoid taking with grapefruit or grapefruit-containing foods.
Eskalith, Eskalith CR	See *lithium*		
Extentabs	See *quinidine*		
Extentabs	See *quinidine*		
Feostat	See *ferrous fumarate*		
Femiron	See *ferrous fumarate*		
Fenofibrate Tricor	See *gemfibrozil*		
Feosol	See *ferrous sulfate*		
Feratab	See *ferrous sulfate*		

Description

Fergon	See *ferrous sulfate*		
Ferrex	See *ferrous sulfate*		
Ferrlecit	See *sodium ferric gluconate*		
Ferrous fumarate Femiron, Feostat	Iron supplement	Constipation, diarrhea, and abdominal discomfort, dark stools. Concurrent use of aluminum hydroxide, sodium citrate, sodium bicarbonate, vitamin E (oral form), or calcium carbonate will decrease level or effect of ferrous fumarate. Ferrous fumarate will decrease absorption of manganese if taken together. When possible, do not consume green tea or green tea extract 1 hour before or 2 hours after giving iron salts.	Emphasize iron-rich food in a well-balanced diet.
Ferrous sulfate Feosol, Feratab, Fergon, Ferrex, Hemocyte, Nephro-Fer, Niferex	Iron supplement	Constipation, diarrhea, vomiting, abdominal discomfort, dark stools, and stomach upset. Concurrent use of iron and zinc may result in decreased gastrointestinal absorption and iron and/or zinc. Concurrent use of iron and dairy foods may result in decreased iron bioavailability. Concurrent use of iron and phytic acid foods (beans, seeds, nuts, some whole grains) may result in reduced iron absorption. Concurrent use of aluminum hydroxide, sodium citrate, sodium bicarbonate, vitamin E (oral form), or calcium carbonate will decrease level or effect of ferrous fumarate. Ferrous fumarate will decrease absorption of manganese if taken together. When possible, do not consume green tea or green tea extract 1 hour before or 2 hours after giving iron salts.	
Ferrous gluconate	See *ferrous sulfate*		

Description

Flagyl	See *metronidazole*		
Fluoxetine Prozac	Antidepressant Selective serotonin reuptake inhibitor (SSRI)	Nausea, diarrhea, decreased appetite, dry mouth, vomiting, constipation, and abdominal pain. Alcohol may increase depression. Concurrent use of fish oil, panax ginseng may increase fluoxetine level or effect by anticoagulation. Concurrent use of green tea or green tree extract may increase risk of bleeding. Concurrent use of pleurisy root decreases the effect of fluoxetine. Concurrent use of SSRIs and St. John's wort, SAMe, or Ginkgo biloba may result in increased risk of serotonin syndrome (hypertension, hyperthermia, myoclonus, mental status changes).	Avoid alcohol. Consider fluid and electrolyte replacement for diarrhea and vomiting. Use ice chips or chew gum for dry mouth. Consider small, frequent meals for decreased appetite.
Fluvastatin	See *atorvastatin*		
Lescol *Furosemide* Lasix	Diuretic Loop diuretic	Loss of appetite, abdominal cramping, nausea, vomiting, diarrhea, constipation, oral irritation, gastric irritation, pancreatitis. Ginkgo biloba may cause increased blood pressure. Shepherd's purse may cause a decrease in blood pressure. Licorice can increase the risk of hypokalemia. May increase blood glucose, uric acid, cholesterol, LDL, calcium, and triglycerides. May decrease urinary excretion of calcium and increase excretion of magnesium, sodium, and potassium levels. Concurrent use of loop diuretics and ginseng or geranium may result in increased risk of diuretic resistance. Concurrent use of loop diuretics and yohimbine may result in reduced diuretic effectiveness. Concurrent use of cornsilk, maitake, agrimony, birch, bitter melon, fo-ti root, forskolin, goldenrod, reishi, increases the effects of furosemide.	Consume foods high in potassium and magnesium. Avoid consumption of natural licorice. Monitor electrolyte levels and consider supplementation. Caution with calcium supplements.
		Concurrent use of geranium decreases the effects of furosemide. Concurrent use of squill, lily of the valley, increases their toxicity by potassium depletion.	

Description

Gemfibrozil Lopid	Fibric acid derivative	Stomach pain, diarrhea, constipation, vomiting, and gas.	Eat a low-cholesterol, low-fat, low-sucrose diet for best results. Avoid alcohol. Consider fluid and electrolyte replacement for diarrhea and vomiting.
Hectorol	See *doxercalciferol*		
Hydralazine Apresoline	Antiarrhythmic Antiprotozoal	Nausea, vomiting, diarrhea, fluid retention, and edema. Impedes metabolism of pyridoxine (vitamin B_6). Concurrent use of yohimbe may result in hypotension. Concurrent use of agrimony, cornsilk, forskolin, maitake, reishi, increases the effects of hydralazine. Use of shepherd's purse may affect blood pressure control. Concurrent use of hydralazine and ma huang may result in reduced hydralazine effectiveness.	Doctor may prescribe a low-salt or low-sodium diet. Take with food. Monitor for pyridoxine deficiency. Consume diet high in pyridoxine. Consider supplementation.
Hemocyte	See *ferrous sulfate*		
Hydrocortisone	See corticosteroid		
Infed	See *iron dextran*		
Iron dextran Infed	IV iron, hematinic	Nausea, vomiting, diarrhea, abdominal pain, and metallic taste. Concurrent use of iron and zinc may result in decreased GI absorption of iron and/or zinc. Concurrent use of iron and phytic acid foods (beans, seeds, nuts, some whole grains) may result in reduced iron absorption. Use of calcium carbonate, sodium carbonate, citric acid/sodium citrate, aluminum hydroxide, calcium chloride, calcium citrate, calcium gluconate, gymnema, manganese, vitamin E, may interfere with absorption.	Avoid taking oral iron. Emphasize iron-rich food in a well-balanced diet.

Description

Iron sucrose Venofer	IV iron, hematinic	Vomiting, diarrhea, constipation. Use of calcium carbonate, sodium carbonate, citric acid/sodium citrate, aluminum hydroxide, calcium chloride, calcium citrate, calcium gluconate, gymnema, manganese, vitamin E, may interfere with absorption.	Avoid taking oral iron. Emphasize iron-rich food in a well-balanced diet.
Isocarboxazid Marplan	Antidepressant Monoamine oxidase inhibitor (MAOI)	May cause nausea, vomiting, constipation. Sudden high blood pressure may occur with ingestion of certain foods. Alcohol may increase depressant effect. Caffeine may increase blood pressure and cardiac arrhythmias. Valerian root may increase sedation. Concurrent use of MAOIs and: ■ Yerba mate, guarana, or bitter orange may result in acute headache and increased blood pressure. ■ Kava, ma huang, licorice, Ginkgo biloba, SAMe, yohimbe, or nutmeg may result in increased risk of adverse effects from excessive monoamine oxidase inhibition. ■ St. John's wort may result in an increased risk of serotonin syndrome (hypertension, hyperthermia, myoclonus, mental status changes) and/or increased risk of hypertensive crisis. ■ Ginseng may result in insomnia, tremor, headache, agitation, and worsening of depression. ■ Pleurisy root may decrease the effectiveness of isocarboxazid.	Avoid foods high in tyramine: cheeses, fava, or broad bean pods; yeast or meat extracts, smoked or pickled meat, poultry, or fish; fermented sausage such as bologna, pepperoni, salami, or other fermented meat; avocados; bananas; beer; wine; and raisins. Avoid excess amounts of caffeine, tea, or chocolate. Avoid alcohol.
Isoptin, Isoptin SR	See *verapamil*		
Keflex	See *cephalexin*		
Kinidine	See *quinidine*		
Lanoxin	See *digoxin*		
Lasix	See *furosemide*		
Lescol	See *fluvastatin*		

Description

Lipitor	See *atorvastatin*		
Lisinopril Prinivil, Zestril	Antihypertensive ACE inhibitor	Nausea, salty or metallic taste, mouth sores. May increase serum potassium. May decrease serum sodium. Aluminum hydroxide, calcium carbonate, sodium citrate, decreases effectiveness of Lisinopril. Citric acid, sodium bicarbonate decreases the effectiveness of Lisinopril; one may also increase the toxicity of the other. Agrimony, cornsilk, maitake, octacosanol, reishi, increases the effects of Lisinopril. Capscium increases ACE inhibitor-induced cough.	Caution with foods high in potassium or potassium supplements. Avoid salt substitutes. Maintain adequate hydration.
Lithane	See *lithium*		
Lithium Eskalith, Eskalith CR, Lithobid, Lithane, Lithonate, Lithotabs	Antimanic agent	Drug interferes with the regulation of sodium and water levels in the body and may lead to dehydration. Toxicity may result from sodium depletion. Caffeine appears to reduce serum lithium concentrations and increase side effects. Loss of appetite, stomach pain or bloating, gas, indigestion, weight gain or loss, dry mouth, excessive saliva in the mouth, tongue pain, change in the ability to taste food, swollen lips, and constipation. Concurrent use of lithium and: ■ Yohimbine may result in increased risk of manic episodes. ■ Psyllium may result in decreased plasma levels and effectiveness of lithium. ■ Guarana or yerba mate may result in alterations in serum lithium levels. ■ St. John's wort—both increase serum serotonin levels.	Maintain steady salt and fluid intake. Avoid salt-free diet or sodium depletion. Avoid caffeine.

Description

Lithobid	See *lithium*		
Lithonate	See *lithium*		
Lithotabs	See *lithium*		
Lopid	See *gemfibrozil*		
Lopressor	See *metoprolol*		
Marplan	See *isocarboxazid*		
Mavacamten Camzyos	cardiac myosin inhibitor	Dizziness, fainting, heart failure Grapefruit juice may increase the plasma concentrations of mavacamten. St. John's Wort (CYP3A4 inducers) (moderate) may decrease the serum concentration of Mavacamten. Avoid combination.	
Methotrexate	Antimetabolite, folate antagonist	Nausea, vomiting, diarrhea, stomach pain, mouth sores, and loss of appetite. Inhibitor of dihydrofolate reductase, thus decreasing availability of active folate. Concurrent use of methotrexate and cola may result in increased methotrexate serum levels and increased risk of toxicity. Concurrent, folic acid, use of caffeine may decrease the effectiveness of methotrexate. Rose hips, willow bark, maitake, will increase the action of methotrexate. Echinacea, maitake, decreases and methotrexate increases immunosuppression. Effect of interaction is unclear; use caution. Tongkat ali, astragalus, also increases immunosuppression and risk of infection.	Consume high-vitamin B_{12} diet. Consider fluid and electrolyte replacement for diarrhea and vomiting. Encourage food intake when patient feels best (e.g., morning). Drink extra fluids to pass more drug through the urine. Leucovorin should be considered to reverse toxic effect of folic acid antagonists.

Description

Metoprolol Betaloc, Lopressor, Toprol XL	β-Blocker, antiadrenergic, antiarrhythmic	Diarrhea, constipation, nausea, and vomiting. Possible hypoglycemia. Signs of sympathetic response to hypoglycemia may be masked. May have a decreased insulin release in response to hyperglycemia. Melatonin reduces the toxicity of metoprolol. Concurrent use of β-adrenergic blockers and: ■ St. John's wort, citric acid, calcium salts, sodium bicarbonate or yohimbine may result in decreased effectiveness of β-adrenergic blockers. ■ Aluminum hydroxide may result in decreased GI absorption of metoprolol. ■ Dong quai may result in low blood pressure. ■ Ma huang may result in reduced hypotensive effect of β-adrenergic blockers. ■ Cornsilk, agrimony, maitake, reishi, may increase the effects of metoprolol. ■ Shepherd's purse may interfere with blood pressure control.	Adherence necessary for those following diabetic diet. Monitor blood glucose levels. Be cautious of nonsympathetic signs of hypoglycemia. Consider fluid and electrolyte replacement for diarrhea and vomiting.
Metronidazole Flagyl	Antibacterial, antiprotozoal	Anorexia, nausea, dry mouth, stomatitis, diarrhea or constipation, vomiting, and metallic taste. Concurrent use of metronidazole and: ■ Milk thistle may result in reduced metronidazole and active metabolite exposure. ■ Eucalyptus increases the level or effect of eucalyptus.	Avoid alcohol and alcohol-containing products during and at least 5 days after treatment. Avoid hot and spicy foods. Consider fluid and electrolyte replacement for diarrhea. Take with food to prevent GI distress. Consider small, frequent meals for anorexia. Use ice chips or chew gum for dry mouth.
Mounjaro	See tirzepatide		
Nardil	See *phenelzine*		
Nephrocaps	See *dialyvite*		
Nephro-Fer	See *ferrous sulfate*		

Description

Nephrovite	See *dialyvite*		
Niacin Niacor, Niasspan, Nicotinex, Slo-Niacin	Nicotinic acid derivative	Occasional gas, nausea, vomiting, and diarrhea. Alcohol may increase side effects of niacin. May increase blood uric acid and glucose levels.	Caution with diabetes. Ensure adherence to diabetic diet if necessary. May consider low-purine diet if necessary. Consider fluid and electrolyte replacement for diarrhea and vomiting.
Niacor	See *niacin*		
Niaspan	See *niacin*		
Nicotinex	See *niacin*		
Nifedical	See *nifedipine*		
Nifedipine Adalat, Nifedical, Procardia	Calcium channel blocker	Upset stomach, heartburn, nausea, and constipation. Concurrent use of nifedipine and: ■ Ginseng or Ginkgo biloba may result in an increased risk of nifedipine side effects. ■ Dong quai may result in low blood pressure. ■ Ma huang may result in reduced hypotensive effect of calcium channel blockers. ■ Yohimbine may result in reduced calcium channel blocker effectiveness. ■ Grapefruit juice or grapefruit-containing foods may result in severe hypotension, myocardial ischemia, and increased vasodilator side effects. ■ Calcium salts, melatonin, St. John's wort decrease the effects of nifedipine. ■ Agrimony, cornsilk, DHEA, fo-ti, maitake, reishi increase the effects of nifedipine. ■ Eucalyptus will increase the level or effect of nifedipine. ■ Shepherd's purse may interfere with blood pressure control.	Avoid drinking grapefruit juice or eating grapefruit 1 h before or for 2 h after taking nifedipine.
Niferex	See *ferrous sulfate*		

Description

Oncovin	See *vincristine*		
Orlistat Xenical	Lipase inhibitor	Flatulence, fatty stools, nausea, diarrhea. Decreased absorption vitamins A, K, and E.	See effects are transient. Monitor nutrient levels.
OsCal	See *calcium salts*		
Ozempic	See semaglutide injection		
Parnate	See *tranylcypromine*		
Paroxetine Pexeva, Paxil, Paxil CR	Antidepressant Selective serotonin reuptake inhibitor (SSRI)	Nausea, dry mouth, constipation, diarrhea, and decreased appetite. Concurrent use of SSRIs and St. John's wort, 5-htp, SAMe, ginseng, or Ginkgo biloba may result in an increased risk of serotonin syndrome (hypertension, hyperthermia, myoclonus, mental status changes). Green tea as a supplement may increase the risk of bleeding. Valerian may increase sedation. Pleurisy root decreases the effectiveness of paroxetine.	Consider fluid and electrolyte replacement for diarrhea. Use ice chips or chew gum for dry mouth. Consider small, frequent meals for decreased appetite.
Pecxeva	See *paroxetine*		
Paxil, Paxil CR	See *paroxetine*		
Penicillin	Antibiotic Kill or prevent growth of bacteria	Mild diarrhea, nausea, vomiting, stomatitis, black or hairy tongue. Some may contain high amounts of sodium or potassium. Rose hips, willow bark when used with penicillin increase the action of the other.	Should be taken 1 h before or 2 h after food to facilitate absorption. Consider fluid and electrolyte replacement for diarrhea. Caution if on low-sodium diet. Some strengths of amoxicillin may contain phenylalanine.

Description

Paricalcitol Zemplar	See *calcitriol*		
Phenelzine Nardil	Antidepressant Monoamine oxidase inhibitor (MAOI)	Sudden high blood pressure may occur with ingestion of certain foods. Alcohol may increase depressant effect. Caffeine may increase blood pressure and risk of cardiac arrhythmias. Concurrent use of MAOIs and: ■ Yerba mate, guarana, or bitter orange may result in acute headache and increased blood pressure. ■ Kava, ma huang, licorice, Ginkgo biloba, SAMe, or nutmeg may result in increased risk of adverse effects from excessive monoamine oxidase inhibition. ■ St. John's wort may result in an increased risk of serotonin syndrome (hypertension, hyperthermia, myoclonus, mental status changes) and/or an increased risk of hypertensive crisis. ■ Ginseng may result in insomnia, tremor, headache, agitation, and worsening of depression. ■ Avocado may result in hypertensive crisis (headache, palpitation, neck stiffness). ■ Pleurisy root decreases the effects of phenelzine. ■ Yohimbe increases risk of hypertensive episode and may increase the effect of phenelzine.	Avoid foods high in tyramine: cheeses, fava, or broad bean pods; yeast or meat extracts, smoked or pickled meat, poultry, or fish; fermented sausage such as bologna, pepperoni, salami, or other fermented meat; avocados; bananas; beer; wine; and raisins. Avoid excess amounts of caffeine, tea, or chocolate. Avoid alcohol.
PhosLo	See *calcium salts*		
Platinol, Platinol-AQ	See *cisplatin*		
Plavix	See *clopidogrel*		
Pravachol	See *pravastatin*		

Description

Pravastatin Pravachol	Inhibitor of HMG-CoA reductase	Constipation, diarrhea, gas, upset stomach, and upper right stomach pain. Coenzyme Q10 may be significantly reduced. Concurrent use of HMG-CoA reductase inhibitors and oat bran may result in reduced effectiveness of HMG-CoA reductase inhibitors. Concurrent use of pravastatin and St. John's wort may result in reduced effectiveness of pravastatin. Use with red yeast rice is contraindicated; each increases toxicity of the other, increased risk of myopathy or rhabdomyolysis. Niacin and pravastatin increase the toxicity of each other.	Avoid intake of large quantities of grapefruit juice or grapefruit-containing foods, for it increases the absorption of statins (>1 quart/d). Eat a low-cholesterol, low-fat diet for best results. GI problems are generally transient.
Prednisolone	See corticosteroid		
Prevalite	See *cholesyramine*		
Prinivil	See *Lisinopril*		
Procardia	See *nifedipine*		
Propanolol Indeal	See *atenolol*		
Prozac	See *fluoxetine*		
Questran	See *cholestyramine*		
Quinidex	See *quinidine*		

Description

Quinidine Kinidine, Quinidex, Extentabs	Antiarrhythmic, antiprotozoal	Abdominal pain and cramps, diarrhea, nausea, and vomiting. May cause hypokalemia, hypomagnesemia, and/or hypocalcemia. Concurrent use of quinidine and grapefruit juice may result in decreased metabolic conversion of quinidine to 3-hydroxyquinidine. Quinidine use will increase the effect of red yeast rice; increased risk of myopathy and rhabdomyolysis. Quinidine increases the toxicity of squill. St. John's wort, eucalyptus will decrease the level or effect of quinidine. Aluminum hydroxide, calcium carbonate, sodium bicarbonate, sodium lactate, DHEA, grapefruit, hawthorn will increase the level or effect of quinidine. Quinidine and lily of the valley each increase the toxicity of the other.	Consider fluid and electrolyte replacement for diarrhea and vomiting. Consume diet adequate in potassium, magnesium, and calcium. Supplementation may be necessary.
Renagel	See *sevelamer*		
Rocaltrol	See *calcitriol*		
Semaglutide injection Ozempic	incretin mimetic	Nausea, vomiting, diarrhea, abdominal pain, constipation, heartburn, burping. Increased risk of hypoglycemia when taking semaglutide injection while taking insulin. Alcohol may affect blood glucose levels in patients with diabetes. Both hypoglycemia (low blood sugar) and hyperglycemia (high blood sugar) may occur, depending on how much and how often you drink.	

Description

Sensipar	See *cinacalcet*		
Sertraline Zoloft	Antidepressant Selective serotonin reuptake inhibitor (SSRI)	Nausea, diarrhea, dry mouth, constipation, altered taste, and dyspepsia. May increase serum triglyceride and total cholesterol. May decrease uric acid levels. St. John's wort may increase adverse side effects. Concurrent use of SSRIs and St. John's wort, SAMe, panax ginseng, or Ginkgo biloba may result in an increased risk of serotonin syndrome (hypertension, hyperthermia, myoclonus, mental status changes). Concurrent use of sertraline and grapefruit juice or grapefruit-containing foods may result in elevated sertraline serum concentrations and an increased risk of adverse side effects. Fish oil triglycerides may increase the effect of sertraline by anticoagulation. Pleurisy root decreases the effect of sertraline. Concurrent use with green tea may increase the risk of bleeding. Sertraline and valerian root both cause sedation; use with caution.	Monitor blood lipid levels. Consider fluid and electrolyte replacement for diarrhea and vomiting. Use ice chips or chew gum for dry mouth. Avoid alcohol. Avoid grapefruit or grapefruit-containing foods.
Sevelamer Renagel	Non–calcium-based phosphorus binder	Diarrhea, dyspepsia, gas, constipation, nausea, and vomiting.	Take with food. Use of aluminum should be limited to <14 days. Monitor bicarbonate, chloride, calcium, and phosphorus levels.

Description

Simvastatin Zocor	Inhibitor of HMG-CoA reductase	Constipation, diarrhea, gas, upset stomach, and upper right stomach pain. Coenzyme Q10 may be significantly reduced. Concurrent use of HMG-CoA reductase inhibitors and oat bran may result in reduced effectiveness of HMG-CoA reductase inhibitors. Concurrent use of simvastatin and cranberry juice may result in increased risk of hepatitis and myopathy/rhabdomyolysis. Concurrent use of HMG-CoA reductase inhibitors and pectin may result in reduced effectiveness of HMG-CoA reductase inhibitors. Simvastatin and red yeast rice increase the toxicity of each other; increased risk of myopathy and rhabdomyolysis. Eucalyptus, St. John's wort will decrease the level or effect of simvastatin.	Avoid intake of large quantities of grapefruit juice, for it increases the absorption of statins (>1 quart/d). Eat a low-cholesterol, low-fat diet for best results. GI problems are generally transient.
Slo-Niacin	See *niacin*		
Sodium ferric Gluconate complex Ferrlecit	See *iron sucrose*		
Spironolactone Aldactone	Diuretic Potassium-sparing diuretic	Hyperkalemia, dehydration, hyponatremia, nausea, vomiting, anorexia, abdominal cramps, and diarrhea. Shepherd's purse may interfere with blood pressure control. Concurrent use of diuretics and licorice may result in increased risk of hypokalemia and/or reduced effectiveness of the diuretic. Concurrent use of potassium-sparing diuretics and ma huang may result in reduced hypotensive effect of potassium-sparing diuretics. Concurrent use of diuretics and yohimbine may result in reduced diuretic effectiveness. Juniper may potentiate or interfere with diuretic therapy. Agrimony, birch, cornsilk, goldenrod, maitake, or reishi increases the effect of spironolactone.	Avoid foods high in potassium, potassium supplements, and salt substitutes.

Description

Tenormin	See *atenolol*		
Tirzepatide Mounjaro	Incretin mimetics GIP/GLP-1 receptor agonist	Nausea, vomiting, diarrhea, abdominal pain, constipation, heartburn, burping.	Alcohol may affect blood glucose levels in patients with diabetes. Both hypoglycemia (low blood sugar) and hyperglycemia (high blood sugar) may occur.
Titralac	See *calcium salts*		
Toprol XL	See *metoprolol*		
Tranylcypromine Parnate	Antidepressant Monoamine oxidase inhibitor (MAOI)	Sudden high blood pressure or increased MAOI activity may occur with ingestion of certain foods and herbals (avocado, yerba mate, guarana, kava, liquorice, ma huang, St. John's wort, 5-htp, SAMe, yohimbe, bitter orange, ginseng, Ginkgo, nutmeg, caffeine). Alcohol may increase depressant effect. Caffeine may increase blood pressure and cardiac arrhythmias. GI upset Tranylcypromine and valerian both increase sedation. Pleurisy root decreases the effect of tranylcypromine.	Avoid foods high in tyramine: cheeses, fava, or broad bean pods; yeast or meat extracts, smoked or pickled meat, poultry, or fish; fermented sausage such as bologna, pepperoni, salami, or other fermented meat; avocados; bananas; beer; wine; and raisins. Avoid excess amounts of caffeine, tea, or chocolate. Avoid alcohol.

Description

Triamterene Dyrenium	Diuretic Potassium-sparing diuretic	Hyperkalemia, dehydration, hyponatremia, nausea, vomiting, anorexia, abdominal cramps, and diarrhea. Concurrent use of diuretics and juniper, or yohimbine may result in reduced diuretic effectiveness. Concurrent use of potassium-sparing diuretics and shepherd's purse orma huang may result in reduced hypotensive effect of potassium-sparing diuretics. Concurrent use of triamterene and potassium foods may result in hyperkalemia. Agrimony, birch, cornsilk, goldenrod, maitake, or reishi may increase the effects of triamterene.	Avoid foods high in potassium, potassium supplements, and salt substitutes.
Tricor	See *fenofibrate*		
Trovafloxacin Trovan	Quinolone	Diarrhea. Iron preparations may increase drug absorption. Dandelion may increase drug effect. Fennel seed may decrease drug effect, resulting in treatment failure.	Consider fluid and electrolyte replacement for diarrhea.
Trovan	See *trovafloxacin*		
Tums	See *calcium salts*		
Vasotec	See *enalapril*		
Velban	See *vinblastine*		
Velsar	See *vinblastine*		

Description

Venlafaxine Effexor, Effexor XR	Antidepressant	Nausea, dry mouth, anorexia, and constipation. St. John's wort, valerian, may increase sedative effect. Concurrent use of venlafaxine and St. John's wort, SAMe. 5-htp, ginseng, may result in an increased risk of serotonin syndrome (hypertension, hyperthermia, myoclonus, mental status changes). Fish oil triglycerides will increase effect by anticoagulation. Concurrent use with green tea may increase risk of bleeding. Pleurisy root decreases the effect of venlafaxine.	Use ice chips or chew gum for dry mouth. Monitor weight.
Venofer	See *iron sucrose*		
Verapamil Calan, Verelan, Verelan PM, Isoptin, Isoptin SR, Covera-HS	Calcium channel blocker	Constipation, upset stomach, heartburn. Concurrent use of verapamil and caffeine or green tea may result in increased caffeine serum concentrations and enhanced CNS stimulation. Eucalyptus, St. John's Wort decreases level or effect of verapamil. Shepherd's purse may interfere with blood pressure control. Verapamil increases the level or effect of eucalyptus or willow bark. Lily of the valley and verapamil affect the toxicity of each other. DHEA, grapefruit, hawthorn, agrimony, cornsilk, fo-ti, maitake, reishi increase the level of effect of verapamil.	Avoid drinking grapefruit juice or eating grapefruit 1 hr before or for 2 hr after taking nifedipine.
Verelan, Verelan PM	See *verapamil*		

Description

Vinblastine Velban, Velsar, vinblastine sulfate, VBL	Plant alkaloid	Nausea, vomiting, stomach pain, constipation, and diarrhea. St. John's Wort or eucalyptus will decrease the effectiveness of vinblastine. DHEA, maitake, or grapefruit, will increase the level or effect of vinblastine.	Drink plenty of fluids to decrease constipation.
Vincristine Oncovin	Plant alkaloid	Nausea, vomiting, stomach pain, stomach cramps, constipation, and diarrhea. St. John's wort or eucalyptus will decrease the effectiveness of vincristine. DHEA, grapefruit will increase level or effect of vincristine.	Consider fluid and electrolyte replacement for diarrhea and vomiting. May consider laxatives for constipation. Mild vomiting remedied with antiemetic. Avoid grapefruit or grapefruit-containing foods.
Warfarin Coumadin	Anticoagulant	Anorexia, nausea, abdominal cramping, and diarrhea. Prevents the conversion of vitamin K to its active form. Garlic, agrimony, alfalfa, ginseng, anamu, cinnamon, devil's claw, DHEA, dong quai, fennel, feverfew, fish oil, ginger, Ginkgo biloba, horse chestnut seed, melatonin, mistletoe, nettle, pau d'arco, reishi, cranberry, horseradish, parsley may increase anticoagulation and the risk of bleeding.	Consider small, frequent meals for anorexia. Consider fluid and electrolyte replacement for diarrhea. Dietary and supplemental intake, particularly vitamin K, must be consistent to achieve steady level of anticoagulation. Those on long-term use should be monitored for bone density.
		Mineralization of newly formed bone may be deterred. Saw Palmetto increases the toxicity of warfarin. St. John's wort, cornsilk, eucalyptus, coenzyme Q, ascorbic acid, rose hips, watercress can decrease the level or effect of warfarin.	

Description

Wellbutrin	See *bupropion*		
Xenical	See *orlistat*		
Zemplar	See *paricalcitol*		
Zestril	See *Lisinopril*		
Zithromax	See *azithromycin*		
Zocor	See *simvastatin*		
Zoloft	See *sertraline*		
Zyban	See *bupropion*		

Description

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Appendix C

Vitamins, Minerals, and Dietary Supplement Facts

Listing of Contents

1. Conversion Tables
2. Vitamin Facts and Nutrient Lists
3. Mineral Facts
4. Dietary Supplements

TABLE C.1 Conversion Tables

A. Length

1 m = 39 in.
1 cm = 0.4 in.
1 in. = 2.5 cm
1 ft = 30 cm

B. Temperature

	Celsius	Fahrenheit
Steam	100°C	212°F
Body temp.	37°C	98.6°F
Ice	0°C	32°F

A. Length

	°C = (°F – 32) × 5/9 °F = (°C × 9/5) + 32	

APPENDIX C.2 C. Volume

1 L	= 1,000 mL	0.26 gal	1.06 qt	2.1 pt
1 mL	= 1,000 L	0.03 fluid oz		
1 gal	= 128 oz	8 cups	3.8 L	
1 qt	= 32 oz	4 cups	0.95 L	
1 pt	= 16 oz	2 cups	0.47 L	
1 cup	= 8 oz	16 Tbsp	~250 mL	0.25 L
1 oz	= 30 mL			
1 tablespoon (Tbsp)	= 3 tsp	15 mL		
1 teaspoon (tsp)	= 5 mL			

Description

APPENDIX C.3 D. Weight

1 kg	**= 1,000 g**	**2.2 lb**	
1 g	= 1/1,000 kg	1,000 mg	0.035 oz
1 mg	= 1/1,000 g	1,000 µg	
1 µg	= 1/1,000 mg		
1 lb	= 16 oz	454 g	0.45 kg
1 oz	= ~28 g		

Description

APPENDIX C.4 E. Energy

1 kJ	**= 0.24 kcal**	
1 mJ	= 240 kcal	

1 kJ	**= 0.24 kcal**	
1 kcal	= 4.2 kJ	
1 g carbohydrate	= 4 kcal	17 kJ
1 g fat	= 9 kcal	17 kJ
1 g protein	= 4 kcal	17 kJ
1 g alcohol	= 7 kcal	29 kJ

TABLE C.2 Vitamin Facts and Nutrient Lists

A. Vitamins			
Vitamin	**Functions**	**Deficiency**	**Toxicity**
A	Maintains integrity of cornea, epithelial cells, and mucous membranes; skin and bone and teeth growth; regulates synthesis of reproductive hormones and immune system; cancer protection	Anemia Blindness, night blindness Bone growth deficit Corneal breakdown Diarrhea Joint pain Kidney stones Infection susceptibility	Amenorrhea Anorexia Bone pain Fatigue Headache Nosebleeds Skin rash
D	Maintains bone tissue by regulating the absorption and excretion of calcium and phosphorus	Defective bone growth (bowed legs, joint pain) Muscle spasms	Anorexia Headaches Excessive thirst Hypercalcemia Kidney stones Nausea Weakness
E	Maintains cell membranes; acts as an antioxidant in fighting disease-causing free radicals and in protecting other important compounds from oxidation	Anemia Breast cysts Leg cramps Weakness	Enhances action of anticoagulants Gastrointestinal distress
K	Synthesis of blood clotting compounds; regulation of calcium levels in blood	Excessive bleeding	Jaundice Interference or anticoagulant drugs
B_1 Thiamin	Coenzyme in energy metabolism; maintenance of appetite and nervous system function	Abnormal heart beat Cardiomegaly/heart failure Fluid retention Mental confusion Muscle pain, weakness, wasting paralysis	None documented
B_2 Riboflavin	Coenzyme in energy metabolism; maintenance of skin and visual function	Corneal abnormalities Dry, cracking at corners of mouth Sensitivity to light Skin rash Tongue abnormalities	None documented

B_3 Niacin, Nicotinamide	Coenzyme in energy metabolism; maintenance of skin, and nervous and digestive systems	Anorexia Diarrhea Skin rash Tongue abnormalities Weakness and dizziness	Diarrhea Dizziness Liver dysfunction Low blood pressure Sweating, flushing
B_6 Pyridoxine	Coenzyme in protein and fat metabolism; synthesis of red blood cells; synthesis of niacin	Anemia Kidney stones Dermatitis Spastic muscles, convulsions Tongue abnormalities	Fluid retention Depression, memory loss Fatigue Weakness
Folate	Coenzyme in cellular synthesis	Anemia Depression, mental confusion Diarrhea, constipation Infection susceptibility Tongue abnormalities	Mask B_{12} deficiency
B_{12} Cobalamine	Cellular synthesis; maintenance of nervous system function	Anemia Fatigue Paralysis Skin abnormalities Tongue abnormalities	None documented
Pantothenic Acid	Coenzyme in energy metabolism	Fatigue Insomnia Vomiting, other intestinal problems	Fluid retention
Biotin	Coenzyme in energy metabolism; synthesis of fat and glycogen	Alopecia Anorexia Depression Fatigue Heartbeat abnormalities Nausea Skin rash	None documented
Choline	Coenzyme in energy metabolism; synthesis of phospholipids and neurotransmitters	Growth failure Kidney failure Liver dysfunction (fat accumulation) Memory abnormalities	Fishy body odor Low blood pressure

C Ascorbic Acid	Collagen synthesis; antioxidant; immune function; enhancement of iron absorption; synthesis of thyroid hormone; protein metabolism	Anemia Bleeding gums, loose teeth Bone fracture susceptibility Depression Infection susceptibility	Abdominal cramps, diarrhea Headache Nausea Skin rash
		Joint pain Muscle pain and wasting Skin problems Wound healing delayed	Interferes with interpretation of some laboratory values

Description

APPENDIX C.6 B. Vitamin C

Food	Portion Size	Vitamin C (mg)	% Daily Value (DV)
Papaya	1, whole fresh	188	313
Orange juice	1 cup, fresh	124	207
Brussel sprouts	1 cup, cooked	96	160
Grapefruit juice	1 cup, fresh	94	157
Green pepper	1, whole	90	150
Strawberries	1 cup, fresh	85	142
Orange	1, medium fresh	80	133
Broccoli	1 cup, cooked	74	123
Cauliflower	1 cup, cooked	72	120
Cantaloupe	1 cup, fresh	68	113
Mango	1, fresh	57	95
Pink grapefruit	½, fresh	47	78
Honeydew melon	1 cup, fresh	42	70
Turnip greens	1 cup, cooked	40	67
Parsley	½ cup, chopped	40	67
Mustard greens	1 cup, cooked	36	60
Tomatoes	1, whole canned	36	60
Cabbage	1 cup, raw	34	57

Sauerkraut	1 cup, canned	34	57
Tomato juice	6 oz, canned	33	55
Raspberries	1 cup, fresh	31	52
Butternut squash	1 cup, boiled	30	50
Sweet potato	1, baked w/skin	28	47
Baked potato	1, whole	26	43
Pineapple chunks	1 cup, fresh	24	40
Asparagus	1 cup, cooked	20	33
Watermelon	1 cup, fresh	15	25
Apple	1, medium fresh	8	13
Milk, 2%	8 oz	2	3

Quick Reference Diet Integration

- Snacks: orange, tomato, and grapefruit juices
- Entrée: fried broccoli, Brussel sprouts, green pepper, and cauliflower
- Mashed, baked, or boiled potatoes
- Fruit salad of strawberries, papaya, mango, watermelon in place of sweets
- Kabob of green peppers, cherry tomatoes, strawberries, and pineapples

Description

APPENDIX C.7 C. Riboflavin

			% Dietary Reference Intake (DRI)	
Food	**Portion Size**	**Riboflavin**	**Women**	**Men**
Brewer's yeast	1 Tbsp	1.21	110	93
Yogurt	1 cup, low fat	0.51	46	39
Mushrooms	1 cup, cooked	0.46	42	35
Ricotta cheese	1 cup, part skim	0.46	42	35
Corn flakes	1 cup	0.43	39	33
Cottage cheese	1 cup, low fat	0.42	38	32
Milk, 2%	8 oz	0.4	36	31
Buttermilk	1 cup	0.38	35	29

Sirloin steak	3.5 oz, broiled	0.29	26	22
Peach halves	10, dried	0.28	25	22
Pork chop	3.5 oz, roasted	0.26	24	20
Ground beef	3.5 oz, lean baked	0.24	22	18
Black-eyed peas	1 cup, cooked	0.24	22	18
Kidney beans	1 cup, canned	0.23	21	18
Asparagus	1 cup, cooked	0.22	20	17
Almonds	1 oz, whole dried	0.22	20	17
Oysters	3 oz, raw	0.2	18	15
Ham	3.5 oz, cooked	0.19	17	15
Turkey	3.5 oz, w/o skin	0.18	16	14
Broccoli	1 cup, cooked	0.16	15	12
Green beans	1 cup, cooked	0.14	13	11
Cheddar cheese	1 oz	0.11	10	8
Spinach	1 cup, cooked	0.1	9	8
Strawberries	1 cup, fresh	0.1	9	8
Chicken breast	½ breast, roasted	0.1	9	8
Sole/flounder	3 oz baked	0.07	6	5
Orange	1, medium fresh	0.06	5	5
Bread	Whole wheat, 1 slice	0.05	5	4
Bean sprouts	1 cup, stir fried	0.04	4	3
Cantaloupe	1 cup, fresh	0.03	3	2
Apple	1, medium fresh	0.02	2	2

Quick Reference Diet Integration

- A bowl of corn flakes with 8 oz milk provides 75% of DRI for women.
- Yogurt as a snack and as a dip for fruits and vegetables.
- Mushrooms on pizza, salads, or stir fries.
- Low-fat cottage or ricotta cheeses in lasagna, ravioli, or other main dish.

Description

APPENDIX C.8 | D. Vitamin B_6

Food	Portion Size	B_6 (mg)	% DRI
Beef liver	3.5 oz, braised	0.91	70
Baked potato	1, whole	0.7	54
Salmon	3 oz, cooked	0.7	54
Banana	1, peeled	0.66	51
Chicken breast	½ breast, w/o skin	0.51	39
Corn flakes	1 cup	0.5	38
Avocado	½ average	0.48	37
Trout	3 oz, broiled	0.46	35
Turkey	3.5 oz, w/o skin	0.46	35
Brewer's yeast	1 oz	0.45	35
Sirloin steak	3.5 oz, broiled	0.45	35
Pork chop	3.5 oz, roasted	0.45	35
Spinach	1 cup, cooked	0.44	34
Soybeans	1 cup, cooked	0.4	31
Wheat germ	¼ cup	0.38	29
Tuna, in water	3 oz, canned	0.3	23
Navy beans	1 cup, cooked	0.3	23
Sunflower seeds	¼ cup, dry	0.3	23
Turnip greens	1 cup, cooked	0.26	20
Cauliflower	1 cup, cooked	0.26	20
Broccoli	1 cup, cooked	0.25	18
Green pepper	1, whole	0.24	18
Watermelon	1 cup, fresh	0.23	18
Ground beef	3.5 oz, lean baked	0.22	17
Asparagus	1 cup, cooked	0.22	17
Figs	5, dried	0.21	16
Cantaloupe	1 cup, fresh	0.18	14
Sole/flounder	3 oz, cooked	0.18	14
Mustard greens	1 cup, cooked	0.16	12
Zucchini	1 cup, cooked	0.14	11
Milk	2%, 8 oz	0.11	8

Description

Quick Reference Diet Integration

- Baked potato for lunch with fat-free sour cream or dressing.
- Banana with corn flakes for breakfast.
- Poach, grill, or broil salmon for a quick meal.

Food	Portion Size	B_{12} (µg)	% DRI
Clams	3 oz, cooked	84.1	3,500
Beef liver	3.5 oz, braised	71	3,000
Oysters	3 oz, cooked	32.5	1,350
Clam chowder	1 cup	10.3	427
Rabbit	3.5 oz, roasted	8.3	346
Braunschweiger	1 oz, tube type	5.2	216
Sirloin steak	3.5 oz, broiled	2.9	119
Salmon	3 oz, cooked	2.7	113
Tuna, in water	3 oz, canned	2.5	106
Lamb chop	3.5 oz, braised	2.3	95
Vegetarian burger	½ cup	2	83
Raisin bran	¾ cup	2	83
Ground beef	3.5 oz, lean baked	1.7	71
Cottage cheese	1 cup, 1% fat	1.4	58
Sole/flounder	3 oz, cooked	1.3	54
Shrimp	3 oz, cooked	1.3	54
Halibut	3 oz, broiled	1.2	50
Milk	2%, 8 oz	0.89	37
Frankfurter	1 beef, 2 oz	0.88	37
Ham	3.5 oz, cooked	0.74	29
Tempeh	½ cup	0.7	29

Pork chop	3.5 oz, roasted	0.6	25
Buttermilk	8 oz, cultured	0.54	23
Egg	1, whole fresh	0.5	21
Turkey	3.5 oz, w/o skin	0.37	15
Canadian bacon	2 slices, cooked	0.36	15
Miso	½ cup	0.29	12
Chicken breast	½ breast, w/o skin	0.29	12
Yogurt	8 oz, low fat	0.24	10
Cheddar cheese	1 oz	0.23	10
Goat's milk	8 oz	0.16	7

Quick Reference Diet Integration

- Raisin bran with low-fat milk for breakfast.
- Low-fat cottage cheese with fruit as a snack.
- 3 oz of salmon, tuna, or rabbit provides 100% DRI of B_{12}.
- Oysters or clams served steamed or in stews and chowders.

Description

APPENDIX C.10 | F. Biotin

Food	Portion Size	Biotin (µg)	% DRI
Egg	1, whole fresh	10	33
Wheat germ	¼ cup, toasted	7	23
Ry-Krisp	½ oz	6.8	23
Granola	¼ cup w/ raisins	5	17
Egg noodles	1 cup, cooked	4	13
Almonds	1 oz, whole natural	1	3
Pistachios	1 oz, red	1	3
Corn meal	1 oz, yellow	1	3

Quick Reference Diet Integration

- Granola as a cereal or snack, or mixed into yogurt
- Wheat germ in casseroles and breads or on cereal
- Ry-Krisp with peanut butter and jelly for a snack
- Egg noodles with veggies, fat-free Italian dressing
- Scrambled, poached, or hardboiled eggs

Description

APPENDIX C.11 | G. Choline

			% DRI	
Food	**Portion Size**	**Choline (μg)**	**Women**	**Men**
Beef liver	3.5 oz	581.1	137	106
Cauliflower	1 cup, cooked	162	38	29
Peanuts	1 oz, dried	127.3	30	23
Peanut butter	2 Tbsp	124.6	29	23
Grape juice	8 oz, canned	120	28	22
Potato	1, whole baked	103.2	24	19
Iceberg lettuce	1 leaf, raw	58.6	14	11
Tomato	1, whole raw	52.9	12	10
Milk	Whole 8 oz	36.6	9	7
Orange	1, fresh medium	28	7	5
Banana	1, whole peeled	27.4	6	5
Bread	Whole wheat 1 slice	24.2	6	4
Cucumber	½ cup, raw	11.3	3	2
Beef steak	3.5 oz	7.5	2	1
Apple	1, fresh medium	3.7	<1	<1
Egg	1, fresh whole	2.1	<1	<1
Ginger ale	12 oz	0.73	<1	<1
Butter	1 tsp	0.21	<1	<1
Margarine	1 tsp, tub type	0.15	<1	<1
Corn oil	1 Tbsp	0.04	<1	<1

Quick Reference Diet Integration Guide

- Dinner: 3.5 oz beef liver, 1 cup cauliflower, baked potato, 8 oz grape juice, banana.
- Add iceberg lettuce to a sandwich.
- Swap straight soda for a "Choline Cocktail"—8 oz grape juice, 12 oz ginger ale.
- Snack: 1 oz peanuts and 8 oz milk.
- Lunch: peanut butter and jelly sandwich with 8 oz grape juice.

Description

H. Folate

APPENDIX C.12

Food	Portion Size	Folate (µg)	% DRI
Pinto beans	1 cup, cooked	294	74
Asparagus	1 cup, cooked	264	66
Spinach	1 cup, cooked	262	66
Navy beans	1 cup, cooked	255	64
Beef liver	3.5 oz, braised	217	54
Black-eyed peas	1 cup, cooked	209	52
Great northern beans	1 cup, cooked	181	45
Turnip greens	1 cup, cooked	170	43
Lima beans	1 cup, cooked	156	39
Kidney beans	1 cup, canned	129	32
Broccoli	1 cup, cooked	104	26
Corn flakes	1 cup	100	25
Parsley	1 cup, chopped	92	23
Beets	1 cup, cooked	90	23
Wheat germ	¼ cup	82	21
Romaine lettuce	1 cup, chopped	76	19
Cauliflower	1 cup, cooked	64	16
Pineapple juice	8 oz, canned	58	15
Orange	1, fresh medium	47	12
Zucchini	1 cup, cooked	30	8
Peanuts	1 oz, dried	29	7
Cantaloupe	1 cup, fresh	27	7
Winter squash	1 cup, cooked	26	7
Strawberries	1 cup, fresh	26	7
Grapefruit juice	1 cup, canned	26	7
Egg	1, whole fresh	23	6
Green beans	1 cup, cooked	22	6
Tofu	½ cup, raw	19	5
Tomato	1, whole raw	18	5
Bread, whole wheat	1 slice	14	4

Quick Reference Diet Integration

Description

- Use dry beans in soups or as a main dish salad.
- Add spinach to lasagna or serve cooked with lemon juice.
- Salad of spinach, romaine, tomato, broccoli, cauliflower, and beets.
- Dinner: 3.5 oz beef liver, 1 cup asparagus, 1 cup lima beans.
- Breakfast: cornflakes in milk; fruit salad of cantaloupe, strawberries, orange.

			% DRI	
Food	**Portion Size**	**Niacin (mg)**	**Women**	**Men**
Chicken breast	½ roasted w/o skin	11.8	84	74
Tuna, in water	3 oz, canned	11.3	81	71
Beef liver	3.5 oz, braised	10.7	76	67
Brewer's yeast	1 oz	10.7	76	67
Salmon	3 oz, broiled	8.6	61	54
Mushrooms	1 cup, cooked	7	50	44
Halibut	3 oz, broiled	6.1	44	38
Peach halves	10, dried	5.7	41	36
Pink salmon	3 oz, canned	5.6	40	35
Pork chop	3.5 oz, roasted	5.5	39	34
Lamb chop	3.5 oz, braised	5.5	39	34
Turkey	3.5 oz w/o skin	5.4	39	34
Sirloin steak	3.5 oz, broiled	4.3	31	27
Ground beef	3.5 oz, lean baked	4.2	30	26
Peanuts	1 oz, dried unsalted	4	29	25
Baked potato	1, whole	3.3	24	21
Sole/flounder	3 oz, baked	2.5	18	16
Kidney beans	1 cup, canned	2.4	17	15
Braunschweiger	1 oz, tube type	2.3	16	14
Shrimp	3 oz, boiled	2.2	16	14
Wheat bran	¼ cup	2	14	13

Asparagus	1 cup, cooked	2	14	13
Oysters	3 oz, raw	1.7	12	11
Sardines	2, sardines in oil	1.3	9	8
Crab meat	3 oz, canned	1.2	9	8
Bread, whole wheat	1 slice	1	8	6
Summer squash	1 cup, cooked	1	8	6
Cantaloupe	1 cup, fresh	0.9	6	6
Peach	1, fresh medium	0.9	6	6
Spinach	1 cup, cooked	0.9	6	6
Broccoli	1 cup, cooked	0.8	6	5

Quick Reference Diet Integration

- Choose tuna, halibut, and salmon instead of red meat.
- Add mushrooms to soups and salads, and stir fry.
- Dinner: half chicken breast, baked potato, 1 cup asparagus.
- Fortified breakfast cereals contain 25% of the DRI for niacin per serving.

Description

APPENDIX C.14 J. Pantothenic Acid

Food	Serving Size	Pantothenic Acid (mg)	% DRI
Beef liver	3.5 oz, braised	4.6	91
Mushrooms	½ cup, boiled	1.7	34
Avocado	1, medium	1.7	34
Salmon	3 oz, cooked	1.4	28
Lentils	1 cup, boiled	1.3	25
Split peas	1 cup, boiled	1.2	23
Potato	1, whole baked	1.1	22
Turkey	3.5 oz w/o skin	0.94	19
Pomegranate	1, medium raw	0.92	18
Chicken breast	½, roasted w/o skin	0.83	17
Peanuts	1 oz, dried	0.79	16
Milk, 2%	8 oz	0.78	16
Sweet potato	1, whole baked	0.74	15

Chickpeas	1 cup, canned	0.72	14
Wheat germ	¼ cup	0.66	13
Pork chop	3.5 oz, roasted	0.65	13
Egg	1, whole fresh	0.63	13
Broccoli	1 cup, cooked	0.5	10
Ham	3.5 oz, cooked	0.47	9
Sole/flounder	3 oz, cooked	0.43	9
Kidney beans	1 cup, canned	0.38	8
Sirloin steak	3.5 oz, broiled	0.37	7
Corn	1 cup, boiled	0.36	7
Orange	1, fresh medium	0.35	7
Watermelon	1 cup, fresh	0.34	7
Ground beef	3.5 oz, baked lean	0.27	5
Spinach	1 cup, cooked	0.26	5
Bread, whole wheat	1 slice	0.18	4
Tuna, in water	3 oz, canned	0.18	4
Macaroni	1 cup, cooked, enriched	0.16	3
Cheddar cheese	1 oz	0.12	2

Quick Reference Diet Integration

- Avocado sliced in a salad or in a dip (guacamole)
- Dinner: 3 oz salmon, 1 baked potato, 8 oz milk, 1 cup broccoli
- Soups with lentils or split peas
- Salad: chicken breast, avocado, spinach, mushrooms, broccoli

Description

APPENDIX C.15 K. Thiamin

			% DRI	
Food	**Portion Size**	**Thiamin (mg)**	**Women**	**Men**
Brewer's yeast	1 oz	4.43	403	369
Pork chop	3.5 oz, roasted	0.91	83	76
Ham	3.5 oz, cooked	0.78	71	65
Green peas	1 cup, cooked	0.42	38	35
Canadian bacon	2 pieces, cooked	0.38	35	32
Corn flakes	1 cup	0.38	35	32
Split peas	1 cup, cooked	0.37	34	31
Macaroni, enriched	1 cup, cooked	0.29	26	24
Kidney beans	1 cup, canned	0.27	25	23
Oatmeal	1 cup, cooked	0.26	24	22
Millet	1 cup, cooked	0.25	23	21
Acorn squash	1 cup, boiled	0.24	22	20
Baked potato	1, whole	0.22	20	18
Asparagus	1 cup, cooked	0.22	20	18
Peanuts	1 oz, dry, unsalted	0.19	17	16
Black eyed peas	1 cup, cooked	0.17	15	14
Watermelon	1 cup, fresh	0.13	12	11
Sirloin steak	3.5 oz, broiled	0.13	12	11
Honeydew melon	1 cup, fresh	0.13	12	11
Orange	1, fresh medium	0.12	11	10
Tofu	½ cup	0.1	9	8
Milk, 2%	8 oz	0.1	9	8
Green beans	1 cup, cooked	0.1	9	8
Broccoli	1 cup, cooked	0.1	9	8
Bread, whole wheat	1 slice	0.09	8	8
Sole/flounder	3 oz, cooked	0.08	7	7
Cauliflower	1 cup, cooked	0.08	7	7
Tomato	1, whole raw	0.07	6	6

Quick Reference Diet Integration

- Include lean pork in weekly menus.

- Use green peas, split peas, black peas, black-eyed peas, kidney beans in soups.
- Choose enriched pasta and bread products.
- Breakfast: oatmeal or fortified cereal.
- Side dish: boiled or baked squash.

Description

APPENDIX C.10 | L. Vitamin A

Food	Portion Size	Vitamin A (RE)	% DRI
Beef liver	3 oz, fried	9,123	1,042
Pumpkin	1 cup, canned	5,424	620
Sweet potato	1, whole baked	2,486	284
Carrot	1, whole fresh	2,024	231
Spinach	1 cup, cooked	1,474	168
Butternut squash	1 cup, baked	1,435	164
Mango	1, medium fresh	806	92
Papaya	1, medium fresh	612	70
Cantaloupe	1 cup, fresh	516	59
Turnip greens	½ cup, chopped	396	45
Collard greens	1 cup, chopped	349	40
Apricot halves	10 halves	253	29
Winter squash	½ cup, cubes	235	27
Mustard greens	½ cup, chopped	212	24
Broccoli	½ cup, frozen	174	20
Parsley	½ cup, chopped	156	18
Milk, 2%	8 oz	140	16
Egg	1, whole fresh	95	11
Cheddar cheese	1 oz	86	10
Watermelon	1 cup, fresh	58	7
Margarine	1 tsp, tub-type	47	5
Sole/flounder	3 oz, cooked	28	3
Orange	1, medium fresh	26	3
Green beans	½ cup, canned	24	3
Corn	½ cup, canned	13	2
Apple	1, medium fresh	7	1
Chicken breast	½, roasted w/o skin	5	0.5
Sirloin steak	3.5 oz, broiled	0	0
Brewer's yeast	1 oz	0	0
Bread, whole wheat	1 slice	0	0

Quick Reference Diet Integration

Description

- Use fresh spinach, romaine lettuce, carrots, and tomatoes in salads.
- Use canned pumpkin in cookies, pies, and desserts.
- Serve baked sweet potatoes in place of baked potatoes.
- Breakfast: fruit salad of mango, papaya, and cantaloupe.
- Snack: dried peaches and apricots.

Food	Portion Size	Vitamin D (μg)	% DRI
Milk, 2%	8 oz	1.6	33
Corn flakes	1 cup	0.81	16
Cod liver oil	1 Tbsp	0.55	11
Egg	1, whole fresh	0.41	8
Margarine	1 tsp, tub type	0.34	7
Frankfurter	1 beef frank	0.18	4
Braunschweiger	1 oz, tube type	0.15	3
Rice, wild	$^{2}/_{3}$ cup, instant	0.07	1
Rice, white	$^{2}/_{3}$ cup, instant	0.03	<1
Cheddar cheese	1 oz	0.02	<1

Quick Reference Diet Integration

- Drink 2–3 eight-oz glasses of milk per day.
- 10–15 minutes of sunlight exposure 3 times per week.
- Breakfast: vitamin D-fortified cereals with milk.
- Snack: pudding made with low-fat milk.
- Use vitamin D-fortified margarine instead of butter.

Description

APPENDIX C.18 N. Vitamin E

Food	Portion Size	Vitamin E (mg)	% DRI
Wheat germ oil	1 Tbsp	20.3	102
Sunflower seeds	1 oz, dry roasted	14.2	71
Mayonnaise	1 Tbsp	11	55
Almonds	1 oz, dried	6.7	34
Dried filberts	1 oz	6.7	34
Sunflower seed oil	1 Tbsp	6.3	32
Sweet potato	1, medium raw	5.9	30
Almond oil	1 Tbsp	5.3	27
Cottonseed oil	1 Tbsp	4.8	24
Safflower oil	1 Tbsp	4.6	23
Wheat germ	¼ cup, toasted	4.1	20
Peanut butter	2 Tbsp	3	15
Shrimp	3 oz, boiled	3	15
Canola oil	1 Tbsp	2.5	13
Asparagus	1 cup, cooked	2.4	12
Mango	1, medium raw	2.3	12
Avocado	½ cup	2.3	12
Peanuts	1 oz, dry roasted	2.2	11
Brazil nuts	1 oz, dried	2.1	11
Salmon	3 oz, baked	2	10
Corn oil	1 Tbsp	1.9	10
Olive oil	1 Tbsp	1.7	8
Peanut oil	1 Tbsp	1.6	8
Soybean oil	1 Tbsp	1.5	8
Apple	1, medium fresh	1.5	8
Brussel sprouts	1 cup, cooked	1.3	7
Spinach	1 cup, raw	1.1	5
Macaroni	1 cup, cooked	1	5
Parsley	1 Tbsp	1	5
Pear	1, medium fresh	0.83	4
Cheddar cheese	1 oz	0.5	3

Quick Reference Diet Integration

- Use wheat germ or sunflower oil in salad dressings or baked goods.
- Serve sweet potato chips instead of regular chips—slice thin and bake.
- Replace cream cheese with peanut butter on bagels.

- Replace cream cheese with peanut butter on bagels.
- Add wheat germ to cereal or yogurt.
- Snack: sunflower seeds, peanuts, or almonds.

Description

APPENDIX C.19 | O. Vitamin K

Food	Portion Size	Vitamin K (μg)	% DRI
Turnip greens	1 cup, chopped raw	364	455
Green tea	1 oz, dry	199	249
Spinach	1 cup, raw	148	185
Broccoli	1 cup, cooked	126	158
Beef liver	3.5 oz, raw	104	104
Cauliflower	1 cup, raw	96	120
Soybean oil	1 Tbsp	76	95
Chickpeas	1 oz, dry	74	93
Asparagus	1 cup, cooked	69	86
Lentils	1 oz, dry	62	78
Soybeans	1 oz	53	66
Cabbage	1 cup, raw shredded	52	65
Mung beans	1 oz, dry	48	60
Green beans	1 cup, boiled	44	55
Wheat flour	Whole wheat, 1 cup	36	45
Tomato	1, whole fresh	25	31
Egg	1, whole fresh	25	31
Peas	1 oz, dry	23	29
Wheat bran	1 oz	23	29
Lettuce	1 leaf iceberg	22	28
Strawberries	1 cup, raw	21	26
Watercress	1 cup, chopped	20	25
Oats	1 oz, dry	18	23
Wheat germ	1 oz	10	13
Carrot	1, medium raw	9	11
Corn oil	1 Tbsp	8	10
Orange	1, medium fresh	7	9
Potato	1, whole baked	6	8
Cucumber	1 cup, raw	6	8

Quick Reference Diet Integration

- Add turnip greens to soup or eat as a side dish.
- Eat [illegible] of broccoli, cauliflower, asparagus, and [illegible]

- Entree: stir fry of broccoli, cauliflower, spinach, carrots, and asparagus.
- Add spinach, watercress, and tomatoes to sandwiches.
- Use soybean oil to make salad dressing.
- Salad: spinach and lettuce with tomatoes and chickpeas.
- Appetizer: coleslaw with low-fat or fat-free dressing.

Description

Mineral/Symbol	Functions	Deficiency	Toxicity
Calcium, Ca	Bone/teeth structure; nerve transmission; blood clotting; muscle contraction	Osteoporosis; osteomalacia; neuromuscular excitability; cramps, tetany; hypoparathyroidism; convulsion	Kidney stones, constipation
Canned sardines, cheese, milk, tofu made with calcium sulfate, yogurt			
Chromium, Cr	Glucose metabolism, an insulin cofactor	Weight loss; diabetes; abnormal glucose metabolism; neuropathy	None known
Broccoli, grape juice, grain products			
Copper, Cu	Iron absorption; component of several metalloenzymes	Menkes disease; microcytic anemia; cognitive deficits; hypothermia; hair depigmentation	Wilson disease; cirrhosis
Liver, clams, oysters, crab meat, cashews, hazelnuts, sunflower seeds, lentils, mushrooms			
Fluoride, F	Dental caries prevention	Dental caries	Dental mottling
Fluoridated water, crab, rice, fish			
Iodine, I	Constituent of thyroid hormones	Hypothyroidism; goiter	Goiter
Iodized salt, cod, milk, potatoes, turkey breast, shrimp			
Iron, Fe	Component of hemoglobin and myoglobin; electron transport	Microcytic anemia; fatigue; skin pallor; glossitis; tachycardia	Hemosiderosis; hemochromatosis
Oysters, beef, chicken (dark meat), blackstrap molasses, prune juice, lentils			

Magnesium, Mg	Component of several enzymes involved in metabolism (including nutrient); muscle contraction; nerve transmission	Muscle spasms, tetany and seizures; hypocalcemia; hypokalemia; paresthesias	Diarrhea; cardiorespiratory failure and abnormal nerve transmission in kidney patients
Bran cereal, brown rice, almonds, spinach, Swiss chard, peanuts, blackstrap molasses, hazelnuts			
Manganese, Mn	Cofactor in several enzymes; gluconeogenesis, metabolism of lipids and mucopolysaccharides; antioxidant as Mn superoxide dismutase	Limited evidence for increased bone remodeling; reduction of serum cholesterol; skin rash; abnormal glucose tolerance	Neurologic damage if inhaled in certain occupations; limited evidence for oral toxicity of neurologic and cognitive problems
Pecans, pineapple, almonds, peanuts, bran cereal, brown rice			
Molybdenum, Mo	Cofactor in enzymes	Tachycardia; coma	Limited evidence for gout-like symptoms
Legumes, lentils, grain products, nuts			
Phosphorus, P	Cell membrane and nucleic acid component; glycolysis; component of ATP in all energy reactions; regulation of acid–base balance	Muscular weakness; glucose intolerance; abnormal cellular function; growth deficit and bone deformation in children	Diarrhea; hyperparathyroidism; soft tissue calcification
Dairy products, cereal products, meat, fish, food additives (phosphates)			
Potassium, K	Intracellular cation; nerve transmission; muscle contraction; blood pressure regulation	Muscular weakness; hypotension; ileus; cardiac arrhythmias; respiratory failure	Cardiac arrhythmia; heart failure
Potatoes, prunes/prune juice, bananas, tomatoes/tomato juice, raisins, lima beans, acorn squash			
Selenium, Se	Antioxidant as glutathione peroxidases and as part of other enzymes; part of amino acid selenocysteine	Susceptibility to physiologic stresses; prolonged deficiency linked to Keshan and Kashin–Beck diseases	Hair and nail brittleness and loss; gastrointestinal distress; skin rash; garlic breath odor; fatigue; irritability; neurologic disorders
Brazil nuts, tuna, oysters, salmon, shrimp, halibut, crab, beef, chicken, rice, sunflower seeds			

Sodium, Na; chloride, Cl	Na and Cl principle extracellular ions; maintenance of membrane potential; nutrient absorption and transport; regulation of blood volume and pressure	Headache; nausea; vomiting; muscle cramps; fatigue; disorientation; dizziness; seizure and coma	Nausea; vomiting; diarrhea; abdominal cramps; edema; coma
Snack foods, processed meats, breads, cereals, canned tomato/vegetable juice, soup, pickles, olives			
Zinc, Zn	Enzyme cofactor in reactions related to growth, sexual maturation, reproduction, immunity, night vision, taste acuity	Hypogonadism; growth failure; impaired taste and smell; impaired wound healing; infection; fatigue; diminished appetite, dry and hyperpigmented skin, acrodermatitis	Interference with copper and iron metabolism; impaired immunity
Oysters, crab, beef, poultry (dark meat), yogurt, cashews			

Description

TABLE C.4 Dietary Supplements

Name	Use	Contraindications/Potential Side Effects
α-Lipoic acid	**Diabetes**, neuropathy, alcoholic liver disease, HIV, kidney disease, pancreatic cancer, wound healing	**Adverse effects:** nausea, vomiting, dizziness **Potential drug interactions:** diabetic medications, thyroid medications, Doxorubicin, Adriamycin, NSAIDs
Ascorbic acid (vitamin C)	**Cold prevention**, scurvy, iron absorption, cancer prevention	**Adverse effects:** nausea, vomiting, heartburn, abdominal pain, flushing, dizziness, headache **Potential drug interactions:** acetaminophen, aluminum antacids, aspirin, barbiturates, fluphenazine, indinavir, levodopa, tetracycline, antibiotics, oral estrogens, anticoagulants, nicotine
β-Carotene	**Cancer**, photosensitivity, age-related macular degeneration, oral leukoplakia, scleroderma, metabolic syndrome,	**Adverse effects:** skin discoloration, loose stools, bruising, joint pain **Potential drug interactions:** statins, cholesterol-lowering medications, orlistat, niacin Patients who smoke or drink heavily or have a history of asbestos exposure

Name	Use	Contraindications/Potential Side Effects
	heart disease, asthma, COPD	should avoid β-carotene supplements because of a possible increase of the risk of heart disease and cancer
Bitter orange	**Obesity/weight loss**	**Adverse effects:** tachycardia, hypertension, fainting, heart attack, stroke, nervousness **Potential drug interactions:** MAOIs, midazolam (Versed), caffeine, dextromethorphan, feldoipine (Plendil), indinavir (Crixivan), CYP450 agents, amiodarone (Cordarone), disopyramide (Norpace), dofetilide (Tikosyn), ibutilide (Corvert), procainamide (Pronestyl), quinidine, sotalol (Betapace), thioridazine (Mellaril), stimulant drugs
Black cohosh	**Postmenopausal symptoms**, arthritis pain, breast cancer, infertility, migraines	**Adverse effects:** gastric discomfort, hepatotoxicity, fatigue, arrhythmia **Potential drug interactions:** anticoagulants, SSRIs, NSAIDs, CYP450 agents, antineoplastics, Antabuse, hormonal agents, antifungals, alcohol, tamoxifen, raloxifene
Chondroitin sulfate	**Osteoarthritis**, urinary incontinence, interstitial cystitis, muscle aches, knee pain	**Adverse effects:** rash, photosensitivity, diarrhea, constipation, dyspepsia, hair loss. **Potential drug interactions:** anticoagulants, NSAIDs, photosensitizing agents. Patients with prostate cancer (family history) and patients with shellfish allergies should avoid.
Chromium	**Diabetes**, hypoglycemia, PCOS, hyperlipidemia, bipolar disorder, depression, Parkinson disease, obesity/weight loss	**Adverse effects:** glucose/insulin metabolism, nausea, vomiting, anemia, headache, insomnia, mood changes, renal failure **Potential drug interactions:** antacids, NSAIDs, SSRI medications, MAOI medications, antidiabetic agents, antihypertensive agents, antilipidemic drugs, steroids, immunomodulators
CoQ10	**CoQ10 deficiency**, hypertension, cancer, chronic fatigue	**Adverse effects:** nausea, rash, flu-like symptoms, headache, photosensitivity, elevated liver enzymes

Name	Use	Contraindications/Potential Side Effects
	syndrome, **CHF, adjunct to statin medication**, migraine headache, diabetes, tinnitus, enhance exercise performance	**Potential drug interactions:** antihypertensive medications, antilipidemic medications, TCAs, Coumadin, antineoplastics, immunosuppressants
Echinacea	**Colds/URIs**, cancer, immune system stimulation, radiation-associated leukopenia, herpes simplex virus	**Adverse effects:** urticarial, rash, nausea, vomiting, leukopenia, dry eyes. **Potential drug interactions:** amoxicillin, anesthetics, CYP450 agents, antifungals, immunosuppressants, steroids, antineoplastics, caffeine. Patients with tuberculosis, leukemia, diabetes, connective tissue disorders, multiple sclerosis, HIV or AIDS, any autoimmune diseases, or, possibly, liver disorders should avoid.
Evening primrose oil	**Postmenopausal symptoms (PMS)**, diabetic neuropathy, allergies, rheumatoid arthritis, ADHD, breast cancer, hypertension, osteoporosis	**Adverse effects:** headache, abdominal pain, nausea, and loose stools. **Potential drug interactions:** anticoagulants, cyclosporine, phenothiazines, chemotherapy agents. Patients with seizure disorders should avoid.
5-HTP (5-hydroxytryptophan)	**Depression**, fibromyalgia, cerebellar ataxia, headache, anxiety, sleep disorders	**Adverse effects:** rash, nausea, diarrhea, heartburn, abdominal pain, irritability, heart palpitations, allergic reactions, hypomania, eosinophilia **Potential drug interactions:** angiotensin receptor blockers, antidepressants, antiepileptics, antipsychotics, CNS depressants, Ultram, lithium, CYP450 agents, β-blockers
Garlic	**Heart disease**, cancer, hypertension, cold/flu	**Adverse effects:** upset stomach, bloating, bad breath, body odor, headache, fatigue, loss of appetite, muscle aches, dizziness, asthmatic reaction, skin rash, skin lesions (topical exposure) **Potential drug interactions:** oral contraceptives, isoniazid, cyclosporine,

Name	Use	Contraindications/Potential Side Effects
		anticoagulants, HIV/AIDS medications (protease inhibitors), NSAIDs
Ginger	**Pregnancy-related nausea and vomiting, chemotherapy, nausea, nausea and vomiting after surgery**, motion sickness, osteoarthritis	**Adverse effects:** mild heartburn, diarrhea, irritation of the mouth. **Potential drug interactions:** anticoagulants, antidiabetic medications, hypertensives. Patients who have a bleeding disorder or are taking blood-thinning medications, including aspirin, should avoid.
Ginkgo biloba	**Dementia and Alzheimer disease**, intermittent claudication, anxiety, glaucoma, macular degeneration, PMS, Raynaud's	**Adverse effects:** stomach upset, headaches, skin reactions, dizziness, internal bleeding. **Potential drug interactions:** NSAIDs, medications broken down by the liver, anticonvulsants, antidepressants, antihypertensives, anticoagulants, alprazolam, cyclosporine, antidiabetic medication, thiazide diuretics. Patients who have epilepsy should not take Ginkgo, because it might cause seizures.
Ginseng (American)	**Postmenopausal symptoms**, boost the immune system, reduce the risk of cancer, improve cognition, well-being, diabetes, colds/flu, ADHD	**Adverse effects:** hypoglycemia, hypertension, bipolar disorder mania, insomnia, restlessness, anxiety, euphoria, diarrhea, vomiting, headache, nosebleeds, breast pain, vaginal bleeding **Potential drug interactions:** antidiabetics, anticoagulants, antipsychotics, MAOIs, stimulants, morphine
Glucosamine sulfate	**Osteoarthritis, rheumatoid arthritis**, leg pain, inflammatory bowel disease	**Adverse effects:** tachycardia, photosensitivity, rash, GI upset, drowsiness, asthma exacerbation. **Potential drug interactions:** acetaminophen, antineoplastics, anticoagulants, diabetic agents, diuretics. Patients with shellfish allergies should avoid.

Name	Use	Contraindications/Potential Side Effects
Hawthorn	**CHF**, anxiety, hypertension, **CAD/angina**	**Adverse effects:** nausea, agitation, headache, dizziness, fatigue, dyspnea, rash, insomnia, diaphoresis, tachycardia **Potential drug interactions:** antihypertensives, metronidazole, Antabuse, ephedrine, anticoagulants
Kava	**Anxiety**, insomnia, Parkinson disease, stress, pain	**Adverse effects:** GI distress, rash, tachycardia, headache, sedation **Potential drug interactions:** opioids, ACE inhibitors, anxiolytics, anesthetics, MAOIs, SSRIs, anticoagulants, benzodiazepines, β-blockers, CYP450 agents, CNS depressants, diuretics, contraceptives, hepatotoxic agents
Lycopene	**Cancer, enlarged prostate (benign prostatic hypertrophy)**, atherosclerosis, asthma, menopausal symptoms, oral mucositis	**Adverse effects:** none reported. **Potential drug interactions:** none known. Patients with a prostate cancer diagnosis should avoid.
Melatonin	**Insomnia**, jet lag, anti-inflammatory, age-related macular degeneration, anxiety, chronic fatigue syndrome, hypertension, menopausal symptoms, seasonal affective disorder, tinnitus, heart disease, fibromyalgia, IBS, ADHD, breast/prostate cancer, benzodiazepine withdrawal, epilepsy, sarcoidosis, PCOS	**Adverse effects:** disorientation, hypotension, hormonal levels, hyperglycemia, nausea, vomiting, diarrhea, fatigue, dizziness, headache, irritability, drowsiness, seizure, mood changes **Potential drug interactions:** anticonvulsants, anesthesia, anxiolytics, antidiabetic medication, antihypertensives, SSRIs, CYP450 agents, CNS depressants, Haldol, HRT, INH, propofol, valproic acid, zolpidem, venlafaxine, antipsychotic medications, benzodiazepines, contraceptives, β-blockers, NSAIDs, interleukin-2, anticoagulants, immunosuppressants, tamoxifen
Milk thistle	**Alcoholic hepatitis/cirrhosis, viral hepatitis**,	**Adverse effects:** stomach upset, diarrhea, nausea and vomiting, rash (topical)

Name	Use	Contraindications/Potential Side Effects
	mushroom poisoning, cancer	**Potential drug interactions:** allergy medications, statins, antianxiety medications, antipsychotics, oral contraceptives, phenytoin (Dilantin), anesthesia, antiplatelet/anticoagulant drugs, CYP450 agents, therapeutic cancer drugs
		Patients with a history of hormone-related cancers, including breast, uterine, and prostate cancer, should avoid.
Omega-3 fish oil	**CAD**, hyperlipidemia, hypertension, stroke	**Adverse effects:** nausea, diarrhea, fish breath/taste, rash, nose bleeds. **Potential drug interactions:** anticoagulants, antihypertensives, antilipidemics, contraceptive drugs. Patients with fish/shellfish allergies should avoid. Patients with HIV or other lowered immune response should avoid.
Red clover	**Postmenopausal symptoms** (PMS), cancer prevention, indigestion, high cholesterol, whooping cough, cough, asthma, bronchitis, osteoporosis	**Adverse effects:** estrogenic effects, reduction of blood clotting **Potential drug interactions:** contraceptives, estrogens, CYHP450 agents, anticoagulants, aspirin, antidiabetics, tamoxifen
Red yeast rice	**CAD**, hyperlipidemia, diabetes	**Adverse effects:** headache, heartburn, abdominal bloating, myopathy, increased liver enzymes, dizziness, rhabdomyolysis. **Potential drug interactions:** NSAIDs, anticoagulants, alcohol, antifungals, antilipidemics, antihypertensives, antidiabetic meds, CYP450 agents, digoxin, thyroid hormones, antibiotics. Patients who have allergies to yeast or molds should avoid.
Saw palmetto	**BPH (benign prostatic hyperplasia)**, alopecia, hypotonic	**Adverse effects:** abdominal pain, diarrhea, fatigue, nausea, headache, decreases libido, rhinitis, hypertension, tachycardia, increased risk of bleeding

Name	Use	Contraindications/Potential Side Effects
	neurogenic bladder, prostatitis, prostate cancer	**Potential drug interactions:** oral contraceptives and hormone replacement therapy, Proscar, Lupron, Casodex, NSAIDs, Antabuse, immunomodulators, antiplatelet and anticoagulant drugs
St. John's wort	**Depression**, anxiety, ADHD, OCD, pain, PMS	**Adverse effects:** anxiety, fatigue, restlessness, dizziness, photosensitivity, sexual dysfunction, headache, dry mouth, HTN, tachycardia, rash, nausea, diarrhea, vomiting, constipation **Potential drug interactions:** anxiolytics, anesthetics, anticoagulants, MAOIs, SSRIs, TCAs, fexofenadine, digoxin, antifungals, immunosuppressants, opiates, statins, theophylline, antineoplatics, contraceptives, omeprazole, Imodium P450 agents
Turmeric	**Cancer**, ulcerative colitis, indigestion/dyspepsia, heart disease, neurodegenerative conditions, osteoarthritis, pruritus	**Adverse effects:** stomach upset, hypoglycemia, bleeding, ulcers, GERD, iron deficiency **Potential drug interactions:** anticoagulants, antacids, diabetic medications
Valerian	**Insomnia**, postmenopausal symptoms, joint pain, ADHD	**Adverse effects:** feeling anxious, restlessness, headache, excitability, sluggish in the morning if taken the previous night **Potential drug interactions:** CYP450 agents, sedatives, antihistamines, statins, antifungals, anesthesia, alprazolam (Xanax), alcohol
Vitamin D	**Osteoporosis**, hypothyroid, hypocalcemia, hypothyroidism, osteomalacia, psoriasis, rickets, muscle pain/weakness, cancer prevention,	**Adverse effects:** hypercalcemia, increased thirst, metal taste in mouth, poor appetite, weight loss, bone pain, tiredness, sore eyes, itchy skin, vomiting, diarrhea, constipation, frequent need to urinate, muscle problems. **Potential drug interactions:** antacids, cholestyramine, orlistat, mineral oil,

Appendix D

Dietary Reference Intakes

TABLE D.1 Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels, Elements
Food and Nutrition Board, Institute of Medicine, National Academies

Life Stage Group	Vitamin A (μg/d)[a]	Vitamin C (mg/d)	Vitamin D (μg/d)[b,c]	Vitamin E (mg/d)[d]	Vitamin K (μg/d)	Thiamin (mg/d)	Riboflavin (mg/d)	Niacin (mg/d)[e]	Vitamin B_6 (mg/d)	Folate (μg/d)[f]	Vitamin B_{12} (μg/d)	Pantothenic Acid (mg/d)	Biotin (μg/d)	Choline (mg/d)[g]
Infants														
0 to 6 mo	400*	40*	10	4*	2.0*	0.2*	0.3*	2*	0.1*	65*	0.4*	1.7*	5*	125*
6 to 12 mo	500*	50*	10	5*	2.5*	0.3*	0.4*	4*	0.3*	80*	0.5*	1.8*	6*	150*
Children														
1–3 y	300	15	15	6	30*	0.5	0.5	6	0.5	150	0.9	2*	8*	200*
4–8 y	400	25	15	7	55*	0.6	0.6	8	0.6	200	1.2	3*	12*	250*
Males														
9–13 y	600	45	15	11	60*	0.9	0.9	12	1.0	300	1.8	4*	20*	375*
14–18 y	900	75	15	15	75*	1.2	1.3	16	1.3	400	2.4	5*	25*	550*
19–30 y	900	75	15	15	75*	1.2	1.3	16	1.3	400	2.4	5*	25*	550*
31–50 y	900	75	15	15	75*	1.2	1.3	16	1.3	400	2.4	5*	25*	550*
51–70 y	900	75	15	15	75*	1.2	1.3	16	1.3	400	2.4[h]	5*	25*	550*
>70 y	900	75	15	15	75*	1.2	1.3	16	1.3	400	2.4[h]	5*	25*	550*
Females														
9–13 y	600	45	15	11	60*	0.9	0.9	12	1.0	300	1.8	4*	20*	375*
14–18 y	700	65	15	15	75*	1.0	1.0	14	1.2	400[i]	2.4	5*	25*	400*
19–30 y	700	75	15	15	90*	1.1	1.1	14	1.3	400[i]	2.4	5*	25*	400*
31–50 y	700	75	15	15	90*	1.1	1.1	14	1.3	400[i]	2.4	5*	25*	400*
51–70 y	700	75	15	15	90*	1.1	1.1	14	1.5	400	2.4[b]	5*	25*	400*
>70 y	700	75	20	15	90*	1.1	1.1	14	1.5	400	2.4[b]	5*	25*	400*
Pregnancy														
14–18 y	750	80	15	15	75*	1.4	1.4	18	1.9	600[j]	2.6	6*	30*	450*
19–30 y	770	85	15	15	90*	1.4	1.4	18	1.9	600[j]	2.6	6*	30*	450*
31–50 y	770	85	15	15	90*	1.4	1.4	18	1.9	600[j]	2.6	6*	30*	450*
Lactation														
14–18 y	1,200	115	15	19	75*	1.4	1.6	17	2.0	500	2.8	7*	35*	550*
19–30 y	1,300	120	15	19	90*	1.4	1.6	17	2.0	500	2.8	7*	35*	550*
31–50 y	1,300	120	15	19	90*	1.4	0.6	17	2.0	500	2.8	7*	35*	550*

National Institutes of Health. Nutrient Recommendations and Databases. Accessed January 6, 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK560681/table/summarytables.t2/?report=objectonly.

Recommended Dietary Allowances (RDAs) are shown in ordinary type and Adequate Intakes (AIs) in ordinary type followed by an asterisk (*). An RDA is the average daily dietary intake level sufficient to meet the nutrient requirements of nearly all (97–98 percent) healthy individuals in a group. It is calculated from an Estimated Average Requirement (EAR). If sufficient scientific evidence is not available to establish an EAR, and thus calculate an RDA, an AI is usually developed. For healthy breast-fed infants, an AI is the mean intake. The AI for other life stage and gender groups is believed to cover the needs of all healthy individuals in the groups, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

[a]As retinol activity equivalents (RAEs). 1 RAE = 1 μg retinol, 12 μg β-carotene, 24 μg α-carotene, or 24 μg β-cryptoxanthin. The RAE for dietary provitamin A carotenoids is two-fold greater than retinol equivalents (REs), whereas the RAE for preformed vitamin A is the same as RE.

[b]As cholecalciferol. 1 μg cholecalciferol = 40 IU vitamin D.

[c]Under the assumption of minimal sunlight.

[d]As α-tocopherol. α-tocopherol includes RRR-α-tocopherol, the only form of α-tocopherol that occurs naturally in foods, and the 2R-stereoisomeric forms of α-tocopherol (RRR-, RSR-, RRS-, and RSS-α-tocopherol) that occur in fortified foods and supplements. It does not include the 2S-stereoisomeric forms of α-tocopherol (SRR-, SSR-, SRS-, and SSS-α-tocopherol), also found in fortified foods and supplements.

[e]As niacin equivalents (NE). 1 mg of niacin = 60 mg of tryptophan; 0–6 months = preformed niacin (not NE).

[f]As dietary folate equivalents (DFE). 1 DFE = 1 μg food folate = 0.6 μg of folic acid from fortified food or as a supplement consumed with food = 0.5 μg of a supplement taken on an empty stomach.

[g]Although AIs have been set for choline, there are few data to assess whether a dietary supply of choline is needed at all stages of the life cycle, and it may be that the choline requirement can be met by endogenous synthesis at some of these stages.

[h]Because 10 to 30 percent of older people may malabsorb food-bound B12, it is advisable for those older than 50 years to meet their RDA mainly by consuming foods fortified with B12 or a supplement containing B12.

[i]In view of evidence linking folate intake with neural tube defects in the fetus, it is recommended that all women capable of becoming pregnant consume 400 μg from supplements or fortified foods in addition to intake of food folate from a varied diet.

[j]It is assumed that women will continue consuming 400 μg from supplements or fortified food until their pregnancy is confirmed and they enter prenatal care, which ordinarily occurs after the end of the periconceptional period—the critical time for formation of the neural tube.

Description

TABLE D.2 Dietary Reference Intakes (DRIs): Recommended Dietary Allowances and Adequate Intakes, Vitamins
Food and Nutrition Board, Institute of Medicine, National Academies

Life Stage Group	Calcium (mg/d)	Chromium (µg/d)	Copper (µg/d)	Fluoride (mg/d)	Iodine (µg/d)	Iron (mg/d)	Magnesium (mg/d)	Manganese (mg/d)	Molybdenum (µg/d)	Phosphorus (mg/d)	Selenium (µg/d)	Zinc (mg/d)	Potassium (g/d)	Sodium (g/d)	Chloride (g/d)
Infants															
0–6 mo	200*	0.2*	200*	0.01*	110*	0.27*	30*	0.003*	2*	100*	15*	2*	0.4*	0.11*	0.18*
7–12 mo	260*	5.5*	220*	0.5*	130*	11	75*	0.6*	3*	275*	20*	3	0.86*	0.37*	0.57*
Children															
1–3 y	700	11*	340	0.7*	90	7	80	1.2*	17	460	20	3	2.0*	.8*	1.5*
4–8 y	1,000	15*	440	1*	90	10	130	1.5*	22	500	30	5	2.3*	1.0*	1.9*
Males															
9–13 y	1,300	25*	700	2*	120	8	240	1.9*	34	1,250	40	8	2.5*	1.5*	2.3*
14–18 y	1,300	35*	890	3*	150	11	410	2.2*	43	1,250	55	11	3.0*	1.5*	2.3*
19–30 y	1,000	35*	900	4*	150	8	400	2.3*	45	700	55	11	3.4*	1.5*	2.3*
31–50 y	1,000	35*	900	4*	150	8	420	2.3*	45	700	55	11	3.4*	1.5*	2.3*
51–70 y	1,000	35*	900	4*	150	8	420	2.3*	45	700	55	11	3.4*	1.5*	2.0*
>70 y	1,200	35*	900	4*	150	8	420	2.3*	45	700	55	11	3.4*	1.5*	1.8*
Females															
9–13 y	1,300	21*	700	2*	120	8	240	1.6*	34	1,250	40	8	2.3*	1.5*	2.3*
14–18 y	1,300	24*	890	3*	150	15	360	1.6*	43	1,250	55	9	2.3*	1.5*	2.3*
19–30 y	1,000	25*	900	3*	150	18	310	1.8*	45	700	55	8	2.6*	1.5*	2.3*
31–50 y	1,000	25*	900	3*	150	18	320	1.8*	45	700	55	8	2.6*	1.5*	2.3*
51–70 y	1,200	20*	900	3*	150	18	320	1.8*	45	700	55	8	2.6*	1.5*	2.0*
>70 y	1,200	20*	900	3*	150	18	320	1.8*	45	700	55	8	2.6*	1.5*	1.8*
Pregnancy															
14–18 y	1,300	29*	1,000	3*	220	27	400	2.0*	50	1,250	60	12	2.6*	1.5*	2.3*
19–30 y	1,000	30*	1,000	3*	220	27	350	2.0*	50	700	60	11	2.9*	1.5*	2.3*
31–50 y	1,000	30*	1,000	3*	220	27	360	2.0*	50	700	60	11	2.9*	1.5*	1.8*
Lactation															
14–18 y	1,300	44*	1,300	3*	290	10	360	2.6*	50	1,250	70	13	2.5*	1.5*	2.3*
19–30 y	1,000	45*	1,300	3*	290	9	310	2.6*	50	700	70	12	2.8*	1.5*	2.3*
31–50 y	1,000	45*	1,300	3*	290	9	320	2.6*	50	700	70	12	2.8*	1.5*	2.3*

National Institutes of Health. Nutrient Recommendations and Databases. Accessed January 6, 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK56068/table/summarytables.t3/?report=objectonly.

Recommended Dietary Allowances (RDAs) are shown in ordinary type and Adequate Intakes (AIs) in ordinary type followed by an asterisk (*). An RDA is the average daily dietary intake level sufficient to meet the nutrient requirements of nearly all (97–98 percent) healthy individuals in a group. It is calculated from an Estimated Average Requirement (EAR). If sufficient scientific evidence is not available to establish an EAR, and thus calculate an RDA, an AI is usually developed. For healthy breast-fed infants, an AI is the mean intake. The AI for other life stage and gender groups is believed to cover the needs of all healthy individuals in the groups, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

Description

TABLE D.3 Dietary Reference Intakes (DRIs): Recommended Dietary Allowances and Adequate Intakes, Total Water and Macronutrients Food and Nutrition Board, Institute of Medicine, National Academies

Food and Nutrition Board, Institute of Medicine, National Academies

Life Stage Group	Total Water[a] (L/d)	Carbohydrate (g/d)	Total Fiber (g/d)	Fat (g/d)	Linoleic Acid (g/d)	α-Linolenic Acid (g/d)	Protein[b] (g/d)
Infants							
0 to 6 mo	0.7*	60*	ND	31*	4.4*	0.5*	9.1*
6 to 12 mo	0.8*	95*	ND	30*	4.6*	0.5*	11.0
Children							
1–3 y	1.3*	130	19*	ND[c]	7*	0.7*	13
4–8 y	1.7*	130	25*	ND	10*	0.9*	19
Males							
9–13 y	2.4*	130	31*	ND	12*	1.2*	34
14–18 y	3.3*	130	38*	ND	16*	1.6*	52
19–30 y	3.7*	130	38*	ND	17*	1.6*	56
31–50 y	3.7*	130	38*	ND	17*	1.6*	56
51–70 y	3.7*	130	30*	ND	14*	1.6*	56
>70 y	3.7*	130	30*	ND	14*	1.6*	56
Females							
9–13 y	2.1*	130	26*	ND	10*	1.0*	34
14–18 y	2.3*	130	26*	ND	11*	1.1*	46
19–30 y	2.7*	130	25*	ND	12*	1.1*	46
31–50 y	2.7*	130	25*	ND	12*	1.1*	46
51–70 y	2.7*	130	21*	ND	11*	1.1*	46
>70 y	2.7*	130	21*	ND	11*	1.1*	46
Pregnancy							
14–18 y	3.0*	175	28*	ND	13*	1.4*	71
19–30 y	3.0*	175	28*	ND	13*	1.4*	71
31–50 y	3.0*	175	28*	ND	13*	1.4*	71
Lactation							
14–18 y	3.8*	210	29*	ND	13*	1.3*	71
19–30 y	3.8*	210	29*	ND	13*	1.3*	71
31–50 y	3.8*	210	29*	ND	13*	1.3*	71

National Institutes of Health. Nutrient Recommendations and Databases. Accessed January 6, 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK56068/table/summarytables.t4/?report=objectonly.

Recommended Dietary Allowances (RDAs) are shown in ordinary type and Adequate Intakes (AIs) in ordinary type followed by an asterisk (*). An RDA is the average daily dietary intake level sufficient to meet the nutrient requirements of nearly all (97–98 percent) healthy individuals in a group. It is calculated from an Estimated Average Requirement (EAR). If sufficient scientific evidence is not available to establish an EAR, and thus calculate an RDA, an AI is usually developed. For healthy breast-fed infants, an AI is the mean intake. The AI for other life stage and gender groups is believed to cover the needs of all healthy individuals in the groups, but lack of data or uncertainty in the data prevent being able to specify with confidence the percentage of individuals covered by this intake.

[a]Total water includes all water contained in food, beverages, and drinking water.
[b]Based on g protein per kg of body weight for the reference body weight, e.g., for adults 0.8 g/kg body weight for the reference body weight.
[c]Not determined.

Description

TABLE D.4 Dietary Reference Intakes (DRIs): Acceptable Macronutrient Distribution Ranges
Food and Nutrition Board, Institute of Medicine, National Academies

Macronutrient	Range (Percent of Energy)		
	Children, 1–3 y	Children 4–18 y	Adults
Fat	30–40	25–35	20–35
n-6 polyunsaturated fatty acids[a] (linoleic acid)	5–10	5–10	5–10
n-3 polyunsaturated fatty acids[a] (α-linolenic acid)	0.6–1.2	0.6–1.2	0.6–1.2
Carbohydrate	45–65	45–65	45–65
Protein	5–20	10–30	10–35

National Institutes of Health. Nutrient Recommendations and Databases. Accessed January 6, 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK56068/table/summarytables.t5/?report=objectonly.

[a]Approximately 10 percent of the total can come from longer-chain *n*-3 or *n*-6 fatty acids.

Description

TABLE D.5 Dietary Reference Intakes (DRIs): Additional Macronutrient Recommendations. Food and Nutrition Board, Institute of Medicine, National Academies

Macronutrient	Recommendation
Dietary cholesterol	As low as possible while consuming a nutritionally adequate diet
Trans fatty acids	As low as possible while consuming a nutritionally adequate diet
Saturated fatty acids	As low as possible while consuming a nutritionally adequate diet
Added sugars[a]	Limit to no more than 25% of total energy

National Institutes of Health. Nutrient Recommendations and Databases. Accessed January 6, 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK56068/table/summarytables.t6/?report=objectonly.
aNot a recommended intake. A daily intake of added sugars that individuals should aim for to achieve a healthful diet was not set.

TABLE D.6 Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels, Vitamins
Food and Nutrition Board, Institute of Medicine, National Academies

Life Stage Group	Vitamin A (μg/d)[a]	Vitamin C (mg/d)	Vitamin D (μg/d)	Vitamin E (mg/d)[b,c]	Vitamin K	Thiamin	Riboflavin	Niacin (mg/d)[c]	Vitamin B_6 (mg/d)	Folate (μg/d)[c]	Vitamin B_{12}	Pantothenic Acid	Biotin	Choline (g/d)	Carotenoids[d]	
Infants																
0 to 6 mo	600	ND[e]	25	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	ND	
6 to 12 mo	600	ND	38	ND	ND	ND		ND	ND	ND	ND	ND	ND	ND	ND	ND
Children																
1–3 y	600	400	63	200	ND	ND		ND	10	30	300	ND	ND	ND	1.0	ND
4–8 y	900	650	75	300	ND	ND		ND	15	40	400	ND	ND	ND	1.0	ND
Males																
9–13 y	1,700	1,200	100	600	ND	ND		ND	20	60	600	ND	ND	ND	2.0	ND
14–18 y	2,800	1,800	100	800	ND	ND		ND	30	80	800	ND	ND	ND	3.0	ND
19–30 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
31–50 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
51–70 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
>70 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
Females																
9–13 y	1,700	1,200	100	600	ND	ND		ND	20	60	600	ND	ND	ND	2.0	ND
14–18 y	2,800	1,800	100	800	ND	ND		ND	30	80	800	ND	ND	ND	3.0	ND
19–30 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
31–50 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
51–70 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
>70 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
Pregnancy																
14–18 y	2,800	1,800	100	800	ND	ND		ND	30	80	800	ND	ND	ND	3.0	ND
19–30 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
31–50 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
Lactation																
14–18 y	2,800	1,800	100	800	ND	ND		ND	30	80	800	ND	ND	ND	3.0	ND
19–30 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND
31–50 y	3,000	2,000	100	1,000	ND	ND		ND	35	100	1,000	ND	ND	ND	3.5	ND

National Institutes of Health. Nutrient Recommendations and Databases. Accessed January 6, 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK56068/table/summarytables.t7/?report=objectonly.

A Tolerable Upper Intake Level (UL) is the highest level of daily nutrient intake that is likely to pose no risk of adverse health effects to almost all individuals in the general population. Unless otherwise specified, the UL represents total intake from food, water, and supplements. Due to a lack of suitable data, ULs could not be established for vitamin K, thiamin, riboflavin, vitamin B_{12}, pantothenic acid, biotin, and carotenoids. In the absence of a UL, extra caution may be warranted in consuming levels above recommended intakes. Members of the general population should be advised not to routinely exceed the UL. The UL is not meant to apply to individuals who are treated with the nutrient under medical supervision or to individuals with predisposing conditions that modify their sensitivity to the nutrient.

[a] As preformed vitamin A only.
[b] As α-tocopherol; applies to any form of supplemental α-tocopherol.
[c] The ULs for vitamin E, niacin, and folate apply to synthetic forms obtained from supplements, fortified foods, or a combination of the two.
[d] β-Carotene supplements are advised only to serve as a provitamin A source for individuals at risk of vitamin A deficiency.
[e] ND = Not determinable due to lack of data of adverse effects in this age group and concern with regard to lack of ability to handle excess amounts. Source of intake should be from food only to prevent high levels of intake.

Description

TABLE D.7 Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels, Elements
Food and Nutrition Board, Institute of Medicine, National Academies

Life Stage Group	Arsenic[a]	Boron (mg/d)	Calcium (mg/d)	Chromium	Copper (µg/d)	Fluoride (mg/d)	Iodine (µg/d)	Iron (mg/d)	Magnesium (mg/d)[b]	Manganese (mg/d)	Molybdenum (µg/d)	Nickel (mg/d)	Phosphorus (mg/d)	Selenium (µg/d)	Silicon[c]	Vanadium (mg/d)[d]	Zinc (mg/d)	Sodium (g/d)	Chloride (g/d)
Infants																			
0 to 6 mo	ND[e]	ND	1,000	ND	ND	0.7	ND	40	ND	ND	ND	ND	ND	45	ND	ND	4	ND	ND
6 to 12 mo	ND	ND	1,500	ND	ND	0.9	ND	40	ND	ND	ND	ND	ND	60	ND	ND	5	ND	ND
Children																			
1–3 y	ND	3	2,500	ND	1,000	1.3	200	40	65	2	300	0.2	3	90	ND	ND	7	1.5	2.3
4–8 y	ND	6	2,500	ND	3,000	2.2	300	40	110	3	600	0.3	3	150	ND	ND	12	1.9	2.9
Males																			
9–13 y	ND	11	3,000	ND	5,000	10	600	40	350	6	1,100	0.6	4	280	ND	ND	23	2.2	3.4
14–18 y	ND	17	3,000	ND	8,000	10	900	45	350	9	1,700	1.0	4	400	ND	ND	34	2.3	3.6
19–30 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	ND	34	2.3	3.6
31–50 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	ND	34	2.3	3.6
51–70 y	ND	20	2,000	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	ND	34	2.3	3.6
>70 y	ND	20	2,000	ND	10,000	10	1,100	45	350	11	2,000	1.0	3	400	ND	ND	34	2.3	3.6
Females																			
9–13 y	ND	11	3,000	ND	5,000	10	600	40	350	6	1,100	0.6	4	280	ND	ND	23	2.2	3.4
14–18 y	ND	17	3,000	ND	8,000	10	900	45	350	9	1,700	1.0	4	400	ND	ND	34	2.3	3.6
19–30 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	1.8	40	2.3	3.6
31–50 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	1.8	40	2.3	3.6
51–70 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	1.8	40	2.3	3.6
>70 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	3	400	ND	1.8	40	2.3	3.6
Pregnancy																			
14–18 y	ND	17	3,000	ND	8,000	10	900	45	350	9	1,700	1.0	3.5	400	ND	ND	34	2.3	3.6
19–30 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	3.5	400	ND	ND	40	2.3	3.6
31–50 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	3.5	400	ND	ND	40	2.3	3.6
Lactation																			
14–18 y	ND	17	3,000	ND	8,000	10	900	45	350	9	1,700	1.0	4	400	ND	ND	34	2.3	3.6
19–30 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	ND	40	2.3	3.6
31–50 y	ND	20	2,500	ND	10,000	10	1,100	45	350	11	2,000	1.0	4	400	ND	ND	40	2.3	3.6

National Institutes of Health. Nutrient Recommendations and Databases. Accessed January 6, 2024. Available at: https://www.ncbi.nlm.nih.gov/books/NBK56068/table/summarytables.t8/?report=objectonly.

A Tolerable Upper Intake Level (UL) is the highest level of daily nutrient intake that is likely to pose no risk of adverse health effects to almost all individuals in the general population. Unless otherwise specified, the UL represents total intake from food, water, and supplements. Due to a lack of suitable data, ULs could not be established for vitamin K, thiamin, riboflavin, vitamin B_{12}, pantothenic acid, biotin, and carotenoids. In the absence of a UL, extra caution may be warranted in consuming levels above recommended intakes. Members of the general population should be advised not to routinely exceed the UL. The UL is not meant to apply to individuals who are treated with the nutrient under medical supervision or to individuals with predisposing conditions that modify their sensitivity to the nutrient.

[a]Although the HYPERLINK "https://www.ncbi.nlm.nih.gov/books/n/nap13050/appendixes.app1/def-item/appendixes.app1.gl1-d104/"UL was not determined for arsenic, there is no justification for adding arsenic to food or supplements.

[b]The ULs for magnesium represent intake from a pharmacological agent only and do not include intake from food and water.

[c]Although silicon has not been shown to cause adverse effects in humans, there is no justification for adding silicon to supplements.

[d]Although vanadium in food has not been shown to cause adverse effects in humans, there is no justification for adding vanadium to food, and vanadium supplements should be used with caution. The HYPERLINK "https://www.ncbi.nlm.nih.gov/books/n/nap13050/appendixes.app1/def-item/appendixes.app1.gl1-d104/"UL is based on adverse effects in laboratory animals, and this data could be used to set a UL for adults but not children and adolescents.

[e]HYPERLINK "https://www.ncbi.nlm.nih.gov/books/n/nap13050/appendixes.app1/def-item/appendixes.app1.gl1-d63/"ND = Not determinable due to lack of data of adverse effects in this age group and concern with regard to lack of ability to handle excess amounts. Source of intake should be from food only to prevent high levels of intake.

Description



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A table is titled Estimating Frame Size Using Wrist Circumference. It is divided into 2 sections. Section 1 is titled Female Wrist Measurements. It has the headers: Blank, Height <5'2", Height 5'2"–5'5", and Height >5'5". Row 1. Blank, Small. Height <5'2", <5.5". Height 5'2"–5'5", <6.0". Height >5'5", <6.25". Row 2. Blank, Medium. Height <5'2", 5.5"–5.75". Height 5'2"–5'5", 6.0"–6.25". Height >5'5", 6.26"–6.5". Row 3. Blank, Large. Height <5'2", >5.75". Height 5'2"–5'5", >6.25". Height >5'5", >6.5". Section 2 is titled Male Wrist Measurements. It has the headers: Blank, Blank, Blank, and Height >5'5". Row 4. Blank, Small. Blank, Blank. Blank, Blank. Height >5'5", 5.5"–6.6". Row 5. Blank, Medium. Blank, Blank. Blank, Blank. Height >5'5", 6.5"–7.5". Row 6. Blank, Large. Blank, Blank. Blank, Blank. Height >5'5", >7.5".

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A table is titled Classification of Overweight and Obesity by BMI, Waist Circumference, and Associated Disease Risk. The table has 3 columns labeled BMI (kg/m2), Weight Status, and Disease Riska Relative to Normal Weight and Waist Circumference. The column labeled Disease Riska Relative to Normal Weight and Waist Circumference is subdivided into 2 columns labeled Men ≤40", Women ≤35" and Men >40",

Women >35". The row entries are as follows. Row 1. BMI (kg/m2), <18.5. Weight Status, Underweight. Disease Riska Relative to Normal Weight and Waist Circumference, Men ≤40", Women ≤35": —. Men >40", Women >35": —. Row 2. BMI (kg/m2), 18.5 to <25. Weight Status, Healthy range. Disease Riska Relative to Normal Weight and Waist Circumference, Men ≤40", Women ≤35": —. Men >40", Women >35": —. Row 3. BMI (kg/m2), 25.0 to <30. Weight Status, Overweight. Disease Riska Relative to Normal Weight and Waist Circumference, Men ≤40", Women ≤35": Increased. Men >40", Women >35": High. Row 4. BMI (kg/m2), 30.0 to <35. Weight Status, Obesity class 1. Disease Riska Relative to Normal Weight and Waist Circumference, Men ≤40", Women ≤35": High. Men >40", Women >35": Very high. Row 5. BMI (kg/m2), 35.0 to <40. Weight Status, Obesity class 2. Disease Riska Relative to Normal Weight and Waist Circumference, Men ≤40", Women ≤35": Very high. Men >40", Women >35": Very high. Row 6. BMI (kg/m2), ≥40.0. Weight Status, Obesity class 3. Disease Riska Relative to Normal Weight and Waist Circumference, Men ≤40", Women ≤35": Extremely high. Men >40", Women >35": Extremely high. Footnote below the table indicates that Disease risk for type 2 diabetes, hypertension, and cardiovascular disease.

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A table is titled Calculation and Interpretation of Creatinine Height Index. It is divided into 2 sections. Section 1 is titled 24-Hour Urine Collection; Meat-Free Diet (Ideally); Compare Creatinine with Standard for Height/Gender. Text below the section reads: % CHI = 24 urinary creatinine × 100/expected 24-hr urinary excretion (mg). Interpret: 60%–80%, mild depletion; 40%–59%, moderate depletion; <40%, severe depletion. Affected by: Renal dysfunction, advanced age, stress, trauma, sepsis, strenuous exercise, use of corticosteroids. Section 2 is titled 24-Hour Urinary Creatinine Excretion in Adults. There are 2 columns labeled Males: 23 mg/kg/IBW, and Females: 18 mg/kg/IBW. Both the tables are subdivided into 2 columns with the headers: Height (cm), Creatinine (mg). Row 1. Males: 23 mg/kg/IBW, Height (cm): 157.5. Creatinine (mg): 1,288. Females: 18 mg/kg/IBW, Height (cm): 147.3. Creatinine (mg): 830. Row 2. Males: 23 mg/kg/IBW, Height (cm): 160.0. Creatinine (mg): 1,325. Females: 18 mg/kg/IBW, Height (cm): 149.9. Creatinine (mg): 851. Row 3. Males: 23 mg/kg/IBW, Height (cm): 162.6. Creatinine (mg): 1,359. Females: 18 mg/kg/IBW, Height (cm): 152.4. Creatinine (mg): 875. Row 4. Males: 23 mg/kg/IBW, Height (cm): 165.1. Creatinine (mg): 1,386. Females: 18 mg/kg/IBW, Height (cm): 154.9. Creatinine (mg): 900. Row 5. Males: 23 mg/kg/IBW, Height (cm): 167.6. Creatinine (mg): 1,426. Females:

18 mg/kg/IBW, Height (cm): 157.5. Creatinine (mg): 925. Row 6. Males: 23 mg/kg/IBW, Height (cm): 170.2. Creatinine (mg): 1,467. Females: 18 mg/kg/IBW, Height (cm): 160.0. Creatinine (mg): 949. Row 7. Males: 23 mg/kg/IBW, Height (cm): 172.7. Creatinine (mg): 1,513. Females: 18 mg/kg/IBW, Height (cm): 162.6. Creatinine (mg): 977. Row 8. Males: 23 mg/kg/IBW, Height (cm): 175.3. Creatinine (mg): 1,555. Females: 18 mg/kg/IBW, Height (cm): 165.1. Creatinine (mg): 1,006. Row 9. Males: 23 mg/kg/IBW, Height (cm): 177.8. Creatinine (mg): 1,596. Females: 18 mg/kg/IBW, Height (cm): 167.6. Creatinine (mg): 1,044. Row 10. Males: 23 mg/kg/IBW, Height (cm): 180.3. Creatinine (mg): 1,642. Females: 18 mg/kg/IBW, Height (cm): 170.2. Creatinine (mg): 1,076. Row 11. Males: 23 mg/kg/IBW, Height (cm): 182.9. Creatinine (mg): 1,691. Females: 18 mg/kg/IBW, Height (cm): 172.7. Creatinine (mg): 1,109. Row 12. Males: 23 mg/kg/IBW, Height (cm): 185.4. Creatinine (mg): 1,739. Females: 18 mg/kg/IBW, Height (cm): 175.3. Creatinine (mg): 1,141. Row 13. Males: 23 mg/kg/IBW, Height (cm): 188.0. Creatinine (mg): 1,785. Females: 18 mg/kg/IBW, Height (cm): 177.8. Creatinine (mg): 1,174. Row 14. Males: 23 mg/kg/IBW, Height (cm): 190.5. Creatinine (mg): 1,831. Females: 18 mg/kg/IBW, Height (cm): 180.3. Creatinine (mg): 1,206. Row 15. Males: 23

mg/kg/IBW, Height (cm): 193.0. Creatinine (mg): 1,891. Females: 18 mg/kg/IBW, Height (cm): 182.9. Creatinine (mg): 1,240. The text below the table indicates that CHI, creatine height index.

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A table is titled Determining Adult Malnutrition. It has the headers: Context of Malnutrition, it is subdivided into Degree of Malnutrition. Chronic Illness, it is subdivided into Malnutrition of Moderate Degree and Severe Protein-Calorie Malnutrition. Acute Illness or Injury, it is subdivided into Malnutrition of Moderate Degree and Severe Protein-Calorie Malnutrition. Social/Environmental Circumstances, it is subdivided into Malnutrition of Moderate Degree and Severe Protein-Calorie Malnutrition. Row 1. Context of Malnutrition, Degree of Malnutrition: Energy intake: Compare recent intake with estimated energy needs and report inadequate intake as a percentage of estimated needs over time. Chronic Illness, Malnutrition of Moderate Degree: <75% of estimated energy requirement for ≥1 mo. Severe Protein-Calorie Malnutrition: ≤75% of estimated energy requirement for ≥1 mo. Acute Illness or Injury, Malnutrition of Moderate Degree: <75% of estimated energy requirement for >7 d. Severe Protein-Calorie Malnutrition: ≤50% of estimated energy requirement for ≥5 d. Social/Environmental Circumstances,

Malnutrition of Moderate Degree: <75% of estimated energy requirement for ≥3 mo. Severe Protein-Calorie Malnutrition: ≤50% of estimated energy requirement for ≥1 mo. Row 2. Context of Malnutrition, Degree of Malnutrition: Weight loss: Evaluate weight in light of other clinical findings including hydration. Report weight change over time as a percentage of weight lost from baseline. Chronic Illness, Malnutrition of Moderate Degree: 5% in 1 mo; 7.5% in 3 mo; 10% in 6 mo; 20% in 12 mo. Severe Protein-Calorie Malnutrition: >5% in 1 mo; >7.5% in 3 mo; >10% in 6 mo; >20% in 12 mo. Acute Illness or Injury, Malnutrition of Moderate Degree: 1–2% in 1 wk; 5% in 1 mo; 7.5% in 3 mo. Severe Protein-Calorie Malnutrition: >2% in 1 wk; >5% in 1 mo; >7.5% in 3 mo. Social/Environmental Circumstances, Malnutrition of Moderate Degree: 5% in 1 mo; 7.5% in 3 mo; 10% in 6 mo; 20% in 12 mo. Severe Protein-Calorie Malnutrition: >5% in 1 mo; >7.5% in 3 mo; >10% in 6 mo; >20% in 12 mo. Row 3. Context of Malnutrition, Degree of Malnutrition: Body fat: Loss of subcutaneous fat (orbital, triceps, fat overlying ribs). Chronic Illness, Malnutrition of Moderate Degree: Mild depletion. Severe Protein-Calorie Malnutrition: Severe depletion. Acute Illness or Injury, Malnutrition of Moderate Degree: Mild depletion. Severe Protein-Calorie Malnutrition: Moderate depletion. Social/Environmental Circumstances, Malnutrition of

Moderate Degree: Mild depletion. Severe Protein-Calorie Malnutrition: Severe depletion. Row 4. Context of Malnutrition, Degree of Malnutrition: Muscle mass: Loss of muscle (temples, clavicles, shoulders, scapula, thigh, calf). Chronic Illness, Malnutrition of Moderate Degree: Mild depletion. Severe Protein-Calorie Malnutrition: Severe depletion. Acute Illness or Injury, Malnutrition of Moderate Degree: Mild depletion. Severe Protein-Calorie Malnutrition: Moderate depletion. Social/Environmental Circumstances, Malnutrition of Moderate Degree: Mild depletion. Severe Protein-Calorie Malnutrition: Severe depletion. Row 5. Context of Malnutrition, Degree of Malnutrition: Fluid accumulation: General or local fluid accumulation (extremities, ascites, or vulva/scrotal edema). Chronic Illness, Malnutrition of Moderate Degree: Mild. Severe Protein-Calorie Malnutrition: Severe. Acute Illness or Injury, Malnutrition of Moderate Degree: Mild. Severe Protein-Calorie Malnutrition: Moderate to severe. Social/Environmental Circumstances, Malnutrition of Moderate Degree: Mild. Severe Protein-Calorie Malnutrition: Severe. Row 6. Context of Malnutrition, Degree of Malnutrition: Functional status: Based on standards supplied by manufacturer of dynamometer. Chronic Illness, Malnutrition of Moderate Degree: Not applicable. Severe Protein-Calorie Malnutrition: Measurably reduced. Acute Illness or Injury,

Malnutrition of Moderate Degree: Not applicable. Severe Protein-Calorie Malnutrition: Not recommended in intensive care setting. Social/Environmental Circumstances, Malnutrition of Moderate Degree: Not applicable. Severe Protein-Calorie Malnutrition: Measurably reduced.

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A table is titled Nutrition-Focused Physical Examination: Malnutrition Assessment. It has four column heads: Fat Area, Normal, Mild to Moderate, and Severe. Row 1. Fat Area, Orbital region—surrounding the eye (fluid retention may mask loss). Normal, Slightly bulged fat pads. Mild to Moderate, Slightly darker circles; somewhat hallow look. Severe, Dark circles; hallow look; depression; loose skin. Row 2. Fat Area, Upper arm region—triceps/biceps. Normal, Large space between fingers when pinching. Mild to Moderate, Some depth pinch but not ample; fingers almost touch. Severe, Very little space between folds, fingers touch. Row 3. Fat Area, Thoracic and lumbar region—ribs, lower back, midaxillary line. Normal, Chest is full; ribs do not show; slight to no protrusion of the iliac crest. Mild to Moderate, Ribs apparent; depressions between them less pronounced; iliac crest somewhat prominent. Severe, Depression between the ribs very apparent; iliac crest very prominent. Under this it has four

column heads: Muscle Area, Normal, Mild to Moderate, and Severe. Row 4. Muscle Area, Temple region—temporalis muscle. Normal, Can see/feel well-defined muscle. Mild to Moderate, Slight depression. Severe, Hollow; scooping depression. Row 5. Muscle Area, Clavicle and acromion bone region—deltoid muscle. Normal, Rounded curves at arm/shoulder/neck. Mild to Moderate, Acromion process may slightly protrude. Severe, Shoulder to arm joint looks square; bones prominent; acromion protrusion very prominent. Row 6. Muscle Area, Clavicle bone region—pectoralis major, deltoid, trapezius muscles. Normal, Not visible in male; visible but not prominent in female. Mild to Moderate, Visible in male; some protrusion in female. Severe, Protruding, prominent bone. Row 7. Muscle Area, Scapular bone region—trapezius, supraspinatus, infraspinatus muscles. Normal, Bones not prominent; no significant depressions. Mild to Moderate, Mild depression or bone may show slightly. Severe, Prominent, visible bones; depressions between ribs/scapula or shoulder/spine. Row 8. Muscle Area, Dorsal hand—interosseous muscle. Normal, Muscle bulges could be flat in some well-nourished people. Mild to Moderate, Slightly depressed. Severe, Depressed area between thumb–forefinger. Row 9. Muscle Area, Patellar region—quadriceps muscle. Normal, Muscles protrude; bones not prominent. Mild

to Moderate, Knee cap less prominent; more rounded. Severe, Bones prominent; little sign of muscle around knee. Row 10. Muscle Area, Anterior thigh region—quadriceps muscles. Normal, Well rounded; no depressions. Mild to Moderate, Mild depression on inner thigh. Severe, Depression/line on thigh; obviously thin. Row 11. Muscle Area, Posterior calf region—gastrocnemius muscle. Normal, Well-developed bulb of muscle. Mild to Moderate, Not well developed. Severe, Thin; minimal to no muscle definition. Row 12. Muscle Area, Edema. Normal, Normal. Mild to Moderate, Mild to Moderate. Severe, Severe. Row 13. Muscle Area, Edema—rule out other causes of edema; patient at dry weight. Normal, No sign of fluid accumulation. Mild to Moderate, Mild to moderate pitting; slight swelling of the extremity; indentation subsides quickly (0–30s). Severe, Deep to very deep pitting; depression lasts a short to moderate time (31–60s); extremity looks swollen (3–4+).

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A table is titled Nutrition-Focused Physical Examination: Nutrient Deficiency Assessment. It has the headers: Body System, Observed Conditions, is sub divided into Healthy and Abnormal, Implications. Row 1. Body System, Hair. Observed Conditions, Healthy: Normal distribution, shiny. Abnormal: Thin,

dull, dry, brittle, corkscrew hairs. Implications, Chemotherapy, protein or biotin deficit, vitamin C deficiency. Row 2. Body System, Eyes. Observed Conditions, Healthy: Bright, clear, shiny, pink conjunctiva. Abnormal: Sunken, dull, pale, dry conjunctiva, photophobia, xerosis. Implications, Deficiency of vitamin A, zinc, riboflavin. Row 3. Body System, Lips. Observed Conditions, Healthy: Moist, good color. Abnormal: Swollen, dry, red, cracked. Implications, Deficiency of riboflavin, pyridoxine, niacin. Row 4. Body System, Gums. Observed Conditions, Healthy: Pink, firm. Abnormal: Sore, spongy, red, swollen, bleed easily. Implications, Vitamin C deficiency. Row 5. Body System, Tongue. Observed Conditions, Healthy: Pink, presence of papillae. Abnormal: Purple, white or gray coating, smooth, slick. Implications, Deficiency of riboflavin, pyridoxine, folic acid, niacin, vitamin B12, iron. Row 6. Body System, Teeth. Observed Conditions, Healthy: Clean, intact, all present. Abnormal: Dentures, missing teeth, loss of tooth enamel. Implications, Calcium deficiency, poor diet. Row 7. Body System, Neck. Observed Conditions, Healthy: No swelling. Abnormal: Presence of nodule(s), goiter. Implications, Excess or deficiency of iodine. Row 8. Body System, Skin. Observed Conditions, Healthy: Smooth, slightly moist, good color. Abnormal: Pale, dry, scaly, bruises easily, pressure ulcers, dermatitis. Implications,

Deficiency of iron, vitamins A or C, zinc, essential fatty acid, protein; excess of niacin. Row 9. Body System, Legs. Observed Conditions, Healthy: Well-developed, firm musculature, no joint or bone pain. Abnormal: Calf tenderness, flaccid muscles, pain, edema, rickets, bone or joint pain. Implications, Deficiency of protein; vitamins A, C, or D; calcium. Row 10. Body System, Abdomen. Observed Conditions, Healthy: No swelling or pain. Abnormal: Mildly edematous, diarrhea, ascites. Implications, Deficiency of protein, niacin, zinc. Row 11. Body System, Hands/Nails. Observed Conditions, Healthy: Smooth. Abnormal: Brittle nails, atrophied fine muscles, spoon-shaped nails. Implications, Deficiency of protein, iron. Row 12. Body System, Musculoskeletal, adipose. Observed Conditions, Healthy: Normal bone, muscle, and fat development. Abnormal: Calf tenderness, loss of subcutaneous fat, emaciated appearance, pain, decreased grip strength, hollow cheeks, fractures, osteoporosis. Implications, Deficiency of protein, thiamin, vitamin C; energy or fluid deficit. Row 13. Body System, Neurologic. Observed Conditions, Healthy: Normal reflexes. Abnormal: CVA, limited reflexes, disorientation, paralysis, convulsions, dementia. Implications, Deficiency of thiamin, niacin, vitamins B6 or B12, folic acid, iodine, phosphorus, calcium,

magnesium. The text below the table indicates that CVA, cerebrovascular accident.

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A table is titled Typical Daily Intake Form. It has the headers: Meal Timing, Food Item, Amount, and Where Eaten. Row 1. Meal Timing, 8:00 a.m. Food Item, Oatmeal. Amount, 1 cup. Where Eaten, Kitchen table at home.

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A table is titled Evaluation of Dietary Intake. It has the headers: Group, Servings/Day, Recommended, and Adequate/Excess. Row 1. Group, Dairy. Servings/Day, Blank. Recommended, Blank. Adequate/Excess, Blank. Row 2. Group, Protein. Servings/Day, Blank. Recommended, Blank. Adequate/Excess, Blank. Row 3. Group, Fruit. Servings/Day, Blank. Recommended, Blank. Adequate/Excess, Blank. Row 4. Group, Vegetable. Servings/Day, Blank. Recommended, Blank. Adequate/Excess, Blank. Row 5. Group, Starch. Servings/Day, Blank. Recommended, Blank. Adequate/Excess, Blank. Row 6. Group, Fat/sweets. Servings/Day, Blank. Recommended, Blank. Adequate/Excess, Blank. Row 7. Overall Diet Adequacy: _____ yes _____ no. The entries under

Specific Nutrients: are as follows. Row 8. Deficit of: _____ kcals _____ PRO _____ fiber _____ Vit A _____ Vit C _____ Fe _____ Ca _____. Row 9. Other. Row 10. Excess of: _____ kcals _____ Fat _____ SFA _____ Chol _____ Sugar _____ Alcohol _____ Na. Row 11. Other: _____. Row 12. Summary:. The text below the table indicates that PRO, protein; Fe, iron; Ca, calcium; SFA, saturated fatty acid; Chol, cholesterol; NA, sodium.

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A table is titled Guidelines for Pregnancy Weight Gain. It has the headers: Category, BMI (WHO), Weight Gain (kg), and Rate of Weight Gain for 2nd and 3rd Trimesters*. Row 1. Category, Underweight. BMI (WHO), <18.5. Weight Gain (kg), 12.5–18. Rate of Weight Gain for 2nd and 3rd Trimesters*, 0.44–0.58 kg/wk. Row 2. Category, Normal weight. BMI (WHO), 18.5–24.9. Weight Gain (kg), 11.5–16. Rate of Weight Gain for 2nd and 3rd Trimesters*, 0.35–0.50 kg/wk. Row 3. Category, Overweight. BMI (WHO), 25.0–29.9. Weight Gain (kg), 7–11.5. Rate of Weight Gain for 2nd and 3rd Trimesters*, 0.23–0.33 kg/wk. Row 4. Category, Obese (all classes). BMI (WHO), ≥30.0. Weight Gain (kg), 5–9. Rate of Weight Gain for 2nd and 3rd Trimesters*, 0.17–0.27 kg/wk. Footnote below the table indicates that Calculations assume a 0.5 to 2 kg weight gain in the first trimester.

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A table is titled Percentage of Increase Over Nonpregnant Female for Daily Recommended Nutrient Intakes. It has the headers: Blank, Female, 14–18 yr, Pregnant, 14–18 yr, % Increase, Female, 19–30 yr, Pregnant, 19–30 yr, % Increase, Female, 31–50 yr, Pregnant, 31–50 yr, % Increase. The row entries are as follows under three sections. Section 1: Macronutrient. Row 1. Carbohydrates. 130 g. 175 g. 35%. 130 g. 175 g. 35%. 130. 175. 35%. Row 2. Protein. 46 g. 71 g. 54%. 46g. 71g. 54%. 46g. 71g. 54%. Row 3. Fibera. 25 g. 31 g. 24%. 28 g. 34 g. 21%. 25 g. 31 g. 24%. Section 2: Vitamin. Row 4. Vitamin A. 700 µg. 750 µg. 7%. 700 µg. 770 µg. 10%. 700 µg. 770 µg. 10%. Row 5. Vitamin D. 600 IU. 600 IU. 0%. 600 IU. 600 IU. 0%. 600 IU. 600 IU. 0%. Row 6. Vitamin C. 65 mg. 80 mg. 24%. 75 mg. 85 mg. 13%. 75 mg. 85 mg. 13%. Row 7. Vitamin E. 15 mg. 15 mg. 0%. 15 mg. 15 mg. 0%. 15 mg. 15 mg. 0%. Row 8. Vitamin K. 75 µg. 75 µg. 0%. 90 µg. 90 µg. 0%. 90 µg. 90 µg. 0%. Row 9. Thiamin B1. 1.0 mg. 1.4 mg. 40%. 1.1 mg. 1.4 mg. 28%. 1.1 mg. 1.4 mg. 28%. Row 10. Riboflavin. 1.0 mg. 1.4 mg. 40%. 1.1 mg. 1.4 mg. 28%. 1.1 mg. 1.4 mg. 28%. Row 11. Niacin. 14 mg. 18 mg. 29%. 14 mg. 18 mg. 29%. 14 mg. 18 mg. 29%. Row 12. Vitamin B6. 1.2 mg. 1.9 mg. 59%. 1.3 mg. 1.9 mg. 46%. 1.3 mg. 1.9 mg. 46%. Row 13. Vitamin B12. 2.4 µg. 2.6 µg. 8%. 2.4 µg. 2.6 µg. 8%.

2.4 µg. 2.6 µg. 8%. Row 14. Folate. 400 µg. 600 µ/day. 50%. 400 µg. 600 µg. 50%. 400 µg. 600 µg. 50%. Row 15. Choline. 400 mg. 450 mg. 12.5%. 425 mg. 450 mg. 6%. 425 mg. 450 mg. 6%. Section 3: Mineral. Row 16. Calcium. 1,300 mg. 1,300 mg. 0%. 1,000 mg. 1,000 mg. 0%. 1,000 mg. 1,000 mg. 0%. Row 17. Iodine. 150 µg. 220 µg. 47%. 150 µg. 220 µg. 47%. 150 µg. 220 µg. 47%. Row 18. Iron. 15 mg. 27 mg. 80%. 18 mg. 27 mg. 50%. 18 mg. 27 mg. 50%. Row 19. Magnesium. 360 mg. 400 mg. 11%. 310 mg. 350 mg. 13%. 320 mg. 360 mg. 13%. Row 20. Phosphorus. 1,250 mg. 1,250 mg. 0%. 700 mg. 700 mg. 0%. 700 mg. 700 mg. 0%. Row 21. Potassium. 2,300 mg. 2,600 mg. 13%. 2,600 mg. 2,900 mg. 12%. 2,600 mg. 2,900 mg. 12%. Row 22. Selenium. 55 µg. 60 µg. 9%. 55 µg. 60 µg. 9%. 55 µg. 60 µg. 9%. Row 23. Sodium. 2,300 mg. 2,300 mg. 0%. 2,300 mg. 2,300 mg. 0%. 2,300 mg. 2,300 mg. 0%. Row 24. Zinc. 9 mg. 12 mg. 34%. 8 mg. 11 mg. 38%. 8 mg. 11 mg. 38%. Footnote below the table indicates that For the 2nd trimester only.

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A table is titled The USDA MyPlate Plan. It has the headers: Food Group, Servings, is sub divided into 1st Trimestera and 2nd and 3rd Trimestersb, Serving Types. Row 1. Food Group, Whole grains. Servings, 1st Trimestera: 6 oz. 2nd and 3rd Trimestersb: 8 oz.

Serving Types, Whole wheat bread and chapati; Whole grain cereals and crackers; Brown and wild rice. Row 2. Food Group, Fruits. Servings, 1st Trimestera: 2 cups. 2nd and 3rd Trimestersb: 2 cups. Serving Types, Fresh, frozen, canned, or dried whole fruits. Row 3. Food Group, Vegetables. Servings, 1st Trimestera: 2.5 cups. 2nd and 3rd Trimestersb: 3 cups. Serving Types, Fresh, frozen, or canned; dark green, red, and orange varieties. Row 4. Food Group, Protein foods. Servings, 1st Trimestera: 5.5 oz. 2nd and 3rd Trimestersb: 6.5 oz. Serving Types, Seafood; Beans, peas and lentils; Unsalted nuts and seeds; Soy products; Eggs; Lean meats and poultry. Row 5. Food Group, Dairy. Servings, 1st Trimestera: 3 cups. 2nd and 3rd Trimestersb: 3 cups. Serving Types, Low-fat or fat-free dairy milk, yogurt and kefir; Lactose free dairy; Fortified soy beverages; Cheese. Row 6. Food Group, Added sugars. Servings, 1st Trimestera: <50 g/day. 2nd and 3rd Trimestersb: <60 g/day. Serving Types, Blank. Row 7. Food Group, Saturated fat. Servings, 1st Trimestera: <22g/day. 2nd and 3rd Trimestersb: <27 g/day. Serving Types, Blank. Row 8. Food Group, Sodium. Servings, 1st Trimestera: <2,300 mg/day. 2nd and 3rd Trimestersb: <2,300 mg/day. Serving Types, Blank. Footnote below the table indicates that aBased on a 2,000 kcal diet (1st trimester). bBased on a 2,400 kcal diet (2nd and 3rd trimesters).

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A table is titled Healthy Lacto-Ovo Vegetarian Eating Patterns. It has the headers: Food Group, Daily Servings (Based on 2,000 Calories), Weekly Servings (Based on 2,000 Calories), and Serving Types. Row 1. Food Group, Vegetables. Daily Servings(Based on 2,000 Calories), 2.5 (cup-eq). Weekly Servings (Based on 2,000 Calories), Blank. Serving Types, Blank. Row 2. Food Group, Dark-green vegetables. Daily Servings (Based on 2,000 Calories), Blank. Weekly Servings (Based on 2,000 Calories), 1.5 cup-eq/wk. Serving Types, Cooked kale, broccoli, bok choy, collard greens; Raw spinach or romaine lettuce. Row 3. Food Group, Red and orange vegetables. Daily Servings (Based on 2,000 Calories), Blank. Weekly Servings (Based on 2,000 Calories), 5.5 cup-eq/wk. Serving Types, Carrots, red pepper, and winter squash. Row 4. Food Group, Beans, peas, and lentils. Daily Servings (Based on 2,000 Calories), Blank. Weekly Servings (Based on 2,000 Calories), 1.5 cup-eq/wk. Serving Types, Black beans, lentils, chickpeas, pinto beans, or edamame. Row 5. Food Group, Starchy vegetables. Daily Servings (Based on 2,000 Calories), Blank. Weekly Servings (Based on 2,000 Calories), 5 cup-eq/wk. Serving Types, Potatoes, sweet potatoes, corn, green peas, or green lima beans. Row 6. Food Group, Other vegetables. Daily Servings (Based on 2,000 Calories), Blank. Weekly Servings (Based on 2,000

Calories), 4 cup-eq/wk. Serving Types, Mushrooms, cabbage, zucchini, or green peppers. Row 7. Food Group, Fruits. Daily Servings (Based on 2,000 Calories), 2 cup-eq. Weekly Servings (Based on 2,000 Calories), Blank. Serving Types, Orange, berries, melon, mangos, banana. Row 8. Food Group, Grains. Daily Servings (Based on 2,000 Calories), 6.5 oz-eq. Weekly Servings (Based on 2,000 Calories), Blank. Serving Types, Blank. Row 9. Food Group, Whole grains. Daily Servings (Based on 2,000 Calories), 3.5 oz-eq. Weekly Servings (Based on 2,000 Calories), Blank. Serving Types, Whole wheat bread, whole grain cereal, brown rice, millet, bulgur, and oatmeal. Row 10. Food Group, Refined grains. Daily Servings (Based on 2,000 Calories), 3 oz-eq. Weekly Servings (Based on 2,000 Calories), Blank. Serving Types, Enriched pasta, white rice, or cereal. Row 11. Food Group, Dairy. Daily Servings (Based on 2,000 Calories), 3 cup-eq. Weekly Servings (Based on 2,000 Calories), Blank. Serving Types, Low-fat milk, cheese, yogurt, or fortified soy beverage/yogurt. Row 12. Food Group, Protein Foods. Daily Servings (Based on 2,000 Calories), 3.5 oz-eq. Weekly Servings (Based on 2,000 Calories), Blank. Serving Types, Blank. Row 13. Food Group, Eggs. Daily Servings (Based on 2,000 Calories), Blank. Weekly Servings (Based on 2,000 Calories), 3 oz-eq/wk. Serving Types, Egg. Row 14. Food Group, Beans, peas, and lentils. Daily Servings

(Based on 2,000 Calories), Blank. Weekly Servings (Based on 2,000 Calories), 6 oz-eq/wk. Serving Types, Black beans, lentils, chickpeas. Row 15. Food Group, Soy products. Daily Servings (Based on 2,000 Calories), Blank. Weekly Servings (Based on 2,000 Calories), 8 oz-eq/wk. Serving Types, Cooked tofu. Row 16. Food Group, Nuts and seeds. Daily Servings (Based on 2,000 Calories), Blank. Weekly Servings (Based on 2,000 Calories), 7 oz-eq/wk. Serving Types, Peanut butter, almonds, sunflower seeds. Row 17. Food Group, Oils. Daily Servings (Based on 2,000 Calories), 27 g. Weekly Servings (Based on 2,000 Calories), Blank. Serving Types, Olive oil, avocado oil, canola oil.

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A table is titled Estimated Energy and Protein Requirements. It has the headers: Category, Age, Reference Weight (kg), DRI kcal/kg, and Pro g/kg. Row 1. Category, Infants (mo). Age, 0–2. 2–3. 4–6. 7–12. 13–35. Reference Weight (kg), N/A. 6. 6. 9. 12. DRI kcal/kg, —. 102. 82. 80. 82. Pro g/kg, 1.52. 1.52. 1.5. 1.2. 1.08. Row 2. Category, Boys (yr). Age, 3. 4–5. 6–7. 8. 9–11. 12–13. 14–16. 17–18. >18. Reference Weight (kg), 12. 20. 20. 20. 36. 36. 61. 61. 70. DRI kcal/kg, 85. 70. 64. 59. 49. 44. 39. 37. 36. Pro g/kg, 1.08. 0.95. 0.95. 0.95. 0.94. 0.94. 0.85. 0.85. 0.8. Row 3. Category, Girls (yr). Age, 3. 4–5. 6–7. 8. 9–11.

12–13. 14–16. 17–18. >18. Reference Weight (kg), 12. 20. 20. 20. 37. 37. 54. 54. 57. DRI kcal/kg, 82. 65. 61. 59. 42. 40. 33. 31. 34. Pro g/kg, 1.08. 0.95. 0.95. 0.95. 0.92. 0.92. 0.85. 0.85. 0.8.

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A table is titled Feeding Guidelines for Infants 0–12 Months Old. It has the headers: Age, Breast Milk and/or Fortified Infant Formula, Cereal, Bread, Grains, and Starches, Fruits and Vegetables, and Meats and Other Protein-Rich Foods. Row 1. Age, Birth–4 mo. Breast Milk and/or Fortified Infant Formula, 8–12 feedings; 2–6 oz/feeding; (18–32 oz/day). Cereal, Bread, Grains, and Starches, None. Fruits and Vegetables, None. Meats and Other Protein-Rich Foods, None. Row 2. Age, 4–6 mo. Breast Milk and/or Fortified Infant Formula, 486 feedings; 4–6 oz/feeding; (27–45 oz/day). Cereal, Bread, Grains, and Starches, None. Fruits and Vegetables, None. Meats and Other Protein-Rich Foods, None. Row 3. Age, 6–8 mo. Breast Milk and/or Fortified Infant Formula, 3–5 feedings; 6–8 oz/feeding; (24–32 oz/day). Cereal, Bread, Grains, and Starches, 2–3 servings of iron-fortified infant cereal, soft cooked breads and starches (serving = 1–2 Tbsp). Fruits and Vegetables, Introduce plain, cooked, mashed, or strained infant vegetables and fruits. Avoid meat-containing dinners. 2–4 oz 100% fruit juice in cup.

Meats and Other Protein-Rich Foods, Begin to offer plain, cooked, pureed meats. Avoid combination dinners. Row 4. Age, 8–10 mo. Breast Milk and/or Fortified Infant Formula, 3–4 feedings; 7–8 oz/feeding; (24–32 oz/day). Cereal, Bread, Grains, and Starches, 2–3 servings of iron-fortified infant cereal, soft cooked breads and starches (serving = 1–2 Tbsp). Fruits and Vegetables, 2–3 servings of soft, cut-up, and mashed vegetables and fruits (serving = 1–2 Tbsp). 3–4 oz 100% fruit juices in cup. Meats and Other Protein-Rich Foods, Offer plain, well-cooked, soft, finely cut, or pureed meats, cheeses, and casseroles. Row 5. Age, 10–12 mo. Breast Milk and/or Fortified Infant Formula, 24–32 oz/day. Cereal, Bread, Grains, and Starches, 4 servings of iron-fortified infant cereal, soft cooked breads and starches (serving = 1–2 Tbsp). Fruits and Vegetables, 4 servings of soft, cut-up, and mashed vegetables and fruits (serving = 1–2 Tbsp). 3–4 oz 100% fruit juices in cup. Meats and Other Protein-Rich Foods, 1–2 oz of soft, finely cut, or chopped meat or other protein foods.

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A table is titled Feeding Guidelines for Toddlers to Adolescents. It has the headers: Blank, Milk and Milk Products, Cereal, Bread, Grains, and Starches, Fruits and Vegetables, Meats and Other Protein-Rich Foods, Fats and Oils, and Sweets, Jellies, Jams, Soft Drinks.

Row 1. Blank, 12–23 mo. Milk and Milk Products, 2 cups/day (whole milk or milk products). Cereal, Bread, Grains, and Starches, 2 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal). Fruits and Vegetables, 1¾ cup/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens). Meats and Other Protein-Rich Foods, 1.5 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts). Fats and Oils, Do not restrict. Sweets, Jellies, Jams, Soft Drinks, Provide small amount if desired. Row 2. Blank, 2–3 yearsa. Milk and Milk Products, 2–2.5 cups/day (1 cup milk, 1.5 oz natural cheese, ½ cup shredded cheese). Cereal, Bread, Grains, and Starches, 3 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal). Fruits and Vegetables, 2 cups/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens). Meats and Other Protein-Rich Foods, 2 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts). Fats and Oils, 3 Tbsp/day (1 Tbsp oil, margarine, butter, or mayonnaise; 1 Tbsp salad dressing; sour cream or light mayonnaise). Sweets, Jellies, Jams, Soft Drinks, 165–170 discretionary calories. Row 3. Blank, 4–8 years. Milk and Milk

Products, 2.5–3 cups/day (1 cup milk, 1.5 oz natural cheese, 2 oz processed cheese, 1/3 cup shredded cheese). Cereal, Bread, Grains, and Starches, 4–5 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal). Fruits and Vegetables, 2½–3 cups/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens). Meats and Other Protein-Rich Foods, 3–4 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts). Fats and Oils, 4 Tbsp/day (1 Tbsp oil, margarine, butter, or mayonnaise; 1 Tbsp salad dressing; sour cream or light mayonnaise). Sweets, Jellies, Jams, Soft Drinks, 170–195 discretionary calories. Row 4. Blank, 9–13 years. Milk and Milk Products, 3 cups/day (1 cup milk, 1.5 oz natural cheese, 2 oz processed cheese, 1/3 cup shredded cheese). Cereal, Bread, Grains, and Starches, 5–6 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal). Fruits and Vegetables, 3½–4 cups/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens). Meats and Other Protein-Rich Foods, 5 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts). Fats and Oils, 5 Tbsp/day (1 Tbsp oil, margarine, butter, or mayonnaise; 1 Tbsp salad dressing; sour cream or

light mayonnaise). Sweets, Jellies, Jams, Soft Drinks, Limit amount, use sparingly (130–140 discretionary calories). Row 5. Blank, 14–18 years. Milk and Milk Products, 3 cups/day (1 cup milk, 1.5 oz natural cheese, 2 oz processed cheese, 1/3 cup shredded cheese). Cereal, Bread, Grains, and Starches, 6–7 oz/day (1 oz equivalent = 1 slice whole grain bread, ½ cup cooked cereal, rice or pasta, 1 cup dry cereal). Fruits and Vegetables, 3½–5 cups/day (1 cup raw or cooked fruit or vegetable or 1 cup fruit or vegetable juice or ½ cup dried fruit, 2 cups raw leafy greens). Meats and Other Protein-Rich Foods, 5–6 oz/day (1 oz beef, poultry, or fish; ¼ cup cooked beans; 1 egg; 1 Tbsp peanut butter; ½ oz nuts). Fats and Oils, 5–6 Tbsp/day (1 Tbsp oil, margarine, butter, or mayonnaise; 1 Tbsp salad dressing; sour cream or light mayonnaise). Sweets, Jellies, Jams, Soft Drinks, Limit amount, use sparingly (265–650 discretionary calories). Footnote below the table indicates that Some foods are considered choking hazards to the developing toddler and should be avoided until the child is >3 years old. Such foods are peanut butter, hot dogs, grapes, hard pieces of fruit and vegetables, and tough or chewy meats.

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A table is titled Increasing Caloric Density of Human Breast Milk (By Adding Powdered Formula to 100 mL

of Expressed Breast Milk). It has the headers: Desired Caloric Concentration (kcal/oz), Similac Advance Powder (g), Enfamil Infant Powder (g), Similac NeoSure Advance Powder (g), and Enfamil EnfaCare Powder (g). Row 1. Desired Caloric Concentration (kcal/oz), 22. Similac Advance Powder (g), 1.3. Enfamil Infant Powder (g), 1.3. Similac NeoSure Advance Powder (g), 1.3. Enfamil EnfaCare Powder (g), 1.4. Row 2. Desired Caloric Concentration (kcal/oz), 24. Similac Advance Powder (g), 2.6. Enfamil Infant Powder (g), 2.7. Similac NeoSure Advance Powder (g), 2.6. Enfamil EnfaCare Powder (g), 2.7. Row 3. Desired Caloric Concentration (kcal/oz), 27. Similac Advance Powder (g), 4.6. Enfamil Infant Powder (g), 4.7. Similac NeoSure Advance Powder (g), 4.5. Enfamil EnfaCare Powder (g), 4.8. Row 4. Desired Caloric Concentration (kcal/oz), 30. Similac Advance Powder (g), 6.5. Enfamil Infant Powder (g), 6.7. Similac NeoSure Advance Powder (g), 6.5. Enfamil EnfaCare Powder (g), 6.8.

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A table is titled Common Pediatric Formulasa by Brand. It has the headers: Formula, Route, Amount Required to Meet DRIs, Caloric Strength (kcal/oz), and Comments. Row 1. Formula, EleCare Junior. Route, Oral or enteral. Amount Required to Meet DRIs, NL.

Caloric Strength (kcal/oz), 30. Comments, Elemental formula, lactose free, gluten free, soy free, milk protein free, fructose free, galactose free. Row 2. Formula, Neocate Junior. Route, Oral or enteral. Amount Required to Meet DRIs, NL. Caloric Strength (kcal/oz), 30. Comments, Elemental formula for children with multiple protein allergies, lactose free, gluten free. Row 3. Formula, Nutren Junior (with or without fiber). Route, Oral or enteral. Amount Required to Meet DRIs, 1,000 mL for 1–8-year-olds; 1,500 mL for 9–13-year-olds. Caloric Strength (kcal/oz), 30. Comments, Lactose free, gluten free, kosher, low residue. Row 4. Formula, PediaSure (with or without fiber). Route, Oral and enteral. Amount Required to Meet DRIs, 1,000 mL for 1–8-year-olds; 1,500 mL for 9–13-year-olds. Caloric Strength (kcal/oz), 30. Comments, Lactose free, gluten free, kosher. Row 5. Formula, Peptamen Junior. Route, Oral and enteral. Amount Required to Meet DRIs, 1,000 mL for 1–8-year-olds; 1,500 mL for 9–13-year-olds. Caloric Strength (kcal/oz), 30. Comments, Elemental formula designed for children with severe GI tract disturbances. Row 6. Formula, Vivonex pediatric. Route, Oral or enteral. Amount Required to Meet DRIs, 1,000 mL for 1–8-year-olds; 1,500 mL for 9–13-year-olds. Caloric Strength (kcal/oz), 24. Comments, Elemental, lactose-free, gluten-free, low-residue formula. The text below the table indicates that GI,

gastrointestinal; NL, not listed by manufacturer. Footnote below the table indicates that The above list is not complete and does not contain all pediatric formulas available. Refer to product labels or manufacturers’ references for most current nutrient values and ingredients. In some cases, it is appropriate to utilize an adult formula for nutrition supplementation. Refer to the guidelines for use provided by the manufacturers of those products.

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"A table is titled Common Blenderized Formulasa by Brand. It has the headers: Formula, Route, Amount Required to Meet DRIs, Caloric Strength (kcal/oz), and Comments. Row 1. Formula, Compleat Pediatric. Route, Enteral. Amount Required to Meet DRIs, 1,400 mL for 1–3-year-olds. Caloric Strength (kcal/oz), 30. Comments, Lactose free, gluten free. Row 2. Formula, Compleat Pediatric Reduce Calorie. Route, Enteral. Amount Required to Meet DRIs, 1–8 years 1,000 mL; 9–13 years 1,200 mL. Caloric Strength (kcal/oz), 18. Comments, Lactose free, gluten free, no corn or soy ingredients. Row 3. Formula, Compleat Organic Blends. Route, Enteral. Amount Required to Meet DRIs, NL. Caloric Strength (kcal/oz), 38. Comments, Lactose-free, Gluten-free; No dairy, soy or corn ingredients. Ingredients vary, plant based available. Row 4. Formula, Pediasure Harvest. Route, Enteral.

Amount Required to Meet DRIs, 1,000 mL for children 1–8 years of age; 1,500 mL for children 9–13 years of age. Caloric Strength (kcal/oz), 30. Comments, Made with no milk-, gluten- or lactose-containing ingredients. Row 5. Formula, Real Food Blends. Route, Enteral. Amount Required to Meet DRIs, NL. Caloric Strength (kcal/oz), Varies. Comments, Suitable for those with nut, soy, gluten, or dairy intolerances. Row 6. Formula, Nourish. Route, Enteral or oral. Amount Required to Meet DRIs, 1,065 mL of Nourish meets 100% of the DRI for 26 key vitamins and minerals for children 4–8 years old. Caloric Strength (kcal/oz), 33. Comments, Dairy free, tree nut free, gluten free, soy free, and corn free. Row 7. Formula, Kate Farms Pediatric Standard 1.2. Route, Enteral or oral. Amount Required to Meet DRIs, 1–3 years: 3.6 cartons or 900 mL (1,080 calories); 4–8 years: 4 cartons or 1,000 mL (1,200 calories); 9–13 years: 6 cartons or 1,500 mL (1,800 calories). Caloric Strength (kcal/oz), 36. Comments, Does not contain whey, lactose, casein, soy,; gluten, nuts or corn. Appropriate for the following diets: lactose free,; vegan, plant based, gluten free. Row 8. Formula, Kate Farms Pediatric Peptide 1.5. Route, Enteral or oral. Amount Required to Meet DRIs, 1–3 years: 2.6 cartons or 650 mL (975 calories); 4–8 years: 3 cartons or 750 mL (1,125 calories); 9–13 years: 4 cartons or 1,000 mL (1,500 calories). Caloric Strength (kcal/oz), 45. Comments,

Does not contain whey, lactose, casein, soy,; gluten, nuts or corn. Appropriate for the following diets:; lactose free, vegan, plant based, gluten free. The text below the table indicates that NL, not listed by manufacturer. Footnote below the table indicates that The above list is not complete and does not contain all pediatric formulas available. Refer to product labels or manufacturers’ references for most current nutrient values and ingredients. In some cases, it is appropriate to utilize an adult formula for nutrition supplementation. Refer to the guidelines for use provided by the manufacturers of those products."

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A table is titled Expert Panel Intervention Stages According to Age and BMI-for-Age Percentile. It has the headers: Counseling Stage, Age (yr), BMI-for-Age Percentile, Presence of Health Risk, and Goal of Intervention. Row 1. Counseling Stage, Stage 1. Age (yr), <18. BMI-for-Age Percentile, <94th. Presence of Health Risk, No. Goal of Intervention, Weight maintenance until BMI-for-age is <85th percentile or a slower rate of weight gain. Row 2. Counseling Stage, Stages 1–2. Age (yr), 2–18. BMI-for-Age Percentile, 85th–94th. Presence of Health Risk, Yes. Goal of Intervention, Weight maintenance until BMI-for-age is <85th percentile or a slower rate of weight gain. Row 3. Counseling Stage, Stages 1–2. Age (yr),

6–18. BMI-for-Age Percentile, 95th–98th. Presence of Health Risk, Yes. Goal of Intervention, Either weight maintenance until BMI-for-age is <85th percentile or gradual weight loss of 1 lb/mo. Row 4. Counseling Stage, Stages 1–3. Age (yr), 2–5. BMI-for-Age Percentile, 95th–98th. Presence of Health Risk, Yes. Goal of Intervention, Weight maintenance until BMI-for age is <85th percentile or a slower rate of weight gain; weight loss should be <1 lb/mo. Row 5. Counseling Stage, Stages 1–3. Age (yr), 2–5. BMI-for-Age Percentile, ≥99th. Presence of Health Risk, Yes. Goal of Intervention, Gradual weight loss of ≤1 lb/mo. Row 6. Counseling Stage, Stages 1–3. Age (yr), 6–11. BMI-for-Age Percentile, >99th. Presence of Health Risk, Yes. Goal of Intervention, Weight loss of 2 lb/mo. Row 7. Counseling Stage, Stages 1–4. Age (yr), 12–18. BMI-for-Age Percentile, >95th. Presence of Health Risk, Yes. Goal of Intervention, Weight loss of ≤2 lb/wk until BMI-for-age is <85th percentile.

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A table is titled Pediatric Malnutrition Criteria (Single Data Point). It has the headers: Primary Indicators, Mild Malnutrition, Moderate Malnutrition, and Severe Malnutrition. Row 1. Primary Indicators, Weight for height z-score. Mild Malnutrition, –1 to –1.9 z-score. Moderate Malnutrition, –2 to –2.9 z-score. Severe Malnutrition, –3 or greater z-score. Row 2. Primary

Indicators, BMI-for-age z-score. Mild Malnutrition, –1 to –1.9 z-score. Moderate Malnutrition, –2 to –2.9 z-score. Severe Malnutrition, –3 or greater z-score. Row 3. Primary Indicators, Length/height z-score. Mild Malnutrition, No data. Moderate Malnutrition, No data. Severe Malnutrition, –3 z-score. Row 4. Primary Indicators, Mid-upper arm circumference. Mild Malnutrition, Greater than or equal to –1 to –1.9 z-score. Moderate Malnutrition, Greater than or equal to –2 to –2.9 z-score. Severe Malnutrition, Greater than or equal to –3 z-score.

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A table is titled Pediatric Malnutrition Criteria (Two or More Data Points). It has the headers: Primary Indicators, Mild Malnutrition, Moderate Malnutrition, and Severe Malnutrition. Row 1. Primary Indicators, Weight gain velocity (<2 years). Mild Malnutrition, <75% of the norm for expected weight gain. Moderate Malnutrition, <50% of the norm for expected weight gain. Severe Malnutrition, <25% of the norm for expected weight gain. Row 2. Primary Indicators, Weight loss (2–20 years). Mild Malnutrition, 5% usual body weight. Moderate Malnutrition, 7.5% usual body weight. Severe Malnutrition, 10% usual body weight. Row 3. Primary Indicators, Deceleration in weight for length/height z-score. Mild Malnutrition, Decline of 1 z-score.

Moderate Malnutrition, Decline of 2 z-score. Severe Malnutrition, Decline of 3 z-score. Row 4. Primary Indicators, Inadequate nutrient intake. Mild Malnutrition, 51–75% estimated energy/protein need. Moderate Malnutrition, 26–50% estimated energy/protein need. Severe Malnutrition, 25% estimated energy/protein need.

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A table is titled Primary Indicators of Neonatal Malnutrition. It has the headers: Indicator, Mild Malnutrition, Moderate Malnutrition, Severe Malnutrition, and Use of Indicator. The entries under Primary Indicators Requiring One Indicator are as follows. Row 1. Decline in weight-for-age z-score. Decline of 0.8 to 1.2 SDb. Decline of >1.2–2 SD. Decline of >2 SD. Not appropriate for first 2 wk of life. Row 2. Weight gain velocitya. <75% of expected rate of weight gain to maintain growth rate. <50% of expected rate of weight gain to maintain growth rate. <25% of expected rate of weight gain to maintain growth rate. Not appropriate for first 2 wk of life. Row 3. Nutrient Intake. 3–5 consecutive days of protein/energy intake 75% of estimated needs. 5–7 consecutive days of protein/energy intake 75% of estimated needs. >7 consecutive days of protein/energy intake 75% of estimated needs. Preferred indicator during the first 2 wk of life.

Footnote below the table indicates that aExpected weight gain velocity, expected linear growth velocity, and z-scores can be determined using the online calculator PediTools (www.peditools.org). bSD = standard deviation.

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A table is titled Primary Indicators of Neonatal Malnutrition. It has the headers: Indicator, Mild Malnutrition, Moderate Malnutrition, Severe Malnutrition, and Use of Indicator. The entries under Primary Indicators Requiring 2 Indicators are as follows. Row 1. Days to regain birth weight. 15–18. 19–21. >21. Use in conjunction with nutrient intake. Row 2. Linear growth velocitya. <75% of expected rate of linear gain to maintain expected growth rate. <50% of expected rate of linear gain to maintain expected growth rate. <25% of expected rate of linear gain to maintain expected growth rate. Not appropriate for first 2 wk of life.; May be deferred in critically ill, unstable infants.; Use in conjunction with another indicator when accurate length measurement available. Row 3. Decline in length-for-age z-score. Decline of 0.8–1.2 SDb. Decline of >1.2–2 SD. Decline of >2 SD. Not appropriate for first 2 wk of life.; May be deferred in critically ill, unstable infants.; Use in conjunction with another indicator when accurate length measurement is available.

Footnote below the table indicates that aExpected weight gain velocity, expected linear growth velocity, and z-scores can be determined using the online calculator PediTools (www.peditools.org). bSD = standard deviation.

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A table is titled Pediatric Nutrition Management for Renal Dysfunction. It has the headers: Nutrient, Nephrotic Syndrome, AKI, No dialysis, AKI, PD or HD, AKI, CRRT, CKD stages 3–5, CKD 5, HD, and CKD 5, PD. Row 1. Nutrient, Energy. Nephrotic Syndrome, EER for age. AKI, No dialysis, EER for age or original disease state. AKI, PD or HD, EER for age or original disease state. AKI, CRRT, EER for age or original disease state. CKD stages 3–5, EER for age. CKD 5, HD, EER for age. CKD 5, PD, EER for age. Row 2. Nutrient, Protein. Nephrotic Syndrome, DRI-do not supplement to replace urinary losses. AKI, No dialysis, DRI or less per BUN monitoring. AKI, PD or HD, DRI with 0.2 g/kg increases for HD, 0.4 g/kg for PD. AKI, CRRT, At least 2.5 g/kg or greater. CKD stages 3–5, CKD 3 100–140% DRI; CKD 4–5 100–120% DRI. CKD 5, HD, 0–6 mo: 1.6 g/kg; 7–12 mo: 1.3 g/kg; 1–3 y: 1.15 g/kg; 4–13 y: 1.05 g/kg; 14–18 y: 0.95 g/kg. CKD 5, PD, 0–6 mo: 1.8 g/kg; 7–12 mo: 1.5 g/kg; 1–3 y: 1.3 g/kg; 4–13 y: 1.1 g/kg; 14–18 y: 1.0. Row 3. Nutrient, Sodium. Nephrotic Syndrome, 1–3

mEq/kg; will vary according to edema or hypertension. AKI, No dialysis, Varies. AKI, PD or HD, Varies. AKI, CRRT, Typically no restriction; may need electrolyte supplementation. CKD stages 3–5, 1–3 mEq/kg; will vary according to edema or hypertension. CKD 5, HD, 1–3 mEq/kg; will vary according to edema or hypertension. CKD 5, PD, 1–3 mEq/kg; will vary according to edema or hypertension. Na supplements should be considered for infants. Row 4. Nutrient, Potassium. Nephrotic Syndrome, Restriction not needed. AKI, No dialysis, <1–2 mEq/kg. AKI, PD or HD, Limit. AKI, CRRT, Typically no restriction; may need electrolyte supplementation. CKD stages 3–5, Most will tolerate 3 mEq/kg/day. CKD 5, HD, 1–3 mEq/kg/day but will vary according to serum levels and age. CKD 5, PD, Generally unrestricted unless low transporter, will need to be monitored closely. Row 5. Nutrient, Phosphorus. Nephrotic Syndrome, Restriction not needed. AKI, No dialysis, Tight limit. AKI, PD or HD, Limit. AKI, CRRT, Typically no restriction; may need electrolyte supplementation. CKD stages 3–5, Blank. 0–6 mo. 7–12 mo. 1–3 y. 4–8 y. 9–18 y. CKD 5, HD, High PTH and Normal Phosphorus. ≤100. ≤275. ≤460. ≤500. ≤1,250. CKD 5, PD, High PTH and High Phosphorus. ≤80. ≤220. ≤370. ≤400. ≤1,000. Row 6. Nutrient, Fluids. Nephrotic Syndrome, Will vary according to urine output. AKI, No dialysis, Will vary

according to urine output. AKI, PD or HD, Will vary according to urine output. AKI, CRRT, May need additional fluid replacements. CKD stages 3–5, Generally unrestricted. CKD 5, HD, Replace urine output, insensible losses, +dialysis ultrafiltrate. CKD 5, PD, Replace urine output, insensible losses, +dialysis ultrafiltrate. Row 7. Nutrient, Micronutrients. Nephrotic Syndrome, 100% DRI, supplementation as needed. AKI, No dialysis, 100% DRI, supplementation will most likely be needed. AKI, PD or HD, Monitor fat-soluble vitamins. AKI, CRRT, 100% DRI, supplement water-soluble vitamins as needed. CKD stages 3–5, 100% DRI, supplementation as needed. CKD 5, HD, CKD 5, PD, 100% DRI water-soluble vitamin supplement is recommended. Monitor vitamin D quarterly and supplement as needed. The text below the table indicates that AKI, acute kidney injury; CKD, chronic kidney disease; CRRT, continuous renal replacement therapy; DRI, daily recommended intake; EER, estimated energy expenditure; HD, hemodialysis; PD, peritoneal dialysis; PTH, parathyroid hormone.

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The table is titled as Dietary Reference Intakes for Older Adults for Vitamins and it has 3 columns Nutrient/Units, Males: DRIa (51–70 yr : 70+ yr), Tolerable UL (51–70 yr : 70+ yr), and Females: DRI:

(51–70 yr : 70+ yr) UL: (51–70 yr : 70+ yr). The row entries are as follows. Row 1. Vitamin A, µg. 900:900. 3,000:3,000. 700:700. 3,000:3,000. Row 2. Vitamin D, µg. 15:20. 100:100. 15:20. 100:100. Row 3. Vitamin E, mg. 15:15. 1,000:1,000. 15:15. 1,000:1,000. Row 4. Vitamin K, µg. 120:120. ND:ND. 90:90. ND:ND. Row 5. Vitamin C, mg. 90:90. 2,000:2,000. 75:75. 2,000:2,000. Row 6. Vitamin B6, mg. 1.7:1.7. 100:100. 1.5:1.5. 100:100. Row 7. Vitamin B12, µg. 2.4:2.4. ND:ND. 2.4:2.4. ND:ND. Row 8. Biotin, µg. 30:30. ND:ND. 30:30. ND:ND. Row 9. Choline, mg. 550:550. 3,500:3,500. 425:425. 3,500:3,500. Row 10. Folate, µg. 400:400. 1,000:1,000. 400:400. 1,000:1,000. Row 11. Niacin mg. 16:16. 35:35. 14:14. 35:35. Row 12. Pantothenic acid, mg. 5:5. ND:ND. 5:5. ND:ND. Row 13. Riboflavin, mg. 1.3:1.3. ND:ND. 1.1:1.1. ND:ND. Row 14. Thiamin, mg. 1.2:1.2. ND:ND. 1.1:1.1. ND:ND. The abbreviation of table is ND, not determined. The foot note a is DRIs represent RDAs except for vitamin K, biotin, choline, and pantothenic acid (AI values).

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The table is titled as Dietary Reference Intakes for Older Adults for Minerals and it has 3 columns Nutrient/Units, Males: DRIa (51–70 yr : 70+ yr), Tolerable UL (51–70 yr : 70+ yr), and Females: DRI: (51–70 yr : 70+ yr) UL: (51–70 yr : 70+ yr). The row

entries are as follows. Row 1. Calcium, mg. 1,000:1,200. 2,000:2,000. 1,200:1,200. 2,000:2,000. Row 2. Chromium, µg. 30:30. ND:ND. 20:20. ND:ND. Row 3. Chloride, mg. 2,000:1,800. 3,600:3,600. 2,000:1,800. 3,600:3,600. Row 4. Copper, µg. 900:900. 10,000:10,000. 900:900. 10,000:10,000. Row 5. Fluoride, mg. 4:4.10:10. 3:3. 10:10. Row 6. Iodine, µg. 150:150. 1,100:1,100. 150:150. 1,100:1,100. Row 7. Iron, mg. 8:8. 45:45. 8:8. 45:45. Row 8. Magnesium, mga. 420:420. 350:350. 320:320. 350:350. Row 9. Manganese, mg. 2.3:2.3. 11:11. 1.8:1.8. 11:11. Row 10. Molybdenum, µg. 45:45. 2,000:2,000. 45:45. 2,000:2,000. Row 11. Nickel, mg. ND:ND. 1:1. ND:ND. 1:1. Row 12. Phosphorus, mg. 700:700. 4,000:3,000. 700:700. 4,000:3,000. Row 13. Potassium, mg. 3,400:3,400. ND:ND. 2,600:2,600. ND:ND. Row 14. Selenium, µg. 55:55. 400:400. 44:44. 400:400. Row 15. Sodium, mgb. 1,500:1,500. 2,300:2,300. 1,500:1,500. 2,300:2,300. Row 16. Vanadium, mg. ND:ND. 1.8:1.8. ND:ND. 1.8:1.8. Row 17. Zinc, mg. 11:11. 40:40. 8:8. 40:40. The abbreviation of table is ND, not determined. DRIs represent RDAs except for chromium, chloride, fluoride, manganese, potassium, and sodium (AI values). The footnote of table a is For magnesium, the UL applies to synthetic forms obtained from drugs or supplements only. The footnote of table b is For

sodium, the upper lever (UL) is replaced with the Chronic Disease Risk Reduction (CDRR) intake level.

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The table is titled as Enteral Access Devices and it has 3 columns Description, Pros, and Cons and Complications. The row entries are as follows. Section 1: Short-term (<4 wk) Row 1. Nasogastric tube (NGT): prepyloric tube. Used for feeding, decompression of stomach, administration of medications, measurement of gastric pH and residuals. At least 10F needed to avoid clogging from enteral formulas. Variety of sizes available (8–24F); Generally easy placement ; Large-bore tube allows more versatility in feeding regimen; Gastric phase of digestion and nutrient metabolism. Contraindicated if nasal/ facial fractures, severe coagulopathy, severe thrombocytopenia, esophageal obstruction; Complications include clogging, esophageal or sinus perforation, nasal mucosal ulceration, pneumothorax, epistaxis, pulmonary aspiration. Row 2. Orogastric tube: prepyloric tube Optional route when tube cannot be placed nasally (facial fracture or head injury). Lower incidence of sinusitis. Tolerated for short periods of time; Complications are the same as with NGT, with exception of nasal-related events. Row 3. Nasoenteric tube: postpyloric tube Tip of tube placed past the pyloric sphincter. Radiologic

verification or electromagnetic image of placement needed before use. Not appropriate to check residuals. Small-bore tubes are more flexible and more comfortable; Less risk of aspiration as patient is able to lay flat, esophageal reflux, delayed gastric emptying if placement past distal third of duodenum. Infusion pumps necessary; Difficulty of postligament of Treitz placement; Difficulty of administering some medications. Section 2. Long-term (>4 wk): Row 1. Gastrostomy tube: prepyloric. Placed surgically, endoscopically (PEG), or radiologically into the stomach. Available sizes from 10 to 28F. Allows bolus feeding; Allows gastric phase of digestion and nutrient metabolism; Low-profile tubes available. Complications may include dislodgment, bleeding, wound infection, tube occlusion, pneumoperitoneum, stomal leakage. Row 2. Jejunostomy tube: postpyloric. Placed surgically, endoscopically (PEJ), or radiologically into the jejunum. Size of 9–12F tube. Not able to check residuals to assess tolerance. Requires infusion via a pump. Reduces aspiration risk if the patient is required to lay flat (several studies have demonstrated no difference in aspiration risk between intragastric and small bowel feedings)3 ; Low-profile tubes available. Does not allow bolus enteral feeding; Complications may include wound dehiscence or infection, bowel obstruction, occlusion, bleeding, dislodgment, volvulus. Section 3. Long-term

(>4 wk): Row 1. Transgastric Jejunostomy. Placed surgically, endoscopically, or radiologically. A jejuna port is placed through gastrostomy tube, allowing infusion of enteral feeding into the small bowel while also suctioning gastric contents. Can be converted to gastrostomy feeding as tolerated. Does not allow bolus enteral feeding; Complications of both jejunostomy and gastrostomy tubes. The abbreviation of table is PEJ, percutaneous endoscopic jejunostomy. The table data from (i) Mueller C (ed.) The A.S.P.E.N. Adult Nutrition Support Core Curriculum. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017; (ii) Compher C, Bingham AL, McCall M, et al. Guidelines for the provision of nutrition support therapy in the adult critically ill patient: The American Society for Parenteral and Enteral Nutrition. J Parenter Enteral Nutr. 2022;46:12-41. https://doi.org/10.1002/jpen.2267; (iii) Koseniecki M, Rebecca F. Enteral nutrition for adults in the hospital setting. Nutr Clin Prac. 2015;30(5):634-651, and (iv) Miller KR, et al. A tutorial on enteral access in adult patients in the hospitalized setting. J Parenter Enteral Nutr. 2014;38(3):282-295.

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The table is titled as Macronutrient Composition of Solutions and it has 4 columns Macronutrient, Concentrations (%), Grams per Liter, and Kilocalories

per Liter under section 1 and also 4 columns Blank. Concentration, Kilocalories per Milliliter, and Kilocalories per Liter under section 2. The row entries are as follows. In section 1: Row 1. Dextrose: 5; 50; 170. 10; 100; 340. 20; 200; 680. 30; 300; 1,020. 40; 400; 1,360. 50; 500; 1,700. 70; 700; 2,380. Row 2. Amino acids: 8.5; 85; 340. 10; 100; 400. 15; 150; 600. 20; 200; 800. Section 2: Row 1. Lipid emulsions: 10; 1.1; 1,100. 20; 2; 2,000. 30; 3; 3,000.

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The table is titled as Standard IV Solutions and it has 8 columns IV solution, Glucose (g/dL), Na+ (mEq/L), Cl- (mEq/L), K+ (mEq/L), Ca2+ (mEq/L), Lactate (mEq/L), Osmolarity (mOsm/L). The row entries are as follows. Row 1. 5% dextrose in water. 5. —. —. —. —. —. 252. Row 2. 10% dextrose in water. 10. —. —. —. —. —. 505. Row 3. 0.45% NaCl. — 77. 77. —. —. —. 154. Row 4. 0.9% NaCl (normal saline). —. 154. 154. —. —. —. 308. Row 5. 5% dextrose in 0.45% NaCl. 5. 77. 77. —. —. —. 406. Row 6. 10% dextrose in 0.9% NaCl. 5. 154. 154. —. —. —. 560. Row 7. Lactated Ringer’s. — 130. 109. 4. 3. 28. 273. The Data from Mueller C (ed.) The A.S.P.E.N. Adult Nutrition Support Core Curriculum. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017.

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The table is titled as Daily Electrolyte Requirements in Formulation of Parenteral Nutrition and it has 5 columns Electrolyte, Daily Requirement (Adult), Causes of Elevated Levels, Causes of Decreased, Levels, and Dosage Form. The row entries are as follows. Row 1. Calcium. 10–15 mEq. Vitamin D excess, renal failure, tumor lysis syndrome, hyperparathyroidism, prolonged immobilization and stress, bone cancer. Decreased vitamin D intake, hypoparathyroidism, hypoalbuminemia, hypomagnesemia, citrate binding of calcium with blood product administration. Ca gluconate. Row 2. Magnesium. 8–20 mEq. Excessive Mg intake in renal insufficiency. Refeeding syndrome, alcoholism, diuretic use, nasogastric suction, diabetic ketoacidosis, elevated stool output, Mg wasting medications. Mg sulfate. Row 3. Phosphorus. 20–40 mmol. Excessive phosphate administration, renal failure. Refeeding syndrome, alcoholism, inadequate intake. Na phosphate; K phosphate. Row 4. Sodium. 1–2 mEq/kg. Inadequate free water, excessive water loss, excessive sodium intake, hyperaldosteronism. Excessive hypotonic fluid, nephritis, adrenal insufficiency, congestive heart failure, syndrome of inappropriate antidiuretic hormones (SIADHs), cirrhosis with ascites. Na chloride; Na acetate; Na lactate. Row 5. Potassium. 1–2 mEq/kg. Renal dysfunction, excessive; K+ intake, metabolic acidosis;

K+-sparing medications. Refeeding syndrome, inadequate K+ intake, excessive losses with diarrhea or intestinal fluids, diuretic medications, hypomagnesemia, metabolic alkalosis. K phosphate; K acetate; K chloride. The Data from (i) Merritt R. (ed) The A.S.P.E.N. Nutrition Support Practice Manual, 2nd ed. American Society of Parenteral and Enteral Nutrition, 2005, and (ii) Mueller C (ed.) The A.S.P.E.N. Adult Nutrition Support Core Curriculum. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017.

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The table is titled as Monitoring for Adult Patients on Parenteral Nutrition and it has 4 columns Parameter, Baseline, Critically Ill Patients, and Stable Patients. The row entries are as follows. Row 1. Electrolytes: Na, K, Cl, CO2, phosphorus, BUN, Cr. Yes. Daily 1–2 × per week. Row 2. ALT, AST, ALP, total bilirubin. Yes. Weekly. Monthly. Row 3. Serum triglycerides. Yes. Weekly. Weekly. Row 4. Complete blood count with differential. Yes. Weekly. Weekly. Row 5.INR, Prothrombin time (PT), partial thromboplastin time (PTT). Yes. Weekly. Weekly. Row 6. Capillary glucose. 3×/day. 3×/day (until consistently <150 mg/dL). As needed. Row 7. Weight. If possible. Daily. 2–3×/wk.Row 8. Intake and output. Daily. Daily. Daily unless fluid status is assessed by physical exam. Row

9. Nitrogen balance. As needed. As needed. As needed. Row 10. Indirect calorimetry. As needed. As needed. As needed. The Data from Mueller C (ed.) The A.S.P.E.N. Adult Nutrition Support Core Curriculum. 3rd ed. American Society of Parenteral and Enteral Nutrition, 2017.

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The table is titled as Bariatric Surgery Procedures and it has 4 columns Procedure, Also Known As, How the Procedure Works, and Potential Nutrient Deficiencies. The row entries are as follows. Row 1. Roux-en-Y gastric bypass. Roux-en-Y" or "Gastric Bypass," "the Bypass". Creating a small stomach pouch approximately 30 cc (30 mL) in size and thus bypassing the majority of the stomach and the first portion of the small intestine, including the duodenum. Iron, vitamin B12, calcium, folate, vitamin D, thiamin. Row 2. Biliopancreatic diversion with (or without) duodenal switch. "the Switch," "BPD-DS" "BPD". Majority of stomach is removed, and the distal part of the small intestine is connected to the pouch, bypassing the duodenum and jejunum. The bypassed small intestine, which carries the bile and pancreatic enzymes, is reconnected to the last portion of the small intestine to eventually mix with the food. The DS keeps the pylorus intact. Vitamins A, D, E, K, zinc, iron, calcium, protein. Row 3. Sleeve gastrectomy.

“Gastric Sleeve,” “the Sleeve,” “Vertical Sleeve Gastrectomy (VSG)”. Approximately 80% of the stomach is removed to leave a long “sleeve” or banana-shaped pouch. Vitamin B12, calcium, iron, thiamin. Row 4. Adjustable gastric band. “the Band,” “Lap-Band”. Silicone band is placed around the top of the stomach, creating restriction; saline may be injected into the band, making it tighter. Folate, vitamin B12, calcium, thiamin. Row 5. Single anastomosis duodenal-ileal bypass with sleeve gastrectomy. SADI. A sleeve gastrectomy, then the first part of the small intestine is divided, a loop of intestine is measured several feet and connected to the stomach. Food goes through the pouch and into the latter portion of the small intestine. Vitamins A, D, E, K, zinc, iron, calcium, protein. The Data from Bariatric Surgery Procedures. American Society for Metabolic and Bariatric Surgery. Updated May 2021. Accessed April 26, 2023. http://asmbs.org /patients/bariatric-surgery-procedures.

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The table is titled as Suggested Postoperative Vitamin Supplementation and it has 5 columns Supplement, AGB, RYGB & SG, BPD/DS, and Comment. The row entries are as follows. Row 1. Multivitamin–multimineral supplement: High potency containing at least 100% DV for 2/3 of nutrients, and 18 mg iron,

400 µg folic acid, at least 50 mg thiamine, and containing selenium and zinc (at least 8 mg) in each serving.; Start with chewable or liquid, and progress to whole tablet/capsule as tolerated.; Avoid children’s or gummy formulas that are not complete.; May separate dosage.; May improve GI tolerance when taken close to food intake.; Do not mix multivitamin containing iron with calcium, take 2 hr apart. 100% of daily value (DV). 200% of DV. 200% of DV. Begin as tolerated. Row 2. Additional cobalamin (B12): Sublingual tablets, liquid drops, mouth spray, or nasal gel/spray (oral).; Intramuscular (IM) injection.; Supplementation after AGB and BPD/DS may be required. None. 350–500 µg/day (oral),1,000 µg/mo (IM). None. Begin 0–3 mo after surgery. Row 3. Additional elemental calcium: Calcium citrate and vitamin D3.; Split into 500–600 mg doses; be mindful of serving size on supplement label.; Suggest a brand that contains magnesium, especially for BPD/DS.; Do not combine calcium with iron.; Promote intake of dairy beverages and/or foods that are significant sources of dietary calcium in addition to recommended supplement, up to3 servings daily.; Combined dietary and supplemental calcium intake >1,700 mg/day may be required to prevent bone loss during rapid weight loss.; 3,000 IU D3 daily until blood levels of 25(OH)D are >30 ng/mL. 1,500 mg/day. 1,500–2,000 mg/day. 1,800–2,400 mg/day.

May begin as tolerated or within 1 mo after surgery. Row 4. Additional elemental iron (above that provided by multivitamin): Recommended for menstruating women and those at risk for anemia (total cumulative intake = 45–60 mg elemental iron/day); males and patients without history of anemia should receive at least 18 mg iron.; No enteric coasting.; Avoid excessive intake of tea owing to tannin interaction.; Encourage foods rich in heme iron.; Vitamin C may enhance absorption of nonheme sources. None. Add a minimum of 27–60 mg/day elemental. Add a minimum of 27–60 mg/day elemental. Begin as tolerated. Row 5. Fat-soluble vitamins: With all procedures, higher maintenance doses may be required for those with a history of deficiency.; Water-soluble preparations of fat-soluble vitamins are available.; Retinol sources of vitamin A should be used to calculate dosage. Vitamin A 5,000 IU/day and Vitamin K 90–120 µg/day. Vitamin E should be taken at a dosage of 15 mg/day. 10,000 IU of vitamin A,3,000 IU of vitamin D, and300 µg of vitamin K. May begin 2–4 wk after surgery. Row 6. Most supplements contain a high percentage of beta carotene, which does not contribute to vitamin A toxicity.; Intake of 3,000 IU vitamin D3 may be achieved with careful selection of multivitamin and calcium supplements.; No toxic effect known for vitamin K1, phytonadione (phyloquinone).; Vitamin K requirement varies with

dietary sources and colonic production.; Caution with vitamin K supplementation for patient receiving coagulation therapy.; Vitamin E deficiency has been suggested but is not prevalent in published studies. Blank. Vitamin A 5,000–10,000 IU/day and Vitamin K 90–120 µg/day. Blank. Blank. Blank. Row 7. Optional B complex: B-50 dosage: Liquid form is available.; Avoid time-released tablets.; No known risk of toxicity.; May provide additional prophylaxis against B-vitamin deficiencies, including thiamin, especially for BPD/DS procedures as water-soluble vitamins are absorbed in the proximal jejunum.; Note: >1,000 ng of supplemental folic acid, provided in combination with multivitamins, could mask B12 deficiency. 1 serving/day. 1 serving/day. 1 serving/day. May begin as tolerated. The AGB, adjustable gastric band; BPD/DS, biliopancreatic diversion with or without duodenal switch; RYGB, Roux-en-Y gastric bypass; SG, sleeve gastrectomy. The Data from (i) Linda Aills, et al., “ASMBS Allied Health Nutritional Guidelines for the Surgical Weight Loss Patient,” Surgery for Obesity and Related Diseases 4, no. 5 (2008): S73-S108, © 2013 by Elsevier, and (ii) Updated with data from Parrott J, Frank L, Rabena R, Craggs-Dino L, Isom KA, Greiman L. American Society for Metabolic and Bariatric Surgery integrated health nutritional guidelines for the surgical weight loss patient 2016 update: Micronutrients. Surg Obes Relat Dis.

2017;13(5): 727-741. doi: 10.1016/j.soard.2016.12.018, and (iii) Updated with data from Mechanick JI, Apovian C, Brethauer S, et al. Endocr Pract 2019 update 2019;25(12):1346-1359. Epub 2019 Nov 4; doi:10.4158/GL -2019-0406P.

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The table is titled as Biochemical Monitoring Tools for Nutrition Status in the Post–Bariatric Surgery Patient and it has 7 Vitamin/Mineral, Screening, Additional Laboratory Indexes, Normal Range, Critical Range, Postoperative Deficiency, and Comment columns Time Frame, Possible Causes, and Interventions. The row entries are as follows. Row 1. B1Thiamin. Serum thiamin. (↓)RBS transketolase(↑)Pyruvate. 10–64 ng/mL. Transketolase activity >20%Pyruvate >1 mg/dL. 15–29% more common in AA and Hispanics; often assoc. with poor hydration. Serum thiamin responds to dietary supplementation, but is poor indicator of total body stores. Row 2. B6Pyridoxine. PLP. RBC glutamic pyruvate oxaloacetic transaminase. 5–24 ng/mL. <3. Unknown. Consider with unresolved anemia; diabetes could influence values. Row 3. B12Cobalamin. Serum B12. (↑)Serum and urinary MMA,(↑)Serum tHcy. 200–1,000 pg/mL. <200. 10–13% may occur with older patients and those taking H2 blockers and PPIs. When symptoms are present and B12 200–250 pg/mL, MMA and tHcy

are useful; serum B12 may miss 25–30% of deficiency cases. Row 4. Folate. RBC folate, serum folate. Urinary FIGLUNormal serum and urinary MMA(↑)Serum tHcy. 280–791 ng/mL. <305 nmol/L. Uncommon. Serum folate reflects recent dietary intake rather than folate status; RBC folate is a more sensitive marker; excessive supplementation can mask B12 deficiency in CBC; neurologic symptoms will persist. Row 5. Iron. Ferritin. (↓)Serum iron(↑)TIBC. Males: 15–200 ng/mLFemales: 12–150 ng/mL. <20. 9–16% of adult women in general population are deficient. Low Hgb and Hct are consistent with iron deficiency anemia in stage 3 or stage 4 anemia; ferritin is an acute phase reactant and will be elevated with illness and/or inflammation; oral contraceptives reduce blood loss for menstruating females. Row 6. Vitamin A. Plasma retinol. RBP. 20–80 µg/dL. <10. Uncommon, up to 7% in some studies. Ocular finding may suggest diagnosis. Row 7. Vitamin D. 25(OH)D. (↓)Serum phosphorus(↑)Alkaline phosphatase(↑)Serum PTH(↓)Urinary calcium. 25–40 ng/mL. <20. Common; 60–70%. With deficiency, serum calcium may be low or normal; serum phosphorus may decrease, serum alkaline phosphatase increase; PTH elevated. Row 8. Vitamin E. Plasma alpha tocopherol. Plasma lipids. 5–20 µg/mL. <5. Uncommon. Low plasma alpha tocopherol to plasma lipids (0.8 mg/g total lipid)

should be used with hyperlipidemia. Row 9. Vitamin K. PT. (↑)DCP(↓)Plasma phylloquinone. 10–13 seconds. Variable. Uncommon. PT is not a sensitive measure of vitamin K status. Row 10. Zinc. Plasma zinc. (↓)RBC zinc. 60–130 μg/dL. <70. Uncommon, but increased risk of low levels associated with obesity. Monitor albumin levels and interpret zinc accordingly, albumin is primary binding protein for zinc; no reliable method of determining zinc status is available; plasma zinc is method generally used; studies cited in this report did not adequately describe methods of zinc analysis. Row 11. Protein. 4–6 g/dL; 6–8 g/dL. <3, <20 mg/dL. Uncommon. Half-life for prealbumin is 2–4 days and reflects changes in nutritional status sooner than albumin, a nonspecific protein carrier with a half-life of 22 days. The Data from Parrott J, Frank L, Rabena R, Craggs-Dino L, Isom KA, Greiman L. American Society for Metabolic and Bariatric Surgery integrated health nutritional guidelines for the surgical weight loss patient 2016 update: Micronutrients. Surg Obes Relat Dis. 2017;13(5):727-741. doi: 10.1016/j.soard.2016.12.018.

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The table is titled as Chemotherapy Medications and it has 7 columns Chemotherapeutic Agent: Generic Name (Trade Name), Nausea and Vomiting Diarrhea,

Xerostomia, Stomatitis and Esophagitis, Anorexia, andTaste Alterations. The row entries are as follows. Row 1. Anastrozole (Arimidex). Mild. No. No. No. No. No. Row 2. Bleomycin (Blenoxane). Mild to moderate. No. Yes. Yes. Yes. No. Row 3. Busulfan (Myleran). Mild. No. No. No. Yes. No. Row 4. Capecitabine (Xeloda). Mild. Yes. No. No. No. No. Row 5. Carboplatin (Paraplatin). Moderate. Yes. No. No. No. No. Row 6 Carmustine (BCNU). Moderate. No. No. Yes. Yes. No. Row 7. Cisplatin (CDDP). Severe. Yes. No. No. Yes. Metallic taste. Row 8 Cyclophosphamide (Cytoxan). Severe. No. Yes. Yes. Yes. No. Row 9. Cytarabine (ARA-C). Severe. Yes. No. Yes. Yes. No. Row 10 Dacarbazine (DTIC-Dome). Severe. Yes. No. Yes. No. Metallic taste. Row 11 Dactinomycin (Actinomycin-D, ACT). Severe. Yes. Yes. Yes. No. Yes. Row 12. Daunorubicin citrate (Daunomycin). Moderate. Yes. Yes. Yes. Yes. Yes. Row 13. Docetaxel (Taxotere). Mild. No. No. No. No. No. Row 14. Doxorubicin (Adriamycin). Moderate. Yes. Yes. Yes. Yes. No. Row 15. Epirubicin HCL (Ellence). Moderate. Yes. No. Yes. No. No. Row 16. Etoposide (VP-16-23). Mild to moderate. Yes. No. Yes. Yes. No. Row 17.Floxuridine (FUDR). Mild. Yes. No. Yes. Yes. No. Row 18. 5-Fluorouracil (5-FU). Moderate. Yes. No. Yes. No. Yes. Row 19 Gemcitabine hydrochloride (Gemzar). Mild. Yes. No. Yes. No. No. Row 20. Hydroxyurea (Hydrea). Mild to moderate. Yes. No. Yes. Yes. No. Row 21 Irinotecan (Camptosar). Severe. Yes. No. No. Yes.

No. Row 22 L-Asparaginase (Elspar). Moderate. No. No. Yes. Yes. No. Row 23. Mechlorethamine (Mustargen). Severe. Yes. No. No. Yes. Metallic taste. Row 24 Methotrexate (MTX). Mild to moderate. Yes. No. Yes. Yes. Yes. Row 25 Mitomycin (Mutamycin). Moderate. Yes. No. Yes. Yes. No. Row 26 Nivolumab (Opdivo). Mild. Yes. No. No. Yes. No. Row 27. Oxaliplatin (Eloxatin)a. Moderate. Yes. No. No. No. No. Row 28. Paclitaxel (Taxol). Mild. No. No. Yes. No. No. Row 29. Pembrolizumab (Keytruda). Mild. Yes. No. No. No. No. Row 30 Procarbazine hydrochloride (Matulane)b. Severe. Yes. No. Yes. Yes. No. Row 31 Streptozocin (Zanosar) Severe. Yes. No. No. No. No. Row 32 Tamoxifen citrate (Nolvadex). Mild. No. No. No. Yes. No. Row 33. Temozolomide (Temodar). Moderate. No. No. No. Yes. No. Row 34 Topotecan hydrochloride (Hycamtin). Mild. Yes. No. No. Yes. No. Row 35 Vinblastine sulfate (Velban). Moderate. Yes. No. Yes. No. No. Row 36. Vincristine sulfate (Vinscar). Mild. Yes. No. Yes. Yes. No. Row 37. Vinorelbine tartrate (Navelbine). Mild. No. No. No. Yes. No. The foot note of a is Sensitivity to cold for 5 or more days following drug administration. The foot note of b is To prevent hypertensive crisis, a low-tyramine diet must be followed while receiving procarbazine hydrochloride and 4 weeks after the final dose. The Data from medication product labeling.

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The table is titled as Medications to Treat Anorexia and it has 4 columns Medication: Generic Name (Trade Name), Dose, Action, and Special Considerations. The row entries are as follows. Row 1. Dronabinol (Marinol). 2.5 mg BID (up to 20 mg/d). Increases appetite, decreases nausea. Drug may be habit-forming. Row 2. Megestrol acetate (Megace). 800 mg daily. Increases appetite, promotes weight gain. May take 8–12 wk to reach maximal weight gain. Row 3. Mirtazapine (Remeron). 15–45 mg daily. Increases appetite. Take at bedtime, causes drowsiness. The Data from medication product labeling.

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The table is titled as Medications to Treat Nausea and Vomiting and it has 4 columns Medication: Generic Name (Trade Name), Dose, Action, and Special Considerations. The row entries are as follows. Row 1. Aprepitant (Emend). Oral: 125 mg on the day of chemo; 80 mg a day after chemo; 80 mg 2 d after chemo; IV: 150 mg over 20–30 min, given 30 min prior to chemo, Prevents N&V. Dose is for ≥12 year olds and ≥30 kg. Row 2. Dolasetron mesylate (Anzemet).Oral: 100 mg; IV: 1.8 mg/kg over 30 sec. Decreases N&V. Give 30–60 min before chemo. Row 3. Granisetron HCl (Kytril). Oral: 1 mg tablet or 5 mL suspension BID; IV: 10 µg/kg. Decreases N&V.Oral

first dose 1 h before chemo and second dose 12 h later; IV: 30 min before chemo. Row 4. Lorazepam (Ativan). Oral: 1–6 mg/d; IV: 1.4 mg/m2. Decreases anxiety, relaxes muscles, decreases N&V. Oral: in divided doses; IV: 30 min before chemo. Row 5. Metoclopramide (Reglan). Oral: 10 mg QID; IV: 1–2 mg/kg q2h. Stimulates GI motility and gastric emptying. May cause diarrhea at high doses. Row 6. Ondansetron (Zofran). Oral: 8 mg q4h for three doses, then q8h for 1–2 d; IV: 32 mg bolus. Decreases N&V. Start oral and IV 30 min before chemo. Row 7. Palonosetron hydrochloride (Aloxi). IV: 0.25 mg over 30 sec, given 30 min prior to chemo. Prevents N&V. Adult dose. Row 8. Perphenazine (Trilafon). Oral: 4 mg q4–6h; IV/IM: 3–5 mg bolus q4–6h. Decreases N&V. May cause dry mouth. Row 9. Prochlorperazine (Compazine). Oral: 5–10 mg QID; Rectal suppository: 25 mg BID. Decreases N&V. Antacids decrease absorption of oral dose—separate by 2 h. GI, gastrointestinal. The Data from medication product labeling.

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The table is titled as Medications to Treat Diarrhea and it has 4 columns Medication: Generic Name (Trade Name), Dose, Action, and Special Considerations. The row entries are as follows. Row 1. Diphenoxylate HCl and atropine (Lomotil). Oral: 5 mg

QID. Slows peristalsis. Can cause nausea and dry mouth. Row 2. Loperamide HCl (Imodium). Oral: 4 mg, followed by 2 mg after each unformed stool. Inhibits peristalsis. Shown to be 2–3 times more potent than Lomotil. The Data from medication product labeling.

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The table is titled as LDL-C Targets for Statin Therapy Combined with Lifestyle Modification and it has 4 columns Targets, Patient Groups, Statin & Non-Statin Therapy, and Intensive Lifestyle Modification. The row entries are as follows. Row 1. Primary prevention. Children aged 0–19 y should be assessed periodically for lifetime riska and calculated 10-year risk of ASCVDb. Children with diagnosis of familial hypercholesterolemia (FH) or abnormal lipid profiles; and fail 3-month lifestyle therapy. Emphasize a heart-healthy lifestyle across the life course for all individuals.; Lifestyle therapy is primary prevention for individuals with metabolic syndrome.Lifestyle therapy is primary prevention for individuals with hypercholesterolemia. Row 2. Hypercholesterolemia (not very high-risk ASCVD). Adults 20–39 y. Consider statin if family history, premature ASCVD and LDL-C ≥160 mg/dL (≥4.1 mmol/L). Encourage lifestyle to reduce ASCVD risk. Adults 40–75 y and LDL-C ≥70 to <190 mg/dL (≥1.8–<4.9 mmol/L) without diabetes

mellitus; Calculated 10-y ASCVD risk indicators are: Low- <5%; Borderline- 5 to <7.5%; Intermediate- ≥7.5–<20%; high risk ≥20%. Coronary artery calcium (CAC) scores may guide statin use decision if risk is still unclear. Clinician-patient risk discussion initiated for borderline, intermediate and high risk ASCVD patients before starting statins. Topics include: risk enhancers; patient's preference; drug costs and adverse effects of statins. Moderate-intensity statins considered for borderline and intermediate-risk patients (30%–49% LDL-C reduction) High-intensity statins for high-risk ASCVD patients to achieve LDL-C lowering ≥50%; If high-intensity statin not tolerated, use moderate intensity statin. If on maximal statin and LDL-C ≥70 mg/dL/1.8 mmol/L, add ezetimibe. Emphasize lifestyle for low to borderline ASCVD risk groups. Note—All individuals receiving statins should be counseled on healthy lifestyle habits. Adults >75 y. Clinical assessment, Risk discussion [as above]. Row 3. Hypercholesterolemia (very high-risk ASCVD): Age 20–75 y with severe hypercholesterolemia (LDL-C ≥190 mg/dL/4.9 mmol/L).; Calculation of ASCVD 10-y risk not required. Initiate high-intensity or maximal statin therapy. Add ezetimibe if LDL-C ≥70 mg/dL (1.8 mmol/L).; A proprotein convertase subtilisin/kexin type 9 inhibitor (PCSK9-I) with LDL-C ≥ 220 mg/dL despite maximally tolerated statins and ezetimibe. Review lifestyle habits for diet, physical activity, body

weight/BMI, and tobacco use.; Endorse healthy lifestyle with counseling and patient education material.; Referral to dietitian required.; Adherence to LM targets should be assessed. Age 20–75 y with severe hypercholesterolemia (LDL-C ≥190 mg/dL/4.9 mmol/L) and triglycerides ≤300 mg/dL/ 3.4 mmol/L. Maximally tolerated statin therapy with ezetimibe if LDL-C lowering < 50% or ≥100 mg/dL (2.6 mmol/L) and addition of bile sequestrants. Evaluate diet as a secondary cause of hypertriglyceridemia: Elevated LDL-C—saturated or trans fats, weight gain, anorexia; Elevated TG—weight gain, very low-fat diets, high intake of refined carbohydrates, excessive alcohol intake; ethnic-based diets should be assessed for carbohydrate and fat consumption levels; Assess sedentary behaviors. Assess less favorable diets. Age 30–75 y with heterozygous FH and LDL-C ≥100 mg/dL (2.6 mmol/L). Maximally tolerated statin therapy with ezetimibe and addition of PCSK9 inhibitor. Review lifestyle habits for diet, physical activity, body weight/BMI, and tobacco use. Age 40–75 y with baseline LDL-C ≥ 220mg/dL (5.7 mmol/L). Maximally tolerated statin therapy with ezetimibe and addition of PCSK9 inhibitor. Review lifestyle habits for diet, physical activity, body weight/BMI, and tobacco use. Row 4. With Clinical ASCVD: Individuals ≤75 y with clinical ASCVD not at high risk. Consider high-intensity statin; (moderate-intensity statin if high-

intensity statin not tolerated).; With maximal statin therapy and LDL-C ≥70 mg/dL (≥1.8mmol/L) ezetimibe may be added. .Counsel on intensive healthy lifestyle habits: Regularly monitor adherence to lifestyle management.; Reinforce healthy lifestyle during follow-ups 3–12 mo. Age >75 y with clinical ASCVD not at high risk. Initiate moderate- or high-intensity statin. Row 5. With Diabetes: Individuals 40–75 y of age with diabetes regardless of ASCVD risk estimation. Moderate-intensity statin. Individuals 40–75 y of age with diabetes and with LDL-C 70–189 mg/dL (1.7 to 4.8 mmol/L) with 1st ASCVD event. Evaluate benefit for high-intensity statin based on individual's global risk (age group, race, and gender) and patient preference, lab values. In adults with diabetes with multiple ASCVD risk factors. High-intensity statin. Age >75 y with diabetes. If not on statins, to initiate statin therapy. If on statin therapy, continue use of statins. Adults with diabetes with 10-y ASCVD risk 20% or higher. Maximally tolerated statin therapy with ezetimibe. Age 20 to 39 yr with long term diabetes and co-morbidities such as albuminuria, eGFR <60ml/min/1.73m2, retinopathy, neuropathy, ankle-brachial index (ABI) <0.9. Initiate statin therapy. High-intensity statin: lower LDL-C by ≥50%, such as Atorvastatin (40)–80 mg; Rosuvastatin 20 (40) mg; for history of hemorrhagic stroke and Asian ancestry. Moderate-intensity statin: lower LDL-C

by 30–49%, such as Atorvastatin 10 (20) mg; Rosuvastatin (5) 10 mg; Simvastatin 20–40 mg; Pravastatin 40 (80) mg; Lovastatin 40 (80) mg; Fluvastatin XL 80 mg; Fluvastatin 40 mg BID; Pitavastatin 1–4 mg. Low-intensity statin: lower LDL-C by <30%, such as simvastatin 10mg, pravastatin 10–20 mg, lovastatin 20 mg, fluvastatin 20–40 mg. The footnote a is Lifetime risk constitutes cigarette smoking, elevated blood pressure, LDL-C, hemoglobin A1C [if indicated]. The footnote b is ASCVD Risk estimate—A spreadsheet enabling estimation of 10-y and lifetime risk for ASCVD and a Web-based calculator are available at http://my.americanheart.org/cvriskcalculator and https://www.acc.org/tools-and-practice - support/mobile-resources/features/2013-prevention-guidelines-ascvd-risk-estimator. The footnote c is ASCVD Risk enhancers—family history of premature ASCVD, persistent elevated LDL-C ≥160 mg/dL (≥4.1 mmol/L), chronic kidney disease, metabolic syndrome, inflammatory diseases, ethnicity (South Asian ancestry), women-specific conditions (preeclampsia, premature menopause), persistently elevated triglycerides (≥175 mg/mL), elevated biomarkers (hsCRP ≥2.0 mg/L, Lp(a) >50 mg/dL or >125 nmol/L, apoB ≥130 mg/dL, ankle-brachial index (ABI) <0.9. The Data from Grundy, Scott M., et al., “2018

AHA/ACC/AACVPR/AAPA/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Blood Cholesterol: A report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines," Circulation 139, no. 25 (2019): e1082-e1143, https://www.ahajournals.org/doi/10.1161/CIR.0000000000000625.

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The table is titled as ACC/AHA 2017 Classification of Blood Pressure and it has 4 columns BP Category, Systolic (mmHg), Blank, and Diastolic (mmHg). The row entries are as follows. Row 1. Normal. <120. And. <80. Row 2. Elevated. 120–129. Or. <80. Row 3. Stage 1 HTN. 130–139. Or. 80–89. Row 4. Stage 2 HTN. ≥140. Or. ≥90. The Data from Paul K. Whelton, et al., "2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines," Hypertension 71, no. 6 (2018): e13-e115, DOI: 10.1161/HYP.0000000000000065.

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The table is titled as Hypertension Treatment Algorithm and it has 3 columns Patient Groups, Antihypertension Therapy a (Indication and Monitoring), and Lifestyle Modificationb. The row entries are as follows. Row 1. Normal BP with systolic BP <120 or diastolic <80 mmHg. No. Reassess annually. Promote optimal lifestyle habits. Row 2. Elevated BP with systolic BP 120–129 or diastolic <80 mmHg. No. Reassess 3 to 6 mo. Non-pharmacological therapyc is preferred. Adherence and impact should be assessed 3 to 6 mo. Row 3. Stage 1 hypertension with systolic BP 130 to 139 mmHg or diastolic BP 80 to 89 mmHg. No Reassess after 6 mo. Non-pharmacological therapy.c. Row 4. With Clinical ASCVD or estimated 10-y CVD risk ≥10% Yes. Reassess in 1 mo. Non-pharmacological therapy .c. Row 5. Stage 2 hypertension with systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg. Yes. Reassess in 1 mo: If goal not met—assess and optimize adherence to therapy, consider intensified therapy. If goal is met—reassess in 3–6 mo. Non-pharmacological therapy.c The footnote of a is Suggested drugs are adjusted as per HTN stage per age, race, and co-morbidities. Stage 1 HTN patients require initial monotherapy with dosage titration with sequential addition of other agents if required to achieve BP target. Stage 2 HTN patients are initiated with combination therapy using 2 first-line agents of different classes or in a fixed-

dose combination. African-American patients with/out diabetes but without heart failure/CKD—initial therapy is thiazide diuretic or CCB. First-line drugs include thiazide diuretic, CCB, ACE-I or ARB. Stable Ischemic Heart Disease: BB, ACE-I, ARB Angina Pectoris: CCB, thiazide diuretic Aortic Disease: BB Atrial fibrillation: ARB Heart Failure: ACE-I or ARB, MRA, thiazide diuretic, BB, ARNI Heart Failure with Preserved Ejection Fraction: ACE-I or ARB, BB, thiazide Diabetes: ACE-I or ARB, thiazide diuretic, CCB Chronic Kidney Disease: ACE-I or ARB Secondary Stroke or TIA: thiazide diuretic, ACE-I, ARB Abbreviations: ACE-I, angiotensin-converting enzyme inhibitor; ARNI, angiotensin receptor-neprilysin inhibitors; ARB, angiotensin II receptor blocker; BB, β-blocker; BP, blood pressure; CCB, calcium channel blocker; HTN, hypertension; MRA, mineralocorticoid receptor antagonist; TIA, transient ischemic attack. The footnote b is Social determinants of health should be considered in lifestyle modification therapy: Diet and sedentary lifestyle, Poverty; Income inequality; Food insecurity; Unhealthy food environment of neighborhoods; Health literacy . The footnote of c is Non-pharmacological therapy: Note-All Recommendations COR:1; LOE:A [i] Weight loss (aim for ideal body weight or—1kg for overweight status) [ii] A heart-healthy diet as per dietary patterns (DASH etc.) [iii] Sodium reduction (optimal is

<1,500 mg/d but aim for 1,000 mg/d) [iv] Potassium supplementation aiming for 3,500–5,000 mg/d (preferable with diet modification) [v] Increased physical activity: Aerobic; 90–150 min/wk; 65%–75% heart rate reserve; Dynamic resistance; 90–150 min/wk; 50%–80% 1 rep maximum; 6 exercises, 3 sets/exercise, 10 repetitions/set Isometric resistance; 4 × 2 min (hand grip), 1 min rest between exercises, 30%–40% maximum voluntary contraction, 3 sessions/wk; 8–10 wk; [vi] Moderate alcohol consumption; women ≤ 1 standard drinks/d; men ≤ 2 standard drinks/d. The Data from (i) Paul K. Whelton, et al., “2017 ACC/AHA/AAPA/ABC/ACPM/AGS/APhA/ASH/ASPC/NMA/PCNA Guideline for the Prevention, Detection, Evaluation, and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines,” Hypertension 71, no. 6 (2018): e13-e115, DOI: 10.1161/HYP.0000000000000065 (ii) Carey, Robert M., Wright Jr, Jackson T., Taler. Sandra J., Whelton, Paul K. Guideline-Driven Management of Hypertension- An Evidence-Based Update. Circulation Research. 2021;128:827-846. https://doi.org/10.1161/CIRCRESAHA.121.318083; and (iii) Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA guideline on primary prevention of

cardiovascular disease: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines. J Am Coll Cardiol. 2019;74:e177-e232.

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The table is titled as DASH Eating Plan Based on Energy Level and it has 4 columns Food Group, Serving Size, Calories per Servinga, and Servings as per Daily Calorie Plan (1,600 kcal; 2,000 kcalb; 2,600 kcal; 3,100 kcal) The row entries are as follows. Row 1. Grains. 1 slice bread; 1 cup ready-to-eat cereal; ½ cup cooked rice, pasta, or cereal. 75. 6–7. 7–8. 9–10. 11–12. Row 2. Vegetables. 1 cup raw leafy vegetable; ½ cup cooked vegetable; 6-oz vegetable juice. Estimated as ~25 kcal. 3–4. 4–5. 5–7. 6–8. Row 3. Fruits. 1 medium fruit; ¼ cup dried fruit; ½ cup fresh, frozen, or canned fruit; 6-oz fruit juice. 60. 3–4. 4–5. 5–7. 6–8. Row 4. Low-fat or fat-free dairy. 8-oz milk; 1 cup yogurt; 1½-oz cheese. 125 of low fat or 90 if fat-free. 2. 2–3. 3–4. 3–5. Row 5. Meats, poultry, fish. 3-oz cooked lean meat, skinless poultry, or fish. 158 if meat/poultry, but 85 if fish. ≤2. 2 or fewer. 3 or fewer. 4 or fewer. Row 6. Nuts, seeds, dry beans, and peas. 13 cup or 1½-oz nuts; 1 Tbsp or ½-oz seeds; ½ cup cooked dry beans. 45. 3–4/wk. 4–5/wk. 5–7/wk 6–8/wk. Row 7. Fats and oilsd. 1 Tsp soft margarine;1 Tbsp low-fat mayonnaise; 2 Tbsp light salad dressing;

1 Tsp vegetable oil. 45. 2. 2–3. 3–4. 3–5. Row 8. Sweets. 1 Tbsp sugar; 1 Tbsp jelly or jam; ½-oz jelly beans; 8-oz lemonade. 60. 4/wk. 5/wk. 7/wk. 8/wk. Row 9. Sodium. Check food labels for sodium content per serving size. 2,300 mg; 1,500 mg for high riske. Blank. The footnotes a to e are Calories estimated from website: http://glycemic.com/DiabeticExchange/The%20Diabetic%20Exchange%20List.pdf. Gray area in the recommended DASH eating plan. Servings are calculated for a 2,000 kcal diet. http://www.nhlbi.nih.gov. Serving sizes vary between ½ cup and 1¼ cups. Check the product's nutrition label. Fat content changes serving counts for fats and oils: For example, 1 Tbsp of regular salad dressing equals 1 serving, 1 Tbsp of low-fat salad dressing equals ½ serving, and 1 Tbsp of fat-free salad dressing equals 0 servings. For African Americans at high risk for hypertension. Lower sodium goal effective at lowering BP. The Data from Government of the United States, National Heart, Lung, and Blood Institute, Your Guide to High Blood Pressure with DASH, NIH Publication No. 06-4082, (Washington, DC: U.S. Department of Health and Human Services, 2006).

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A table is titled Diagnostic Criteria for Diabetes and Prediabetesa. It has the headers: Blank, Fasting Plasma Glucose (mg/dL), A1C (%), and 2-Hour Plasma Glucose in the OGTT (mg/dL). Row 1. Blank, Normal. Fasting Plasma Glucose (mg/dL), <100. A1C (%), ≤5.6. 2-Hour Plasma Glucose in the OGTT (mg/dL), ≤139. Row 2. Blank, Prediabetes. Fasting Plasma Glucose (mg/dL), 100–125. A1C (%), 5.7–6.4. 2-Hour Plasma Glucose in the OGTT (mg/dL), 140–199. Row 3. Blank, Diabetes. Fasting Plasma Glucose (mg/dL), ≥126. A1C (%), ≥6.5. 2-Hour Plasma Glucose in the OGTT (mg/dL), ≥200. Footnote below the table indicates that Each criterion must be confirmed by repeat testing unless unequivocal symptoms of hyperglycemia are present.

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A table is titled Exchange Lists for Meal Planning. It has the headers: Food Groups, Kilocalories, Carbohydrate, Protein, and Fat. The entries under Carbohydrates are as follows. Row 1. Starch. 80. 15. 3. 0–1. Row 2. Milk—skim and low-fat. Blank. Blank. Blank. Blank. Row 3. 2%. 120. 12. 8. 5. Row 4. Whole. 150. 12. 8. 8. Row 5. Fruit. 60. 15. —. —. Row 6. Vegetables. 25. 5. 2. —. The entries under Meat and Meat Substitutes are as follows. Row 7. Very lean. 35. —. 7. 1. Row 8. Lean. 55. —. 7. 2–3. Row 9. Medium-

fat. 75. —. 7. 5. Row 10. High-fat. 100. —. 7. 8. Row 11. Fat. 45. —. —. 5.

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A table is titled Insulin and Insulin Analogs. It has the headers: Type of Insulin: Brand Name (Generic Name), Onset, Peak, Duration (h), and When to Administer. Row 1. Type of Insulin: Brand Name (Generic Name), Rapid-acting; Humalog, Admelog, Lyumjev (insulin lispro) NovoLog, Fiasp (insulin aspart) Apidra (insulin glulisine). Onset, 5–15 min. Peak, 30–90 min. Duration (h), 3–5. When to Administer, 0–15 min before meals. Row 2. Type of Insulin: Brand Name (Generic Name), Rapid-acting by inhalation; Afrezza (technosphere insulin). Onset, 12–15 min. Peak, 60 min. Duration (h), 2–3. When to Administer, Blank. Row 3. Type of Insulin: Brand Name (Generic Name), Short-acting; Humulin R, Novolin R (Regular human [R]). Onset, 30–60 min. Peak, 2–4 h. Duration (h), 5–8. When to Administer, 30–45 min before meals. Row 4. Type of Insulin: Brand Name (Generic Name), Intermediate-acting; Humulin N, Novolin N (NPH human [N]). Onset, 1–3 h. Peak, 6–10 h. Duration (h), 12–16. When to Administer, Before a.m. and p.m. meal or before a.m. meal and at bedtime. Row 5 has the header Long-acting. Row 6. Type of Insulin: Brand Name (Generic Name), Basaglar, Lantus, Toujeo (insulin glargine).

Onset, 1 h. Peak, No peak. Duration (h), Up to 24. When to Administer, In a.m. or at bedtime. Row 7. Type of Insulin: Brand Name (Generic Name), Semglee (insulin glargine-yfgn). Onset, 1 h. Peak, No peak. Duration (h), Up to 24. When to Administer, In a.m. or at bedtime. Row 8. Type of Insulin: Brand Name (Generic Name), Levemir (insulin detemir). Onset, 1 h. Peak, No peak. Duration (h), Up to 24. When to Administer, Once or twice daily. Row 9. Type of Insulin: Brand Name (Generic Name), Tresiba (insulin degludec). Onset, Blank. Peak, Blank. Duration (h), Up to 42. When to Administer, Once daily. Row 10 has the header Combinations/Mixtures. Row 11. Type of Insulin: Brand Name (Generic Name), Humulin 70/30, Novolin 7/30 (NPH/Regular). Onset, 30–60 min. Peak, 2–12. Duration (h), 14–24. When to Administer, Before breakfast and dinner. Row 12. Type of Insulin: Brand Name (Generic Name), Humalog Mix 50/50 (protamine/lispro). Onset, 25–30 min. Peak, .5–3 h. Duration (h), 14–24. When to Administer, Blank. Row 13. Type of Insulin: Brand Name (Generic Name), Humalog Mix 75/25 (protamine/lispro). Onset, 15–30 min. Peak, .5–2.5 h. Duration (h), 14–24. When to Administer, Before breakfast and dinner. Row 14. Type of Insulin: Brand Name (Generic Name), NovoLog Mix 70/30 (protamine/aspart). Onset, 5–15 min. Peak, 1–4.

Duration (h), 18–24. When to Administer, Before breakfast and dinner.

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A table is titled AACE Lipid and Blood Pressure Targets for Patients with Type 2 Diabetes. It has the headers: Risk Levels, High DM but no Other Major Risk and ≤40 YOA, Very High DM + Major ASCVD Risksa, and Extreme DM + Established Clinical CVD. Row 1. Risk Levels, LDL cholesterol (mg/dL). High DM but no Other Major Risk and ≤40 YOA, <100. Very High DM + Major ASCVD Risksa, <70. Extreme DM + Established Clinical CVD, <55. Row 2. Risk Levels, Non-HDL cholesterol (mg/dL). High DM but no Other Major Risk and ≤40 YOA, <130. Very High DM + Major ASCVD Risksa, <100. Extreme DM + Established Clinical CVD, <80. Row 3. Risk Levels, Triglycerides (mg/dL). High DM but no Other Major Risk and ≤40 YOA, <150. Very High DM + Major ASCVD Risksa, <150. Extreme DM + Established Clinical CVD, <150. Row 4. Risk Levels, Apo B (mg/dL). High DM but no Other Major Risk and ≤40 YOA, <90. Very High DM + Major ASCVD Risksa, <80. Extreme DM + Established Clinical CVD, <70. Row 5. Risk Levels, Blood Pressure. High DM but no Other Major Risk and ≤40 YOA, <130/80 mm Hg. Very High DM + Major ASCVD Risksa, <130/80 mm Hg. Extreme DM + Established Clinical CVD, <120/70 mm Hg.

Footnote below the table indicates that Hypertension, family history of ASCVD (atherosclerotic cardiovascular disease), low HDL-C, smoking, CKD3,4. The text below the table indicates that YOA, years of age; CVD, cardiovascular disease; LDL, low-density lipoprotein; HDL, high-density lipoprotein; Apo B, apolipoprotein B.

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A table is divided into 2 sections. Section 1 is titled Foods High in FODMAPs: Limit or Avoid. It has the headers: Fructans, Fructose, Galactans, Lactose, and Polyols. Row 1. Fructans, Fruits: persimmon and watermelon; Grains: rye and wheat products in high amounts; Vegetables: artichokes, asparagus, beets, broccoli, Brussels sprouts, cabbage, eggplant, fennel, garlic, leek, okra, onions, and shallots; Other: pistachios and products containing chicory, inulin. Fructose, Fruits: apple, mango, pear, and watermelon; Sweeteners: agave, corn syrup, fructose, high-fructose corn syrup, and honey. Galactans, Legumes: baked beans, chickpeas, kidney beans, lentils, soybeans. Lactose, Cheeses: cottage cheese, cream cheese, ricotta, and other soft, unripened cheeses; Milk: cow’s, goat’s, and sheep’s milk, and products from milk such as ice cream and yogurt. Polyols, Fruits: apple, apricot, avocado, blackberries, cherries, nectarine, peach, pear, plum,

and watermelon; Sweeteners: isomalt, maltitol, mannitol, sorbitol, xylitol; Vegetables: cauliflower, corn, green bell pepper, and mushrooms. Section 2 is titled Foods Low in FODMAPs: Appropriate for Diet. It has the headers: Fruits, Dairy, Grains, Vegetables, and Miscellaneous. Row 2. Fruits, Banana, blueberries, boysenberries, cantaloupe, cranberries, grapes, grapefruit, honeydew, kiwi, lemon, lime, orange, passion fruit, raspberries, rhubarb, strawberries, and tangerines. Dairy, Cheese: brie, camembert, and hard cheeses; Ice cream: gelato and sorbet; Milk: lactose-free milk, oat, rice, and soy milk (and tofu); Yogurt: lactose-free products. Grains, Arrowroot, corn products (polenta), gluten-free products, millet, oats, psyllium, quinoa, rice, spelt, and tapioca. Vegetables, Alfalfa and bean sprouts, bamboo shoots, bok choy, carrot, celery, endive, ginger, green beans, lettuce, olives, parsnip, potato, pumpkin, red bell pepper, spinach, squash, sweet potato, tomato, turnip, yam, and zucchini. Miscellaneous, Sweeteners: any artificial sweetener not ending in “ol,” glucose, sucrose (table sugar).

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A table is titled Nutrition Intervention and Monitoring in Acute Kidney Injury. It has the headers: Nutrient Rationale, Recommended Amounts, it is subdivided into ASPEN and KDIGO, and Monitor. Row 1. Nutrient

Rationale, Protein: Avoid net negative nitrogen balance associated with hypercatabolism; Sufficient to reduce serum urea and improve urinary output12; Sufficient to maintain somatic and visceral protein status; Replace protein losses from KRT; Correct hyperkalemia. Recommended Amounts, ASPEN: 1.2–2.0 g/kg/d; On hemodialysis or CRRT: additional 0.2 g/kg/d up to 2.5 g/kg/d. KDIGO: Not on dialysis: 0.8–1.0 g/kg/d; On HD or PD: 1.0–1.5 g/kg/d; On CRRT: up to 1.7 g/kg/d. Monitor, BUN; Serum creatinine; Serum chloride; Urine output; GI tolerance. Row 2. Nutrient Rationale, Energy: Sufficient to spare protein for synthesis; Sufficient to prevent tissue protein catabolism; Titrate for stress-induced hyperglycemia. Recommended Amounts, ASPEN: 25–30 kcal/kg/d (use usual body weight for normal weight patients; use ideal body weight for obese or critically ill); Indirect calorimetry is preferred. KDIGO: 20–30 kcal/kg/d in patients with any stage of AKI; Indirect calorimetry is preferred. Monitor, Blood glucose; Insulin dose. Row 3. Nutrient Rationale, Electrolytes: Sodium—restriction during oliguric phase; Potassium—hyperkalemia from tissue catabolism; control to prevent cardiac arrhythmias. Recommended Amounts, ASPEN: Sodium—20–40 mEq/d (2–3 g) during oliguric phase; Potassium—30–50 mEq/d during oliguric phase; Phosphorus—limit as needed; Restriction not required on dialysis. KDIGO: Not

stated. Monitor, Blood levels of sodium, potassium, and phosphorus. Row 4. Nutrient Rationale, Fluid: Control in early oliguric phase; Net intake must balance output; Maintain normal blood pressure. Recommended Amounts, ASPEN: 500 mL + previous days output; Restriction not required on dialysis. KDIGO: Not stated. Monitor, I/O charts; Weight change; Blood pressure; IVs; Parenteral or enteral fluid volumes. The text below the table indicates that ASPEN, American Society for Parenteral and Enteral Nutrition; BUN, blood urea nitrogen; CRRT, continuous renal replacement therapy; GI, gastrointestinal; I/O, in-out charting; IV, intravenous.

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A table with 5 columns labeled Stage, Description, GFRb, Symptoms, and Action. The row entries are as follows: Row 1. Stage, Pre-CKD. Description, At increased risk. GFRb, >90 with CKD risk factors. Symptoms, Blank. Action, Screening for CKD; CKD risk reduction. Row 2. Stage, 1. Description, Kidney damage with normal or increased GFR. GFRb, ≥90. Symptoms, Albuminuria, proteinuria, hematuria. Action, Diagnosis and treatment; Treatment of comorbid conditions; Slowing progression; CVD risk reduction. Row 3. Stage, 2. Description, Kidney damage with mild decrease in GFR. GFRb, 60–89. Symptoms, Albuminuria, proteinuria, hematuria.

Action, Estimating progression. Row 4. Stage, 3a. Description, Moderate decrease in GFR. GFRb, 30–59. Symptoms, Early to chronic renal insufficiency. Action, Evaluating and treating complications; Start nutrition intervention. Row 5. Stage, 4. Description, Severe decrease in GFR. GFRb, 15–29. Symptoms, Chronic renal insufficiency, late renal insufficiency, pre-ESKD. Action, Preparation for RRT; Nutrition counseling. Row 6. Stage, 5. Description, Kidney failure. GFRb, <15. Symptoms, Renal failure, uremia, ESKD. Action, RRT if uremia is present. The text below the table indicates that CVD, cardiovascular disease; RRT, renal replacement therapy. Footnote below the table indicates that KDIGO guidelines2 established in 2012 further divide stage 3 CKD into the following categories, with concurrence from NKF-KDOQI: 3a (GFR 45–59): mild to moderate decrease in GFR or early renal insufficiency. 3b (GFR 30–44): moderate to severe decrease in GFR or chronic renal insufficiency. Glomerular filtration rate (mL/min/1.72 m2).

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A table with 5 columns labeled Dialysate Dextrose Concentration, Grams of Dextrose/L, kcal/L from Dextrose, kcal/L with CCPD (40%), and kcal/L with CAPD (60%). The row entries are as follows: Row 1. Dialysate Dextrose Concentration, 1.5%. Grams of Dextrose/L, 15 g. kcal/L from Dextrose, 51 kcal. kcal/L

with CCPD (40%), 20–26 kcal. kcal/L with CAPD (60%), 31–36 kcal. Row 2. Dialysate Dextrose Concentration, 2.5%. Grams of Dextrose/L, 25 g. kcal/L from Dextrose, 85 kcal. kcal/L with CCPD (40%), 34–43 kcal. kcal/L with CAPD (60%), 51–60 kcal. Row 3. Dialysate Dextrose Concentration, 4.25%. Grams of Dextrose/L, 42.5 g. kcal/L from Dextrose, 145 kcal. kcal/L with CCPD (40%), 58–73 kcal. kcal/L with CAPD (60%), 87–102 kcal. Row 4 has the text Example: A CAPD patient uses 4 L of 1.5% dialysate and 4 L of 4.25% dialysate daily.; 4 L of 1.5% = 124 kcal (31 kcal/L × 4 L); 4 L of 4.25% = 348 kcal (87 kcal/L × 4 L); Total kcal absorbed from dextrose = 472.

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Table lists common medications that have significant nutritional implications. The list is alphabetical by generic name and cross-referenced by brand or trade name.

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A table is titled C. Volume. The row entries are as follows. Row 1. 1 L. = 1,000 mL. 0.26 gal. 1.06 qt. 2.1 pt. Row 2. 1 mL. = 1,000 L. 0.03 fluid oz. Blank. Blank. Row 3. 1 gal. = 128 oz. 8 cups. 3.8 L. Blank. Row 4. 1 qt. = 32 oz. 4 cups. 0.95 L. Blank. Row 5. 1

pt. = 16 oz. 2 cups. 0.47 L. Blank. Row 6. 1 cup. = 8 oz. 16 Tbsp. ~250 mL. 0.25 L. Row 7. 1 oz. = 30 mL. Blank. Blank. Blank. Row 8. 1 tablespoon (Tbsp). = 3 tsp. 15 mL. Blank. Blank. Row 9. 1 teaspoon (tsp). = 5 mL. Blank. Blank. Blank.

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A table is titled D. Weight. The row entries are as follows. Row 1. 1 kg. = 1,000 g. 2.2 lb. Blank. Row 2. 1 g. = 1/1,000 kg. 1,000 mg. 0.035 oz. Row 3. 1 mg. = 1/1,000 g. 1,000 µg. Blank. Row 4. 1 µg. = 1/1,000 mg. Blank. Blank. Row 5. 1 lb. = 16 oz. 454 g. 0.45 kg. Row 6. 1 oz. = ~28 g. Blank. Blank.

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A table is titled Vitamin Facts and Nutrient Lists. It has the header A. Vitamins. Under it has four columns labeled Vitamin, Functions, Deficiency, and Toxicity. Row 1. Vitamin, A. Functions, Maintains integrity of cornea, epithelial cells, and mucous membranes; skin and bone and teeth growth; regulates synthesis of reproductive hormones and immune system; cancer protection. Deficiency, Anemia; Blindness, night blindness; Bone growth deficit; Corneal breakdown; Diarrhea; Joint pain; Kidney stones; Infection susceptibility. Toxicity, Amenorrhea; Anorexia; Bone pain; Fatigue; Headache; Nosebleeds; Skin rash. Row

2. Vitamin, D. Functions, Maintains bone tissue by regulating the absorption and excretion of calcium and phosphorus. Deficiency, Defective bone growth (bowed legs, joint pain); Muscle spasms. Toxicity, Anorexia; Headaches; Excessive thirst; Hypercalcemia; Kidney stones; Nausea; Weakness. Row 3. Vitamin, E. Functions, Maintains cell membranes; acts as an antioxidant in fighting disease-causing free radicals and in protecting other important compounds from oxidation. Deficiency, Anemia; Breast cysts; Leg cramps; Weakness. Toxicity, Enhances action of anticoagulants; Gastrointestinal distress. Row 4. Vitamin, K. Functions, Synthesis of blood clotting compounds; regulation of calcium levels in blood. Deficiency, Excessive bleeding. Toxicity, Jaundice; Interference or anticoagulant drugs. Row 5. Vitamin, B1 Thiamin. Functions, Coenzyme in energy metabolism; maintenance of appetite and nervous system function. Deficiency, Abnormal heart beat; Cardiomegaly/heart failure; Fluid retention; Mental confusion; Muscle pain, weakness, wasting paralysis. Toxicity, None documented. Row 6. Vitamin, B2 Riboflavin. Functions, Coenzyme in energy metabolism; maintenance of skin and visual function. Deficiency, Corneal abnormalities; Dry, cracking at corners of mouth; Sensitivity to light; Skin rash; Tongue abnormalities. Toxicity, None documented.

Row 7. Vitamin, B3 Niacin, Nicotinamide. Functions, Coenzyme in energy metabolism; maintenance of skin, and nervous and digestive systems. Deficiency, Anorexia; Diarrhea; Skin rash; Tongue abnormalities; Weakness and dizziness. Toxicity, Diarrhea; Dizziness; Liver dysfunction; Low blood pressure; Sweating, flushing. Row 8. Vitamin, B6 Pyridoxine. Functions, Coenzyme in protein and fat metabolism; synthesis of red blood cells; synthesis of niacin. Deficiency, Anemia; Kidney stones; Dermatitis; Spastic muscles, convulsions; Tongue abnormalities. Toxicity, Fluid retention; Depression, memory loss; Fatigue; Weakness. Row 9. Vitamin, Folate. Functions, Coenzyme in cellular synthesis. Deficiency, Anemia; Depression, mental confusion; Diarrhea, constipation; Infection susceptibility; Tongue abnormalities. Toxicity, Mask B12 deficiency. Row 10. Vitamin, B12 Cobalamine. Functions, Cellular synthesis; maintenance of nervous system function. Deficiency, Anemia; Fatigue; Paralysis; Skin abnormalities; Tongue abnormalities. Toxicity, None documented. Row 11. Vitamin, Pantothenic Acid. Functions, Coenzyme in energy metabolism. Deficiency, Fatigue; Insomnia; Vomiting, other intestinal problems. Toxicity, Fluid retention. Row 12. Vitamin, Biotin. Functions, Coenzyme in energy metabolism; synthesis of fat and glycogen. Deficiency, Alopecia; Anorexia; Depression; Fatigue; Heartbeat

abnormalities; Nausea; Skin rash. Toxicity, None documented. Row 13. Vitamin, Choline. Functions, Coenzyme in energy metabolism; synthesis of phospholipids and neurotransmitters. Deficiency, Growth failure; Kidney failure; Liver dysfunction (fat accumulation); Memory abnormalities. Toxicity, Fishy body odor; Low blood pressure. Row 14. Vitamin, C Ascorbic Acid. Functions, Collagen synthesis; antioxidant; immune function; enhancement of iron absorption; synthesis of thyroid hormone; protein metabolism. Deficiency, Anemia; Bleeding gums, loose teeth; Bone fracture susceptibility; Depression; Infection susceptibility. Toxicity, Abdominal cramps, diarrhea; Headache; Nausea; Skin rash. Row 15. Vitamin, C Ascorbic Acid. Functions, Blank. Deficiency, Joint pain; Muscle pain and wasting; Skin problems; Wound healing delayed. Toxicity, Interferes with interpretation of some laboratory values.

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A table is titled B. Vitamin C. It has the headers: Food, Portion Size, Vitamin C (mg), and % Daily Value (DV). Row 1. Food, Papaya. Portion Size, 1, whole fresh. Vitamin C (mg), 188. % Daily Value (DV), 313. Row 2. Food, Orange juice. Portion Size, 1 cup, fresh. Vitamin C (mg), 124. % Daily Value (DV), 207. Row 3. Food, Brussel sprouts. Portion Size, 1 cup, cooked. Vitamin C (mg), 96. % Daily Value (DV), 160. Row 4.

Food, Grapefruit juice. Portion Size, 1 cup, fresh. Vitamin C (mg), 94. % Daily Value (DV), 157. Row 5. Food, Green pepper. Portion Size, 1, whole. Vitamin C (mg), 90. % Daily Value (DV), 150. Row 6. Food, Strawberries. Portion Size, 1 cup, fresh. Vitamin C (mg), 85. % Daily Value (DV), 142. Row 7. Food, Orange. Portion Size, 1, medium fresh. Vitamin C (mg), 80. % Daily Value (DV), 133. Row 8. Food, Broccoli. Portion Size, 1 cup, cooked. Vitamin C (mg), 74. % Daily Value (DV), 123. Row 9. Food, Cauliflower. Portion Size, 1 cup, cooked. Vitamin C (mg), 72. % Daily Value (DV), 120. Row 10. Food, Cantaloupe. Portion Size, 1 cup, fresh. Vitamin C (mg), 68. % Daily Value (DV), 113. Row 11. Food, Mango. Portion Size, 1, fresh. Vitamin C (mg), 57. % Daily Value (DV), 95. Row 12. Food, Pink grapefruit. Portion Size, ½, fresh. Vitamin C (mg), 47. % Daily Value (DV), 78. Row 13. Food, Honeydew melon. Portion Size, 1 cup, fresh. Vitamin C (mg), 42. % Daily Value (DV), 70. Row 14. Food, Turnip greens. Portion Size, 1 cup, cooked. Vitamin C (mg), 40. % Daily Value (DV), 67. Row 15. Food, Parsley. Portion Size, ½ cup, chopped. Vitamin C (mg), 40. % Daily Value (DV), 67. Row 16. Food, Mustard greens. Portion Size, 1 cup, cooked. Vitamin C (mg), 36. % Daily Value (DV), 60. Row 17. Food, Tomatoes. Portion Size, 1, whole canned. Vitamin C (mg), 36. % Daily Value (DV), 60. Row 18. Food, Cabbage. Portion Size, 1 cup, raw. Vitamin C (mg),

34. % Daily Value (DV), 57. Row 19. Food, Sauerkraut. Portion Size, 1 cup, canned. Vitamin C (mg), 34. % Daily Value (DV), 57. Row 20. Food, Tomato juice. Portion Size, 6 oz, canned. Vitamin C (mg), 33. % Daily Value (DV), 55. Row 21. Food, Raspberries. Portion Size, 1 cup, fresh. Vitamin C (mg), 31. % Daily Value (DV), 52. Row 22. Food, Butternut squash. Portion Size, 1 cup, boiled. Vitamin C (mg), 30. % Daily Value (DV), 50. Row 23. Food, Sweet potato. Portion Size, 1, baked w/skin. Vitamin C (mg), 28. % Daily Value (DV), 47. Row 24. Food, Baked potato. Portion Size, 1, whole. Vitamin C (mg), 26. % Daily Value (DV), 43. Row 25. Food, Pineapple chunks. Portion Size, 1 cup, fresh. Vitamin C (mg), 24. % Daily Value (DV), 40. Row 26. Food, Asparagus. Portion Size, 1 cup, cooked. Vitamin C (mg), 20. % Daily Value (DV), 33. Row 27. Food, Watermelon. Portion Size, 1 cup, fresh. Vitamin C (mg), 15. % Daily Value (DV), 25. Row 28. Food, Apple. Portion Size, 1, medium fresh. Vitamin C (mg), 8. % Daily Value (DV), 13. Row 29. Food, Milk, 2%. Portion Size, 8 oz. Vitamin C (mg), 2. % Daily Value (DV), 3. Row 30 has the following text Quick Reference Diet Integration: Snacks: orange, tomato, and grapefruit juices; Entrée: fried broccoli, Brussel sprouts, green pepper, and cauliflower; Mashed, baked, or boiled potatoes; Fruit salad of strawberries, papaya, mango, watermelon in place of sweets; Kabob of green

peppers, cherry tomatoes, strawberries, and pineapples.

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A table is titled C. Riboflavin. It has the headers: Food, Portion Size, Riboflavin, and % Dietary Reference Intake (DRI), it is subdivided into Women and Men. Row 1. Food, Brewer’s yeast. Portion Size, 1 Tbsp. Riboflavin, 1.21. % Dietary Reference Intake (DRI), Women: 110. Men: 93. Row 2. Food, Yogurt. Portion Size, 1 cup, low fat. Riboflavin, 0.51. % Dietary Reference Intake (DRI), Women: 46. Men: 39. Row 3. Food, Mushrooms. Portion Size, 1 cup, cooked. Riboflavin, 0.46. % Dietary Reference Intake (DRI), Women: 42. Men: 35. Row 4. Food, Ricotta cheese. Portion Size, 1 cup, part skim. Riboflavin, 0.46. % Dietary Reference Intake (DRI), Women: 42. Men: 35. Row 5. Food, Corn flakes. Portion Size, 1 cup. Riboflavin, 0.43. % Dietary Reference Intake (DRI), Women: 39. Men: 33. Row 6. Food, Cottage cheese. Portion Size, 1 cup, low fat. Riboflavin, 0.42. % Dietary Reference Intake (DRI), Women: 38. Men: 32. Row 7. Food, Milk, 2%. Portion Size, 8 oz. Riboflavin, 0.4. % Dietary Reference Intake (DRI), Women: 36. Men: 31. Row 8. Food, Buttermilk. Portion Size, 1 cup. Riboflavin, 0.38. % Dietary Reference Intake (DRI), Women: 35. Men: 29. Row 9. Food, Sirloin steak. Portion Size, 3.5 oz, broiled. Riboflavin, 0.29. %

Dietary Reference Intake (DRI), Women: 26. Men: 22. Row 10. Food, Peach halves. Portion Size, 10, dried. Riboflavin, 0.28. % Dietary Reference Intake (DRI), Women: 25. Men: 22. Row 11. Food, Pork chop. Portion Size, 3.5 oz, roasted. Riboflavin, 0.26. % Dietary Reference Intake (DRI), Women: 24. Men: 20. Row 12. Food, Ground beef. Portion Size, 3.5 oz, lean baked. Riboflavin, 0.24. % Dietary Reference Intake (DRI), Women: 22. Men: 18. Row 13. Food, Black-eyed peas. Portion Size, 1 cup, cooked. Riboflavin, 0.24. % Dietary Reference Intake (DRI), Women: 22. Men: 18. Row 14. Food, Kidney beans. Portion Size, 1 cup, canned. Riboflavin, 0.23. % Dietary Reference Intake (DRI), Women: 21. Men: 18. Row 15. Food, Asparagus. Portion Size, 1 cup, cooked. Riboflavin, 0.22. % Dietary Reference Intake (DRI), Women: 20. Men: 17. Row 16. Food, Almonds. Portion Size, 1 oz, whole dried. Riboflavin, 0.22. % Dietary Reference Intake (DRI), Women: 20. Men: 17. Row 17. Food, Oysters. Portion Size, 3 oz, raw. Riboflavin, 0.2. % Dietary Reference Intake (DRI), Women: 18. Men: 15. Row 18. Food, Ham. Portion Size, 3.5 oz, cooked. Riboflavin, 0.19. % Dietary Reference Intake (DRI), Women: 17. Men: 15. Row 19. Food, Turkey. Portion Size, 3.5 oz, w/o skin. Riboflavin, 0.18. % Dietary Reference Intake (DRI), Women: 16. Men: 14. Row 20. Food, Broccoli. Portion Size, 1 cup, cooked. Riboflavin, 0.16. % Dietary Reference Intake (DRI), Women: 15. Men: 12.

Row 21. Food, Green beans. Portion Size, 1 cup, cooked. Riboflavin, 0.14. % Dietary Reference Intake (DRI), Women: 13. Men: 11. Row 22. Food, Cheddar cheese. Portion Size, 1 oz. Riboflavin, 0.11. % Dietary Reference Intake (DRI), Women: 10. Men: 8. Row 23. Food, Spinach. Portion Size, 1 cup, cooked. Riboflavin, 0.1. % Dietary Reference Intake (DRI), Women: 9. Men: 8. Row 24. Food, Strawberries. Portion Size, 1 cup, fresh. Riboflavin, 0.1. % Dietary Reference Intake (DRI), Women: 9. Men: 8. Row 25. Food, Chicken breast. Portion Size, ½ breast, roasted. Riboflavin, 0.1. % Dietary Reference Intake (DRI), Women: 9. Men: 8. Row 26. Food, Sole/flounder. Portion Size, 3 oz baked. Riboflavin, 0.07. % Dietary Reference Intake (DRI), Women: 6. Men: 5. Row 27. Food, Orange. Portion Size, 1, medium fresh. Riboflavin, 0.06. % Dietary Reference Intake (DRI), Women: 5. Men: 5. Row 28. Food, Bread. Portion Size, Whole wheat, 1 slice. Riboflavin, 0.05. % Dietary Reference Intake (DRI), Women: 5. Men: 4. Row 29. Food, Bean sprouts. Portion Size, 1 cup, stir fried. Riboflavin, 0.04. % Dietary Reference Intake (DRI), Women: 4. Men: 3. Row 30. Food, Cantaloupe. Portion Size, 1 cup, fresh. Riboflavin, 0.03. % Dietary Reference Intake (DRI), Women: 3. Men: 2. Row 31. Food, Apple. Portion Size, 1, medium fresh. Riboflavin, 0.02. % Dietary Reference Intake (DRI), Women: 2. Men: 2. Row 32 has the following text Quick Reference Diet

Integration: A bowl of corn flakes with 8 oz milk provides 75% of DRI for women.; Yogurt as a snack and as a dip for fruits and vegetables.; Mushrooms on pizza, salads, or stir fries.; Low-fat cottage or ricotta cheeses in lasagna, ravioli, or other main dish.

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A table is titled D. Vitamin B6. It has the headers: Food, Portion Size, B6 (mg), and % DRI. Row 1. Food, Beef liver. Portion Size, 3.5 oz, braised. B6 (mg), 0.91. % DRI, 70. Row 2. Food, Baked potato. Portion Size, 1, whole. B6 (mg), 0.7. % DRI, 54. Row 3. Food, Salmon. Portion Size, 3 oz, cooked. B6 (mg), 0.7. % DRI, 54. Row 4. Food, Banana. Portion Size, 1, peeled. B6 (mg), 0.66. % DRI, 51. Row 5. Food, Chicken breast. Portion Size, ½ breast, w/o skin. B6 (mg), 0.51. % DRI, 39. Row 6. Food, Corn flakes. Portion Size, 1 cup. B6 (mg), 0.5. % DRI, 38. Row 7. Food, Avocado. Portion Size, ½ average. B6 (mg), 0.48. % DRI, 37. Row 8. Food, Trout. Portion Size, 3 oz, broiled. B6 (mg), 0.46. % DRI, 35. Row 9. Food, Turkey. Portion Size, 3.5 oz, w/o skin. B6 (mg), 0.46. % DRI, 35. Row 10. Food, Brewer’s yeast. Portion Size, 1 oz. B6 (mg), 0.45. % DRI, 35. Row 11. Food, Sirloin steak. Portion Size, 3.5 oz, broiled. B6 (mg), 0.45. % DRI, 35. Row 12. Food, Pork chop. Portion Size, 3.5 oz, roasted. B6 (mg), 0.45. % DRI, 35. Row 13. Food, Spinach. Portion Size, 1 cup, cooked. B6 (mg), 0.44. % DRI, 34. Row

14. Food, Soybeans. Portion Size, 1 cup, cooked. B6 (mg), 0.4. % DRI, 31. Row 15. Food, Wheat germ. Portion Size, ¼ cup. B6 (mg), 0.38. % DRI, 29. Row 16. Food, Tuna, in water. Portion Size, 3 oz, canned. B6 (mg), 0.3. % DRI, 23. Row 17. Food, Navy beans. Portion Size, 1 cup, cooked. B6 (mg), 0.3. % DRI, 23. Row 18. Food, Sunflower seeds. Portion Size, ¼ cup, dry. B6 (mg), 0.3. % DRI, 23. Row 19. Food, Turnip greens. Portion Size, 1 cup, cooked. B6 (mg), 0.26. % DRI, 20. Row 20. Food, Cauliflower. Portion Size, 1 cup, cooked. B6 (mg), 0.26. % DRI, 20. Row 21. Food, Broccoli. Portion Size, 1 cup, cooked. B6 (mg), 0.25. % DRI, 18. Row 22. Food, Green pepper. Portion Size, 1, whole. B6 (mg), 0.24. % DRI, 18. Row 23. Food, Watermelon. Portion Size, 1 cup, fresh. B6 (mg), 0.23. % DRI, 18. Row 24. Food, Ground beef. Portion Size, 3.5 oz, lean baked. B6 (mg), 0.22. % DRI, 17. Row 25. Food, Asparagus. Portion Size, 1 cup, cooked. B6 (mg), 0.22. % DRI, 17. Row 26. Food, Figs. Portion Size, 5, dried. B6 (mg), 0.21. % DRI, 16. Row 27. Food, Cantaloupe. Portion Size, 1 cup, fresh. B6 (mg), 0.18. % DRI, 14. Row 28. Food, Sole/flounder. Portion Size, 3 oz, cooked. B6 (mg), 0.18. % DRI, 14. Row 29. Food, Mustard greens. Portion Size, 1 cup, cooked. B6 (mg), 0.16. % DRI, 12. Row 30. Food, Zucchini. Portion Size, 1 cup, cooked. B6 (mg), 0.14. % DRI, 11. Row 31. Food, Milk. Portion Size, 2%, 8 oz. B6 (mg), 0.11. % DRI, 8. Row 32 has the following text Quick

Reference Diet Integration: Baked potato for lunch with fat-free sour cream or dressing.; Banana with corn flakes for breakfast.; Poach, grill, or broil salmon for a quick meal.

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A table is titled E. Vitamin B12. It has the headers: Food, Portion Size, B12 (µg), and % DRI. Row 1. Food, Clams. Portion Size, 3 oz, cooked. B12 (µg), 84.1. % DRI, 3,500. Row 2. Food, Beef liver. Portion Size, 3.5 oz, braised. B12 (µg), 71. % DRI, 3,000. Row 3. Food, Oysters. Portion Size, 3 oz, cooked. B12 (µg), 32.5. % DRI, 1,350. Row 4. Food, Clam chowder. Portion Size, 1 cup. B12 (µg), 10.3. % DRI, 427. Row 5. Food, Rabbit. Portion Size, 3.5 oz, roasted. B12 (µg), 8.3. % DRI, 346. Row 6. Food, Braunschweiger. Portion Size, 1 oz, tube type. B12 (µg), 5.2. % DRI, 216. Row 7. Food, Sirloin steak. Portion Size, 3.5 oz, broiled. B12 (µg), 2.9. % DRI, 119. Row 8. Food, Salmon. Portion Size, 3 oz, cooked. B12 (µg), 2.7. % DRI, 113. Row 9. Food, Tuna, in water. Portion Size, 3 oz, canned. B12 (µg), 2.5. % DRI, 106. Row 10. Food, Lamb chop. Portion Size, 3.5 oz, braised. B12 (µg), 2.3. % DRI, 95. Row 11. Food, Vegetarian burger. Portion Size, ½ cup. B12 (µg), 2. % DRI, 83. Row 12. Food, Raisin bran. Portion Size, ¾ cup. B12 (µg), 2. % DRI, 83. Row 13. Food, Ground beef. Portion Size, 3.5 oz, lean baked. B12 (µg), 1.7. % DRI, 71. Row 14. Food, Cottage

cheese. Portion Size, 1 cup, 1% fat. B12 (µg), 1.4. % DRI, 58. Row 15. Food, Sole/flounder. Portion Size, 3 oz, cooked. B12 (µg), 1.3. % DRI, 54. Row 16. Food, Shrimp. Portion Size, 3 oz, cooked. B12 (µg), 1.3. % DRI, 54. Row 17. Food, Halibut. Portion Size, 3 oz, broiled. B12 (µg), 1.2. % DRI, 50. Row 18. Food, Milk. Portion Size, 2%, 8 oz. B12 (µg), 0.89. % DRI, 37. Row 19. Food, Frankfurter. Portion Size, 1 beef, 2 oz. B12 (µg), 0.88. % DRI, 37. Row 20. Food, Ham. Portion Size, 3.5 oz, cooked. B12 (µg), 0.74. % DRI, 29. Row 21. Food, Tempeh. Portion Size, ½ cup. B12 (µg), 0.7. % DRI, 29. Row 22. Food, Pork chop. Portion Size, 3.5 oz, roasted. B12 (µg), 0.6. % DRI, 25. Row 23. Food, Buttermilk. Portion Size, 8 oz, cultured. B12 (µg), 0.54. % DRI, 23. Row 24. Food, Egg. Portion Size, 1, whole fresh. B12 (µg), 0.5. % DRI, 21. Row 25. Food, Turkey. Portion Size, 3.5 oz, w/o skin. B12 (µg), 0.37. % DRI, 15. Row 26. Food, Canadian bacon. Portion Size, 2 slices, cooked. B12 (µg), 0.36. % DRI, 15. Row 27. Food, Miso. Portion Size, ½ cup. B12 (µg), 0.29. % DRI, 12. Row 28. Food, Chicken breast. Portion Size, ½ breast, w/o skin. B12 (µg), 0.29. % DRI, 12. Row 29. Food, Yogurt. Portion Size, 8 oz, low fat. B12 (µg), 0.24. % DRI, 10. Row 30. Food, Cheddar cheese. Portion Size, 1 oz. B12 (µg), 0.23. % DRI, 10. Row 31. Food, Goat’s milk. Portion Size, 8 oz. B12 (µg), 0.16. % DRI, 7. Row 32 has the following text Quick Reference Diet Integration: Raisin bran with low-fat

milk for breakfast.; Low-fat cottage cheese with fruit as a snack.; 3 oz of salmon, tuna, or rabbit provides 100% DRI of B12.; Oysters or clams served steamed or in stews and chowders.

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A table is titled F. Biotin. It has the headers: Food, Portion Size, Biotin (µg), and % DRI. Row 1. Food, Egg. Portion Size, 1, whole fresh. Biotin (µg), 10. % DRI, 33. Row 2. Food, Wheat germ. Portion Size, ¼ cup, toasted. Biotin (µg), 7. % DRI, 23. Row 3. Food, Ry-Krisp. Portion Size, ½ oz. Biotin (µg), 6.8. % DRI, 23. Row 4. Food, Granola. Portion Size, ¼ cup w/ raisins. Biotin (µg), 5. % DRI, 17. Row 5. Food, Egg noodles. Portion Size, 1 cup, cooked. Biotin (µg), 4. % DRI, 13. Row 6. Food, Almonds. Portion Size, 1 oz, whole natural. Biotin (µg), 1. % DRI, 3. Row 7. Food, Pistachios. Portion Size, 1 oz, red. Biotin (µg), 1. % DRI, 3. Row 8. Food, Corn meal. Portion Size, 1 oz, yellow. Biotin (µg), 1. % DRI, 3. Row 9 has the following text Quick Reference Diet Integration: Granola as a cereal or snack, or mixed into yogurt; Wheat germ in casseroles and breads or on cereal; Ry-Krisp with peanut butter and jelly for a snack; Egg noodles with veggies, fat-free Italian dressing; Scrambled, poached, or hardboiled eggs.

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A table is titled G. Choline. It has the headers: Food, Portion Size, Choline (µg), and % DRI, it is subdivided into Women and Men. Row 1. Food, Beef liver. Portion Size, 3.5 oz. Choline (µg), 581.1. % DRI, Women: 137. Men: 106. Row 2. Food, Cauliflower. Portion Size, 1 cup, cooked. Choline (µg), 162. % DRI, Women: 38. Men: 29. Row 3. Food, Peanuts. Portion Size, 1 oz, dried. Choline (µg), 127.3. % DRI, Women: 30. Men: 23. Row 4. Food, Peanut butter. Portion Size, 2 Tbsp. Choline (µg), 124.6. % DRI, Women: 29. Men: 23. Row 5. Food, Grape juice. Portion Size, 8 oz, canned. Choline (µg), 120. % DRI, Women: 28. Men: 22. Row 6. Food, Potato. Portion Size, 1, whole baked. Choline (µg), 103.2. % DRI, Women: 24. Men: 19. Row 7. Food, Iceberg lettuce. Portion Size, 1 leaf, raw. Choline (µg), 58.6. % DRI, Women: 14. Men: 11. Row 8. Food, Tomato. Portion Size, 1, whole raw. Choline (µg), 52.9. % DRI, Women: 12. Men: 10. Row 9. Food, Milk. Portion Size, Whole 8 oz. Choline (µg), 36.6. % DRI, Women: 9. Men: 7. Row 10. Food, Orange. Portion Size, 1, fresh medium. Choline (µg), 28. % DRI, Women: 7. Men: 5. Row 11. Food, Banana. Portion Size, 1, whole peeled. Choline (µg), 27.4. % DRI, Women: 6. Men: 5. Row 12. Food, Bread. Portion Size, Whole wheat; 1 slice. Choline (µg), 24.2. % DRI, Women: 6. Men: 4. Row 13. Food, Cucumber. Portion Size, ½ cup, raw. Choline (µg), 11.3. % DRI, Women: 3. Men: 2. Row 14. Food, Beef steak. Portion Size, 3.5

oz. Choline (µg), 7.5. % DRI, Women: 2. Men: 1. Row 15. Food, Apple. Portion Size, 1, fresh medium. Choline (µg), 3.7. % DRI, Women: <1. Men: <1. Row 16. Food, Egg. Portion Size, 1, fresh whole. Choline (µg), 2.1. % DRI, Women: <1. Men: <1. Row 17. Food, Ginger ale. Portion Size, 12 oz. Choline (µg), 0.73. % DRI, Women: <1. Men: <1. Row 18. Food, Butter. Portion Size, 1 tsp. Choline (µg), 0.21. % DRI, Women: <1. Men: <1. Row 19. Food, Margarine. Portion Size, 1 tsp, tub type. Choline (µg), 0.15. % DRI, Women: <1. Men: <1. Row 20. Food, Corn oil. Portion Size, 1 Tbsp. Choline (µg), 0.04. % DRI, Women: <1. Men: <1. Row 21 has the following text Quick Reference Diet Integration Guide: Dinner: 3.5 oz beef liver, 1 cup cauliflower, baked potato, 8 oz grape juice, banana.; Add iceberg lettuce to a sandwich.; Swap straight soda for a “Choline Cocktail”—8 oz grape juice, 12 oz ginger ale.; Snack: 1 oz peanuts and 8 oz milk.; Lunch: peanut butter and jelly sandwich with 8 oz grape juice.

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A table is titled H. Folate. It has the headers: Food, Portion Size, Folate (µg), and % DRI. Row 1. Food, Pinto beans. Portion Size, 1 cup, cooked. Folate (µg), 294. % DRI, 74. Row 2. Food, Asparagus. Portion Size, 1 cup, cooked. Folate (µg), 264. % DRI, 66. Row 3. Food, Spinach. Portion Size, 1 cup, cooked. Folate

(µg), 262. % DRI, 66. Row 4. Food, Navy beans. Portion Size, 1 cup, cooked. Folate (µg), 255. % DRI, 64. Row 5. Food, Beef liver. Portion Size, 3.5 oz, braised. Folate (µg), 217. % DRI, 54. Row 6. Food, Black-eyed peas. Portion Size, 1 cup, cooked. Folate (µg), 209. % DRI, 52. Row 7. Food, Great northern beans. Portion Size, 1 cup, cooked. Folate (µg), 181. % DRI, 45. Row 8. Food, Turnip greens. Portion Size, 1 cup, cooked. Folate (µg), 170. % DRI, 43. Row 9. Food, Lima beans. Portion Size, 1 cup, cooked. Folate (µg), 156. % DRI, 39. Row 10. Food, Kidney beans. Portion Size, 1 cup, canned. Folate (µg), 129. % DRI, 32. Row 11. Food, Broccoli. Portion Size, 1 cup, cooked. Folate (µg), 104. % DRI, 26. Row 12. Food, Corn flakes. Portion Size, 1 cup. Folate (µg), 100. % DRI, 25. Row 13. Food, Parsley. Portion Size, 1 cup, chopped. Folate (µg), 92. % DRI, 23. Row 14. Food, Beets. Portion Size, 1 cup, cooked. Folate (µg), 90. % DRI, 23. Row 15. Food, Wheat germ. Portion Size, ¼ cup. Folate (µg), 82. % DRI, 21. Row 16. Food, Romaine lettuce. Portion Size, 1 cup, chopped. Folate (µg), 76. % DRI, 19. Row 17. Food, Cauliflower. Portion Size, 1 cup, cooked. Folate (µg), 64. % DRI, 16. Row 18. Food, Pineapple juice. Portion Size, 8 oz, canned. Folate (µg), 58. % DRI, 15. Row 19. Food, Orange. Portion Size, 1, fresh medium. Folate (µg), 47. % DRI, 12. Row 20. Food, Zucchini. Portion Size, 1 cup, cooked. Folate (µg), 30. % DRI, 8. Row 21. Food,

Peanuts. Portion Size, 1 oz, dried. Folate (µg), 29. % DRI, 7. Row 22. Food, Cantaloupe. Portion Size, 1 cup, fresh. Folate (µg), 27. % DRI, 7. Row 23. Food, Winter squash. Portion Size, 1 cup, cooked. Folate (µg), 26. % DRI, 7. Row 24. Food, Strawberries. Portion Size, 1 cup, fresh. Folate (µg), 26. % DRI, 7. Row 25. Food, Grapefruit juice. Portion Size, 1 cup, canned. Folate (µg), 26. % DRI, 7. Row 26. Food, Egg. Portion Size, 1, whole fresh. Folate (µg), 23. % DRI, 6. Row 27. Food, Green beans. Portion Size, 1 cup, cooked. Folate (µg), 22. % DRI, 6. Row 28. Food, Tofu. Portion Size, ½ cup, raw. Folate (µg), 19. % DRI, 5. Row 29. Food, Tomato. Portion Size, 1, whole raw. Folate (µg), 18. % DRI, 5. Row 30. Food, Bread, whole wheat. Portion Size, 1 slice. Folate (µg), 14. % DRI, 4. Row 31 has the following text Quick Reference Diet Integration: Use dry beans in soups or as a main dish salad.; Add spinach to lasagna or serve cooked with lemon juice.; Salad of spinach, romaine, tomato, broccoli, cauliflower, and beets.; Dinner: 3.5 oz beef liver, 1 cup asparagus, 1 cup lima beans.; Breakfast: cornflakes in milk; fruit salad of cantaloupe, strawberries, orange.

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A table is titled I. Niacin. It has the headers: Food, Portion Size, Niacin (mg), and % DRI, it is subdivided into Women and Men. Row 1. Food, Chicken breast.

Portion Size, ½ roasted w/o skin. Niacin (mg), 11.8. % DRI, Women: 84. Men: 74. Row 2. Food, Tuna, in water. Portion Size, 3 oz, canned. Niacin (mg), 11.3. % DRI, Women: 81. Men: 71. Row 3. Food, Beef liver. Portion Size, 3.5 oz, braised. Niacin (mg), 10.7. % DRI, Women: 76. Men: 67. Row 4. Food, Brewer’s yeast. Portion Size, 1 oz. Niacin (mg), 10.7. % DRI, Women: 76. Men: 67. Row 5. Food, Salmon. Portion Size, 3 oz, broiled. Niacin (mg), 8.6. % DRI, Women: 61. Men: 54. Row 6. Food, Mushrooms. Portion Size, 1 cup, cooked. Niacin (mg), 7. % DRI, Women: 50. Men: 44. Row 7. Food, Halibut. Portion Size, 3 oz, broiled. Niacin (mg), 6.1. % DRI, Women: 44. Men: 38. Row 8. Food, Peach halves. Portion Size, 10, dried. Niacin (mg), 5.7. % DRI, Women: 41. Men: 36. Row 9. Food, Pink salmon. Portion Size, 3 oz, canned. Niacin (mg), 5.6. % DRI, Women: 40. Men: 35. Row 10. Food, Pork chop. Portion Size, 3.5 oz, roasted. Niacin (mg), 5.5. % DRI, Women: 39. Men: 34. Row 11. Food, Lamb chop. Portion Size, 3.5 oz, braised. Niacin (mg), 5.5. % DRI, Women: 39. Men: 34. Row 12. Food, Turkey. Portion Size, 3.5 oz w/o skin. Niacin (mg), 5.4. % DRI, Women: 39. Men: 34. Row 13. Food, Sirloin steak. Portion Size, 3.5 oz, broiled. Niacin (mg), 4.3. % DRI, Women: 31. Men: 27. Row 14. Food, Ground beef. Portion Size, 3.5 oz, lean baked. Niacin (mg), 4.2. % DRI, Women: 30. Men: 26. Row 15. Food, Peanuts. Portion Size, 1 oz, dried unsalted. Niacin (mg), 4. %

DRI, Women: 29. Men: 25. Row 16. Food, Baked potato. Portion Size, 1, whole. Niacin (mg), 3.3. % DRI, Women: 24. Men: 21. Row 17. Food, Sole/flounder. Portion Size, 3 oz, baked. Niacin (mg), 2.5. % DRI, Women: 18. Men: 16. Row 18. Food, Kidney beans. Portion Size, 1 cup, canned. Niacin (mg), 2.4. % DRI, Women: 17. Men: 15. Row 19. Food, Braunschweiger. Portion Size, 1 oz, tube type. Niacin (mg), 2.3. % DRI, Women: 16. Men: 14. Row 20. Food, Shrimp. Portion Size, 3 oz, boiled. Niacin (mg), 2.2. % DRI, Women: 16. Men: 14. Row 21. Food, Wheat bran. Portion Size, ¼ cup. Niacin (mg), 2. % DRI, Women: 14. Men: 13. Row 22. Food, Asparagus. Portion Size, 1 cup, cooked. Niacin (mg), 2. % DRI, Women: 14. Men: 13. Row 23. Food, Oysters. Portion Size, 3 oz, raw. Niacin (mg), 1.7. % DRI, Women: 12. Men: 11. Row 24. Food, Sardines. Portion Size, 2, sardines in oil. Niacin (mg), 1.3. % DRI, Women: 9. Men: 8. Row 25. Food, Crab meat. Portion Size, 3 oz, canned. Niacin (mg), 1.2. % DRI, Women: 9. Men: 8. Row 26. Food, Bread, whole wheat. Portion Size, 1 slice. Niacin (mg), 1. % DRI, Women: 8. Men: 6. Row 27. Food, Summer squash. Portion Size, 1 cup, cooked. Niacin (mg), 1. % DRI, Women: 8. Men: 6. Row 28. Food, Cantaloupe. Portion Size, 1 cup, fresh. Niacin (mg), 0.9. % DRI, Women: 6. Men: 6. Row 29. Food, Peach. Portion Size, 1, fresh medium. Niacin (mg), 0.9. % DRI, Women: 6. Men: 6. Row 30. Food, Spinach. Portion Size, 1 cup,

cooked. Niacin (mg), 0.9. % DRI, Women: 6. Men: 6. Row 31. Food, Broccoli. Portion Size, 1 cup, cooked. Niacin (mg), 0.8. % DRI, Women: 6. Men: 5. Row 32 has the following text Quick Reference Diet Integration: Choose tuna, halibut, and salmon instead of red meat.; Add mushrooms to soups and salads, and stir fry.; Dinner: half chicken breast, baked potato, 1 cup asparagus.; Fortified breakfast cereals contain 25% of the DRI for niacin per serving.

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A table is titled J. Pantothenic Acid. It has the headers: Food, Serving Size, Pantothenic Acid (mg), and % DRI. Row 1. Food, Beef liver. Serving Size, 3.5 oz, braised. Pantothenic Acid (mg), 4.6. % DRI, 91. Row 2. Food, Mushrooms. Serving Size, ½ cup, boiled. Pantothenic Acid (mg), 1.7. % DRI, 34. Row 3. Food, Avocado. Serving Size, 1, medium. Pantothenic Acid (mg), 1.7. % DRI, 34. Row 4. Food, Salmon. Serving Size, 3 oz, cooked. Pantothenic Acid (mg), 1.4. % DRI, 28. Row 5. Food, Lentils. Serving Size, 1 cup, boiled. Pantothenic Acid (mg), 1.3. % DRI, 25. Row 6. Food, Split peas. Serving Size, 1 cup, boiled. Pantothenic Acid (mg), 1.2. % DRI, 23. Row 7. Food, Potato. Serving Size, 1, whole baked. Pantothenic Acid (mg), 1.1. % DRI, 22. Row 8. Food, Turkey. Serving Size, 3.5 oz w/o skin. Pantothenic Acid (mg), 0.94. % DRI, 19. Row 9. Food, Pomegranate. Serving Size, 1, medium

raw. Pantothenic Acid (mg), 0.92. % DRI, 18. Row 10. Food, Chicken breast. Serving Size, ½, roasted w/o skin. Pantothenic Acid (mg), 0.83. % DRI, 17. Row 11. Food, Peanuts. Serving Size, 1 oz, dried. Pantothenic Acid (mg), 0.79. % DRI, 16. Row 12. Food, Milk, 2%. Serving Size, 8 oz. Pantothenic Acid (mg), 0.78. % DRI, 16. Row 13. Food, Sweet potato. Serving Size, 1, whole baked. Pantothenic Acid (mg), 0.74. % DRI, 15. Row 14. Food, Chickpeas. Serving Size, 1 cup, canned. Pantothenic Acid (mg), 0.72. % DRI, 14. Row 15. Food, Wheat germ. Serving Size, ¼ cup. Pantothenic Acid (mg), 0.66. % DRI, 13. Row 16. Food, Pork chop. Serving Size, 3.5 oz, roasted. Pantothenic Acid (mg), 0.65. % DRI, 13. Row 17. Food, Egg. Serving Size, 1, whole fresh. Pantothenic Acid (mg), 0.63. % DRI, 13. Row 18. Food, Broccoli. Serving Size, 1 cup, cooked. Pantothenic Acid (mg), 0.5. % DRI, 10. Row 19. Food, Ham. Serving Size, 3.5 oz, cooked. Pantothenic Acid (mg), 0.47. % DRI, 9. Row 20. Food, Sole/flounder. Serving Size, 3 oz, cooked. Pantothenic Acid (mg), 0.43. % DRI, 9. Row 21. Food, Kidney beans. Serving Size, 1 cup, canned. Pantothenic Acid (mg), 0.38. % DRI, 8. Row 22. Food, Sirloin steak. Serving Size, 3.5 oz, broiled. Pantothenic Acid (mg), 0.37. % DRI, 7. Row 23. Food, Corn. Serving Size, 1 cup, boiled. Pantothenic Acid (mg), 0.36. % DRI, 7. Row 24. Food, Orange. Serving Size, 1, fresh medium. Pantothenic Acid (mg), 0.35. %

DRI, 7. Row 25. Food, Watermelon. Serving Size, 1 cup, fresh. Pantothenic Acid (mg), 0.34. % DRI, 7. Row 26. Food, Ground beef. Serving Size, 3.5 oz, baked lean. Pantothenic Acid (mg), 0.27. % DRI, 5. Row 27. Food, Spinach. Serving Size, 1 cup, cooked. Pantothenic Acid (mg), 0.26. % DRI, 5. Row 28. Food, Bread, whole wheat. Serving Size, 1 slice. Pantothenic Acid (mg), 0.18. % DRI, 4. Row 29. Food, Tuna, in water. Serving Size, 3 oz, canned. Pantothenic Acid (mg), 0.18. % DRI, 4. Row 30. Food, Macaroni. Serving Size, 1 cup, cooked, enriched. Pantothenic Acid (mg), 0.16. % DRI, 3. Row 31. Food, Cheddar cheese. Serving Size, 1 oz. Pantothenic Acid (mg), 0.12. % DRI, 2. Row 32 has the following text Quick Reference Diet Integration: Avocado sliced in a salad or in a dip (guacamole); Dinner: 3 oz salmon, 1 baked potato, 8 oz milk, 1 cup broccoli; Soups with lentils or split peas; Salad: chicken breast, avocado, spinach, mushrooms, broccoli.

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A table is titled K. Thiamin. It has the headers: Food, Portion Size, Thiamin (mg), and % DRI, it is subdivided into Women and Men. Row 1. Food, Brewer’s yeast. Portion Size, 1 oz. Thiamin (mg), 4.43. % DRI, Women: 403. Men: 369. Row 2. Food, Pork chop. Portion Size, 3.5 oz, roasted. Thiamin (mg), 0.91. % DRI, Women: 83. Men: 76. Row 3. Food,

Ham. Portion Size, 3.5 oz, cooked. Thiamin (mg), 0.78. % DRI, Women: 71. Men: 65. Row 4. Food, Green peas. Portion Size, 1 cup, cooked. Thiamin (mg), 0.42. % DRI, Women: 38. Men: 35. Row 5. Food, Canadian bacon. Portion Size, 2 pieces, cooked. Thiamin (mg), 0.38. % DRI, Women: 35. Men: 32. Row 6. Food, Corn flakes. Portion Size, 1 cup. Thiamin (mg), 0.38. % DRI, Women: 35. Men: 32. Row 7. Food, Split peas. Portion Size, 1 cup, cooked. Thiamin (mg), 0.37. % DRI, Women: 34. Men: 31. Row 8. Food, Macaroni, enriched. Portion Size, 1 cup, cooked. Thiamin (mg), 0.29. % DRI, Women: 26. Men: 24. Row 9. Food, Kidney beans. Portion Size, 1 cup, canned. Thiamin (mg), 0.27. % DRI, Women: 25. Men: 23. Row 10. Food, Oatmeal. Portion Size, 1 cup, cooked. Thiamin (mg), 0.26. % DRI, Women: 24. Men: 22. Row 11. Food, Millet. Portion Size, 1 cup, cooked. Thiamin (mg), 0.25. % DRI, Women: 23. Men: 21. Row 12. Food, Acorn squash. Portion Size, 1 cup, boiled. Thiamin (mg), 0.24. % DRI, Women: 22. Men: 20. Row 13. Food, Baked potato. Portion Size, 1, whole. Thiamin (mg), 0.22. % DRI, Women: 20. Men: 18. Row 14. Food, Asparagus. Portion Size, 1 cup, cooked. Thiamin (mg), 0.22. % DRI, Women: 20. Men: 18. Row 15. Food, Peanuts. Portion Size, 1 oz, dry, unsalted. Thiamin (mg), 0.19. % DRI, Women: 17. Men: 16. Row 16. Food, Black eyed peas. Portion Size, 1 cup, cooked. Thiamin (mg), 0.17. % DRI, Women: 15. Men:

14. Row 17. Food, Watermelon. Portion Size, 1 cup, fresh. Thiamin (mg), 0.13. % DRI, Women: 12. Men: 11. Row 18. Food, Sirloin steak. Portion Size, 3.5 oz, broiled. Thiamin (mg), 0.13. % DRI, Women: 12. Men: 11. Row 19. Food, Honeydew melon. Portion Size, 1 cup, fresh. Thiamin (mg), 0.13. % DRI, Women: 12. Men: 11. Row 20. Food, Orange. Portion Size, 1, fresh medium. Thiamin (mg), 0.12. % DRI, Women: 11. Men: 10. Row 21. Food, Tofu. Portion Size, ½ cup. Thiamin (mg), 0.1. % DRI, Women: 9. Men: 8. Row 22. Food, Milk, 2%. Portion Size, 8 oz. Thiamin (mg), 0.1. % DRI, Women: 9. Men: 8. Row 23. Food, Green beans. Portion Size, 1 cup, cooked. Thiamin (mg), 0.1. % DRI, Women: 9. Men: 8. Row 24. Food, Broccoli. Portion Size, 1 cup, cooked. Thiamin (mg), 0.1. % DRI, Women: 9. Men: 8. Row 25. Food, Bread, whole wheat. Portion Size, 1 slice. Thiamin (mg), 0.09. % DRI, Women: 8. Men: 8. Row 26. Food, Sole/flounder. Portion Size, 3 oz, cooked. Thiamin (mg), 0.08. % DRI, Women: 7. Men: 7. Row 27. Food, Cauliflower. Portion Size, 1 cup, cooked. Thiamin (mg), 0.08. % DRI, Women: 7. Men: 7. Row 28. Food, Tomato. Portion Size, 1, whole raw. Thiamin (mg), 0.07. % DRI, Women: 6. Men: 6. Row 29 has the following text Quick Reference Diet Integration: Include lean pork in weekly menus.; Use green peas, split peas, black peas, black-eyed peas, kidney beans in soups.; Choose enriched pasta and bread products.;

Breakfast: oatmeal or fortified cereal.; Side dish: boiled or baked squash.

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A table is titled L. Vitamin A. It has the headers: Food, Portion Size, Vitamin A (RE), and % DRI. Row 1. Food, Beef liver. Portion Size, 3 oz, fried. Vitamin A (RE), 9,123. % DRI, 1,042. Row 2. Food, Pumpkin. Portion Size, 1 cup, canned. Vitamin A (RE), 5,424. % DRI, 620. Row 3. Food, Sweet potato. Portion Size, 1, whole baked. Vitamin A (RE), 2,486. % DRI, 284. Row 4. Food, Carrot. Portion Size, 1, whole fresh. Vitamin A (RE), 2,024. % DRI, 231. Row 5. Food, Spinach. Portion Size, 1 cup, cooked. Vitamin A (RE), 1,474. % DRI, 168. Row 6. Food, Butternut squash. Portion Size, 1 cup, baked. Vitamin A (RE), 1,435. % DRI, 164. Row 7. Food, Mango. Portion Size, 1, medium fresh. Vitamin A (RE), 806. % DRI, 92. Row 8. Food, Papaya. Portion Size, 1, medium fresh. Vitamin A (RE), 612. % DRI, 70. Row 9. Food, Cantaloupe. Portion Size, 1 cup, fresh. Vitamin A (RE), 516. % DRI, 59. Row 10. Food, Turnip greens. Portion Size, ½ cup, chopped. Vitamin A (RE), 396. % DRI, 45. Row 11. Food, Collard greens. Portion Size, 1 cup, chopped. Vitamin A (RE), 349. % DRI, 40. Row 12. Food, Apricot halves. Portion Size, 10 halves. Vitamin A (RE), 253. % DRI, 29. Row 13. Food, Winter squash. Portion Size, ½ cup, cubes. Vitamin A (RE), 235. % DRI, 27. Row 14. Food,

Mustard greens. Portion Size, ½ cup, chopped. Vitamin A (RE), 212. % DRI, 24. Row 15. Food, Broccoli. Portion Size, ½ cup, frozen. Vitamin A (RE), 174. % DRI, 20. Row 16. Food, Parsley. Portion Size, ½ cup, chopped. Vitamin A (RE), 156. % DRI, 18. Row 17. Food, Milk, 2%. Portion Size, 8 oz. Vitamin A (RE), 140. % DRI, 16. Row 18. Food, Egg. Portion Size, 1, whole fresh. Vitamin A (RE), 95. % DRI, 11. Row 19. Food, Cheddar cheese. Portion Size, 1 oz. Vitamin A (RE), 86. % DRI, 10. Row 20. Food, Watermelon. Portion Size, 1 cup, fresh. Vitamin A (RE), 58. % DRI, 7. Row 21. Food, Margarine. Portion Size, 1 tsp, tub-type. Vitamin A (RE), 47. % DRI, 5. Row 22. Food, Sole/flounder. Portion Size, 3 oz, cooked. Vitamin A (RE), 28. % DRI, 3. Row 23. Food, Orange. Portion Size, 1, medium fresh. Vitamin A (RE), 26. % DRI, 3. Row 24. Food, Green beans. Portion Size, ½ cup, canned. Vitamin A (RE), 24. % DRI, 3. Row 25. Food, Corn. Portion Size, ½ cup, canned. Vitamin A (RE), 13. % DRI, 2. Row 26. Food, Apple. Portion Size, 1, medium fresh. Vitamin A (RE), 7. % DRI, 1. Row 27. Food, Chicken breast. Portion Size, ½, roasted w/o skin. Vitamin A (RE), 5. % DRI, 0.5. Row 28. Food, Sirloin steak. Portion Size, 3.5 oz, broiled. Vitamin A (RE), 0. % DRI, 0. Row 29. Food, Brewer’s yeast. Portion Size, 1 oz. Vitamin A (RE), 0. % DRI, 0. Row 30. Food, Bread, whole wheat. Portion Size, 1 slice. Vitamin A (RE), 0. % DRI, 0. Row 31 has the following

text Quick Reference Diet Integration: Use fresh spinach, romaine lettuce, carrots, and tomatoes in salads.; Use canned pumpkin in cookies, pies, and desserts.; Serve baked sweet potatoes in place of baked potatoes.; Breakfast: fruit salad of mango, papaya, and cantaloupe.; Snack: dried peaches and apricots.

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A table is titled M. Vitamin D. It has the headers: Food, Portion Size, Vitamin D (µg), and % DRI. Row 1. Food, Milk, 2%. Portion Size, 8 oz. Vitamin D (µg), 1.6. % DRI, 33. Row 2. Food, Corn flakes. Portion Size, 1 cup. Vitamin D (µg), 0.81. % DRI, 16. Row 3. Food, Cod liver oil. Portion Size, 1 Tbsp. Vitamin D (µg), 0.55. % DRI, 11. Row 4. Food, Egg. Portion Size, 1, whole fresh. Vitamin D (µg), 0.41. % DRI, 8. Row 5. Food, Margarine. Portion Size, 1 tsp, tub type. Vitamin D (µg), 0.34. % DRI, 7. Row 6. Food, Frankfurter. Portion Size, 1 beef frank. Vitamin D (µg), 0.18. % DRI, 4. Row 7. Food, Braunschweiger. Portion Size, 1 oz, tube type. Vitamin D (µg), 0.15. % DRI, 3. Row 8. Food, Rice, wild. Portion Size, 2/3 cup, instant. Vitamin D (µg), 0.07. % DRI, 1. Row 9. Food, Rice, white. Portion Size, 2/3 cup, instant. Vitamin D (µg), 0.03. % DRI, <1. Row 10. Food, Cheddar cheese. Portion Size, 1 oz. Vitamin D (µg), 0.02. % DRI, <1. Row 11 has the following text Quick Reference Diet

Integration: Drink 2–3 eight-oz glasses of milk per day.; 10–15 minutes of sunlight exposure 3 times per week.; Breakfast: vitamin D-fortified cereals with milk.; Snack: pudding made with low-fat milk.; Use vitamin D-fortified margarine instead of butter.

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A table is titled N. Vitamin E. It has the headers: Food, Portion Size, Vitamin E (mg), and % DRI. Row 1. Food, Wheat germ oil. Portion Size, 1 Tbsp. Vitamin E (mg), 20.3. % DRI, 102. Row 2. Food, Sunflower seeds. Portion Size, 1 oz, dry roasted. Vitamin E (mg), 14.2. % DRI, 71. Row 3. Food, Mayonnaise. Portion Size, 1 Tbsp. Vitamin E (mg), 11. % DRI, 55. Row 4. Food, Almonds. Portion Size, 1 oz, dried. Vitamin E (mg), 6.7. % DRI, 34. Row 5. Food, Dried filberts. Portion Size, 1 oz. Vitamin E (mg), 6.7. % DRI, 34. Row 6. Food, Sunflower seed oil. Portion Size, 1 Tbsp. Vitamin E (mg), 6.3. % DRI, 32. Row 7. Food, Sweet potato. Portion Size, 1, medium raw. Vitamin E (mg), 5.9. % DRI, 30. Row 8. Food, Almond oil. Portion Size, 1 Tbsp. Vitamin E (mg), 5.3. % DRI, 27. Row 9. Food, Cottonseed oil. Portion Size, 1 Tbsp. Vitamin E (mg), 4.8. % DRI, 24. Row 10. Food, Safflower oil. Portion Size, 1 Tbsp. Vitamin E (mg), 4.6. % DRI, 23. Row 11. Food, Wheat germ. Portion Size, ¼ cup, toasted. Vitamin E (mg), 4.1. % DRI, 20. Row 12. Food, Peanut butter. Portion Size, 2 Tbsp. Vitamin E (mg), 3. % DRI,

15. Row 13. Food, Shrimp. Portion Size, 3 oz, boiled. Vitamin E (mg), 3. % DRI, 15. Row 14. Food, Canola oil. Portion Size, 1 Tbsp. Vitamin E (mg), 2.5. % DRI, 13. Row 15. Food, Asparagus. Portion Size, 1 cup, cooked. Vitamin E (mg), 2.4. % DRI, 12. Row 16. Food, Mango. Portion Size, 1, medium raw. Vitamin E (mg), 2.3. % DRI, 12. Row 17. Food, Avocado. Portion Size, ½ cup. Vitamin E (mg), 2.3. % DRI, 12. Row 18. Food, Peanuts. Portion Size, 1 oz, dry roasted. Vitamin E (mg), 2.2. % DRI, 11. Row 19. Food, Brazil nuts. Portion Size, 1 oz, dried. Vitamin E (mg), 2.1. % DRI, 11. Row 20. Food, Salmon. Portion Size, 3 oz, baked. Vitamin E (mg), 2. % DRI, 10. Row 21. Food, Corn oil. Portion Size, 1 Tbsp. Vitamin E (mg), 1.9. % DRI, 10. Row 22. Food, Olive oil. Portion Size, 1 Tbsp. Vitamin E (mg), 1.7. % DRI, 8. Row 23. Food, Peanut oil. Portion Size, 1 Tbsp. Vitamin E (mg), 1.6. % DRI, 8. Row 24. Food, Soybean oil. Portion Size, 1 Tbsp. Vitamin E (mg), 1.5. % DRI, 8. Row 25. Food, Apple. Portion Size, 1, medium fresh. Vitamin E (mg), 1.5. % DRI, 8. Row 26. Food, Brussel sprouts. Portion Size, 1 cup, cooked. Vitamin E (mg), 1.3. % DRI, 7. Row 27. Food, Spinach. Portion Size, 1 cup, raw. Vitamin E (mg), 1.1. % DRI, 5. Row 28. Food, Macaroni. Portion Size, 1 cup, cooked. Vitamin E (mg), 1. % DRI, 5. Row 29. Food, Parsley. Portion Size, 1 Tbsp. Vitamin E (mg), 1. % DRI, 5. Row 30. Food, Pear. Portion Size, 1, medium fresh. Vitamin E (mg), 0.83. % DRI, 4. Row

31. Food, Cheddar cheese. Portion Size, 1 oz. Vitamin E (mg), 0.5. % DRI, 3. Row 32 has the following text Quick Reference Diet Integration: Use wheat germ or sunflower oil in salad dressings or baked goods.; Serve sweet potato chips instead of regular chips—slice thin and bake.; Replace cream cheese with peanut butter on bagels.; Add wheat germ to cereal or yogurt.; Snack: sunflower seeds, peanuts, or almonds.

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A table is titled O. Vitamin K. It has the headers: Food, Portion Size, Vitamin K (µg), and % DRI. Row 1. Food, Turnip greens. Portion Size, 1 cup, chopped raw. Vitamin K (µg), 364. % DRI, 455. Row 2. Food, Green tea. Portion Size, 1 oz, dry. Vitamin K (µg), 199. % DRI, 249. Row 3. Food, Spinach. Portion Size, 1 cup, raw. Vitamin K (µg), 148. % DRI, 185. Row 4. Food, Broccoli. Portion Size, 1 cup, cooked. Vitamin K (µg), 126. % DRI, 158. Row 5. Food, Beef liver. Portion Size, 3.5 oz, raw. Vitamin K (µg), 104. % DRI, 104. Row 6. Food, Cauliflower. Portion Size, 1 cup, raw. Vitamin K (µg), 96. % DRI, 120. Row 7. Food, Soybean oil. Portion Size, 1 Tbsp. Vitamin K (µg), 76. % DRI, 95. Row 8. Food, Chickpeas. Portion Size, 1 oz, dry. Vitamin K (µg), 74. % DRI, 93. Row 9. Food, Asparagus. Portion Size, 1 cup, cooked. Vitamin K (µg), 69. % DRI, 86. Row 10. Food, Lentils. Portion

Size, 1 oz, dry. Vitamin K (µg), 62. % DRI, 78. Row 11. Food, Soybeans. Portion Size, 1 oz. Vitamin K (µg), 53. % DRI, 66. Row 12. Food, Cabbage. Portion Size, 1 cup, raw shredded. Vitamin K (µg), 52. % DRI, 65. Row 13. Food, Mung beans. Portion Size, 1 oz, dry. Vitamin K (µg), 48. % DRI, 60. Row 14. Food, Green beans. Portion Size, 1 cup, boiled. Vitamin K (µg), 44. % DRI, 55. Row 15. Food, Wheat flour. Portion Size, Whole wheat, 1 cup. Vitamin K (µg), 36. % DRI, 45. Row 16. Food, Tomato. Portion Size, 1, whole fresh. Vitamin K (µg), 25. % DRI, 31. Row 17. Food, Egg. Portion Size, 1, whole fresh. Vitamin K (µg), 25. % DRI, 31. Row 18. Food, Peas. Portion Size, 1 oz, dry. Vitamin K (µg), 23. % DRI, 29. Row 19. Food, Wheat bran. Portion Size, 1 oz. Vitamin K (µg), 23. % DRI, 29. Row 20. Food, Lettuce. Portion Size, 1 leaf iceberg. Vitamin K (µg), 22. % DRI, 28. Row 21. Food, Strawberries. Portion Size, 1 cup, raw. Vitamin K (µg), 21. % DRI, 26. Row 22. Food, Watercress. Portion Size, 1 cup, chopped. Vitamin K (µg), 20. % DRI, 25. Row 23. Food, Oats. Portion Size, 1 oz, dry. Vitamin K (µg), 18. % DRI, 23. Row 24. Food, Wheat germ. Portion Size, 1 oz. Vitamin K (µg), 10. % DRI, 13. Row 25. Food, Carrot. Portion Size, 1, medium raw. Vitamin K (µg), 9. % DRI, 11. Row 26. Food, Corn oil. Portion Size, 1 Tbsp. Vitamin K (µg), 8. % DRI, 10. Row 27. Food, Orange. Portion Size, 1, medium fresh. Vitamin K (µg), 7. % DRI, 9. Row 28. Food, Potato. Portion

Size, 1, whole baked. Vitamin K (µg), 6. % DRI, 8. Row 29. Food, Cucumber. Portion Size, 1 cup, raw. Vitamin K (µg), 6. % DRI, 8. Row 30 has the following text Quick Reference Diet Integration: Add turnip greens to soup or eat as a side dish.; Entrée: stir fry of broccoli, cauliflower, spinach, carrots, and asparagus.; Add spinach, watercress, and tomatoes to sandwiches.; Use soybean oil to make salad dressing.; Salad: spinach and lettuce with tomatoes and chickpeas.; Appetizer: coleslaw with low-fat or fat-free dressing.

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A table is titled Mineral Facts. It has the headers: Mineral/Symbol, Functions, Deficiency, and Toxicity. Row 1. Mineral/Symbol, Calcium, Ca. Functions, Bone/teeth structure; nerve transmission; blood clotting; muscle contraction. Deficiency, Osteoporosis; osteomalacia; neuromuscular excitability; cramps, tetany; hypoparathyroidism; convulsion. Toxicity, Kidney stones, constipation. Row 2 has the text Canned sardines, cheese, milk, tofu made with calcium sulfate, yogurt. Row 3. Mineral/Symbol, Chromium, Cr. Functions, Glucose metabolism, an insulin cofactor. Deficiency, Weight loss; diabetes; abnormal glucose metabolism; neuropathy. Toxicity, None known. Row 4 has the text Broccoli, grape juice, grain products. Row 5.

Mineral/Symbol, Copper, Cu. Functions, Iron absorption; component of several metalloenzymes. Deficiency, Menkes disease; microcytic anemia; cognitive deficits; hypothermia; hair depigmentation. Toxicity, Wilson disease; cirrhosis. Row 6 has the text Liver, clams, oysters, crab meat, cashews, hazelnuts, sunflower seeds, lentils, mushrooms. Row 7. Mineral/Symbol, Fluoride, F. Functions, Dental caries prevention. Deficiency, Dental caries. Toxicity, Dental mottling. Row 8 has the text Fluoridated water, crab, rice, fish. Row 9. Mineral/Symbol, Iodine, I. Functions, Constituent of thyroid hormones. Deficiency, Hypothyroidism; goiter. Toxicity, Goiter. Row 10 has the text Iodized salt, cod, milk, potatoes, turkey breast, shrimp. Row 11. Mineral/Symbol, Iron, Fe. Functions, Component of hemoglobin and myoglobin; electron transport. Deficiency, Microcytic anemia; fatigue; skin pallor; glossitis; tachycardia. Toxicity, Hemosiderosis; hemochromatosis. Row 12 has the text Oysters, beef, chicken (dark meat), blackstrap molasses, prune juice, lentils. Row 13. Mineral/Symbol, Magnesium, Mg. Functions, Component of several enzymes involved in metabolism (including nutrient); muscle contraction; nerve transmission. Deficiency, Muscle spasms, tetany and seizures; hypocalcemia; hypokalemia; paresthesias. Toxicity, Diarrhea; cardiorespiratory failure and abnormal nerve transmission in kidney

patients. Row 14 has the text Bran cereal, brown rice, almonds, spinach, Swiss chard, peanuts, blackstrap molasses, hazelnuts. Row 15. Mineral/Symbol, Manganese, Mn. Functions, Cofactor in several enzymes; gluconeogenesis, metabolism of lipids and mucopolysaccharides; antioxidant as Mn superoxide dismutase. Deficiency, Limited evidence for increased bone remodeling; reduction of serum cholesterol; skin rash; abnormal glucose tolerance. Toxicity, Neurologic damage if inhaled in certain occupations; limited evidence for oral toxicity of neurologic and cognitive problems. Row 16 has the text Pecans, pineapple, almonds, peanuts, bran cereal, brown rice. Row 17. Mineral/Symbol, Molybdenum, Mo. Functions, Cofactor in enzymes. Deficiency, Tachycardia; coma. Toxicity, Limited evidence for gout-like symptoms. Row 18 has the text Legumes, lentils, grain products, nuts. Row 19. Mineral/Symbol, Phosphorus, P. Functions, Cell membrane and nucleic acid component; glycolysis; component of ATP in all energy reactions; regulation of acid–base balance. Deficiency, Muscular weakness; glucose intolerance; abnormal cellular function; growth deficit and bone deformation in children. Toxicity, Diarrhea; hyperparathyroidism; soft tissue calcification. Row 20 has the text Dairy products, cereal products, meat, fish, food additives (phosphates). Row 21. Mineral/Symbol, Potassium, K. Functions, Intracellular

cation; nerve transmission; muscle contraction; blood pressure regulation. Deficiency, Muscular weakness; hypotension; ileus; cardiac arrhythmias; respiratory failure. Toxicity, Cardiac arrhythmia; heart failure. Row 22 has the text Potatoes, prunes/prune juice, bananas, tomatoes/tomato juice, raisins, lima beans, acorn squash. Row 23. Mineral/Symbol, Selenium, Se. Functions, Antioxidant as glutathione peroxidases and as part of other enzymes; part of amino acid selenocysteine. Deficiency, Susceptibility to physiologic stresses; prolonged deficiency linked to Keshan and Kashin–Beck diseases. Toxicity, Hair and nail brittleness and loss; gastrointestinal distress; skin rash; garlic breath odor; fatigue; irritability; neurologic disorders. Row 24 has the text Brazil nuts, tuna, oysters, salmon, shrimp, halibut, crab, beef, chicken, rice, sunflower seeds. Row 25. Mineral/Symbol, Sodium, Na; chloride, Cl. Functions, Na and Cl principle extracellular ions; maintenance of membrane potential; nutrient absorption and transport; regulation of blood volume and pressure. Deficiency, Headache; nausea; vomiting; muscle cramps; fatigue; disorientation; dizziness; seizure and coma. Toxicity, Nausea; vomiting; diarrhea; abdominal cramps; edema; coma. Row 26 has the text Snack foods, processed meats, breads, cereals, canned tomato/vegetable juice, soup, pickles, olives. Row 27. Mineral/Symbol, Zinc, Zn. Functions, Enzyme

cofactor in reactions related to growth, sexual maturation, reproduction, immunity, night vision, taste acuity. Deficiency, Hypogonadism; growth failure; impaired taste and smell; impaired wound healing; infection; fatigue; diminished appetite, dry and hyperpigmented skin, acrodermatitis. Toxicity, Interference with copper and iron metabolism; impaired immunity. Row 28 has the text Oysters, crab, beef, poultry (dark meat), yogurt, cashews.

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Table is detailing Dietary Reference Intakes (DRIs): Recommended Dietary Allowances and Adequate Intakes, Vitamins Food and Nutrition Board, Institute of Medicine, National Academies.

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Table is detailing Dietary Reference Intakes (DRIs): Recommended Dietary Allowances and Adequate Intakes, Elements Food and Nutrition Board, Institute of Medicine, National Academies.

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A table is titled Dietary Reference Intakes (DRIs): Recommended Dietary Allowances and Adequate Intakes, Total Water and Macronutrients Food and Nutrition Board, Institute of Medicine, National

Academies. It has the headers: Life Stage Group. Total Watera (L/d), Carbohydrate (g/d), Total Fiber (g/d), Fat (g/d), Linoleic Acid (g/d), α-Linolenic Acid (g/d), Proteinb (g/d). Row 1 has the header Infants. Row 2. Life Stage Group, 0 to 6 mo. Total Watera (L/d), 0.7*. Carbohydrate (g/d), 60*. Total Fiber (g/d), ND. Fat (g/d), 31*. Linoleic Acid (g/d), 4.4*. α-Linolenic Acid (g/d), 0.5*. Proteinb (g/d), 9.1*. Row 3. Life Stage Group, 6 to 12 mo. Total Watera (L/d), 0.8*. Carbohydrate (g/d), 95*. Total Fiber (g/d), ND. Fat (g/d), 30*. Linoleic Acid (g/d), 4.6*. α-Linolenic Acid (g/d), 0.5*. Proteinb (g/d), 11.0. Row 4 has the header Children. Row 5. Life Stage Group, 1–3 y. Total Watera (L/d), 1.3*. Carbohydrate (g/d), 130. Total Fiber (g/d), 19*. Fat (g/d), NDc. Linoleic Acid (g/d), 7*. α-Linolenic Acid (g/d), 0.7*. Proteinb (g/d), 13. Row 6. Life Stage Group, 4–8 y. Total Watera (L/d), 1.7*. Carbohydrate (g/d), 130. Total Fiber (g/d), 25*. Fat (g/d), ND. Linoleic Acid (g/d), 10*. α-Linolenic Acid (g/d), 0.9*. Proteinb (g/d), 19. Row 7 has the header Males. Row 8. Life Stage Group, 9–13 y. Total Watera (L/d), 2.4*. Carbohydrate (g/d), 130. Total Fiber (g/d), 31*. Fat (g/d), ND. Linoleic Acid (g/d), 12*. α-Linolenic Acid (g/d), 1.2*. Proteinb (g/d), 34. Row 9. Life Stage Group, 14–18 y. Total Watera (L/d), 3.3*. Carbohydrate (g/d), 130. Total Fiber (g/d), 38*. Fat (g/d), ND. Linoleic Acid (g/d), 16*. α-Linolenic Acid (g/d), 1.6*. Proteinb (g/d), 52. Row 10. Life Stage

Group, 19–30 y. Total Watera (L/d), 3.7*. Carbohydrate (g/d), 130. Total Fiber (g/d), 38*. Fat (g/d), ND. Linoleic Acid (g/d), 17*. α-Linolenic Acid (g/d), 1.6*. Proteinb (g/d), 56. Row 11. Life Stage Group, 31–50 y. Total Watera (L/d), 3.7*. Carbohydrate (g/d), 130. Total Fiber (g/d), 38*. Fat (g/d), ND. Linoleic Acid (g/d), 17*. α-Linolenic Acid (g/d), 1.6*. Proteinb (g/d), 56. Row 12. Life Stage Group, 51–70 y. Total Watera (L/d), 3.7*. Carbohydrate (g/d), 130. Total Fiber (g/d), 30*. Fat (g/d), ND. Linoleic Acid (g/d), 14*. α-Linolenic Acid (g/d), 1.6*. Proteinb (g/d), 56. Row 13. Life Stage Group, >70 y. Total Watera (L/d), 3.7*. Carbohydrate (g/d), 130. Total Fiber (g/d), 30*. Fat (g/d), ND. Linoleic Acid (g/d), 14*. α-Linolenic Acid (g/d), 1.6*. Proteinb (g/d), 56. Row 14 has the header Females. Row 15. Life Stage Group, 9–13 y. Total Watera (L/d), 2.1*. Carbohydrate (g/d), 130. Total Fiber (g/d), 26*. Fat (g/d), ND. Linoleic Acid (g/d), 10*. α-Linolenic Acid (g/d), 1.0*. Proteinb (g/d), 34. Row 16. Life Stage Group, 14–18 y. Total Watera (L/d), 2.3*. Carbohydrate (g/d), 130. Total Fiber (g/d), 26*. Fat (g/d), ND. Linoleic Acid (g/d), 11*. α-Linolenic Acid (g/d), 1.1*. Proteinb (g/d), 46. Row 17. Life Stage Group, 19–30 y. Total Watera (L/d), 2.7*. Carbohydrate (g/d), 130. Total Fiber (g/d), 25*. Fat (g/d), ND. Linoleic Acid (g/d), 12*. α-Linolenic Acid (g/d), 1.1*. Proteinb (g/d), 46. Row 18. Life Stage

Group, 31–50 y. Total Watera (L/d), 2.7*. Carbohydrate (g/d), 130. Total Fiber (g/d), 25*. Fat (g/d), ND. Linoleic Acid (g/d), 12*. α-Linolenic Acid (g/d), 1.1*. Proteinb (g/d), 46. Row 19. Life Stage Group, 51–70 y. Total Watera (L/d), 2.7*. Carbohydrate (g/d), 130. Total Fiber (g/d), 21*. Fat (g/d), ND. Linoleic Acid (g/d), 11*. α-Linolenic Acid (g/d), 1.1*. Proteinb (g/d), 46. Row 20. Life Stage Group, >70 y. Total Watera (L/d), 2.7*. Carbohydrate (g/d), 130. Total Fiber (g/d), 21*. Fat (g/d), ND. Linoleic Acid (g/d), 11*. α-Linolenic Acid (g/d), 1.1*. Proteinb (g/d), 46. Row 21 has the header Pregnancy. Row 22. Life Stage Group, 14–18 y. Total Watera (L/d), 3.0*. Carbohydrate (g/d), 175. Total Fiber (g/d), 28*. Fat (g/d), ND. Linoleic Acid (g/d), 13*. α-Linolenic Acid (g/d), 1.4*. Proteinb (g/d), 71. Row 23. Life Stage Group, 19–30 y. Total Watera (L/d), 3.0*. Carbohydrate (g/d), 175. Total Fiber (g/d), 28*. Fat (g/d), ND. Linoleic Acid (g/d), 13*. α-Linolenic Acid (g/d), 1.4*. Proteinb (g/d), 71. Row 24. Life Stage Group, 31–50 y. Total Watera (L/d), 3.0*. Carbohydrate (g/d), 175. Total Fiber (g/d), 28*. Fat (g/d), ND. Linoleic Acid (g/d), 13*. α-Linolenic Acid (g/d), 1.4*. Proteinb (g/d), 71. Row 25 has the header Lactation. Row 26. Life Stage Group, 14–18 y. Total Watera (L/d), 3.8*. Carbohydrate (g/d), 210. Total Fiber (g/d), 29*. Fat (g/d), ND. Linoleic Acid (g/d), 13*. α-Linolenic Acid (g/d), 1.3*. Proteinb (g/d), 71. Row

27. Life Stage Group, 19–30 y. Total Watera (L/d), 3.8*. Carbohydrate (g/d), 210. Total Fiber (g/d), 29*. Fat (g/d), ND. Linoleic Acid (g/d), 13*. α-Linolenic Acid (g/d), 1.3*. Proteinb (g/d), 71. Row 28. Life Stage Group, 31–50 y. Total Watera (L/d), 3.8*. Carbohydrate (g/d), 210. Total Fiber (g/d), 29*. Fat (g/d), ND. Linoleic Acid (g/d), 13*. α-Linolenic Acid (g/d), 1.3*. Proteinb (g/d), 71.

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A table is titled Dietary Reference Intakes (DRIs): Acceptable Macronutrient Distribution Ranges Food and Nutrition Board, Institute of Medicine, National Academies. It has the headers: Macronutrient, and Range (Percent of Energy), it is subdivided into Children, 1–3 y, Children 4–18 y, and Adults. Row 1. Macronutrient, Fat. Range (Percent of Energy), Children, 1–3 y: 30–40. Children 4–18 y: 25–35. Adults: 20–35. Row 2. Macronutrient, n-6 polyunsaturated fatty acidsa (linoleic acid). Range (Percent of Energy), Children, 1–3 y: 5–10. Children 4–18 y: 5–10. Adults: 5–10. Row 3. Macronutrient, n-3 polyunsaturated fatty acidsa (α-linolenic acid). Range (Percent of Energy), Children, 1–3 y: 0.6–1.2. Children 4–18 y: 0.6–1.2. Adults: 0.6–1.2. Row 4. Macronutrient, Carbohydrate. Range (Percent of Energy), Children, 1–3 y: 45–65. Children 4–18 y: 45–65. Adults: 45–65. Row 5. Macronutrient, Protein.

Range (Percent of Energy), Children, 1–3 y: 5–20. Children 4–18 y: 10–30. Adults: 10–35. Footnote below the table indicates that Approximately 10% of the total can come from longer-chain n-3 or n-6 fatty acids.

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Table is detailing Dietary Reference Intakes (DRIs): Tolerable Upper Intake Levels, Vitamins Food and Nutrition Board, Institute of Medicine, National Academies.

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Table is detailing Dietary Reference Intakes (DRIs): Recommended Dietary Allowances and Adequate Intakes, Elements Food and Nutrition Board, Institute of Medicine, National Academies.

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Two pyramids show the diet continuum for IDDSI dysphasia. The inverted pyramid at the top of the figure is for foods from regular at the top to liquidized at the bottom. From top to bottom, the foods get softer. The second pyramid is for drinks from extremely thick at the top of the pyramid to the thinnest at the bottom. The consistencies of the

drinks get less and less thick going down to the bottom of the pyramid.

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Flow chart on CVD and nutrition intervention targets. There are two boxes at the top of the chart: systolic/diastolic abnormalities and diet. The boxes below systolic/diastolic abnormalities are hypertension and below that, CCF and stroke. The boxes below the diet box are lipd abnormalities, can lead to atherosclerosis, then stroke, myocardial infarction, myocardial/peripheral ischemia, and CCF at the bottom.

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Diagram of complete blood count, including, from left to right, WBC, Hgb, Hcl, segs/bands/lymph/monos/baso/EOS, MCV/MCH/MCHC, and platelet count. The second diagram is an illustration of the electrolytes and includes sodium, potassium, cloride, carbon dioxide, BUN, Cr, and glucose.

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This diagram is an illustration of the electrolytes and includes sodium, potassium, cloride, carbon dioxide, BUN, Cr, and glucose.

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