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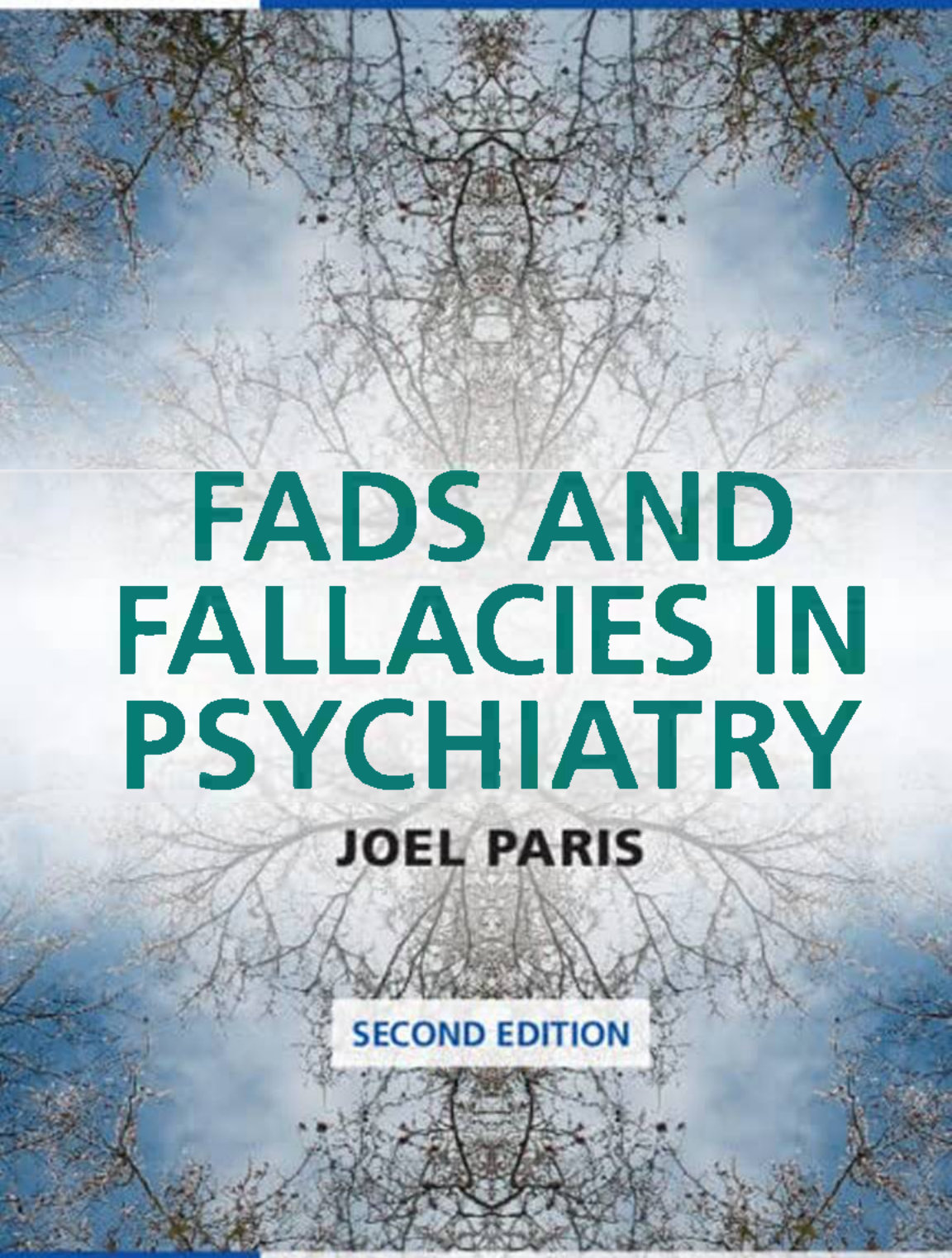

FADS AND FALLACIES IN PSYCHIATRY

JOEL PARIS

SECOND EDITION

CAMBRIDGE
Medicine

Fads and Fallacies in Psychiatry

Second Edition

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This book is dedicated to family and friends who encouraged me to go into psychiatry.

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Preface to the Second Edition

A decade has passed since the publication of the first edition of this book. Although many notable research findings have emerged since then, psychiatry has yet to solve the enigma of the causes of mental illness. Our treatment methods are largely effective, and we do well with most patients. Yet, like physicians of earlier times, we suffer from the absence of a valid theory, and are forced to target symptoms rather than mental illness as a whole. But uncertainty can be difficult to accept. This lack of knowledge opens the door to fads and fallacies.

The first edition of this book was relatively brief, but this second edition will examine psychiatry's fads and fallacies in somewhat more detail, reviewing recent research and expanding the scope of the critique. The recommendations will be the same—that there is a need for a more scientific practice, and for greater humility in the face of complexity.

Acknowledgments

Peter Tyrer has been consistently supportive of both the first edition and the expanded second edition of this book.

Introduction

Mental illness is, and always has been, a mystery. Despite active research, we may have to wait many decades for answers to important questions. Yet it is precisely because mental illness is enigmatic that I chose psychiatry as a career, and why I still love it. Sixty years later, I can say that becoming a psychiatrist was one of the best decisions I ever made, and I have little interest in retirement.

Over time, my perspective on psychiatry has changed, in the direction of greater skepticism. As a student, I did not understand why I needed to be taught the history of medicine. Once ideas go out of date, why bother to learn about them? Yet, as I grew older, I became more interested in the history of my specialty, and came to realize that progress is not linear. Impeded by false beliefs, medical science can go off on serious tangents. Understanding past mistakes can help us to be appropriately skeptical about current theories and practices—some of which may be remembered as dangerous errors.

I have always been the type of person who questions everything. When I was young, this attitude got me into trouble. I asked tough questions that threatened the convictions of my teachers. Since I did not accept received wisdoms without question, they saw me as a rebellious young man. Now, in old age, I have been called a curmudgeon for saying many of the same things. Although psychiatrists do a lot of good, it remains important to criticize contemporary practice, with its susceptibility to fads and its penchant for fallacies. That is the passion that drives this book.

The title of this volume is a deliberate paraphrase of a classic volume by Martin Gardner, *Fads and Fallacies in the Name of Science* (Gardner, 1957). *Fads* are ideas and practices based on temporary bursts of enthusiasm, whereas *fallacies* reflect cognitive errors or wishful thinking. When we think of fads, bizarre ideas come to mind, and Gardner's book focused on some of the strangest and most pseudoscientific theories of the time. However, fads in psychiatry have occurred not only on the fringe, but also in the very mainstream of theory and practice. Some of the trendiest theoretical paradigms turn out to be unsupported by data. In diagnosis, currently faddish approaches to classification lack a secure base in etiology, and many are unlikely to last. In treatment, both psychopharmacology and psychotherapy have embraced interventions that have a weak evidence base and that have made unjustified claims about their efficacy. These errors run the risk of doing harm to patients.

Should we be surprised or discouraged that psychiatry does not yet understand mental illness? No. The brain is the most complicated structure in the known universe. Neuroscience has not solved these problems fast enough to be applied to practice. We are often told that answers lie just around the corner, but that is where they tend to stay. The most important questions remain unanswered.

Given that psychiatrists still have so much to learn, they need to remain humble. For example, the idea of reducing everything about mental illness to a molecular or a neuronal level is currently all the rage, but is over-simplistic and hubristic. I am not criticizing basic research. But for clinical application, the biological processes behind disorders can only be understood in the context of interactions with psychological adversities and sociocultural stressors—that is, within a *biopsychosocial model* (Engel, 1980). Although multifactorial models can be intimidatingly complex, they explain why research on the origins and treatment of mental illness is so difficult.

Ironically, the main source of psychiatric fads is the strong desire of practitioners to help patients. Human nature being what it is, clinicians are uncomfortable with doubt and seek certainty. They have trouble maintaining a cautious stance in the face of scientific ignorance. Practitioners do not want to wait 100 years for answers, and are tempted to believe they know enough to practice in the present. Yet that is the main reason why psychiatry has been infected by fads and fallacies. This book will document how and why this happens.

Why I Have Written This Book

I began my career as a clinician and an educator. Despite my contrarian temperament, I largely accepted the point of view of my teachers. Yet, with time, I came to realize that the older generation was wrong about many things. Quite a few relied on clinical judgment to support theories, and were barely familiar with empirical data. I gradually became committed to a scientific and empirical perspective, and, with the help of colleagues, trained myself to become a researcher. I became a passionate convert to evidence-based medicine. I no longer take clinical experience, including my own, for granted. Despite the current fad for "lived experience," I am not prepared to accept ideas that fail to be supported by quantitative data.

For this reason, I have taken care in this book to ensure that its conclusions are consistent with the scientific literature, and I have referred the reader to relevant studies, comprehensive reviews, or meta-analyses. But since the subject of psychiatry as a whole is so vast, I have had to be selective about references.

This book will also draw on my 50 years of work as a consultant. Although one cannot base practice on clinical experience, the latter can be used to illustrate points that are confirmed by empirical data. Since 1972, I have run a hospital clinic that sees hundreds of patients every year referred from primary care. I also worked in a university health service for 25 years and saw thousands of troubled students. In 2001 and 2007, I founded two specialty clinics for the treatment of personality disorders, and over the last two decades I have conducted many thousands of consultations on patients with these conditions. Although the patients I treat myself are highly symptomatic, like many of my colleagues I now spend more time on consultations to primary care providers.

In total, I estimate that I have seen at least 30,000 patients over the last 50 years. When my students ask me how I manage to reach conclusions fairly rapidly, I tell them that things get easier after the first 30,000 cases. Yet even the most extensive experience does not automatically make you right. You could be making the same mistakes thousands of times. That is why I strongly support evidence-based psychiatry.

If you want to practice scientific medicine, you have to give up certainty and embrace doubt. In the first 10 years of my career, I aimed for radical change. With experience,

I learned that although I could help many or most patients, psychiatry lacks the tools to achieve consistent and stable remissions of serious mental disorders. The field is only at the beginning of a very long journey. Once I recognized that my specialty still has a thin knowledge base, I went into research to do my part in broadening it.

This second career in research started rather late, in my mid-forties. For that reason, I could not reach the same level as those who had started earlier. Moreover, I am only one soldier in a vast army. Yet I benefited from having more clinical experience, which some of my research colleagues—tied to labs and desks—lacked. Being an active clinician also helped me to ask more relevant questions. In turn, conducting research affected my practice. The doubt that characterizes the scientific culture is the best antidote to fads. I brought its worldview back to my clinical work and my teaching.

The clinical trenches are far from the ivory tower of academia. Although I aim to practice, as much as possible, in an evidence-based way, some of the most crucial questions cannot yet be answered by empirical data. Thus, when I treat patients, I keep in mind what I can and cannot do. And although I teach students to follow the research literature, I advise them to remain cautious about generalizing from just one or a few published studies. Unfortunately, not all of my colleagues share this perspective. Some jump on bandwagons and pretend to have an unjustified certainty. Most simply follow the crowd, and join in a consensus, however uncertain, that is shared by their colleagues.

Psychiatric Fads: Then and Now

When I was young, two major theoretical models shaped psychiatry, and both became sources of orthodoxy. One was the psychoanalytic model. I began training in the late 1960s, towards the end of the heyday of psychoanalysis in North America. At many universities, including my own, analysts were the leaders of academic psychiatry. Trainees revered them, mainly because they were eloquent and seemed to have an answer for everything. Some analysts may have been rather arrogant, but students tend to be attracted to confidence and certainty. As teachers, analysts could provide plausible (or not so plausible) explanations for symptoms of all kinds. They also insisted, without evidence, that their treatment method was highly effective. When it did not work, that was only because therapy had not lasted long enough, or was not conducted with sufficient skill.

The psychoanalytic fad was never as powerful in Europe. It had a good deal of influence in the UK, but never dominated psychiatry there. Disinterest in research ultimately proved to be the downfall of psychoanalysis. Neither the theory nor the method could stand up to empirical scrutiny. Today, although the analytic movement remains alive, it plays a rather marginal role in psychiatry, both in North America and in Europe. I have written two books about its decline (Paris, 2005, 2019).

Although psychoanalysis was a fad, one cannot say that it was *only* a fad. Many of its concepts and methods have been incorporated into other forms of psychotherapy that have since undergone clinical testing, and have been shown to be effective. Even cognitive–behavioral therapy (CBT) was founded by an analyst (Aaron Beck), and one can see a few surviving elements of his previous training in the method. But the key issue concerns the length of treatment, which leads to therapy being expensive and unavailable to most patients. As I shall show in this book, research supports *brief* courses of both psychodynamic psychotherapy and CBT, but does not support seeing patients regularly for years on end.

This having been said, psychoanalysis has a legacy. My training promoted an ability to listen empathically to patients and to understand what they might be thinking and feeling. (This is also a skill that cannot be entirely turned off, even in private life.)

Following the move away from psychoanalysis, modern psychiatry returned to its medical roots. Even in my student days, biological psychiatry had become an alternative way of looking at psychopathology. But it had not yet become an orthodoxy, and did not yet take psychopharmacology to an extreme, as it does now. Although drugs are often effective (and occasionally miraculous), clinicians who only treat symptoms with medications tend to lose interest in people and their life histories. Yet research shows that in disorders such as depression and anxiety, the effects of psychological treatment are more durable (Cuijpers et al., 2020). This is why I expect and hope that psychotherapy, much of which is well supported by research, will make a comeback.

The 1960s was the golden age of psychopharmacology. The dramatic success of drug treatment for severe mental disorders gave biological psychiatry an enormous boost. Healy (2002) described the medical management of psychosis as one of the most inspiring moments in human history. I entirely agree. I visited a large mental hospital as an undergraduate student, and saw what psychotic patients were like before drugs were available to control their symptoms. Only a few years later, psychiatrists had effective treatments for most of the psychoses and severe mood disorders. I saw patients being discharged and maintained in the community after years of serious illness. This was indeed a time of miracles.

Biological psychiatrists may be less colorful than psychoanalysts, but they have kept psychiatry within the scientific mainstream. Instead of tradition and authority, they rely on research studies and clinical trials. Gradually, neuroscience became the dominant force in psychiatry, even if clinicians did not understand why the drugs they prescribed are effective. Unfortunately, the neuroscience community has taken a narrowly biological approach, claiming that mental disorders are "nothing but" brain disorders. That mantra is both true and untrue. There can be no mind without brain. But psychiatry needs to study mind at its own level, not as something that can be reduced to neural connections. Moreover, neuroscience should not ignore (or pay lip service to) the powerful effects of psychological and social forces, which interact with biological factors, and which can also shape the structure and function of the brain.

In this way, biological psychiatry, when associated with an almost total dependence on drug treatment, can be as dogmatic as psychoanalysis ever was. Its ideas are based on a core of truth that has been stretched to the point of faddishness. Drugs are useful tools, but rarely *cure* severe mental disorders, many of which tend to remain chronic. Psychiatrists, rushing to gain the respect of their medical colleagues, embraced an ideology that is triumphant for now, but covers vast ignorance with a gloss of science.

Thus, despite the progress of recent decades, neuroscience is still in its infancy. Brain research has not even begun to explain how psychological symptoms develop. It should eventually do better. But it will never be possible to reduce all mental phenomena and symptoms to a cellular level, or to neural networks. Unless psychiatry embraces a broader model, it will suffer from a cripplingly narrow perspective.

Fads in Contemporary Psychiatry

I wish I could say that psychiatry has outgrown the fads and fallacies of my youth. But it has not. This book will focus on three areas that remain problematic.

The first is the diagnostic system used by psychiatrists. The *Diagnostic and Statistical Manual of Mental Disorders (DSM)* is now in its fifth edition, and although developed by American psychiatrists, it is used in most places in the world. I shall examine this system and outline its limitations. The latest revision, the DSM-5-Text Revision (DSM-5-TR; American Psychiatric Association, 2022) is a tool that works best for the most severe illnesses, but is much less useful for the common disorders that are most prevalent in practice. I shall also examine several of the alternatives to DSM, including the *International Classification of Diseases (ICD)*, now in its eleventh edition (ICD-11; World Health Organization, 2018). Some of these alternatives are based on quantitative dimensions rather than qualitative categories. But none of these has been shown to provide a solution to the lack of precision in categorization. They are still limited by the absence of etiologically based models, and the lack of data describing the endophenotypes that lie behind clinical disorders. This book will conclude that although DSM-5 is problematic, we currently lack the knowledge to replace it with something radically different or better.

The second focus of the book will be a critical examination of the currently popular view that neuroscience, by itself, can provide the answers to all the dilemmas of psychiatry. Clinicians have often been promised breakthroughs, each of which has been touted as being "just around the corner." But since that is where solutions remain, these promises remain unfulfilled.

In some ways, the more we learn, the more we realize how difficult will be the task of understanding mind and brain. The brain is an incredibly complex system consisting of about 85 billion neurons, with trillions of connections between them. Reading the 20,000 or so genes on the genome, and using genome-wide association studies (GWAS), has not yet been of much help. As we shall see, this research shows that every phenotype is rooted in small effects from hundreds or thousands of interacting alleles. We can add up these correlations to produce a "polygenic risk score," but that measure only accounts for a small part of the outcome (Tam et al., 2019).

Progress in biological research continues to be more relevant to basic science than to clinical needs. Unfortunately, the National Institute of Mental Health (NIMH) in the USA mainly funds studies of neuroscience, and provides little support for research that examines issues of direct clinical relevance. Perhaps research funders believe in making long-term investments at the expense of current needs. But this fallacy is based on hope, not facts, and is a disservice to the millions of people who suffer from mental illness. We have the tools to help many or most of our patients, but suffer from serious underfunding and lack of human resources to make our efforts count for more.

This book will argue that we need to avoid reductionism and adhere to a biopsychosocial model that acknowledges the complexity of the interactions that carry a risk for mental disorders. The bias in favor of neuroscience has greatly affected clinical practice. Psychiatrists these days may spend only 15 minutes with each patient, just enough time to check on symptoms and write a prescription. And when medication fails to help, the result is often more medication (i.e., polypharmacy).

The third, closely related, issue concerns the marginalization of psychotherapy in the practice of psychiatry. Only a minority of psychiatrists are seriously committed to making talking therapy part of their clinical activities, and that field is now dominated by clinical psychologists. This would not be a problem if psychological treatment was properly insured and readily available, but it is not. In the USA, psychotherapy remains expensive, with most insurance covering only a few sessions. The result is that patients

with common mental disorders (anxiety and depression) routinely receive medication without access to evidence-based forms of psychological treatment. In the UK, there has been a serious effort to make psychotherapy for these problems available within the National Health Service (Clark et al., 2018). But this welcome initiative only scratches the surface of the problem, especially for patients with severe and disabling mental disorders.

The Antipsychiatry Movement

A book like this, which focuses on how psychiatrists can go wrong, might suggest to readers that its author supports what has been called "antipsychiatry." That is most definitely not the case. I am a mainstream thinker and practitioner who is only asking my colleagues to slow down and exercise more scientific caution.

The antipsychiatry movement dates back at least to the 1960s. It was always political. Right-wingers and radical libertarians like Thomas Szasz claimed that mental illness was a myth, and that nobody should ever be treated involuntarily (Szasz, 1961). This point of view could only be maintained by individuals who were safely protected by academia, and who never spent time working in hospital emergency rooms or wards. But it appealed to those who felt threatened by the idea that any of us can lose our minds, and that we may have to be looked after against our will.

On the left wing of politics, Ronald Laing took a similar view, romanticizing mental illness as an exciting journey into the boundaries of the mind (Laing, 1967). Laing, unlike Szasz, did treat seriously ill patients, but with little success. Instead of providing medication to psychotic patients, he offered psychotherapy, pretending to be their guru on a "trip." His personal life was marked by alcoholism and abandonment of his many children, several of whom died while still young (Burston, 1998).

The opposition of antipsychiatrists to psychopharmacology is a marker of the irrationality of their viewpoint. In recent years, the American journalist Robert Whitaker has claimed to be a spokesman for the supposedly oppressed mentally ill (Whitaker, 2001). The British psychologist Richard Bentall, who emphasizes trauma as a cause of psychosis (Bentall, 2010), has played a similar role.

A few other sticks have been used to beat psychiatry. One that appears often is a so-called "experiment" reported 50 years ago by Rosenhan (1973) in the journal *Science*. Rosenhan described sending eight volunteers to mental hospitals, having instructed them to pretend to be psychotic, resulting in their admission. Needless to say, anyone can be admitted to a hospital—whether medical or psychiatric—if they fake the symptoms of an illness. But the truth of this story is even darker. Careful investigation by a journalist showed that the entire study was a fraud (Cahalan, 2019). Rosenhan seems to be the only person who pretended to be ill, and the other participants were figments of his imagination. This story shows how fraudulent research with sensational results can reach the pages of the most respected scientific journals. And it has been told and retold in books and the media ever since.

My reaction to antipsychiatrists is that they seem not only to hate the idea of mental illness, but also to be prepared to undermine the treatment of people who suffer from it. Fortunately, these critics have had little influence on the practice of psychiatry. Antipsychiatrists prefer to stand on the sidelines, and to add the mentally ill to the long list of oppressed sufferers who require the service of social justice warriors.

Antidotes to Fads

Faddish clinical practices derive from overly simplistic theories. Given our lack of knowledge about the causes and potential cure of mental illness, it is not surprising that clinicians have often failed to adopt practices based on evidence-based medicine. Contemporary views about the etiology of mental disorders have favored the idea that mental symptoms are due to a "chemical imbalance" or aberrant neural circuits. These theories could turn out to be at least partly correct, but are currently unsupported by good evidence. Even so, many practitioners, and quite a few patients, believe these ideas to be scientific truth. The result is that treatment aims to correct putative imbalances with a "cocktail" of drugs. Many patients are being given prescriptions that they do not need.

The enterprise of science encourages debate and doubt, which are the best correctives for faddish ideas. In the basic sciences, even the most powerful paradigms decline when the weight of evidence fails to support them, but change is slower in medicine. Sick people can be desperate, and physicians may seek desperate remedies. I have great sympathy for front-line clinicians who deal with highly distressed patients. But that is why psychiatry, which deals with poorly understood illnesses that cause profound suffering, is so susceptible to faddish ideas. A scientific worldview implies a commitment to test all theories before accepting them, and to subject all treatments to clinical trials. Practitioners can practice virtues such as patience, humility, and caution.

The antidote to fads consists of thinking scientifically and conducting evidence-based practice (Evidence-Based Medicine Working Group, 1992). This influential concept, developed by the British physician Archie Cochrane, in whose name guidelines to treatment are still being published, is a principle to which we all pay lip service. But clinicians have preconceptions that make them see the world in a way that confirms their point of view. These *confirmation biases* (Kahneman, 2011) lie at the heart of fallacious thinking in clinical work. Close attention to the scientific literature helps to keep such biases in check, and it also leads to a more cautious and conservative way of working with patients. Adopting an evidence-based perspective helps us to be comfortable with uncertainty, and it makes us less likely to harm patients and more likely to help them.

Chapter 1

Fads and Fallacies in Science, Medicine, and Psychology

Defining Fads

Fads are novel ideas that are rapidly adopted and enthusiastically followed—at least for a time. Fads are also based on bad theories. Science moves slowly, and to make progress more certain, proceeds with caution. Yet since fads can appear new and attractive, they initially gain much attention. Most end by disappearing from view, sometimes with barely a trace. The American sociologist Joel Best described these phases as "emerging, surging, and purging" (Best, 2006).

Not every new idea is a fad. There can be real breakthroughs in knowledge, but it takes years to determine how they actually pan out. As a rule, it is best to remain cautious about concepts that spread too rapidly, and to be more welcoming to those that gain support gradually, that prove to be replicable, and that withstand the test of time. In the end, fads are addictive ideas that short-circuit the slow advance of science. They lead to mistaken conclusions that can be embraced incautiously, but do not bear close inspection.

Fallacies and Cognitive Errors

Fallacies are cognitive errors that can be described in research (Kahneman, 2011). Most people assume that however foolish others may be, they themselves are more rational and have good judgment. Thus a lack of critical perspective on the self is the most prevalent of all fallacies. It is related to what has been called a *fundamental attribution error*. This term refers to the tendency to attribute other people's mistakes to their character, but to attribute one's own mistakes to circumstance.

One would like to assume that intelligent clinicians and scientists are less susceptible to fallacies, which only appeal to uneducated non-professionals. If only that were so! This book will show how stubbornly wrong ideas can be held, even by brilliant people. It also takes time for them to decline and disappear, often only after the death of influential founders of schools of thought and their disciples. In a witticism attributed to the physicist Max Planck, science advances one funeral at a time.

One of the earliest books on this subject was Charles Mackay's *Extraordinary Popular Delusions and the Madness of Crowds* (Mackay, 1841/1980), still in print after almost 200 years. Mackay made fun of faddish ideas, but implicitly assumed that his readers would be immune to them. Over a century later, Martin Gardner's *Fads and Fallacies in the Name of Science* (Gardner, 1957) showed how science, or at least popular science, can also be infected by fads. Most of Gardner's examples were fringe ideas that have since died out, but a few remain current nearly 70 years later, including extra-sensory perception (ESP), homeopathy, food fads, and Scientology.

If Gardner were still with us, he would no doubt want to write about the latest twists in the story. Fads and fallacies remain a problem, even in mainstream science. For example, a well-known psychologist published a paper some years ago claiming to prove the existence of a form of ESP called precognition (Bem, 2011). Attempts at replication of these findings consistently failed (Ritchie, 2020). Yet, as often happens in science, it was more difficult to publish failures to replicate than sensational findings that turn out be incorrect. This is one reason why we have a "replication crisis" that affects both medical and social sciences. Or as the internist John Ioannidis famously described the matter, most research findings turn out to be false (Ioannidis, 2005). Thus the progress of science generally involves two steps forward and at least one step backward.

Even so, some ideas have a tendency to "go viral." The evolutionary theorist Richard Dawkins made a useful contribution to the understanding of fads by introducing the term "meme" (Dawkins, 1976), a concept that was later expanded by the psychologist Susan Blackmore (Blackmore, 1999). Dawkins and Blackmore suggest that ideas can spread through society rather like genes, and that they are replicated even more rapidly. The difference is that the mechanism is entirely social and cultural. The concept of a meme goes some way toward explaining how false ideas can spread rapidly.

To explain why people are attracted to fads, we can begin by considering fallacious mechanisms of thought that promote incorrect conclusions. Fads gain adherents because they seem promising, even when based on false reasoning. These errors have been the subject of a large body of scientific research, particularly in the new disciplines of behavioral economics (Ariely, 2008; Thaler, 2015) and cognitive science (Kahneman, 2011).

Fads and fallacies can also lead to people losing a great deal of money, as demonstrated by the regular periods of financial turmoil that have been driven by misjudgments and unjustified optimism (Taleb, 2007). What research most often shows is that many opinions and judgments are based on emotion, not reason, and that arguments are used to justify conclusions that have already been reached. This explains why it can be a waste of time to try to change another person's mind by arguing—whether in politics and religion, or in scientific debate.

Some of the most important cognitive errors derive from preconceived beliefs. The idea that we discover the truth from reason is beloved of philosophers. Yet there is good evidence that people adopt a view of the world based on intuition, not data, and that preconceived ideas shape their perceptions of reality (Haidt, 2012).

Many decades ago, Festinger (1957) introduced the term *cognitive dissonance* to account for how people explain away discrepancies between their expectations and unwelcome facts. He studied how followers of a failed prophet became even more fanatical when their prediction about the end of the world did not come about. Once they were committed to a point of view, it was hard for them to admit they had been wrong or foolish. Instead, they "doubled down," holding on to their original opinions more strongly than ever. (They explained the failure of the prophecy as being the result of their intense prayers.)

Strangely, scientists sometimes do the same thing. When presented with contrary evidence, they may find a way to explain why the data prove they were right in the first place, or why contrary data cannot be relied on because of methodological flaws (Ritchie, 2020). Of course, since hardly any study is definitive, one can easily play that game. And if highly trained researchers can sometimes be fanatical, those trained as clinicians are

even more likely to be credulous. Practitioners with strong beliefs about the effectiveness of certain treatment methods can be very good at finding ways to explain away contradictory evidence.

The general term used to describe these phenomena is *confirmation bias* (Oswald and Grosjean, 2004). Once you have already made up your mind, new information is interpreted in the light of preconceived ideas. One might think that this kind of error should not happen in research, where data, at least in principle, should be the final arbiter. As the nineteenth-century biologist Thomas Huxley is thought to have said, "many a beautiful theory is killed by an ugly fact." Unfortunately, some scientists hold on to favorite theories with religious fervor.

Many researchers will have had the experience of encountering difficulty in publishing results that challenge a current consensus or paradigm. Peer review is a necessary part of science, but can sometimes be used by experts who do not want data contradictory to their own views to be published. Thus when a submitted scientific paper challenges a broadly held consensus (i.e., is "counter-intuitive"), the immediate reaction of a peer reviewer could be negative, an intuition that can easily be backed up by pointing out inevitable shortcomings in research methods. When I was a journal editor, I sometimes made the mistake of asking colleagues with fixed ideas to review papers that they disagreed with, requiring me to search out more balanced opinions. I have seen peer reviewers demonstrate their scientific potency by tearing apart papers that do not support their own ideas (or that simply fail to quote their work).

Similarly, anyone who has ever attended a scientific congress can attest to the way that researchers hold on to favorite ideas for dear life. During the wait for older scientists to be replaced by younger ones, incorrect conclusions can linger on through simple inertia.

Mahoney and DeMonbreun (1977) carried out a striking empirical study of confirmation bias in the peer review of scientific papers. They sent the same submission to 75 expert readers, modifying only what the data showed. The results revealed that reviewers had a much more favorable opinion of studies with findings that confirmed their own theoretical views, and a poorer opinion of those that disconfirmed them. In another provocative study, researchers sent out several classical high-quality research papers from years in the past under different names (Peters and Ceci, 1982). Only a few journals recognized the deception, and 89% of the submissions were rejected on methodological grounds.

The same process occurs in grant submissions. I have known researchers who spend almost as much time predicting who their reviewers will be as on writing a grant proposal. If a hypothesis seems too controversial, they may withhold the submission. (Some colleagues have told me that they prefer to transfer funds from another grant.)

Kahneman (2011) published a widely read book that described a very broad range of cognitive biases, one of which is the *availability heuristic*. In that scenario, error results from depending on what comes easily to mind, rather than on what is most probable. Even the most intelligent people tend to be impressed by a lively anecdote or a recent personal experience. But as the witticism goes, "the plural of anecdote is not data."

This type of cognitive bias tends to afflict clinical practice. For example, practitioners may remember something that happened to a recent patient, but fail to bear in mind that the most striking observations tend to be rare. If you have just seen a series of patients with a particular diagnosis and have given them a certain therapy, you may be tempted to

view future patients as having the same condition and requiring the same intervention. I shall show later in this book how many patients receive incorrect diagnoses, such as major depression, bipolar disorder, post-traumatic stress disorder, or attention-deficit hyperactivity disorder (ADHD), leading to incorrect forms of treatment.

The human mind is programmed to find patterns in the world (Bloom, 2004), a phenomenon that Shermer (2012) has described as "patternicity." Sometimes people see hidden faces in natural landmarks. In medicine, any explanation tends to be better than none. When I was a young teacher of psychiatry, I passed on many of these "just-so stories" to my trainees. Since I believed them myself, my enthusiasm made me a popular teacher. Today, embracing a scientific culture of doubt, I find myself telling students that we have a very limited idea of why our patients fall ill—and we often don't quite know what to do for them. The price I pay for greater humility is being less popular as a teacher. Colleagues who have an answer for everything are more attractive.

Establishing Cause and Effect

The simplest cognitive error concerns the nature of cause and effect. The basic fallacy is *post hoc, ergo propter hoc* (i.e., "after this, therefore because of this"). In plain English, correlation does not prove causation. Although everyone understands this principle, it is surprising how often it is flouted. As a journal editor, I (along with my peer reviewers) have often had to remind authors to tone down their conclusions and avoid making causal inferences from simple associations. This happens all too often in research, and is an even more serious problem in practice. It is one of the main reasons for the "replication crisis" in medical and psychological research (Witkowski, 2019).

The same problems arise in clinical work. Consider one of the most common errors in medicine—attributing change in a patient's condition to the most recent intervention. A physician prescribes a drug, after which the patient rapidly gets better. Is that not good enough? Unfortunately not. There can be other reasons for patient improvement. One is spontaneous remission. Another is the natural course of disease. Still another is a change in life circumstances or the removal of a risk factor. Physicians make this mistake because they want to believe that what they do is of value.

Patients also like to think in this way. If they get better after taking a medicine, they assume cause and effect. If they get worse after taking (or stopping) a medicine, the same assumption is made. Maybe the drug worked, but it is also possible that all you are seeing is a coincidence or a placebo effect. You cannot be sure about causation unless you conduct an experiment with proper controls. That is why clinical trials are so essential for guiding the practice of medicine, and, given the complexity of the questions asked, one should always wait for replications and meta-analyses.

Physicians want to believe that clinical problems are treatable, and they tend to stick with the treatments they know. They like to make diagnoses that offer a basis for providing such treatments. In other words, if all you have is a hammer, everything looks like a nail. On a broader level, problems in determining cause and effect lead to a profound misunderstanding of the causes of illness.

The idea of a single cause for a single disease is attractive. That model has been based on infectious diseases, in which Koch's postulates for identifying a specific organism led to the discovery of many effective treatments for infections. In fact even that example is misleading, as it fails to take into account resistance factors that determine whether even

the most virulent infections become pathogenic. Only a few diseases in medicine have a single cause.

The multifactorial nature of illness gives physicians trouble. The human mind is programmed to favor single causes and single effects. But the real world is different. To take multiple factors into account, there has been a vast change in the way that research data are analyzed statistically. When I was an undergraduate, we learned to carry out *t*-tests and chi-squares. Today, journals may not accept submissions unless the analyses are multivariate. This is because we need to know how much of the variance in a study is captured by each variable. I have come to the conclusion that the world as a whole may be a kind of multiple regression. Everything has many causes, and few effects are easily predictable.

Over-simplification of complexity also affects research. When a single risk factor is identified, whether it is biological or psychosocial, it can sometimes be seen as the cause of an illness. That conclusion is usually wrong. A good example concerns attempts to find viral infections in patients with chronic fatigue, which may not play any causative role in the syndrome, but can be triggers that contribute to the overall burden of risk, are secondary effects, or are incidental findings (Afari and Buchwald, 2003). This illustrates the danger involved when medicine views every disorder in the light of biomedical reductionism.

Another example, closer to my own area of research, is the idea that if a patient has a history of childhood trauma, that must be the main cause of adult psychopathology. But, as I shall discuss in Chapter 2, this conclusion fails to consider either that traumatic events tend to occur when other risk factors are present, or that most children who are exposed to trauma do not develop a mental disorder as adults.

In summary, disease is not a software glitch that only needs to be tweaked. Complex interactions between multiple risk factors and protective factors require complex forms of treatment.

Fads, Fallacies, and Good Intentions

Many years ago, when I was in training on an inpatient ward, I suggested to one of my teachers that since schizophrenia has a strong genetic basis, psychotherapy for psychosis can have only limited therapeutic value. His reply was "If I accepted your view, I would have to consider the condition hopeless." But my teacher's assumption that genes strictly determine outcomes was wrong. Even in the most severe illnesses, genes are rarely the only determinant of outcome, but can be best understood in a gene–environment interaction model (Paris, 2022). Moreover, research in epigenetics describes mechanisms by which the environment can switch genes on or off (Szyf et al., 2009).

My teacher's comment also made me realize how important hope is for clinicians. He was a psychoanalyst who, unlike most of his colleagues, had spent his life treating some of the sickest patients. He sincerely believed that if one had enough skill, anything was possible. But this led him to make mistakes, such as treating two colleagues suffering from bipolar disorder with psychological therapy.

By and large, medical fads arise when practitioners have good intentions but lack knowledge. For reasons of training, idealism, and professional pride, physicians passionately want to help their patients. Although they often succeed, most are less comfortable

with chronic illness than with acute disease. Managing chronicity requires patience and an acceptance of limitations.

When I was a medical student, a painting was prominently displayed at the entrance to the faculty building. It showed a heroic physician at the bedside of a child, physically struggling with a skeleton representing death. This is an image that many medical graduates want to believe in. Doctors aim to conquer disease, even as they learn that this is not the only service they can provide to patients. In a famous saying (attributed, like so many other sayings, to Hippocrates), the role of the physician is to cure sometimes, to treat often, but to care always.

Medicine and Science

The idea that medicine should be practiced on scientific principles is relatively new. Over most of history, clinical work was more of an art than a science. In the past, bleeding and purging killed many patients, and people were much better off avoiding physicians than seeking their advice.

Then, in the late nineteenth century, pathology and bacteriology developed methods to confirm many medical diagnoses. But treatment methods in medicine were still not particularly evidence based. Practice was based on clinical experience, or on the consensus of experts. There was no formal concept of evidence-based medicine (EBM) until the mid-twentieth century. Even today, it is not possible to conduct practice entirely on the basis of empirical data. The evidence we have is rarely conclusive, and quite a few important clinical questions have never been studied.

Scientific medicine is associated with advances that have reduced the burden of disease and increased the human lifespan. But it is still necessary to ensure that empiricism sets the rules for further progress. Medical journals set standards for what is accepted as scientific. But as empirical data became required for deciding what diagnoses are valid and what treatments are effective, the bar was constantly being raised. Peer review is much more critical today. Many (if not most) articles that were published 25 years ago would be rejected today. We expect larger, more representative samples, and journals have statistical consultants to ensure that the most advanced methods of analysis have been used. The top journals pride themselves on a high rejection rate, which can approach 80–90%.

When perusing medical journals of previous generations, I observe a very different standard. Journals in the past were replete with papers whose methods were almost entirely unscientific and unreliable. One still sees these kinds of publications in lower-impact journals—case series without control groups, associations reported as percentages, with few hypotheses and a lack of formal statistical testing. For many years, prominent journals still published reports of single cases, from which one can conclude almost nothing. (Case reports can occasionally be heuristic if they generate hypotheses that stimulate more systematic research, but most journals refuse to publish them, and those that do may put them in the letters column.)

Yet 50 years ago the case series was one of the commonest types of article published in journals. A physician would describe and report outcomes for a number of patients. There would be no control group for comparison, and no effort to show that the samples were in any way representative of larger clinical populations. If the article went on to

propose an association between an etiological factor and a disease, one had no way of determining whether the observations were of real significance, could be explained in some other way, or were only chance findings. If the article described a new method of treatment and claimed it was effective, it tended not to be replicated, since so many such reports were based on unrepresentative samples, or in fact described placebo effects. This change has produced angst among researchers who now have more papers rejected. But setting a high bar is good for both medical science and clinical practice.

The stakes of publishing misleading data in medical journals are higher than they are in the basic sciences. If physics, chemistry, biology, or academic psychology produce faulty research findings, no one other than the authors ends up being adversely affected. But patients can suffer real harm when medical researchers get things wrong. Sometimes, as in the claim that certain vaccines cause autism (Taylor et al., 1999), public health can be put at risk. To prevent incorrect and dangerous conclusions, peer review must assure that research methods were properly followed, and that conclusions are justified.

Even under the stringent standards of modern medical journals, papers will be published that eventually prove to have misleading findings. In most cases, unrepresentative and/or insufficiently large samples are the problem. This may be why, as Ioannidis (2005) has shown, most articles published in medical journals are never replicated. Dr. Ioannidis may have shocked the world of medicine, but he helped to support a review of practices that have led to more doubt as to whether findings from a single scientific paper reflect what one would see in larger and more representative samples.

Failure to replicate results is also the main reason why the media so often get medical science wrong. They are looking for a story, particularly anything that looks like a dramatic breakthrough. When a new finding comes out in a top journal, medical reporters tend to jump on it. We are unlikely to learn that, later on, the results were not replicable. About 30 years ago, one of my colleagues became briefly famous for finding a gene that he claimed strongly determined a major personality trait. The finding was duly written up in *Time Magazine*, but no one was ever able to replicate it. The media did not consider non-replication to be a story, and never returned to the subject.

For the same reason, we should be cautious about the expert consensus that lies behind formal treatment guidelines, even the most high-quality recommendations. In my opinion, the guidelines published by the National Institute for Health and Care Excellence (NICE) in the UK are more reliable than anything produced in North America. (This could be because British culture has historically, at least until recently, valued empiricism and common sense over hype.) The other major contribution of British medical experts (in collaboration with Canadian ones) has been a series of Cochrane reports that are considered to be a gold standard for evaluating treatment. But Cochrane is so rigorous that it typically concludes that not enough research is available to lead to strong conclusions. Moreover, although NICE guidelines and Cochrane reports represent the best we can do at any given time, they have to be regularly updated to serve as a guide to practice. Many of them become dated within a decade or so.

An internist once remarked: "the consensus of experts has been a traditional source of all the errors that have been established throughout medical history" (Feinstein, 1988). But imagine the plight of physicians of the past, who had nothing to rely on but the opinions of senior clinicians and unscientific reports in medical journals. Let us go back

in history and consider examples of how practice, when not evidence based, went seriously astray.

Medical Fads in Historical Context

When I was a student, working at a summer camp in a remote area of Canada, and reading by kerosene lamp, I encountered a copy of William Osler's *The Principles and Practice of Medicine*. This famous textbook, first published in 1892, eventually went into an eighth edition (Osler, 1916), and was still an instructive read in the 1970s. Osler acknowledged how little was then known about illness, or how to treat it. That made him ahead of his time in many ways. He criticized the fads of his era, and he did not approve of a "shotgun" approach in which every symptom was treated with a separate drug. (Unfortunately, that kind of practice remains common today.)

Reading a book originally published before the First World War made me wonder what our own textbooks will look like 50 or 100 years hence. This is an example of how the history of medicine offers a useful perspective on present errors. Future readers may shake their heads, wondering how physicians of the twenty-first century could have so frequently misunderstood and mismanaged disease.

Physicians of the past were foolish to rely on bleeding and purging as mainstays of therapy for so many diseases. But they lived in a climate of opinion in which the most respected members of their profession promoted these practices. American psychiatrists still admire Benjamin Rush, a signer of the Declaration of Independence. But Rush advocated bleeding and purging to the very end of his days, killing many patients along the way (Fruchtman, 2005). This was down to ignorance, but the failure on the part of medicine to challenge such ideas is a chilling historical fact.

In another cognitive error, called "group-think" (Janis, 1972), people adjust their views to those of peers with whom they work. It is difficult to stand against received wisdoms without being branded a renegade. Practitioners generally accept the consensus of their colleagues unless they are familiar with alternative options.

A good example is pharmacology. Even in the past, drug treatment was aggressive, despite the fact that most agents available in the nineteenth century were not effective. An American physician of the time, Oliver Wendell Holmes, is believed to have once said, "If the entire pharmacopeia were thrown into the ocean, it would be much better for mankind and much worse for the fishes." Only a few drugs from the twentieth century have stood the test of time, the most prominent being digitalis and morphine. Even today physicians may prefer to prescribe ineffective agents than to stand around helplessly in the face of serious illness.

Once diseases are understood, therapy becomes more rational. Even prior to the era of antibiotics, once the organisms that cause infectious diseases were identified, patients were less likely to receive treatment that could make them worse. Often, once effective therapy became available, fads died out entirely. That was the ultimate reason for the disappearance of bleeding and purging. And most drugs of unproven value disappeared once physicians had access to a modern pharmacopeia.

Diagnostic Fads in Medicine

The most common and most intractable symptoms with which patients present to physicians are those that are particularly likely to attract fads. I need not discuss the

endless number of diets that have been promoted by physicians over the years. Food fads have never entered mainstream practice, although some physicians have made fortunes recommending them. One could also write volumes on the various methods used to manage chronic insomnia, some of which also lie beyond the boundaries of medicine. But let us narrow our focus to two of the most frequent presentations seen in any physician's office: unexplained fatigue and unexplained chronic pain. These common but often intractable problems tend to elicit faddish remedies.

Chronic fatigue syndrome (CFS) has been the subject of intensive research and serious controversy for decades, and the diagnosis remains controversial (Holgate et al., 2011). As defined by the Centers for Disease Control and Prevention in the USA, CFS is characterized by persisting or relapsing fatigue for at least 6 months, cannot be explained in other ways, and is associated with four other symptoms from a list that includes post-exertional malaise, impaired memory or concentration, unrefreshing sleep, muscle pain, multi-joint pain without redness or swelling, tender cervical or axillary lymph nodes, sore throat, and headache.

Attempts to define CFS in other ways, such as by the term "myalgic encephalopathy (ME)," assume that a specific biological etiology has been found. But these claims have never been proven or replicated. Viral infection need not be the main cause of the syndrome, although it seems to be a trigger. Holgate et al. (2011) concluded that post-viral fatigue ends up being chronic, either because of psychosocial stressors or due to other unknown factors. Thus chronic fatigue is not an infectious disease, as claimed by some patient advocates who want to legitimize their suffering, but an abnormal response of the immune system to infection, leading to a failure to recover.

No evidence-based treatment for CFS has ever been established. This is not surprising, given that the syndrome is quite heterogeneous. For a time, the idea that fatigue might be due to low blood sugar, even in the absence of diabetes, affected practice (Bennion, 1983). Later, Abbey and Garfinkel (1991) concluded that CFS includes cases with unknown organic causes, and others that represent depression, or what nineteenth-century psychiatry called "neurasthenia" (Shorter, 1993), as well as what DSM-5-TR now calls "somatic symptom disorders" (American Psychiatric Association, 2022).

Another patient symptom that physicians struggle with, namely chronic pain, can take many forms. Today *fibromyalgia* is a common diagnosis in primary care. This syndrome has a specific definition (Chakrabarty and Zoroob, 2007): the presence of widespread pain for a period of at least 3 months, as well as tender points at 11 out of 18 specific anatomic sites. However, no lesions can be found at the tender points, and there is no evidence of any etiological factor—or of any consistently effective evidence-based method of treatment. Like chronic fatigue, fibromyalgia overlaps with somatic disorders (Shorter, 1993), with the important difference that it is more widely accepted within the medical profession. Even so, the concept remains controversial. Like many mental disorders, it presents with symptoms but without signs, and is not associated with biological markers or organic changes. It remains possible that fibromyalgia will eventually go down in history as a medical fad.

Surgical Fads

In the history of medicine, surgery developed many heroic and effective interventions for illness. But some of its procedures have been faddish. I shall focus on one that for decades was firmly in the mainstream of clinical practice, namely *radical mastectomy*.

The history of therapy for breast cancer is a complex story (Lerner, 2001). This fairly common disease can be fatal. It has therefore attracted powerful treatment methods, as well as strong emotions. For over 100 years, the first line of treatment has been surgery. But it was never clear how extensive these procedures should be. Because cancer spreads to lymph nodes and beyond, many surgeons felt that one should go beyond removing the tumor itself.

William Stewart Halsted, a prominent American professor of surgery at Johns Hopkins University, was a pioneer in surgical technique, antisepsis, and effective anesthesia (Nuland, 1988). He developed the technique of radical mastectomy, in which axillary lymph nodes, as well as chest muscles, were also removed. This method was still standard when I was a medical student 60 years ago. But it was already becoming apparent that simple mastectomy, accompanied by radiotherapy and/or chemotherapy, could be equally effective. Long-term follow-up studies that lasted as long as 25 years later confirmed this conclusion (Fisher and Wolmark, 2002).

Why was an ineffective and disfiguring surgical procedure popular for so long? One factor was the prestige of Halsted, a pioneer working at one of America's top medical schools. Despite having an addiction to cocaine and morphine (revealed years after his death), Halsted (1961) convinced the surgical community of the effectiveness of his methods by publishing highly descriptive surgical papers. Yet if control groups had been required, as they would be today, radical mastectomy might never have gained support. Perhaps Halsted's aggressive approach reflected the "can-do" beliefs of American culture—if a disease is dangerous, you just have to fight harder to beat it.

Was radical mastectomy—a mainstream treatment used for many thousands of patients and a standard approach supported by clinical consensus—a fad? I would say yes, because it persisted into an era when better evidence could have been made available. Despite poor scientific support, radical mastectomy spread rapidly and became wildly popular, but eventually disappeared. Perhaps it was the best that medicine had to offer at the time, but Halsted's operation could easily have been challenged in his own time, by following up patients who received it and comparing their outcomes with those who received less radical treatment. It was bad judgment to stick with a procedure that was both aggressive and naively ambitious.

One can readily find other examples of surgical fads. One is tonsillectomy for children, considered a routine procedure for many years. As confirmed by a Cochrane review (Burton et al., 2014), it should retain only a marginal role in practice. The concept that chronic untreated infections anywhere in the body can lead to serious consequences also had some impact on psychiatry, and there was even a brief fad for surgical procedures in mental hospitals, based on the idea that psychosis is due to chronic focal infection or autointoxication. This led to the removal of teeth and even portions of the colon (Scull, 2005). Today, such procedures are unthinkable, and any surgeon wanting to carry them out would lose hospital privileges.

Unfortunately, the problem of determining whether a surgical procedure is effective or necessary is hard to resolve. Even now, surgical practice depends much more on clinical experience than on randomized clinical trials (Balch, 2006). It is much more difficult to carry out research on surgical procedures than on drug treatment. But without randomized controlled trials, accompanied by careful and extended follow-up of patients, one cannot be sure that any surgical method is superior to a less invasive alternative, or is not simply a placebo.

The Challenge of Chronicity

Many medical fads have gone into decline, but new ones continue to appear. The reason is that many diseases are incurable, whereas others are chronic and can only be palliated. Moreover, advances in acute treatment mean that most of medical practice involves the management of chronic illness.

When diseases are progressive but remitting, fads are likely to develop. Multiple sclerosis (MS) provides an excellent example. Most patients have remissions, but over time they get gradually worse, and the ultimate outcome tends to be fatal. This course of illness has led to a range of faddish treatments for MS, including one that involved an untested form of vascular surgery (Kolber et al., 2011). None of these therapies has ever been shown to affect the course of the disease. The fluctuating course of MS, with sudden and surprising periods of improvement, can fool physicians into thinking that their interventions are responsible for changes. As we shall see, many mental disorders have a similar course, leading both patients and their physicians to explain any improvement on the basis of the most recent intervention. This is another example of how cognitive biases can affect medical judgment.

Patient Advocacy

In the contemporary world, patient advocacy groups, working through the Internet, aim to raise awareness of chronic diseases and attract funds for research. This is a positive development, and I have been involved with one such group (for borderline personality disorder). We live in an era in which patients are more actively involved in treatment decisions. That is also is a good thing. Many patients are looking up their diagnoses online, which gives them a chance to understand their illness and its treatment in more detail.

The problem is that the Internet allows uninformed groups of consumers and patients with a strong agenda to "flood" search engines with dubious ideas, sometimes supported by instant "experts" (some of whom are celebrities rather than professionals). This is what happened in the case of the anti-vaccination campaign, based on a claim by the British physician Andrew Wakefield that measles, mumps, rubella, and pertussis vaccines can cause autism (Goldberg, 2010). The story became an international scandal when, after systematic research, it became clear that no such relationship exists. Unfortunately, several physicians became involved in this malignant fad. Moreover, many parents refused vaccination for their children, which led to an outbreak of several entirely preventable diseases. Thus, like so many other things, advocacy can be double-edged. When conducted with professional support, it offers help to patients and families in need. When linked to a fad, getting information from the Internet can do real harm.

How the Pharmaceutical Industry Promotes Fads

The pharmaceutical industry has played an important role in promoting medical fads (Goldacre, 2013). To understand why, we need to understand the relationship between "Big Pharma" and practitioners. Ideally, they should work in partnership. Both aim to use drugs to treat disease. Positive results benefit patients and please physicians. In such cases, drug companies should make a legitimate and well-deserved profit. However, this rosy scenario is far from current reality. One reason for this is the high margin of profit for drugs.

Moreover, medicine—particularly academic medicine—has suffered from being too close to industry (Angell, 2004). Pharmaceutical corporations, which used to be small, are now large and among the most profitable companies in the world. Although some drugs have benefited large numbers of patients, "Big Pharma" is reluctant to invest billions in developing new agents, preferring to make "copycat" drugs that resemble those already available. Even if only a single atom is changed in the molecule, once government approval is granted a new agent can be marketed to physicians, supported by an aggressive campaign. The industry spends large amounts of money on marketing, much more than on research. It also benefits from new diagnoses that are associated with drug treatment, so that its influence may also extend into diagnosis.

The dynamic behind the relationship between the pharmaceutical industry and physicians depends on the way new drugs make money for companies. Older drugs, particularly when they are out of patent and "go generic," yield little profit. Thus pharmaceutical representatives have the task of convincing practitioners to adopt the latest agents. They provide physicians with gifts (Wazana, 2000), and they recruit medical opinion leaders to influence prescription practices (Angell, 2004). These academics may receive "consultant fees" to actively promote a product. Moreover, an army of pharmaceutical representatives establishes personal relationships with practitioners, sometimes paying for dinners at fine restaurants.

Yet older drugs are often as good as (or better than) newer agents. Twenty years ago, a large-scale study (ALLHAT Collaborative Research Group, 2002) showed that the classical diuretic chlorthalidone is more effective for hypertension than any of the current favorites (ACE inhibitors or calcium-channel blockers). Similarly, acetylsalicylic acid may be as effective as any current alternative in reducing the risk of developing cardiovascular disease (Gaziano et al., 2006). (In fact, none of the alternatives are that effective.) These findings have been published in top medical journals, but have had little impact on practice. Physicians seem to have an almost irresistible attraction to "the latest thing" in drug therapy.

Some authors defend a close relationship between industry and academic medicine on the grounds that it promotes research into the development of new drugs (Goldberg, 2010). But although effective collaborations do occur, they are relatively rare. Some academic physicians conduct clinical trials run by industry, which designs protocols that are most likely to support its products (Goldacre, 2013). But industry would rather spend its money on drug promotion than on more research. One result is that practicing physicians are constantly encouraged to embrace the latest diagnosis and the latest treatment.

This is not to say that every new idea is wrong, but that medicine is being practiced in a climate that almost inevitably promotes fads. We cannot blame industry for this problem. It is the result of our own unjustified enthusiasm. The next few chapters will show how these problems have infected the theory and practice of psychiatry.

Chapter 2

Psychiatric Fads: Past and Present

Psychiatry may be somewhat more prone to fads than other specialties in medicine. I don't have data to prove that this is true, and it is possible that I only see things this way because I know more about my own specialty. However, this perception has been shared by noted colleagues (Frances, 2009; Carlat, 2010), and also by well-informed observers outside the field (e.g., Horwitz, 2002, 2021).

If psychiatry *is* prone to fads, why should that be the case? One explanation could be that even as the demand for our services remains high, the origins of mental illness remain poorly understood. Although some domains of medicine can be equally mysterious, disorders of the mind have a unique complexity that will take decades to unravel. Another explanation is that since mental disorders can be hard to treat, they are more open to faddish "quick fixes."

Yet, as shown by Leucht et al. (2012), effect sizes in psychiatric practice (derived from meta-analyses) compare favorably with those in other medical specialties. Moreover, we are not the only physicians who deal with chronic illness. The problem is that because not all of our patients do well, mental health practitioners can be too keen to find remedies that promise much but do not deliver.

Moreover, even though current treatment methods help many patients, we do not have a good understanding of how or why they work. Given that the etiology of most mental illnesses is unknown, it is rare (or impossible) to develop interventions based on specific chains of causality. Since causality in psychiatry is highly complex, the field is vulnerable to people with too many bright ideas. And usually one cannot prove them wrong, at least not initially.

In this light, it should not be surprising that the history of twentieth-century psychiatry is replete with theories and treatment methods that claimed to be breakthroughs, but that did not stand up to scrutiny. Moreover, each time a diagnostic fad, an etiological fad, or a treatment fad died out, a new one appeared. To shed light on the current state of psychiatry, it is instructive to study the history of this specialty to see how these fads developed and disappeared.

Psychiatry in the Early Twentieth Century

To understand the problems facing psychiatry today, let us go back more than a century, to the year 1912. In an annual report to the *British Journal of Psychiatry*, an American psychiatrist, William MacDonald, described only one major area of progress, the impact of which was much greater than was originally thought. This was the observation that new testing procedures had identified *Treponema pallidum* in the brains of many

patients in mental hospitals (MacDonald, 1913). Now psychiatrists could more accurately separate syphilis from "functional" psychoses, a finding that later—in the age of antibiotics—provided a basis for specific treatment. Yet it did not affect the diagnosis and treatment of most mental disorders, whose fundamental nature remained unknown.

Psychiatry itself was not yet a well-developed discipline. It only became independent from neurology at the end of the nineteenth century. For many decades, mentally ill patients were segregated from other patients (Berrios and Porter, 1995). Unfortunately, these mental hospitals, which were originally designed to be more humane than the older general hospitals, quickly became overcrowded and dysfunctional. Moreover, patients received little treatment there—at best they could wait for a spontaneous remission, but some stayed for years, or even a lifetime.

From the beginning, many practitioners hoped that mental disorders could eventually be understood as diseases affecting the brain (Shorter, 1997; Wallace and Gach, 2008; Harrington, 2019). Yet research failed to demonstrate biomarkers or lesions specific to any diagnosis. At the beginning of the twentieth century, an unbiased observer might have concluded that much of psychiatry fell outside the mainstream of modern medicine. William Osler's *The Principles and Practice of Medicine* (Osler, 1916) included a short chapter on mental illness, mainly to say how little was known. Thus therapeutics was limited by ignorance. Meanwhile, treatment methods in asylums were primitive and mostly consisted of sedation.

To be fair, although medicine was ahead of psychiatry in its theoretical understanding of disease, its practice (outside of surgical procedures) was not always effective. Even so, the nineteenth century was a time when the science of medicine was making dramatic progress. By the beginning of the twentieth century, the understanding of many diseases was moving briskly forward.

Now advances in practice could be based on research. Medicine in 1912 could take advantage of X-rays, bacteriological culture, and biochemical tests. But since no mental disorder was associated with biomarkers, psychiatry could not make use of any of these tools. It was forced to rely on a tradition of "phenomenology"—that is, the detailed description of clinical symptoms (Jaspers, 1913/1997). Like their neurological colleagues, psychiatrists were better at describing illness than at treating it.

Mental hospitals were large institutions, usually built outside major cities. As mentioned earlier, patients could spend years on these wards, and sometimes remained there for the rest of their life. Then, as now, the main reason for admission was psychosis. But the classification of severe mental disorders was confused and undeveloped. A German academic, Emil Kraepelin (1856–1926), hoped to improve diagnosis through systematic clinical observation and follow-up. At that time the world's leading psychiatrist, Kraepelin wrote standard textbooks that promoted this perspective. But given the tools at hand, he could make only limited progress.

In the nineteenth century, pathologists had learned how to slice and stain tissues so that they could examine them under the microscope. This method proved crucial to the understanding of many diseases. But histopathology failed to account for the etiology or pathogenesis of mental illness (Wallace and Gach, 2008). (In those cases where it could, as in syphilis, the disease moved to another specialty.) Pathologists failed to find any consistent pattern of lesions or cellular changes in the brain that were similar to those identified in other organs of the body. And although psychosis was understood to be

hereditary, too little was known about genetics to be of practical value. Imaging, at that time confined to skull X-rays, also added little to the understanding of psychopathology.

The treatment of psychoses has always been mainly biological. But the best drugs available in 1912 were sedatives—bromides, chloral hydrate, and paraldehyde (Shorter, 2009). The introduction of barbiturates did not add greatly to this armamentarium. All of these agents calmed patients but had no direct effect on psychotic symptoms. Thus practice was characterized by an acceptance of chronicity and a fair degree of therapeutic nihilism. There was little to offer to severely ill patients, other than institutionalization while awaiting remission.

In summary, the psychiatry of 1912 lagged behind the mainstream of medicine. We can feel sorry about the relative ignorance of our predecessors. Yet even today we still do not know the causes of most forms of mental illness, and we cannot identify cellular, genetic, or imaging findings specific to any diagnosis. It is possible that by 2112 our descendants will be doing better.

I shall now illustrate the history of fads and fallacies in psychiatry through examples drawn from three different domains, namely psychoanalysis, psychosurgery, and electroconvulsive therapy (ECT).

Psychoanalysis became a fad in the mid-twentieth century, despite the lack of evidence for its theory or practice. It was mistakenly offered by some practitioners as a cure-all. Still, as we shall see, the efficacy of brief psychodynamic therapy, in which patients are treated using some of the same principles, but seen once weekly over several months, has been supported by research (Abbass et al., 2014).

Psychosurgery in its original form (frontal lobotomy) was a very dangerous fad. It has now mostly disappeared, but there are still a few indications for brain surgery (Mahoney and Green, 2020), or for the implantation of electrodes (Bormann et al., 2021), that are supported by evidence.

ECT is a special case. If used properly, it is one of the most effective treatments in psychiatry (Abrams, 2002). But in its early days, before the appearance of antidepressant drugs, ECT temporarily morphed into a fad.

Psychoanalysis As a Fad

Sigmund Freud was trained in neurology, but his clientele and his interests belonged to another tradition, dating back to the time when practice was conducted in offices. He developed a wide-ranging and ambitious theory and therapy to treat "neurosis" (i.e., symptoms that are troubling but not disabling), which became the niche for psychoanalysis (Freud, 1916/1958). His ideas were received with cautious interest by other physicians, and even earned a page in Osler's *The Principles and Practice of Medicine* (Osler, 1916).

Neurotic patients, if they had the money, paid for their treatment. But this kind of office practice, isolated from the feedback provided by academic institutions and critical colleagues, was a place where fads could thrive. Since the time of the Austrian physician Franz Anton Mesmer (1734–1815), charismatic clinicians have promoted all kinds of therapies for neurotic problems, including hypnosis, "rest cures," and the harnessing of willpower (Ellenberger, 1970). Freud thought he had something better. Yet, in most ways, he turned out to be wrong (Paris, 2019).

Psychoanalysis was based on an all-embracing theory of human psychology, and it made great claims as a therapeutic method (Hale, 1971). Developed by a charismatic leader, it generated a movement that attracted followers who actively promoted it, both to mental health clinicians and to the educated public. After an initial period of resistance, psychoanalysis became powerful and influential, forming part of the intellectual climate of modernity. Since psychoanalytic theory had implications for the humanities, Freud was often attractive to intellectuals.

In psychiatry, psychoanalysis had its greatest success in the USA (Hale, 1995), which might be explained, at least in part, by some aspects of American culture. Based on its experience of immigration and internal migration, American society was created by people who believed that individuals could change themselves and start a new life. They could be more readily attracted to fads than the British, who have a longer historical tradition. The psychoanalytic movement offered new and radical ideas, which after a few decades reached a position of great influence in academic psychiatry in the USA.

Since early psychoanalytic theories often seemed rather fantastical, many leading European psychiatrists rejected Freud's ideas. Emil Kraepelin wrote:

> Here we meet everywhere the characteristic fundamental feature of the Freudian method of investigation, the representation of arbitrary assumptions and conjectures as assumed facts, which are used without hesitation for the building up of always new castles in the air, ever towering higher, and the tendency to generalizations beyond measure from single observations. I must finally confess that with the best will I am not able to follow the trains of thought of this "metapsychiatry," which, like a complex, sucks up the sober method of clinical observation. As I am accustomed to walk on the sure foundation of direct experience, my Philistine conscience of natural science stumbles at every step on objections, considerations, and doubts, over which the lightly soaring power of imagination of Freud's disciples carries them without difficulty. (Kraepelin, 1921, p. 250)

This was a prescient critique. Psychoanalysis declined when its theories failed to fit into mainstream psychology, which came to demand psychometric precision for its constructs. The movement also declined when it became obvious that its treatment methods were inefficient and had no evidence for their efficacy. By the late 1970s and the 1980s, analysis had fallen out of favor, to be replaced by a new psychiatry based on reliable diagnosis, with roots in neuroscience and psychopharmacology, and by other, more practical methods of psychotherapy.

The question is how psychoanalysis came to gain so much influence. I have written two books about this subject (Paris, 2005, 2019), and will not repeat the details of this complex story here. But let me consider briefly why these ideas became a fad. One of the reasons lay in their intellectual appeal. Like Karl Marx, Freud thought that behind the appearance of phenomena lay a hidden reality that only the initiated could comprehend. The impenetrability of analytic theories and methods, like a mystery that required initiation to explore, was also part of its attraction. Arcane procedures—a couch, a silent analyst, and a high frequency of sessions over a period of years—gave it the aura of a religion. Psychoanalysis was also the subject of intense public fascination, almost from the beginning, perhaps because so much of it was about sex. Finally, psychoanalysis, like Marxism, offered a comprehensive worldview that addressed modern alienation. This was a time when organized religion had fallen into decline. For those who adhered to the movement, it provided a community of believers and a source of hope for the future.

Freud needs to be understood in the context of his times. Although psychoanalysis was based on unproven theoretical assumptions, and offered treatment methods that were never tested, such problems were not particularly unusual. The medicine of 100 years ago was far from evidence based. Even 60 years ago, when I was a student, the authority of professors, relying on clinical experience and personal charisma, took precedence over systematic investigation.

One of the most faddish aspects of the analytic movement was the way that it explained its failures. This is a frequent characteristic of pseudoscience (Popper, 2002; Lilienfeld et al., 2015). Unsuccessful therapy could be attributed to the resistance of the patient and/or not carrying out treatment properly or for long enough. The analytic movement had some typical features of quack medicine—failures do not lead to questioning of one's methods, but prove that one was right all along. This is a classic example of cognitive dissonance and confirmation bias. Analysts were not put off by the lack of evidence that their method was consistently successful. One is reminded of the observations of Festinger (1957) about what people do when they prepare for the end of the world—and when the world refuses to end.

Finally, we live in an era when the demand for mental health care is at a historical high, and therefore we need to focus on interventions that are briefer, are less expensive, and have strong empirical support. But only the wealthy can afford a course of classical psychoanalysis, and many of those who practice this technique see their patients once or twice a week.

The peak of hegemony for American psychoanalysis was between 1945 and 1975 (Hale, 1995). I trained in psychiatry towards the end of that era, and this experience gave me a bird's-eye view of the landscape. Many academic chairs had had training in psychoanalysis, even if few actively practiced it. Freudians had clever strategies for creating and maintaining adherence. Analysts are trained in a way that maximizes belief. In addition to course work and supervised cases, they have to undergo their own treatment, usually for 5 years. Once you make that kind of commitment, it is very difficult to admit that you have not made good use of several years of your life. It is also difficult to turn against a training analyst who has devoted so much time to your personal care. Finally, it is difficult to give up strongly held beliefs, and if you disagreed with the theory, you might be accused of being insufficiently analyzed. These methods of thought control parallel those that have been used by religious and political movements throughout history.

Yet despite all of these reasons for accepting a kind of brainwashing, many psychoanalysts rebelled and rejected the movement. Quite a few of the leaders of neo-Kraepelinian psychiatry (such as Robert Spitzer, the editor of DSM-III), and the developers of alternative psychotherapies (such as Aaron Beck, the creator of cognitive–behavioral therapy, or CBT), were former analysts. Many therapists have kept the best part of Freud (listening to people attentively) and dropped the worst part (unproven theories and methods).

I work in a department where a few psychoanalysts still practice, although most of them are now well into middle age. Few young psychiatrists are interested in analytic training. Yet some of my students, despite the stronger scientific education now provided in medical schools, are still fascinated by these ideas. They want to understand the human mind in ways that biological psychiatry does not address. Moreover, analytic

teachers spend more time with students, and show more personal interest than other faculty members.

It was much the same in my time. Like many of my contemporaries, I spent a couple of years lying on an analytic couch. But I was a fellow traveler, not a party member. I did not make a clean break with psychoanalysis until later, when I came to question one of its basic paradigms—that adult problems are largely rooted in childhood experiences.

I had seen many patients who were troubled despite having had a fairly normal childhood, and many who were functioning well despite having had a terrible childhood. I began to read the research literature on developmental psychopathology, and was deeply influenced by the ideas of the British child psychiatrist Michael Rutter, who saw psychological development as complex, interactive, and responsive to a multitude of vulnerabilities and life events (Rutter and Rutter, 1993). I wrote a book on why the theory of childhood determinism is mistaken (Paris, 2000), and another on what research actually shows about the relationship between early experience and adult functioning (Paris, 2022). Even so, it was difficult to give up concepts that had guided my work for so long, and that seemed to explain so much.

When one writes a critique of Freud and his system, editors tend to assign the book review to a psychoanalyst, virtually guaranteeing negative comments. This happened to me when Jeremy Holmes reviewed one of my books for the *British Journal of Psychiatry* (Holmes, 2005), and suggested that I sounded like someone who had emerged from a cult. But psychoanalysis *was* a cult. It offered a quasi-religious faith that provided an explanation of the human condition, and a cure for the angst of modernity. These great expectations could only invite disappointment. Moreover, an expensive and unproven treatment that continues for years on end cannot survive in the current climate.

I have to ask myself why my younger self took psychoanalysis so seriously. Although my teachers considered me to be a rebel, I still thought they were older and wiser than me. It was rather like growing up in a religious family. Even if I never became a psychoanalyst, I was surrounded by a powerful climate of opinion supporting these theories. In short, I fell victim to the fad, and it took me years to see the world differently. After all, psychoanalysis did survive for almost a century.

One reason why the movement lasted for so long was that there were few better options. When I was a student, even if one was ambivalent about Freud, if one was interested in talking to patients the only alternative was behavior therapy. But that approach, at least in its original form, was intellectually sterile. Behaviorists denied the existence of the mind, a view that eventually doomed their discipline. That method eventually went into a steep decline, and was gradually absorbed into CBT. Moreover, once psychiatrists had effective drugs to treat mental illness, psychopharmacology and neuroscience became the exciting leading edge of the discipline.

Although later versions of psychoanalysis have generated new ideas, most of these are not much better than Freud's own speculations, and several have already disappeared with their originators. The most lasting offshoot has been *attachment theory*, a testable scientific model developed by John Bowlby (Bowlby, 1973), a psychiatrist at the Tavistock Clinic in London. This model of human development, unlike Freud's, is linked to academic psychology and empirical research, and has attracted a large research literature (Cassidy and Shaver, 2016). Attachment theory is an admirable model in many ways, but it needs to give greater consideration to interactions between genes and environment.

As a treatment, classical psychoanalysis survives today only as a remnant—in enclaves such as the Tavistock Clinic in London and the Menninger Clinic in Texas, as well as in a few private practice settings in large cities. Moreover, psychoanalysis is rarely practiced in its original form. But there is one evidence-based method to which it *has* given birth. This is brief psychodynamic therapy (Leichsenring et al., 2004), which is markedly different. It uses the same theoretical principles, but treats patients over a period of months instead of years. It has been shown to have the same efficacy as the now more standard method of CBT (Abbass et al., 2014; Fonagy, 2015).

The decline of the traditional forms of psychoanalysis is instructive for another reason. Psychiatry had to reject Freud unless it was willing to continue to be treated with contempt by other specialties in medicine, most of whose practitioners viewed psychoanalysis with dismay. Thus my specialty returned to its biological and phenomenological roots, adopting a model that has been described as "neo-Kraepelinian" (Klerman, 1986).

Even so, psychoanalysis has left a legacy, namely the importance of empathic listening. Almost all therapies have made use of this principle. It is no accident that evidence-based psychotherapies, such as CBT, were developed by clinicians who originally trained as psychoanalysts. Unfortunately, this kind of listening is at risk of becoming a forgotten skill among psychiatrists.

Biological Fads: Frontal Lobotomy

Let us now move into a completely different world, namely the application of psychosurgery to the treatment of refractory psychosis. Although one can hardly compare neurosurgery to asking the patient to lie on a couch, some of the reasons why this fad was adopted, and later rejected, mirror the story of psychoanalysis.

Egas Moniz (1874–1955), a Portugese neurologist, is one of the few physicians involved in the treatment of mental disorders to have won a Nobel Prize. But the method he promoted—frontal lobotomy—turned out to be a fad that has long since been discredited. Valenstein (1986) documented this story in detail, and the title of his book, *Great and Desperate Remedies*, nicely summarizes the climate that made lobotomy possible. Later books on the subject (Pressman, 1998; El-Hai, 2007) have come to very similar conclusions.

Sixty years ago, large numbers of psychotic patients could only be managed by confinement in mental hospitals. This desperate situation certainly attracted desperate remedies, and almost anything seemed worth trying. Walter Freeman, an American physician with great ambitions but weak scientific training, toured the country demonstrating a method of frontal lobotomy that could be carried out by clinical psychiatrists (El-Hai, 2007). He would operate with an ice-pick, using a transorbital technique that did not require general anesthesia. He met plenty of opposition, but claimed to get good results and gained adherents. Since lobotomy was offered to patients whose condition was otherwise hopeless, the surgery became widely used—even the hospitalized sister of John F. Kennedy underwent this procedure.

How did psychiatrists, or at least many of them, become convinced that lobotomy was an effective treatment for psychosis? The lesson lies, once again, in the absence of scientific procedures that have now become standard. As discussed in Chapter 1, at that time one could publish articles in prominent medical journals describing a small number

of patients with a given condition who seemed to get better with treatment. Without a true control group, as well as psychometrically valid measures of outcome, one could conclude little. This way of reporting clinical experiences led generations of surgeons to carry out interventions that later proved quite unnecessary. (Even today, surgical procedures are supported by fewer clinical trials than are drug treatments, which require approval by government agencies, whereas hospital colleagues are responsible for regulating the quality of surgical treatments.) The lesson is that medical enthusiasm, even under desperate circumstances, needs to be curbed by accountability. Frontal lobotomy never underwent clinical trials. If psychiatrists had responded to Freeman's claims with the now common retort, "Show me your data," lobotomy might never have become a fad.

It is interesting to compare this story with another that developed at the same time, namely the use of insulin coma to treat psychosis. That intervention was widespread in the 1940s and 1950s, and I saw it used when I was a medical student. Patients were given enough insulin to render them unconscious, and were then woken up with intravenous glucose. The procedure was far from simple, and required intensive nursing for it to be safe. Then, in a paper published in *The* Lancet, Ackner et al. (1957) showed that insulin therapy had no sustained effect on schizophrenia. Insulin coma turned out to be little more than a very expensive placebo. Once psychiatrists had access to antipsychotic drugs, this practice disappeared completely. These drugs were also the main reason for the disappearance of frontal lobotomy.

There are still a few narrow indications for neurosurgery in psychiatry. Such interventions can be helpful for some patients with non-psychotic conditions. The best known example is the use of more limited psychosurgery (focusing on the cingulate gyrus) to reduce the intensity of symptoms in patients with obsessive–compulsive disorder (OCD) (Brown et al., 2016). Although operating on patients with OCD is not a first-line treatment, it can be used when all other treatment options have failed, but targets in the brain are much more restricted (Dougherty et al., 2002). We should also keep in mind that the brain can be altered without removing any neurons. An example of this is the still experimental work on the treatment of intractable depression using implanted electrodes to modify neural networks using deep brain stimulation (Wu et al., 2021).

ECT: How an Effective Treatment Became a Fad

ECT had been developed in the 1930s to treat psychosis, but was more effective for depression, for which it became the primary treatment in the 1940s and 1950s. It is still used today, mainly for severe depression or melancholia, in which it can be dramatically effective, and is often superior to drug treatment (Fink, 1999). ECT can also be used for some cases of schizophrenia and bipolar disorder, and it has advantages over drugs in terms of safety for the treatment of depression in the elderly (Abrams, 2002).

In some ways, ECT is an "anti-fad," in that it is much *more* effective than most psychiatrists realize. They have been seduced by the romance of psychopharmacology to favor treatments that are designed to alter brain chemistry. We still have little idea why ECT works at the neural level, other than viewing it as something like restarting a computer.

ECT became popular when there were no effective drugs for depression. Once tricyclic antidepressants had been introduced, ECT was less frequently prescribed.

However, when it was almost the only option, ECT had been used indiscriminately, and in the 1950s, patients with depressive symptoms of any kind might receive this treatment (Shorter, 2009).

Thus ECT was not a fad, but a good treatment that was used faddishly. This is also a common problem in psychopharmacology, where good drugs can be made bad through overuse. Part of the story of psychiatric fads is that whenever a treatment is effective for some patients, clinicians want to try it on everyone. Today antipsychotics are being prescribed for a wide range of patients who are not in the least psychotic, in the hope of benefit—practices that are not evidence based (Maher et al., 2011).

ECT works dramatically when given to patients who really need it. As a psychiatric trainee, I was deeply impressed by its effectiveness. Patients who barely spoke a word because they were severely depressed or catatonic would "come out of it," sometimes after the very first treatment. I began to think that this was psychiatry's penicillin. Today, as ECT has been largely replaced by antidepressants as first-line treatment, it is more likely to be used when drugs fail. And it continues to suffer from stigma among those who fear what psychiatrists can do to the mind.

ECT was at the center of a serious scandal at my own medical faculty (McGill University in Montreal). McGill is one of the leading medical schools in Canada, where physicians from William Osler to Wilder Penfield have made their reputation. When I served as a department chair, over 40 years after the event, the media were still coming around to ask how such terrible things could have happened. Several books have been written about this story, but the best is Anne Collins' *In the Sleep Room* (Collins, 2002), which places the scandal in historical perspective. I shall closely follow her narrative here. Collins showed that when few effective treatment options are available, as was the case before the development of effective drugs, fads are more likely to take hold.

The man behind this story was D. Ewen Cameron (1901–1967), a Scottish-American physician who founded the McGill Department of Psychiatry in 1943. Cameron became one of the world's most eminent specialists, serving as President of the American Psychiatric Association, and a founder of the World Psychiatric Association. He was an ambitious man who wanted to make his mark in his field, aspiring to a Nobel Prize in Medicine.

In the 1950s, Cameron began to treat patients with a method he called "de-patterning." This involved giving massive doses of ECT (up to 100 treatments). Cameron's idea was to turn a side effect of ECT (memory loss) into a therapeutic effect. He thought that doing so could remove memories and close down dysfunctional brain circuits, to be replaced by new and healthier patterns of thought and behavior, created by "psychic driving"—a procedure in which patients were exposed to hours of recorded messages.

Cameron's therapy was bizarre and unscientific, even by the standards of his time. At the first World Psychiatric Association meeting in Canada in 1961, when I was a medical student, visitors were already criticizing the "Montreal horrors." In fact, de-patterning has never been shown to have *any* long-term effect on the course of patients' lives and illness, other than wasting time and failing to provide effective therapy (Schwartzman and Termansen, 1967). But following Cameron's "mad scientist" procedures, numerous lawsuits against the hospital were filed, leading to large payouts for compensation.

The story took a more bizarre twist in 1977, 10 years after Cameron's death, when it was revealed that some of the funds for the project had been provided by the Central

Intelligence Agency (CIA). Naturally, the media loved this angle. The American government of the time was concerned about the possibility that captured soldiers might be "brainwashed" into revealing secrets, and it was claimed that this had happened to prisoners of war in Korea. (A fictional treatment of that scenario gained currency in the film *The Manchurian Candidate*.) But since de-patterning did not actually do anything, it would have been of no use to spies. Nonetheless, the story was kept alive for decades by litigation, while the media gobbled up the CIA connection.

When psychiatrists had little to offer many of their patients, de-patterning, like frontal lobotomy, proposed a radical cure that aroused radical expectations. Even in the context of the time, none of this would have happened if proper scientific procedures had been followed. After the Second World War, randomized controlled trials were already being conducted on medical treatments. But Cameron, a notably arrogant man, would confront his critics by saying "Are you suggesting I don't know how to do research?" In fact, Cameron had no idea how to do proper scientific work. But neither did most of his colleagues. However, he was too powerful to be effectively challenged. The psychopharmacology pioneer Heinz Lehmann, one of my great teachers, kept out of Cameron's way by working at a mental hospital several miles away.

One of the lessons of this story is about the abuse of power. Because Cameron was a master of medical politics, he became a psychiatric superstar, and this position enabled him to promote a malignant fad. A final irony is that Cameron, like most of his contemporaries, believed in the environmental causation of mental illness, but proposed an aggressive biological intervention to remove maladaptive behavioral patterns and replace them with new ones.

Another lesson of the story concerns the dangers of impatience. Cameron wanted to solve the most serious problems of psychiatry, and to do so rapidly. Ironically, the development of effective psychopharmacology made his efforts at least partially redundant. Today, drugs, evidence-based psychotherapies, and rehabilitation programs have greatly improved the prognosis of even the most serious mental disorders.

Like other medical fads, de-patterning ended in failure. Cameron's hubris met its nemesis, and he himself gave up on the procedure. He left McGill, and returned to the USA in 1963, where he died 4 years later from a heart attack. With poetic justice, Cameron collapsed while climbing a mountain that was higher than he had anticipated.

Even today, this story continues to be used as a stick to beat psychiatry. If only we could say that we have grown out of falling for simple and dangerous answers to the problems of mental illness. Unfortunately, we have not.

Past and Present Fads in Psychiatry

Fads continue to afflict contemporary psychiatry. I shall devote several chapters of this book to fads and fallacies in etiology, epidemiology, diagnosis, and prevention. But the most important fads continue to lie in treatment.

Psychiatrists are reluctant to admit that their therapeutic methods, although sometimes successful, leave much to be desired. We need to carry out more sophisticated clinical trials of existing treatment methods. But to find more consistent therapies, we will need to understand the causes of mental illness.

Psychiatric expertise may not be necessary to treat mild disorders in which symptoms improve naturalistically over a reasonably short time. These patients can be sent to other

professionals, or can be followed with watchful waiting. Another important element in ensuring that these cases are treated effectively is the availability of a multidisciplinary team (Paris, 2008).

However, a specialist is usually required to manage complex and chronic problems, such as severe depression, bipolar disorder, schizophrenia, eating disorders, severe personality disorders, and severe substance abuse. These are the conditions that psychiatrists spend most time on today. But to provide consistently effective treatment for such patients, we will need much more research.

Unfortunately, treatment research in psychiatry does not always address the chronicity and relapsing pattern of many mental illnesses. Clinical trials of drugs are often conducted within a time frame of weeks—not the months or years required to find out how well they work. Moreover, the patients who are selected for clinical trials are often atypical and unrepresentative of the populations that psychiatrists are asked to treat. The same problems arise in psychotherapy research, almost all of which concerns patients with less severe problems who are being treated for short periods of time. However, there is an evidence base for some more lengthy interventions, such as rehabilitation for psychiatric patients (Pratt et al., 2006).

The reason why research that follows sicker patients for longer periods is rare is fairly obvious—the problem comes down to money. Yet we should not fool ourselves into believing that either our drugs or our psychological interventions can be applied to the real-world and highly complex problems we face every day. When a new treatment—either the latest drug on the market or the latest brand of talking therapy—is developed, we should assess it cautiously and not jump on a bandwagon. Without that degree of caution, we will end up being just as wrong as Sigmund Freud, Walter Freeman, or D. Ewen Cameron.

Chapter 3

Etiological Fads

The Two Cultures of Psychiatry

Psychiatry has long been a house divided. Many decades ago, in a study of psychiatric practice in New Haven, Connecticut, Hollingshead and Redlich (1958) described how psychiatrists fell into two categories—"directive–organic" types, who wore white coats and whose therapies consisted mostly of biological treatment, and "analytic–psychological" types, who wore jackets and whose treatment consisted of talking therapy. This dichotomy has always reminded me of C. P. Snow's concept of "the two cultures," in which academics are divided into those who work in the hard sciences, and those who fall within the humanities (Snow, 1959), and how these cultures are cut off from each other.

The dichotomy between biological and psychological models continues to afflict psychiatry to this day. But the main change occurred over the last few decades as biological psychiatry emerged triumphant. Many psychiatrists now treat patients almost exclusively with drugs, and either do not refer patients to therapy, or leave that task to clinical psychologists. It takes less time to quickly revise a prescription, and one can make more money by seeing four patients in an hour instead of just one. Yet a minority of psychiatrists remain active therapists, and I am one of them. But I have a special reason. I only work with patients with borderline personality disorder (BPD), who are unpopular with other mental health clinicians because of their chronic suicidality.

This split in psychiatry reflects another dichotomy, called the *nature–nurture problem* (Paris, 2020a, 2022). Clinicians who believe that psychiatry is not different from other branches of medicine see psychopathology as biological, and will almost exclusively use biological methods in treatment. In contrast, clinicians who believe that symptoms are due to life experiences and/or cognitive errors will focus on talking therapy. Thus different models of the etiology of mental illness can lead to important differences in practice.

Fads and fallacies can emerge from both domains. A commitment to biological psychiatry leads to the belief that mental disorders are the result of neuronal connections or neurotransmitters, and that answers to clinical problems lie almost entirely in pharmacology. This view is partially supported by diagnostic manuals such as DSM-5 (American Psychiatric Association, 2013). That is not to say that DSM has specific guidelines for treatment (it does not), but that the system is based on symptoms that have come to be seen as targets for medications. In contrast, a commitment to psychological models (whether psychodynamic or cognitive–behavioral) targets the way patients think about their symptoms. Both points of view fail to understand that causality

in mental disorders reflects complex interactions between multiple risk factors. And that requires a biopsychosocial model (Engel, 1980).

Psychiatrists may not like to admit it, but they do not always understand why their patients are sick. To be fair, this problem is not unusual in medicine. Knowledge about the etiology of disease, especially of chronic diseases, can lag behind treatment efficacy. Yet although psychiatrists spend much of their time managing patients with poorly understood illnesses, they still manage to help most patients who come their way. In some respects, they are ahead of neurologists, who tend to be better at pinpointing pathology than at doing something about it.

Even so, human nature being what it is, it is difficult to remain in a state of doubt. Although rapid responses often lead to wrong conclusions, our minds prefer closure. Faced with suffering patients who are desperate for help, physicians prefer confidence to uncertainty. This cognitive bias encourages pretensions to unrealistic and unreachable knowledge. We hold on to the illusion that we already understand the causes of mental illness, even when research is limited or absent.

Throughout my career in psychiatry, I have struggled against these orthodoxies. The idea that mental disorders are brain disorders, and that the only way to understand them is by studying the activity of neurons and neurotransmitters, does not take into account psychosocial factors in mental illness, and supports a mindless psychiatry. The idea that mental disorders are entirely the result of life experiences, particularly in childhood, does not take into account the strong genetic and biological components in most mental disorders, and supports a brainless psychiatry. Both views are associated with narrow approaches to treatment—by medication alone, or by psychotherapy alone.

There is a way to reconcile these views. To do so, we need to embrace an interactive approach using a biopsychosocial model. In my own writings, teaching, and research I have promoted the idea that one can best understand psychopathology as the result of gene–environment interplay (Paris, 2020b, 2022). These ideas reflect the profound influence of the British child psychiatrist Michael Rutter (Rutter, 2006). Some of the fads and fallacies that afflict psychiatry come from a lack of understanding of the complexity of the pathways to mental illness. Unfortunately, since an interactive approach requires more mental effort, it is not always adopted.

Biological Reductionism

The more a theory tries to explain everything, the more likely it is to be wrong. Psychoanalysis was a good example. It had an explanation for every kind of outcome, but in the end it accounted for little. Biological psychiatry has the same problem. It claims to explain psychopathology by reducing the complexity of mind and brain to the level of neural networks, but, lacking firm data, it relies on promises that the answer is close at hand. This view of the relationship between mind and brain is both oversimplistic and reductionistic.

A scientific strategy of *reductionism* refers to a process in which complex phenomena cam be accounted for by mechanisms operating at a simpler level (Gold, 2009). I am not criticizing methods in which complex phenomena or observations are divided into smaller components. Reductionism has been an essential and productive method in many domains. Some of the best examples are the standard model in particle physics and the periodic table of elements in chemistry. Reductionism has also been highly

successful in medicine. Little progress was made until physicians understood how human cells and tissues are structured and how they function. Although medical students are still expected to think of patients as a whole, research at the level of organs, tissues, and cells has been responsible for most of the progress in medical practice during the last 100 years.

What I *am* criticizing is the idea that complexity can always be reduced to a simpler level of analysis. That brand of reductionism fails to allow for the fact that large complex systems have properties that are not accounted for by their smallest elements. It does not allow for multi-level analyses in which higher levels (like the mind) are studied in their own right, not just as a network of neurons.

Moreover, reductive strategies have serious limitations. Today, with DNA in mind, medical research focuses on genetic mechanisms that have a link to abnormal chemistry. Yet aberrant genes and molecules account for only a few rare diseases. Research finds that most genes account for a very small percentage of outcome variance, in all but a few classical Mendelian conditions (Uher and Rutter, 2012). Few illnesses or mental illnesses can be reduced to simple genetic, biochemical, or connective pathways.

The brain is the most complex structure known to science. We are not looking at an organ such as the liver in which most cells do much the same thing. Instead, the brain has 85 billion neurons and trillions of connections, each of which may be functionally unique. Although some academic psychiatrists (e.g., Zorumski and Rubin, 2011) have suggested that research on neural circuits will solve some of the most important problems in understanding mental disorders, that is, once again, only a promise. It is not a project for a decade, but for a century.

In summary, the crucial problem with reductionism is that complex structures have emergent properties that cannot be explained by simpler components (Kendler, 2005; Gold, 2009). Emergence means that the whole is more than the sum of its parts. That is why medicine is not a branch of chemistry. Even in the basic sciences, one would not attempt to explain physical phenomena at the level of atoms, quarks, or quanta. Moreover, many biological phenomena, although shaped by natural selection, only make sense at the level of the organism as a whole (Kirmayer and Gold, 2011).

In psychiatry, researchers who are seeking to understand the mind need to study thought, emotion, and behavior at a mental level. Thus even though mental processes are illuminated by neurochemistry and neural networks, they cannot be reduced to cellular processes. This does not mean that reductionistic methods are not useful in any way, but these approaches need to be put in a larger perspective. Psychiatry must study mental disorders at every level, from molecules to social networks.

Unfortunately, it is rare to find practicing psychiatrists or researchers who are knowledgeable enough, or sophisticated enough, to think in this way. Doing so requires an understanding of and respect for complexity, and of multiple interactions between many factors that increase or decrease the likelihood of a mental illness. Even though psychiatrists pay lip service to a biopsychosocial model, they tend to focus on only one aspect, using treatment methods that target one of many possible pathways.

Fallacies about Psychiatric Genetics

We now know that all mental disorders have a heritable component. This principle derives from the science of *behavioral genetics* (Plomin, 2018). This method usually

depends on data obtained from the study of twins, in which heritability can be quantified by examining differences in concordance for traits between monozygotic and dizygotic twins. In this way, heritability can be measured as a percentage of variance in outcome. Differences in concordance for traits of all kinds are higher in monozygotic than in dizygotic twins, allowing a quantitative calculation of heritability (Jang, 2005). These coefficients usually find that nearly 50% of the variance is heritable. In severe mental disorders, genes tend to account for even more than this.

Yet behavioral genetics does not tell us where heritable factors lie on the genome. The discovery of the structure of DNA, which led to the unraveling of the genetic code, followed by the success of the Human Genome Project, is among the greatest triumphs in the history of science. In medicine, great hopes were held that genetics would provide a breakthrough in understanding the causes of disease. Yet, in psychiatry, progress has been unimpressive, and many in the mental health field who had hoped for more have been disappointed.

One reason for this is that very few of the disorders seen by clinicians can be attributed to changes in one gene, or in just a few. On the contrary, genome-wide association studies (GWAS) show that mental disorders are associated with variations in hundreds or even thousands of sites, each with a very small effect (Tam et al., 2019). Moreover, although many mental disorders are associated with a genetic risk, most also have an environmental component and/or a relationship to gene–environment interactions. For most illnesses, heritability is only one factor in a complex, multiple, and interactive set of risks.

The problem lies in a discrepancy between behavioral genetics and genome-wide association studies (GWAS), in which the whole genome is under study. One can add up small effects from many genes, yielding a *polygenic risk score (PCR)*. But PCRs only account for 5–10% of total variance, much less than what behavioral genetics suggests, leading to the coining of the term "missing heritability" (Zuk et al., 2012). We need to look to interactions with other genes and with the environment to account for this gap.

One type of interaction that has been widely studied in recent research is *epigenetics*. Gene expression is mediated by chemical tags (methyl groups and histones) that surround molecules of DNA and serve as dimmer switches for gene activity. Environmental stressors can influence methylation, producing changes in gene activation that can be passed down to the next generation (Szyf et al., 2009). This mechanism can be a pathway by which the environment can modify heritable traits. But although this is a fascinating line of research, its clinical application is uncertain, and epigenetics does not, on its own, account for missing heritability.

Behavioral genetics also allows us to measure how the environment affects the development of traits and symptoms (Jang, 2005). These involve two pathways. The first is due to similarities in outcome related to the family in which people are raised ("shared environment"), whereas the second derives from effects that do not depend on growing up in a particular family ("non-shared environment"). For traits and symptoms, effects are almost exclusively *non-shared*, which is something of a surprise and a mystery. (The strongest effects of shared environment are found for intelligence and antisocial behavior, not for most mental disorders.)

We still need to understand why the effects of growing up in dysfunctional families, a relationship that has been well documented in research (Paris, 2022), do not appear in behavioral genetics findings. The most likely explanation is that temperamentally

vulnerable people react more strongly to environmental adversity, and these are the people who are most affected by stressors. Some prominent researchers in behavioral genetics (e.g., Plomin, 2018) have been dismissive of the role of families, and seem to favor a kind of genetic reductionism, but other scientists (e.g., Jang and Choi, 2020) are more open to interactive models.

By and large, the idea that genes necessarily shape our destiny is not supported by research. Scientists have come to realize that DNA is a recipe, not a blueprint (Mukherjee, 2018). Genetic effects "bend the twig" but do not determine the shape of the tree. That is the logic behind research on gene–environment interactions in psychiatry (Rutter, 2006). In most disorders, neither genetic nor environmental risks are sufficient by themselves to account for etiological pathways.

A standard joke is that no matter how many corners we turn in science, breakthroughs usually remain "just around the corner." That is what happens when enthusiasm raises unrealistically high hopes, failing to consider complexity. As we shall see, this story can be told about every faddish development in the history of psychiatry.

Another idea promoted by genetics enthusiasts is "personalized medicine." In this scenario, one would prescribe drugs on the basis of a reading of the patient's genome. This idea has had some success in treating cancer. But in psychiatry we are looking at phenomena that are much more complex and unpredictable. Again, we do not find relationships between psychopathology and single genes, or even a clear relationship to the genome as a whole. It may therefore be impossible to base treatment on genetic patterns targeting the hundreds or thousands of genes identified by GWAS research. Progress in these domains of research has been suggestive, but still has a very long way to go.

Fallacies about Neurotransmitters

Neurotransmitters are chemical messengers that link neurons throughout the brain. Much research has attempted to explain mental processes at this level of analysis. Glutamate and gamma-aminobutyric acid (GABA) mediate transmission at the vast majority of synapses, but monoamines (particularly serotonin, dopamine, and norepinephrine) modulate this activity throughout the brain. Research has shown that antipsychotic drugs can block dopamine receptors, and that selective serotonin reuptake inhibitor (SSRI) antidepressants block the reuptake of serotonin. These findings suggest a mechanism for the development of psychopathology, but this hypothesis has not been borne out by evidence.

These observations have been postulated to support etiological theories. One such theory is that schizophrenia is caused by abnormal dopamine transmission (Howes et al., 2012). As little evidence was found to support this hypothesis, more recent research has focused on glutamate (McCutcheon et al., 2020). In fact it is doubtful whether any category of mental disorder can be explained by a single neurotransmitter. Neurochemical theories of any kind, even if they turn out to tell part of the story, are seriously over-simplistic.

The idea that depression is caused by underactivity (or imbalance) of serotonin or norepinephrine has even less evidence to support it (Nemeroff, 2020). The long but futile search by psychiatry for a chemical theory and a chemical cure has been described in a recent book (Harrington, 2019). Certainly, no one has ever demonstrated imbalances of

monoamines in depressed patients (Moncrieff, 2008). Yet, under the influence of the pharmaceutical industry, many clinicians continue to assume that these mechanisms have strong scientific support, and that they can explain why drugs for depression work.

It is difficult to generalize from mechanisms of drug action to causes of disease. For example, acetylsalicylic acid (ASA) is known to block prostaglandins, but these molecules are not responsible for the symptoms relieved by ASA. In the same way, even if antipsychotics block dopamine, it does not follow that the symptoms of schizophrenia are due to abnormal dopamine receptors. Similarly, even if most antidepressants are agonists for monoamines, it does not follow that patients suffering from depression have abnormal neuronal receptors. Ultimately, the theory of chemical imbalances is fallacious, even if it has had an almost irresistible appeal to clinicians and patients.

Neurochemical theories have been popular because they seem to make the practice of psychiatry more "scientific." Based on these models, some practitioners adjust medication at every visit, rather like balancing electrolytes in patients who are on intravenous infusions. This kind of treatment has been compared to preparing a cocktail. Some patients love the theory, since it makes them victims of chemistry rather than of bad life choices.

Although neurotransmitters play a role in mental disorders, they are only one link in a complex chain. Instead of being the cause of illness, levels of chemical messengers may well be an epiphenomenon. Yet the concept of chemical imbalances is a potential basis of treatment fads, in which antidepressants and antipsychotics are used to manage mental disorders of all kinds. (This practice is also confusing due to the overdiagnosis of depression, as will be discussed in Chapter 4.) It would be better if psychiatrists were willing to acknowledge that although their drugs can be useful and effective for the right indications, we just do not know how they work (Moncrieff et al., 2022). That perspective could help them to avoid unnecessarily aggressive pharmaceutical treatment, and open the door to combining drugs with psychotherapy.

Fallacies about Neuroimaging

For decades, psychiatrists have lacked the tools (such as imaging and blood tests) that guide other physicians to diagnosis and treatment. A good deal of progress has been made in scanning, which is now one of the most popular ways to measure brain changes in research. We can use functional magnetic resonance imaging (fMRI) to examine brain structure and activity in more detail, and these brightly colored images provide a window on brain activity. However, the clinical application of imaging remains unclear.

What this method tells us is whether blood flow is increasing in given brain regions. But when specific areas "light up" on a scan, that is still an indirect way to assess the billions of interacting neurons that lie behind every observation. The computerized image is based on average blood flow to one region (compared with others), and it does not account for the wide distribution of neural networks across the brain. Satel and Lilienfeld (2013) assessed fMRI as a useful but ambiguous representation of a highly complex system. Another problem is the expense of scans. That is why most published neuroimaging studies have samples that are too small to allow generalization or replication of their findings (Ioannidis, 2005).

This is not to say that the situation may not change over time. Recently there has been some excitement about a method called *optogenetics*, in which the activity of single

neurons can be modified using lasers (Deisseroth, 2015). However, these technical advances need much more research, and are currently not sufficient to bridge the gap between the levels of neurons and larger-scale brain function.

The Limitations of Biological Psychiatry

Some years ago, I was asked to sit on a committee for the Canadian government that was reviewing grant proposals related to psychiatry. I was honored, but I found the experience somewhat disheartening. On the one hand, since the Canadian Institute of Health Research only approves about 10% of all applications, the ones that were approved were clearly the best. On the other hand, I found that only rarely did applications in basic research have any serious implications for understanding or managing psychiatric patients.

To consider an example, one of the grant proposals proposed the study of three models of schizophrenia—in mice! Needless to say, there is no such thing as psychosis in a rodent. Other animals can get depressed but, as far as we know, only human brains can have psychotic symptoms. I was not disappointed to see that idea go unfunded. But when I later shared my reaction with the committee chair, he disagreed, saying that only basic research on brain functions will be able to answer the questions that psychiatry raises. Meanwhile, grants rarely support research on better ways to treat patients, or examine the public health implications of severe mental illness.

Some years ago, Allen Frances commented on the proposal of the National Institute of Mental Health to replace standard diagnosis with a new system, the Research Domain Criteria (RDoC), which claims to be based on neuroscience (Frances, 2014). He described the issue well, and I cannot say it better:

> . . . because the brain is so much more complicated than other organs, psychiatry confronts by far the most challenging of all translational leaps. . . . It is amazing that a machine with so many moving parts works as flawlessly as usually it does. By comparison, the breast is the most straightforward of organs, many orders of magnitude simpler than the brain. If, despite decades of intensive research, we are still early days in understanding breast cancer, why be surprised that we haven't yet gotten much of a handle on schizophrenia.
>
> . . . NIMH has had its attention so distracted by glorious dreams of a future research revolution that it has completely lost touch with the desperate suffering of schizophrenic patients in the present. It pays no attention to, and takes no responsibility for, the mess that is US mental health care. During the same fifty years that witnessed a basic science research revolution, the US has closed one million psychiatric hospital beds. But having provided too little care and housing in the community, we have been forced to open one million prison beds for psychiatric patients who were arrested for nuisance crimes, preventable had they received adequate community services and housing. These patients are suffering greatly not so much for lack of knowledge on how to care for them, but because of a lack of attention and inadequate resources. (Frances, 2014, p. 48)

These conclusions suggest that the fallacies of biological psychiatry may be even more serious in practice than in theory. Psychiatry's wish to be a medical specialty like any other has not addressed the problem of making treatment accessible to people who have severe mental disorders. And that is a subject that badly needs research funding.

Fads and Fallacies in Psychological Theories

The role of psychological factors in mental illness has been supported by research, but has also been afflicted by fads and fallacies. Some of the most serious misconceptions have been reviewed by Arkowitz and Lilienfeld (2017), who examined false beliefs about each of the major forms of psychopathology. Here I shall focus on the relationship between psychological theories of the etiology of psychopathology and clinical practice.

Fewer psychiatrists offer psychotherapy these days, and many see patients for just enough time to update a DSM diagnosis and write another prescription. (Since I do not practice in that way, I have to consider myself to be a member of a beleaguered minority group.) Psychiatrists should be prescribing fewer drugs and referring more patients to psychotherapists, even if we do not do this kind of work ourselves. Moreover, as we shall see in Chapter 7, common mental disorders may respond better to psychotherapies than to medication alone. And in several groups of high-prevalence mental disorders, the most effective treatment is a form of evidence-based psychotherapy. Specifically, that applies to personality disorders (my sub-specialty), substance use disorders, and eating disorders.

However, the theoretical models behind psychological treatment are something of a muddle. Psychotherapy needs to be based on a theory, but has suffered from having too many theories. That may be why there are hundreds of therapy methods, each identified by a memorable acronym. In this domain, as long as there is an unfilled demand and an uncertain outcome, there will be fads and fallacies that affect both theory and practice.

This crowded and competitive field of psychotherapy is a problem that reminds me of the competition in pharmacology between different drugs which all do pretty much the same thing. Yet the factors to be found in all effective therapies are not very specific, but common to all (Wampold, 2001; Barkham et al., 2021). A possible exception is Linehan's biosocial theory of BPD (Linehan, 1993), which is the basis of a specialized and evidence-based method, namely dialectical behavior therapy, which has been used for a range of conditions that involve emotional dysregulation.

Fallacies of Childhood Trauma

Let us now consider theories based on currently fashionable ideas about a crucial role for trauma, both in childhood and in adulthood.

Childhood trauma has been a particularly hot topic. We live in a society that is concerned about the protection of children, and particularly the possibility of harm being inflicted on the innocent. The worldwide scandal about child abuse in the Catholic Church was certainly a wake-up call. But we also live in a society where, in most families, both parents need to work, leaving their children in the care of others. These tensions help to explain why accusations of child abuse in day-care centers, many of which turned out to be false, became common 20 to 30 years ago. At the peak of these accusations, aggressive prosecutors convinced children that they had been abused, and a job in day care became a high-risk occupation (McHugh, 2008). But the blame for that scandal (one of the worst ever to afflict the mental health field) mainly lay with psychotherapists (both psychiatrists and psychologists).

The idea that child abuse is very common, and that it accounts for the origin of several mental disorders, became influential during this period. These life adversities are indeed more common than many had previously realized. But several issues concerning

their role became highly charged and emotional. If one criticized the assumption that abuse is a crucial cause of mental disorder, one could be accused of protecting offenders and invalidating victims. These ideas have now gone out of favor, but are still espoused by a minority of psychotherapists.

The evidence shows that there is a statistical, but not predictable, relationship between childhood experiences and adult outcome (Paris, 2020a, 2022). Although reports of abuse are frequent in several clinical populations, community studies show that the vast majority of those affected never develop mental disorders (Fergusson and Mullen, 1999). Also, since child abuse co-varies with parental neglect and other forms of family dysfunction, long-term effects are not due to any specific kind of experience, but to problematic rearing as a whole (Paris, 2020a, 2022). Moreover, the most consistent effects of childhood adversity occur in those who are temperamentally vulnerable. This is where we need to invoke the central role of gene–environment interactions.

The idea that adult symptoms can be the outcome of early childhood experiences was a central idea for psychoanalysis that remains influential. This theory is not a complete fallacy, but a half-truth or a quarter-truth. As we saw in Chapter 2, there is a serious problem with recovered memories, in which patients are convinced by their therapists that they have been traumatized, even if they have not been. Yet childhood trauma does happen and is all too real, and these events are almost certain to be remembered.

It is better to have a happy childhood than an unhappy one. A large body of literature demonstrates that childhood adversities are a risk factor for a range of mental disorders (Rutter and Rutter, 1993; Paris, 2022). However, an equally large body of literature demonstrates that resilience to adversity, both in childhood and in adulthood, is ubiquitous (Paris, 2022, 2023). Thus psychological risk factors lower thresholds for pathology, but do not lead to predictable outcomes. People with severe adversities, such as dysfunctional or abusive families, can become perfectly normal adults. And many people who have experienced no significant early adversities still develop mental disorders.

It makes sense for resilience to be the rule in development. Human life, especially in the past, can be replete with trauma and loss. If people were as vulnerable as some psychological theories suggest, our species would have long since become extinct. Fortunately, child abuse has greatly decreased in recent decades in countries such as the USA (Children's Bureau, 2020). But mental disorders that can be linked to child abuse, such as post-traumatic stress disorder (PTSD) and BPD, have not decreased at all, and in fact are being diagnosed much more frequently.

A psychotherapist who only sees a few hundred patients in a lifetime may get a different impression. All patients have stories to tell, and many of them are poignant. It is easy to make links between past trauma and present dysfunction. Early in my career I became adept at that game, making facile "formulations" that attributed current symptoms to childhood maltreatment or neglect.

Experience with thousands of consultations, supported by a large body of research on resilience, has led me to a very different conclusion. Once I dropped my cognitive bias in favor of making links between childhood and adult experiences, and read the research literature more carefully, it became clear to me that the connection was weak. This is certainly the case for long-term effects of childhood sexual abuse—an adversity that has been the focus of longitudinal studies to examine its impact (Fergusson and Mullen, 1999). It is true that a significant minority are affected by these experiences, but it is equally true that the majority rise above them. That is the difference between a risk factor and a cause.

An inconsistent relationship between past and present shows the need for a broader model. Some people are more vulnerable by temperament to adversity, whereas others are less vulnerable. These principles help to explain the somewhat unpredictable effects of trauma. Adverse events, even highly traumatic ones, do not usually lead to PTSD, or to any mental disorder at all.

Moreover, PTSD itself emerges from an interaction between exposure to stressors and vulnerability, with high levels of trait neuroticism leading to more intense emotional reactions to adversity (Breslau et al., 1991). Yet this important finding remains largely unknown to many psychotherapists, who are all too quick to attribute a wide range of symptoms to traumatic experiences and to diagnose their patients with PTSD.

This is a good example of a cognitive error (*post hoc, ergo propter hoc*). Psychotherapists listen to and sympathize with patients' life histories, but we must keep in mind that adverse life events do not, by themselves, cause mental disorders. This idea may have remained popular because it is simple, and because, like the theory of chemical imbalances, it places blame on outside forces rather than within the self.

Fallacies about Trauma in Adulthood

PTSD, with its prominent symptoms, has been a magnet for purely psychological theories of trauma (Horwitz, 2018). However, although trauma is a *necessary* condition (by definition) for PTSD, it is not a *sufficient* condition. The frequency of this outcome varies with the severity of traumatic exposure, with most incidents leading to PTSD in about 5–10% of cases, and more severe incidents, such as rape, leading to it in up to 20% of cases. Even so, the vast majority of those in all these scenarios will be resilient to trauma.

Other non-traumatic factors help to account for this discrepancy. The most important for trauma are high levels of trait neuroticism, a past history of exposure to adverse events or of other mental disorders, and a lack of social support (Paris, 2023). Thus PTSD will be seen in those who are most vulnerable, in terms of both their temperament (which determines the intensity of reaction to life events) and previous exposure to adverse events.

It follows that seeing PTSD *only* as a response to trauma is fallacious. It also follows that "trauma-focused" psychotherapies for PTSD that deal almost exclusively with processing adverse life experiences are at best incomplete, because their etiological theory is mistaken (Paris, 2023). It may be useful to add a component that addresses underlying traits, particularly neuroticism. These methods would focus less on re-experiencing the trauma than on reframing it. This conclusion is consistent with research that shows few differences between standard CBT and methods that claim to be more specific.

Moreover, the concepts behind PTSD have supported a fad that has been widely used, not only by clinicians and patients, but also by university professors. This occurs when students claim to be easily "triggered" by anything that reminds them of past trauma. This fad has also become a part of the "cancel culture" that now afflicts the academic world.

Ultimately, the overdiagnosis of PTSD is an example of what has been called "concept creep" (Haslam, 2016), in which concepts that describe harm gradually expand to include more and more phenomena. It is also related to the "psychiatrization" of normal experiences (Frances, 2013). Thus trauma has become a term that is being used to

describe adversities of any kind, such as neglect or an unhappy childhood. In this case, adhering to DSM-5 criteria could come to our rescue, since it defines traumatic events as having to be life-threatening. But clinicians love the idea so much that they are willing to apply it to all kinds of adversities.

Fallacies of Social Risk

Social factors, ranging from the state of the economy to the strength of social networks, can have a profound influence on the development of mental disorders (Kirmayer et al., 2007). But mental health professionals are not in the business of promoting social change or advocating for a political program to make a better world. We are not trained to prescribe for society as a whole—not in our own country, and certainly not around the globe.

We live in a world where the young, and many older people, yearn for social justice. Unfortunately, this quest has often proved perilous in the past. All too often, radical social change ends up being change for the worse. The failures of Marxism are the most obvious example. We need to learn from these mistakes and not repeat them. The community psychiatry movement of the 1960s, which I am old enough to remember, is another example, for it raised great expectations that could not be met.

This is not to deny that social stressors can lower the threshold for mental symptoms. If patients are socially isolated, poor, or unemployed, no antidepressant in the world will make them happy, and psychotherapy may well fail. But psychiatrists do not have a therapy to make society work better. Symptoms that are brought on by social stressors cannot always be treated with the interventions that psychiatrists know best how to provide.

Social psychiatry attempts to take the pathology of society into account, but it has had its own share of fads and fallacies (Krupzinksi, 1992). When I was a student, some psychiatrists promoted the "primary prevention" of mental illness. One of them was the Boston psychiatrist Gerald Caplan (Caplan, 1964). My hospital invited him to give a talk, and he spoke about his work in Jerusalem that aimed to bring Israelis and Palestinians together. (We all know how that turned out.) Caplan was fairly charismatic, and I must admit that I was impressed at the time. But he had no data to support his ideas, just a serious overdose of good will.

The idea that clinicians who work in mental health know how the world should be run is absurd. On a similar note, in 1968 I heard the well-known Chicago psychiatrist Roy Grinker claim that if city officials had consulted him about how to handle the famous riots of that year, he would have told them how to prevent them.

The fad for social psychiatry reflects much about the 1960s. This was a time when, under the influence of radicals such as R. D. Laing (Laing, 1967), psychotic patients were seen as victims of society. Today, hardly anyone remembers Laing, but academics in the humanities still retain a strong sympathetic interest in the ideas of Michel Foucault (Foucault, 1961). Foucault was a French philosopher who knew almost nothing about psychosis, but was able to convince many readers that psychiatric patients are misunderstood rebels or outsiders stigmatized by society. Even today, one hears this idea, mostly promoted by left-wing critics of psychiatry.

These examples of the early stages of social psychiatry show how its ideas were based on hope, not on science. We need to face some painful realities. No one has ever shown

that major mental disorders can be prevented by interventions at a societal level. How could they work if we do not understand what causes psychopathology? These ideas are not evidence based.

The Need for a Biopsychosocial Model

In middle age, when I embarked on a second career as a researcher, I needed to upgrade my knowledge of statistics. I quickly learned that the *t*-tests and chi-squares of my undergraduate days were out of date, and that when they were used they were a signal that samples were too small. Most research papers now make use of multivariate analyses. Multiple and logistic regression, path analysis, model fitting, and hierarchical modeling can measure what proportion of the variance is uniquely related to any one risk factor. These procedures more closely correspond to the real world, in which no single risk leads to any predictable outcome. Also, the relationship between a risk factor and an outcome can be quantified, rather than treated as an absolute relationship.

We generally prefer to think in a linear fashion, but the real world is multivariate. Therefore psychiatrists need to think in this way when assessing and treating patients. A wide range of biological variations and adverse life experiences can increase risk for disorder. Only a combination of all these factors brings people to a tipping point at which they fall ill. Applying these models can be a useful antidote to faddish thinking.

This point of view underlines the central importance of gene–environment interactions in psychiatry. Most broadly, researchers and clinicians are now beginning to think in these terms. Again, genes govern vulnerability and resilience, whereas the environment presents challenges that those with the most problematic temperaments have most difficulty handling.

For all these reasons, I am a strong defender of the biopsychosocial model in psychiatry (Engel, 1980). Although some psychiatrists (e.g., Ghaemi, 2009a) have criticized this model, that is probably because they do not have a good understanding of the psychosocial side—either of the psychological challenges that can impair development, or of the social challenges that can affect anyone.

Chapter 4

Diagnostic Fads

Diagnosis is an essential part of medical practice. When diagnosis is used to guide treatment, it is important to get it right. Classifying disease also helps physicians to communicate with each other. A category that is easily recognizable packs a great deal of useful information.

Ideally, diagnostic categories should be *valid*—that is, they should describe illnesses that correspond to natural disease entities, and that have a specific etiology, a likely prognosis, and a predictable response to treatment. These features of validity were first described half a century ago (Robins and Guze, 1970). Current diagnoses in psychiatry are too inexact to meet such criteria. In most cases, their etiology and pathogenesis remain unknown. However, there are diagnoses that, once made, require specific interventions, such as lithium for bipolar disorder, or specialized psychotherapy for borderline personality disorder (BPD).

In medicine, clinical observations can be confirmed using biopsies, laboratory tests, imaging, or genetic testing. Since psychiatry lacks these tools, and since none of the disorders it treats have any biomarkers, we are not yet in a position to define true diseases (Uher and Rutter, 2012). The absence of a "gold standard" also leaves diagnosis open to faddish ideas.

It follows that classification systems are not necessarily a guide to treatment. Yet diagnoses can be useful clinical tools. Some categories can summarize vast amounts of clinically relevant information. I tell my students to make diagnoses, but not to believe in them.

The DSM Classification System

Sixty years ago, when I was a medical student, psychiatrists did not take diagnosis very seriously. Classifications had been developed by the American Psychiatric Association, namely the first edition of the *Diagnostic and Statistical Manual of Mental Disorders*, DSM-I (American Psychiatric Association, 1952), later revised as DSM-II (American Psychiatric Association, 1968). as well as by the World Health Organization, namely the seventh edition of the *International Classification of Diseases*, ICD-7 (World Health Organization, 1957). Yet for many years these systems were mainly used for record keeping, not for treatment planning.

In the 1960s, psychiatry came under attack for its diagnostic system. How could anyone consider the field scientific if practitioners could not agree on what was wrong with most patients? Reliability for even the most important diagnoses was found to be unacceptably low. For example, it had been reported that British psychiatrists tended to

label as manic-depressive the same patients who were described as schizophrenic by American psychiatrists (Cooper et al., 1972). It turned out that mistakes of this kind were even more serious than was thought at the time. The introduction of lithium, which is effective for bipolar disorder but not for schizophrenia, made differential diagnosis between the two disorders clinically important. (Even so, the procedure for doing so remains problematic.) This was an example, still unfortunately rare, of specific responses to specific treatment methods contributing to the definition of a major mental disorder.

Moreover, there is much overlap between most of the categories listed in manuals such as DSM-5 and ICD-11. That is why many researchers have suggested that we should study psychopathology from a *transdiagnostic* perspective (Fusar-Poli et al., 2019b). It is not a given that the different forms of mental illness fall into sharply defined categories, as opposed to lying on the peak of a bell curve with gradual gradations to normality.

Even so, 40 years ago the medical view of diagnosis taken by the DSM system was a major step forward. Psychiatry was under siege from several directions. The critiques of antipsychiatrists, although almost entirely invalid, were hard to answer if practitioners could not agree about the basic categories of illness. To address this criticism, and to gain more respect from medical colleagues who looked down on psychiatric diagnoses and treatments, something had to be done.

The third revision of the *Diagnostic and Statistical Manual*, DSM-III (American Psychiatric Association, 1980), was a serious attempt to create a more reliable and valid classification. In the ICD manual, diagnosis was (and still is) based on complex prototypical descriptions, whereas DSM-III's innovation was to make diagnosis *algorithmic*. To qualify for a given category, the patient had to fulfill several operational criteria from a whole list of such criteria. At least half of the listed criteria had to be present, and some were required. Listing criteria and asking clinicians to count them can also be a user-friendly procedure. However, clinicians do not often open the manual, so there remains a problematic tendency to rely on a single predominant feature to make a diagnosis. Clinicians can make inaccurate diagnoses due to a preference for linear thinking.

Even so, the DSM system quickly became popular all over the world, even in countries where the ICD remained standard. A category could have reasonable *reliability* if clinicians accurately observe symptoms and follow standard procedures. But in practice, reliability still tends to be rather low. One shocking example came from a field trial of DSM-5 (Regier et al., 2013), in which the reliability of the diagnosis of major depression was found to be only 0.20.

By itself, reliability is insufficient to prove *validity*—that is, the extent to which one is measuring what one is supposed to measure. In medicine and psychology, validity is often supported by showing that different methods lead to the same conclusion. Almost none of the current diagnoses in diagnostic manuals meet that criterion.

I shall focus here on the DSM system, which is still the most influential classification of diagnoses across the globe. Since the publication of the monumental third edition, the following editions have been produced: DSM-III-R (American Psychiatric Association, 1987), DSM-IV (American Psychiatric Association, 1994), DSM-IV-TR (American Psychiatric Association, 2000), DSM-5 (American Psychiatric Association, 2013), and a minor update in DSM-5-TR (American Psychiatric Association, 2022).

All of these editions suffer from the same problems. First and foremost, categories are based entirely on clinical observation and/or patient self-report, not on laboratory

findings. Although medicine always begins with signs and symptoms, the science of diagnosis has greatly benefited from blood tests, specific imaging findings, and genetic findings. Since psychiatry lacks all of these biomarkers, it is in more or less the same position that medicine was in during the nineteenth century.

Crucially, many if not most DSM categories overlap with each other to a great extent. This is because they are defined by common symptoms, so the more symptoms a patient has, and the more severely ill they are, the more likely they are to meet the criteria for more than one disorder. There are very few criteria in the manual that are absolutely required, or that rule out alternative diagnoses. The massive overlap between disorders has been called "comorbidity." But since patients need not actually have two separate illnesses, this phenomenon might be better termed "co-occurrence." It is an artifact of a system that created a list of categories without clear boundaries.

Finally, the DSM system has never produced a clear definition of mental disorder that can distinguish psychopathology from normality. (It does have one, but if you look it up I defy you to make sense of it.) Instead, categories are "fuzzy," fading into subclinical (but common) symptoms at the edges. This problem, which has been addressed in a whole book (Frances, 2013), makes it all too easy to "medicalize" universal experiences of psychological distress (Conrad, 2007).

Most of the diagnoses that psychiatrists use in practice are not diseases in the usual sense of modern medicine. In the absence of a known etiological mechanism, one cannot say whether phenomenological clusters describe unique pathological processes. These DSM categories are syndromes—symptoms that tend to be seen together. Yet although research in psychiatry has been limited by using DSM manuals to describe patients, in the absence of deeper knowledge, this may be the best we can do for now.

The editors of DSM-III saw their manual as provisional, and thought that as research progressed, most of these problems would eventually be worked out. That expectation proved far too optimistic. Over 40 years on, despite significant progress in neuroscience, research has not yet shed light on the causes of the most important mental disorders, and has not yielded data that can validate psychiatric diagnoses. That is why only minor changes to the DSM system have been made over the last four decades.

DSM-5 initially aimed to be a "paradigm shift" (Kupfer and Regier, 2011). But this version of the manual could not offer a new paradigm as long as its criteria remained almost entirely dependent on clinical observation. For this reason, the manual can only tinker with its definitions. These problems will not be resolved until much more is known. It may well take decades for an alternative to be developed, when research on mental disorders has become more mature.

For now, I recommend following the classifications as written in the DSM manuals, on the grounds that clinicians need a common language, while remaining open to the use of "transdiagnostic" measures. But I also suggest that we need to avoid reifying our diagnoses, and that we should regard them only as provisional constructs. This message can be difficult for many to accept. Most teachers encourage students of psychiatry to make diagnoses based on manuals, and to build their treatment plans on these criteria. Note that all versions of the DSM have warned clinicians *not* to use diagnoses to guide treatment. But the temptation to do so has proved irresistible.

In summary, although the DSM system was definitely a move in the right direction, what we call diagnosis in psychiatry is a collection of syndromes whose origins remain

unknown. In some ways, classification has not advanced greatly since the time of Kraepelin. Some believe that neuroscience will solve all of these problems, but we know too little to base our classification system on that kind of research (Hyman, 2007).

In the end, diagnostic manuals are rough-and-ready guides to the classification of the highly complex phenomena of psychopathology. Current systems are based almost entirely on observable signs and symptoms, and are not confirmed by laboratory tests as happens in the rest of medicine. Some categories of disorder are faddish and can expect a short life.

At the same time, psychiatric diagnosis has been continuously expanding, sometimes threatening to medicalize the human condition. Mental disorders are being seriously overdiagnosed, leading to inappropriate treatment and unnecessary stigma. We lack a basis for establishing the true boundaries of illness, and as time goes on, diagnosis tends to cross into normal variation, leaving hardly anyone not diagnosed with mental disorder at some point in their life (Frances, 2013). This is a story of overdiagnosis, punctuated by diagnostic epidemics.

Research in psychiatry is advancing, albeit slowly. But because the existing data were so inadequate, the editors of DSM-5 had to give up their hopes of making radical changes based on advances in genetics and neuroscience. Thus the new version ended up being not that different from previous editions. Nine years later, the publication of a revised text revision, the DSM-5-TR (American Psychiatric Association, 2022), made few substantive changes, and we shall probably have to wait another decade for the DSM-6.

I suggest that clinicians should withhold judgment as to whether the DSM manual corresponds to reality. Its diagnoses are *heuristics* that aid communication, but they are not a reliable guide to the choice of treatment. Unfortunately, contrary to the intention of the manual, many clinicians consider DSM diagnoses to perform precisely that function (e.g., the almost universal but ill-advised habit of prescribing antidepressants for every form of depression). Moreover, due to the overlap of symptoms between categories, patients tend to receive multiple DSM diagnoses, each potentially targeted by a pharmacological intervention. This practice remains one of the major fallacies of modern psychiatry.

Alternatives to DSM

By and large, DSM disorders describe symptoms and syndromes, not illnesses. They are not "real" in the same sense as most medical diagnoses. Yet despite serious problems with their validity, these categories have become reified by constant use.

Several alternative systems have been developed that aim to deal with the limitations of the DSM manuals. I shall first consider one that uses medicine as a model and also depends almost entirely on categories. This is the International Classification of Diseases (ICD), published by the World Health Organization. It is the oldest diagnostic system in medicine, and its eleventh edition (ICD-11; World Health Organization, 2018) remains the official classification of disease across the globe, and applies to all of medicine. Although the ICD is more often used in Europe, the DSM system has been favored in most countries. This preference is reflected in almost all research papers in which diagnosis is part of the design. ICD-11 does not in fact, with some exceptions, differ greatly from DSM. (The main exceptions are a new system for diagnosing personality disorders using dimensions instead of categories, and a new diagnosis of complex

post-traumatic stress disorder.) The categories in ICD-11 are also based on observable symptoms, not disease mechanisms. These limitations inevitably affect research, much of which is based on heterogeneous and fuzzy categories.

ICD-11 does not use algorithms, although it has to be said that those in the DSM manuals are fairly arbitrary. Many only require more than half of the items on a list. This means that patients with very different symptoms can meet criteria for the same disorder. Yet expository paragraphs may not provide enough guidance for reliable diagnosis. Thus a major study of ICD-10 found it to be so wanting in reliability that it was not considered suitable for use by primary care physicians (Wockenfuss et al., 2009).

Moreover, the older practice of describing illnesses in paragraphs is harder to follow (and to remember) than the algorithms used in DSM. This may be one reason why previous editions of ICD have been overshadowed by the DSM manuals. Another reason could be the domination of the USA in most areas of science, with the majority of all medical research coming from that country. Finally, the ICD system is actually designed to be similar to DSM-5, which is why these systems use common codes so that their categories can be translated from one system to the other.

The domain which differs most between the two systems is that of personality disorders. This group of diagnoses in the DSM system has long been criticized for lacking evidence for its categories, and for a serious level of overlap between disorders (Skodol and Oldham, 2021). To address that problem, ICD-11 offers a purely dimensional approach, in which clinicians score patients on five trait domains and on severity of dysfunction. The British psychiatrist Peter Tyrer, the main researcher behind this new system, once welcomed me to the UK by declaring "Be cautious, you are now entering a DSM-free zone."

However, at the eleventh hour, the dimensionality of the ICD system for personality disorders was diluted. European researchers who have for decades been studying BPD as a category protested strongly against the ICD-11 proposal (Herpertz et al., 2017). BPD is the most researched personality disorder, mainly because of its high clinical prevalence and problematic clinical features, such as suicidality. You can find thousands of papers on BPD on the Internet, and it seems radical to dismiss this rich literature. The compromise that emerged was to allow clinicians to identify and score a "borderline pattern" that closely resembles the DSM-5 definition.

Meanwhile, a work group for the DSM-5 had proposed a different dimensional system for personality disorder diagnosis, in which fewer categories would be retained. but in which categories would be built up from ratings of personality trait profiles. This is the Alternative Model for Personality Disorders (AMPD; Hopwood et al., 2019). This system also involves scoring of both traits and a level of personality functioning. But it is a "hybrid," in that one can construct categorical diagnoses from dimensional data using a set of algorithms. The AMPD was not included in the final version of the DSM-5 because it was thought to need more research before it could be formally adopted, and it was relegated to Section III (i.e., categories that require further study). But since then, hundreds of papers have been published on the AMPD, and it seems likely that this system will eventually be included in the main text of DSM-6.

The idea of dimensionalizing personality disorder diagnosis has also been considered as a test of whether *all* psychiatric diagnoses could benefit from such an approach. Many years ago, Achenbach and Ruffle (2000) had proposed a system like that for diagnoses in child psychiatry (whose current categories are particularly "comorbid"). This would

involve two broad spectra—internalizing (including anxiety and depression) and externalizing (including conduct disorder and some forms of attention-deficit hyperactivity disorder). These domains have been shown to account for a wide range of common disorders in adult psychopathology (Krueger, 1999), but do not account for psychoses or thought disorder, which require a separate dimension.

Quite a few researchers favor using dimensions as the basis of *all* diagnoses in psychiatry. The most recent incarnation of this idea is the Hierarchical Taxonomy of Psychopathology (HiTOP) system (Kotov et al., 2017), which attempts to replace *all* categories with scores on various dimensions of psychopathology. This system has some promising aspects, but the research behind it remains sketchy, and it does not seem ready to be adopted by clinicians, even though its ideas are challenging and potentially helpful. Thus researchers have been able to identify an overall psychopathology ("p") factor associated with *all* mental disorders, topping a hierarchy of several levels that moves from general psychopathology to specific symptoms.

Like the DSM, HiTOP is based on clinical observations, but uses sophisticated forms of factor analysis to define its dimensions. In some ways, this system parallels the Five Factor Model of personality, which describes five domains (neuroticism, extraversion, conscientiousness, agreeableness, and openness to experience). HiTOP describes higher levels of psychopathology under the p factor as follows: internalizing, disinhibited externalizing, antagonistic externalizing, detachment, thought disorder, and somatoform. In principle, these relationships could be studied using a method of network analysis, which measures the links between different symptoms (McNally, 2021).

We need something new, and these ideas could be promising. What is missing is a basis in biology for any of the alternatives. The editors of DSM-III had hoped that biomarkers would emerge from a more systematic classification. But given that this has not happened, one has to question whether the DSM approach was ever likely to be related to biomarkers.

Another alternative system, developed at the National Institute of Mental Health (NIMH) in the USA, and promoted as an eventual alternative to DSM, is the Research Domain Criteria (RDoC; Insel et al., 2010). Here a hierarchy focuses on putative etiological factors, ranging from "neuron to neighborhood," placed on a matrix with dimensions described as negative valence systems, positive valence systems, cognitive systems, social processes, arousal and regulatory systems, and sensorimotor systems. However, the RDoC system is rather vague, and up to now has not led to any major changes in research priorities. It has also not been adopted in any serious way by clinicians. This may be due to its highly unfamiliar terminology as well as the lack of a user-friendly method for scoring its domains. If it had not been developed by the NIMH, RDoC would have received little attention. For now, the DSM system rules, at least outside the UK.

Another issue is that although the RDoC system differs more radically from both the DSM and ICD systems, and claims to be broad in coverage, it favors neuroscience over psychosocial factors in mental illness. Yet it lacks the data that would be needed to support such an ambitious proposal (Paris and Kirmayer, 2016). In the years since its introduction, the RDoC system has been applauded by researchers in neuroscience, but has not found any place in clinical practice. At best, it remains a rough draft that will require many decades of further work.

None of these alternative systems have been shown to improve outcomes for patients. My colleague Mark Zimmerman, who has done extensive research on diagnosis, insists that diagnostic systems should not be revised unless the new version leads to better treatment (Zimmerman, 2021).

Currently, while psychiatric treatment continues to be symptom focused, and despite a limited understanding of the causes of mental disorders, psychiatrists still do a reasonable job of helping most patients. We just need to be patient and await the results of the next few decades of research. In the meantime, we should avoid kidding ourselves that we know more than we actually do.

Overdiagnosis

Diagnostic classifications have a practical value, in that they are written to include almost every kind of case that clinicians see in clinical practice, whether the symptoms constitute a true disorder or not. No fundamental distinction is made between categories that describe brain disorders (e.g., schizophrenia, bipolar disorder, melancholic depression) and those that describe exaggerated reactions to life circumstances (Horwitz, 2002). The expansion of diagnosis into the vicissitudes of normal life is one of the main reasons for faddish diagnoses. Not all patients whom psychiatrists see are truly ill, but they tend to be labeled as if they were. Yet, to justify our interventions, we almost inevitably enter the terrain of overdiagnosis (Frances, 2013; Paris, 2020c).

The problem is not necessarily that clinicians create categories de novo. Instead, existing disorders are extended into broad spectra that overlap with normal variations. Another reason for overdiagnosis is that categories which are valid in prototypical cases are affected by "concept creep"—that is, the tendency to expand boundaries (Haslam, 2016). This is particularly notable in disorders that can be treated with specific pharmaceutical agents (e.g., bipolar II disorder, attention-deficit hyperactivity disorder).

Several forces have driven the process of overdiagnosis and unfounded claims for the validity of current categories (Frances, 2013). First, the DSM system made psychiatric diagnosis interesting enough to be accessible to the general public. Some categories have become popular because of media attention, rather than being rooted in a solid base of research. Second, psychiatrists are encouraged to diagnose conditions that are common in the general population, and which may or may not deserve "caseness." At the same time, many categories that are seen in adulthood have been extended into childhood, and thus children with similar symptoms can receive diagnoses traditionally reserved for adults. All of this creates a systemic problem which Frances (2013) describes as "diagnostic inflation."

The pharmaceutical industry plays a vital role in promoting diagnostic inflation, particularly in diagnoses for which they have products that they want to sell. With direct-to-consumer advertising (not allowed in most countries but endemic in the USA), industry interests have promoted overdiagnosis of various categories. In North America, one can hardly avoid seeing ads in the media that instruct people to wonder if they have depression or bipolar disorder, and to ask their physicians to prescribe drugs for these conditions. One can also see advertisements encouraging clinicians to make more frequent diagnoses of attention-deficit hyperactivity disorder (ADHD) (Hinshaw and Scheffler, 2014).

In addition, patient and family advocacy groups tend to encourage the use of categories in which they have a particular interest. Although this could be a good idea in principle, it almost always supports overdiagnosis. Also, the Internet encourages and facilitates diagnostic inflation, which the media then highlight. Many patients (or their families) have become convinced that a diagnosis is present as a result of learning about it on television or online. Finally, people who believe in the possibility of perfect health are intolerant of normal unhappiness and life's inevitable vicissitudes (Elliott, 2003).

Diagnostic Fads and Fallacies

Psychiatry borrows some of the prestige of medicine. Both physicians and their patients have come to believe that diagnoses of mental disorder are as real as cancer or cardiovascular disease. That is more or less true for the most severe disorders (e.g., schizophrenia, bipolar disorder, melancholic depression). But many common forms of psychopathology (e.g., substance use, eating disorders, personality disorders) can be better understood as dysfunctional exaggerations of normal variations.

Nonetheless, many of the patients whom I see describe the diagnoses they have been given, and hold on to them for dear life. In recent years, I have seen many patients who insist that they have a diagnosis of ADHD. This is a real clinical syndrome, but the diagnosis is often made with insufficient care (Hinshaw and Scheffler, 2014). ADHD has no biomarkers, and although psychological testing can be suggestive, it is never definitive. But patients who believe that taking a stimulant will solve most of their problems are hard to convince about these facts. Several of them have stormed out of my office when I declined to recommend this prescription.

Almost any explanation for suffering can make people feel better (Frank and Frank, 1991). Instead of feeling inadequate or at fault, patients can see themselves as struggling with forces that lie outside their control. What they do not know is that a diagnosis may sometimes reflect little more than a weakly supported opinion. Some patients also ask me if they can have a blood test or a brain scan. This might help to explain the popularity of self-report questionnaires that are used to "confirm" conditions with unclear boundaries. But since the gold standard for all such measures is still the signs and symptoms on which categories are based, even when converted into self-report questionnaires, these procedures are illusory, and they only offer the appearance of science. The history of medicine shows that treatments which are based on appearances, rather than on an understanding of pathological mechanisms, are less likely to be effective. What looks the same is not necessarily the same.

The most favored diagnoses, and the categories most likely to become fads, are those that lead to a specific treatment intervention. Thus, in patients who are feeling low for any reason, major depression may be diagnosed if practitioners want to prescribe pharmacological agents. This hammer quite easily finds its nail. Similarly, in patients who are moody or irritable, bipolar disorder may be diagnosed with the hope that mood stabilizers will be helpful. In reality, they have no effect on the mood instability that is seen in personality disorders (Crawford et al., 2018). Almost any patient who has problems with attention or concentration may receive a diagnosis of ADHD, after which stimulants can be prescribed. In reality, the same symptoms can be produced by anxiety or depression (Paris, 2015a, 2015b). Almost any patient with a difficult developmental history tends to be diagnosed as having PTSD. In reality, trauma is only one of many risk

factors that can lead to that syndrome (Paris, 2023). The problem is that all of these diagnostic labels are heterogeneous, and we have no biological markers to determine their boundaries.

Another set of diagnostic illusions arises from beliefs about the causes of mental disorders. The theory that mental disorders are due to "chemical imbalances" has been particularly attractive. It was widely promulgated by pharmaceutical companies who are selling agents that claim to restore these balances. I cannot recall how many lectures I have attended that showed beautiful slides of the synapse, with tiny balls of neurotransmitters moving gracefully across the gap. This is of course little more than marketing, offering the appearance of science, but without substance.

Neurotransmitters such as serotonin, dopamine, and norepinephrine, which are important mediators of neural processes, have been shown to be related to a wide variety of human behaviors, and to have activity that can be altered by psychiatric drugs. But this does not prove that an imbalance of neurotransmitters is the *primary cause* of mental disorders. These chemical changes could be secondary to other processes. One would have to show that patients with depression, bipolar disorder, or schizophrenia have a deficit (or abnormal production) of neurotransmitters. In the absence of that kind of evidence, one cannot base diagnoses on responses to pharmacological agents.

Simplistic ideas support fads, but are hard to give up. They offer a safe haven of certainty in uncharted waters. It could take another 100 years before psychiatrists understand the causes of mental illness and are in a position to use that knowledge to develop a valid classification. However, one can readily understand why clinicians want answers now.

Lack of knowledge about the nature of mental disorders has not prevented the emergence of fads, and has in fact made their appearance more likely. Most involve a major expansion in scope for diagnoses that are already in existence. Fads tend to develop in areas of psychiatry that have been inadequately studied, and are applied to patient groups who do not respond to standard therapy. Like many errors in medicine, they reflect good intentions gone wrong.

Almost any symptom pattern can be turned into a diagnosis. This kind of fad takes an established diagnostic entity, and then expands its boundaries to account for symptoms in a much broader spectrum of patients. The process of diagnostic inflation and concept creep applies particularly to major depression, bipolar disorders, ADHD, autism spectrum disorders, and post-traumatic stress disorder (PTSD). There are many other examples in the DSM manuals. Moreover, the entire manual is compromised by concept creep. Few experts who have attempted to do research on psychiatric diagnosis seriously believe that there are really hundreds of separate categories of mental illness.

Major Depression

Depression lies at the heart of psychiatry. So how can it constitute a fad? It is hard to convince many psychiatrists that there is even a problem. Some years ago, at an annual departmental party in which residents present "skits" that satirize their teachers, I was portrayed as saying, "I don't believe in depression, but I still feel depressed." Perhaps what did not come across to my trainees was that I was not questioning the existence of depressive mood itself, but the validity of the diagnosis of a "major depressive episode" as defined in DSM.

People do feel low and suffer from altered mood. But depression can be a symptom, a syndrome, or a disease. The Australian psychiatrist Gordon Parker has been the most convincing critic of the concept of major depression, and he has argued that melancholia is the form of depression that best qualifies as a disease (Parker, 2011). These patients can have a severe lowering of mood associated with prominent physical symptoms, and they can sometimes be psychotic. These are also the patients who respond best to antidepressant therapy (Kirsch et al., 2008), and in whom pharmacological augmentation is most likely to be successful.

In contrast, patients with milder depression may not actually have a disease, but can be better described as *unhappy*. There are many reasons for being unhappy in life, so feeling low from time to time can be considered as part of the human condition, rather than something to be routinely "medicalized." Horwitz and Wakefield (2007) have strongly argued that psychiatry has confused sadness with illness.

The diagnosis of a major depressive episode, a term originally introduced in DSM-III, describes a very broad concept. The description of depression in ICD-11 is similar. In DSM-5, one only needs to meet five out of nine listed criteria to receive this diagnosis. (Again, it was arbitrarily decided that more than half of the clinical features should be sufficient.)

Crucially, the duration of symptoms required is only 2 weeks. It is hard to understand the adoption of such a time scale (which is also found in ICD-11). Almost anyone who is consistently unhappy for a fortnight, and has other symptoms, such as insomnia and loss of concentration, can be diagnosed as having a major depressive episode. This low bar makes the prevalence of the condition very high. In fact, data suggest that over a lifetime up to 50% of people meet these criteria (Moffitt et al., 2010). Although it is true that we also have a high lifetime prevalence of viral infection, infectious diseases can be identified by biomarkers.

Unfortunately, the diagnosis of a major depressive episode is not held in reserve if patients become depressed as a result of adverse life events. DSM-5 removed these exclusions, particularly the one for grief. And in DSM-5-TR, a diagnosis of "prolonged grief disorder" has been added for those who do not recover from a loss, although it is not clear what implications this change will have for treatment. The assumption is that any set of symptoms that lasts for a specific time period may reflect an abnormal response to a life circumstance. Perhaps the researchers who wrote these criteria had never suffered a severe loss. Arthur Kleinman, a well-known cultural psychiatrist, took a very different view in an article published in *The Lancet*, in which he described his own experience of profound grief after the death of his wife (Kleinman, 2012).

Psychiatrists consider depression to be a single disorder, not a group of disorders. Fifty years ago, Akiskal and McKinney (1973) argued in an influential review paper published in *Science* that the traditional distinctions between depressive subtypes were invalid. They pointed out that patients with melancholia can have first-degree relatives with milder depression, and that some patients with mild symptoms have a family history of severe depression. Akiskal and McKinney also claimed that all types of depression respond to antidepressants. Their ideas influenced the definition of major depressive episode that appeared in DSM-III. The symptom picture that it describes still applies to all forms of depression in DSM-5, although clinicians are invited to code severity.

Akiskal and McKinney had a point. There is no absolute separation between melancholia and mild to moderate depression. That does not, however, prove that one can describe points on a smoothly varying continuum. Moreover, conflating different syndromes has had problematic clinical implications, in that all patients with "major depression" would be offered antidepressants—which is, of course, exactly what happened.

The unitary theory of depression is rooted in the worldview of biological psychiatry. It has convinced practitioners that milder disorders can be treated with the same drugs as severe mental illness—that is, with antidepressants and antipsychotics. We have long established that antidepressants are effective drugs, and that their efficacy is higher than that of placebo (Gillett et al., 2020). But although some patients with mild depression do respond to antidepressants, initial severity is the best predictor (Kirsch, 2009). Psychotherapy may take more time (and requires longer appointments), but its short-term effects are as robust, and its long-term effects tend to be superior to those of antidepressants (Cuijpers et al., 2020). These findings show that the routine diagnosis of major depression, followed by an immediate prescription, is not good clinical practice.

Let us consider the analogy of a common cold and pneumonia. Colds derive from a viral infection, but patients sometimes develop pneumonia as a complication. That does not make them one and the same disease. Similarly, we do not have evidence for concluding that all depressions are milder or more severe versions of the same illness. There are very good reasons, particularly treatment efficacy, for separating off melancholia from milder depression (Parker et al., 2010).

Today psychiatrists are being trained to treat *all* depressions with antidepressants. This practice takes it for granted that once you have made that diagnosis, the patient should require medical treatment. Some practitioners are even afraid that lawsuits could ensue if they fail to prescribe. That actually happened in a famous case in the USA (Healy, 1997). The patient was a physician who had been hospitalized for melancholic depression, but was misdiagnosed as only having a personality disorder, leading to treatment with psychodynamic therapy. However, psychiatrists can correctly prescribe psychotherapy for outpatients with depression, and would be best advised to try therapy first for all but the most severe depressions, and to use antidepressants as a back-up. They should not have to worry about lawsuits, as this practice is well supported by evidence (National Institute for Health and Care Excellence, 2022).

Antidepressants do not always relieve symptoms, and even when patients seem to respond to these agents, one cannot be sure whether or not one is observing a placebo effect. This could explain why so many patients describe feeling better after receiving a new prescription, but later finding that these effects mysteriously disappear. This sequence is a cardinal feature of placebo responses (Benedetti, 2008). Similarly, it helps to explain why some patients feel worse when their drugs are stopped, even within as short a period as 24 hours (a time scale inconsistent with the half-life of these agents).

Nevertheless, the wish to prescribe drives a rush to diagnosis. And even though the finding that antidepressants can act as placebos has been widely publicized, this knowledge has not had much effect on practice. A survey of prescription rates for antidepressants in the USA (Pratt and Brody, 2011) found that 11% of the entire population (and 16% of women) were currently taking one of these drugs. Although antidepressants are also used for anxiety and insomnia, the most common reason for prescribing them is a

diagnosis of major depression. Treatment of this kind also tends to be interminable, based on a fear of relapse.

In summary, although melancholia is a disease that requires a biological approach (antidepressants and/or ECT and/or antipsychotics) for effective treatment, depression without melancholia is a heterogeneous syndrome that may or may not benefit from pharmacotherapy. Calling both by the same name (and treating them in the same way) was a fallacy that became a fad. By setting the bar too low, and by diagnosing depression on the basis of symptoms that last for short periods, or on the basis of criteria that closely resemble unhappiness or sadness, psychiatrists are overdiagnosing the disorder, leading millions to be given ineffective prescriptions. A unitary theory leads to unitary therapy. We need to think of mental illness as a set of spectra, each of which fades gradually into normal variation (Grinker, 2021). Until we have biological markers to identify those cases who are most likely to respond to pharmacological intervention, these problems will continue to plague practice.

Major depressive disorder is a paradigmatic example of the problems of psychiatric diagnosis. Younger clinicians may not realize that the category is relatively new, introduced by DSM-III in 1980 (Shorter, 2009). In DSM-II, psychiatrists distinguished between "endogenous" and "reactive" depression. The endogenous type was equivalent to melancholia, a severe and sometimes life-threatening illness that tends to come "out of the blue" (Parker, 2005). In contrast, reactive depressions are brought on by stressors, and remit when circumstances change for the better. Perhaps no one should diagnose depression if symptoms remit when patients find better lovers or become unexpectedly wealthy.

The reason why the lifetime prevalence of major depression could be as high as 50% is that depression is not always that "major." Most patients with mild depressive symptoms never come for treatment, and usually recover spontaneously (Patten, 2008). Thus current prevalence estimates reflect a low bar for diagnosis of depression (Horwitz, 2002), and do not distinguish between disabling pathology and brief episodes of lowered mood, particularly when associated with sadness or grief (Horwitz and Wakefield, 2007). This is not to say that patients who do seek help for less severe symptoms do not have a problem. But they need not be classified as having a disorder called major depression.

Inflated prevalence also creates a problem for mental health policy. Epidemiological findings can be used to guide the way in which resources are allocated. Even though mental illnesses are highly prevalent, only a minority of potential patients will seek treatment (Kessler et al., 2005b). This gap has often been seen as a cause for concern. Yet we need to be respectful of people who choose to solve problems on their own. Moreover, patients with milder depression are a mainstay of psychotherapy practice for clinical psychologists, whose tools are often sufficient to manage these problems.

Depression screening programs have a weak rationale—we should not be screening the population to find unhappy people, and treating those who are only unhappy is a poor use of scarce medical resources (Patten, 2008; Thombs et al., 2012). These methods identify people who are distressed enough to meet the criteria for major depression, but who only have mild or transient symptoms. We should be making it easier for *severely* ill people to seek treatment, rather than focusing on people with milder symptoms who are less likely to need our services. Thus concept creep has seriously expanded the definition

of major depression, with a loss of older ideas about the normality of sadness and the human condition.

Bipolar Spectrum Disorders

I shall now examine a diagnosis that is perfectly valid in "classical" cases, but that has also been stretched to the point of invalidity by concept creep.

Bipolar disorder has been a major category of mental illness for over a century (even after undergoing a name change from "manic depression"). But like many categories in psychiatry, bipolarity has fuzzy edges, particularly in patients who experience mood swings. A new category of bipolar II was introduced in DSM-IV to describe those who have never had a manic episode, but who have hypomanic periods (i.e., elevated mood and high energy persisting for at least 4 days). Since then, the idea of a bipolar spectrum has been promoted for all kinds of cases in which mood instability is a feature.

Some years ago, I wrote a book-length critique of this idea (Paris, 2012a). The problem is that unstable mood is a feature of a broad range of mental illnesses, most particularly personality disorders and substance use. But these patients do not respond either to lithium or to the anti-epileptic drugs that have been called "mood stabilizers." For this reason, the concept of a bipolar spectrum leads to prescriptions of pharmacological treatments that patients do not need.

Bipolar I disorder is one of the few conditions in psychiatry that closely resembles a medical illness. There is nothing faddish or fallacious about that construct. Although we do not know the etiology of classical bipolarity, it has, as described by Kraepelin (1921), a characteristic course and outcome. Diagnosis in this case also points to a specific approach to treatment, using mood stabilizers such as lithium (Goodwin and Jamison, 2007).

Bipolar II disorder, which is characterized by hypomania rather than full mania, is somewhat more controversial, but when used cautiously it is also fairly well validated, at least when the requirement for hypomanic episodes is carefully observed (Parker, 2019). However, this condition is being seriously overdiagnosed. One reason for this is that the criteria for a hypomanic episode, listed in both the DSM and ICD, may be stretched or ignored.

I am surprised how many of my colleagues are unaware that hypomania requires 4 days of *consistently* abnormal mood. Although that time scale is arbitrary, one has to draw the line somewhere. Otherwise, patients who describe mood swings of any kind, including euphoric episodes that last for only a few hours, can be given a bipolar II diagnosis. Also, DSM-5 allows clinicians to give patients with briefer mood swings a diagnosis of "bipolar disorder, not otherwise specified," which allows for a vast expansion of the concept. Moreover, physicians have been primed to consider bipolarity in an all-too-common clinical scenario—when antidepressants fail to help patients.

The expansion of bipolarity has been justified by the concept of a "bipolar spectrum," which is thought to explain all patterns of mood instability. There may be some kind of spectrum here, but that does not mean that everyone who falls within it has the same disorder. This fad has reached the point where a survey using the spectrum concept identified over 30% of depressed patients in one clinic as having some form of bipolarity (Angst et al., 2011), and it was estimated that close to half of all psychiatric patients could meet the broadened bipolar criteria (Akiskal, 2006). This is concept creep on steroids.

Some patients with melancholia do "convert" to bipolarity, but this outcome is less common in milder depressions (Parker, 2019). The problem is that when patients fail to respond to antidepressant therapy and are labeled "treatment resistant," bipolarity is suspected, and they may be treated as bipolar in the absence of any clinical features. Moreover, patients who are placed on a putative bipolar spectrum for mood swings, such as those seen for BPD, do not respond to mood stabilizers at all, and certainly not in the way that classical cases do (Crawford et al., 2018).

Claims that a large number of outpatients in psychiatry have an occult form of bipolarity are based entirely on the presence of "soft bipolar" symptoms, namely mood instability and irritability. But these features are also very common in other conditions, particularly personality disorders. Irritability is even more non-specific. Common symptoms such as these are no substitute for biological markers. In the absence of markers, bipolarity has usually been assessed with self-report measures or standard interviews. But these scales measure not bipolar disorder, but "soft bipolarity." For example, the popular Mood Disorder Questionnaire (Hirschfeld et al., 2000), which is sometimes used to "confirm" bipolar diagnoses, has been shown to be more sensitive to BPD than to bipolarity (Zimmerman et al., 2010).

The fad for a bipolar spectrum also spread to child psychiatry, a development that has aroused serious concern among clinicians. Bipolar disorder, long considered to begin only at or after puberty, has been diagnosed in young children to account for severe behavioral symptoms. The alarm was raised in North America when it was realized that some pre-pubertal children were being prescribed mood stabilizers and antipsychotics for years. For this reason, DSM-5 added a new diagnosis of "disruptive mood dysregulation disorder," which was specifically designed to keep young children out of bipolar therapy (Margulies et al., 2012). Moreover, children with irritable and unstable mood do not develop classical bipolar disorder over time, and do not respond to treatments developed for adult patients with bipolar disorder (Duffy, 2007). When these children are followed into adolescence, they do not develop classical bipolar disorder, but continue to have "soft bipolar" symptoms (Geller et al., 2008).

The bipolar spectrum fad would not be so worrying if it were not for the fact that making these diagnoses leads to the prescription of drugs associated with significant side-effect burdens. The long-term use of mood stabilizers and antipsychotics for patients who do not have classical forms of bipolar disorder could be a tragedy. These patients do have a mental disorder, but, in adults, rapidly shifting mood (over hours instead of weeks) is more likely to point to BPD (Koenigsberg, 2010). In children, most putative bipolar cases also meet the criteria for conduct disorder, oppositional defiant disorder, or ADHD (Geller et al., 2008), and DSM-5 has added a new (non-bipolar) category of "disruptive mood dysregulation disorder" to account for this clinical picture. All of these diagnoses have their own problems with validity. But diagnosing *all* unstable mood as bipolar is misguided in theory and potentially harmful in practice.

Attention-Deficit Hyperactivity Disorder (ADHD)

ADHD begins in childhood, but its symptoms can persist into the adult years in up to 50% of cases (Hechtman, 2016; Sibley et al., 2022). This disorder may reflect, at least in part, an "evolutionary mismatch." If schooling was not required by modern society, there might be no such syndrome (Hinshaw and Scheffler, 2014). Many children are overly

excited and distractable at times, and boys in particular tend to have trouble sitting quietly for hours in a schoolroom.

Overdiagnosis of ADHD, particularly of the inattentive type, in adults has also become a major problem, since it leads to the long-term prescription of stimulants, possibly for life (Paris et al., 2015). Although these agents are safer than many drugs that are commonly given to patients these days, we do not know what effects they might have over a period of decades.

One of the most striking fads in contemporary psychiatry has been the extension of the ADHD diagnosis into adult populations. But to diagnose adult ADHD, a definite history in childhood has to be established, which may require a careful review of school records. Unfortunately, clinicians who are evaluating adults tend to ignore this requirement. This is increasingly likely to happen when patients arrive in clinics convinced that their "lack of focus" is a symptom of ADHD, and demanding to be put on stimulants. Physicians may be reluctant to argue with patients in these circumstances.

What too often happens is that ADHD is identified by problems with attention, whatever the cause. But many other disorders, including depression, anxiety, and personality disorder, can also lead to attention difficulties. Using the current low bar for diagnosis, the National Comorbidity Survey in the USA found a community prevalence of 4.4% for adult ADHD (Kessler et al., 2009). That is a very high figure for a disorder that was rarely diagnosed in adults 20 years ago.

The frequency with which stimulants are now prescribed to both children and adults has increased dramatically, doubling in a decade (Piper et al., 2018). Many or most of these patients will not have had similar symptoms prior to puberty, as required by DSM-5. (However, patients may insist that they did have these problems, but that no one noticed.)

This discrepancy was highlighted by a report from Moffitt et al. (2015), who studied a birth cohort in Dunedin, New Zealand, that has been followed for decades. The results showed that the majority of those who met the adult criteria for ADHD had *not* had the same problems in childhood, which suggests that, at best, adult ADHD could be a separate illness. In other words, longitudinal data show that most adults with ADHD symptoms did not have them as children, whereas most children with such symptoms lose them in adulthood. There is some evidence to the contrary, provided by the longitudinal follow-up of children in a major research program, which showed that about 50% of them continue to be symptomatic in adulthood (Sibley et al., 2022). But an intensively studied population of well-diagnosed patients may not be typical of the wider community. The most likely explanation for the increasing prevalence of adult ADHD is that misdiagnosis arises from other problems that also lead to a "lack of focus."

ADHD has been the subject of a vast research literature, and the use of stimulants to treat children with classic features of the disorder has a strong evidence base (Barkley, 2014). As with bipolar I, classic cases can be uncontroversial. However, many problems remain in the definition, particularly in adults. ADHD exists in one form in which hyperactivity is prominent, and in a second, more poorly defined form that is characterized by inattention (Carlson and Mann, 2000). Treatment is more effective in the presence of the clinical picture in which hyperactivity is predominant (Leung and Lemay, 2003). But these are not the adults for whom these agents are being prescribed. ADHD has become a cure-all for lack of focus and attention.

Given the promise of a quick fix with a stimulant prescription, one can understand why clinicians may give in to the temptation to see every problem with attention or lack

of focus as justifying a diagnosis of ADHD. Moreover, children and adults with these symptoms may not routinely need stimulants but can be treated with cognitive therapy (Hinshaw and Scheffler, 2014).

Worryingly, the diagnosis is often "confirmed" by psychological testing. But these procedures do not yield conclusive results, since they lack the specificity and sensitivity that physicians expect from blood tests or imaging (Berger, 2011). Questionnaires such as the Conners' Parent Rating Scale (Conners et al., 1998), which ask parents to tally up symptoms described in the manual, are also suggestive but non-specific to the diagnosis.

ADHD is real, but the drastic extension of its boundaries as a diagnosable disorder has reached a point of serious faddishness. The community prevalence in children has steadily increased, rising as high as 9.5%, with a particularly striking increase among adolescents (Zuvekas and Vitiello, 2012). The high prevalence in the USA may be due to the fact that clinicians in that country have a lower threshold for identifying the disorder, or it may reflect a difference in the social context, or failure to recognize the syndrome in other countries (Faraone et al., 2003). Faraone and Biederman (2005) identified 2.9% of the population as meeting the criteria for ADHD, but stated (ominously) that a broader definition could support a prevalence of 16.4%. Meanwhile, the number of diagnoses of ADHD has continuously increased over the years. Fairman et al. (2020) reported an increase of 36% in adults and 18% in clinical populations of young people between 2008 and 2013.

The hyperactive type of ADHD is easier to identify, and is more responsive to stimulants. But when the diagnosis was expanded to include an inattentive subtype, treatment was less effective (Barkley, 2014). Moreover, the long-term results are not as dramatic as many clinicians think. Although the short-term effects of stimulants are well established, one of the largest-scale multi-site studies of treatment (Molina et al., 2009) found that when children were followed up over 6 to 8 years, the outcome was no better than with non-pharmacological interventions.

It was once thought that one could diagnose ADHD by a positive response to stimulants. Yet even healthy people have better attention when they take these agents (Rapoport et al., 1978); students may use stimulants to improve their performance in examinations. Thus stimulants might be compared to eyeglasses that correct normal but problematic variations (Hinshaw and Scheffler, 2014). Helping people to be more focused through drugs has been called "cosmetic psychopharmacology" (Kramer, 1993), a practice in which people take drugs in order to be "better than normal."

Once patients become attached to an ADHD diagnosis, they can be tempted to explain all their problems on that basis. This is part of a much vaster trend—the medicalization of all kinds of life problems. Today university students may be offered extra time to write examinations because they have a "disability." The case may be "proven" by psychological testing procedures that can identify a profile of strengths and weaknesses in anyone. If you perform poorly in school or at work, it is not because you need to try harder. It is because your brain is malfunctioning, and you need to take a stimulant.

My view is that future psychiatrists may well regard the diagnostic epidemic of ADHD with regret. It is not the phenomenon itself that is questionable, but the concept creep, leading to instant recourse to the prescription pad for complex problems with complex causes.

Autism Spectrum Disorders

In DSM-5, autism and Asperger's syndrome have been folded into a broader autistic spectrum. As often happens when psychopathology is seen in terms of spectra, diagnoses have increased from being rare to being relatively common (Fombonne, 2018). A survey in South Korea (Kim et al., 2011), which garnered much attention from the media, found autism spectrum diagnoses in over 3% of the general population.

Again, there could be several explanations. One possibility is that there could be better detection of these disorders. Another is that there could be a re-diagnosis of other conditions, perhaps driven by a need for access to specialized services. (Although in contrast to bipolar disorder or ADHD there is no specific drug therapy, autistic children do need psychosocial education.)

We may be seeing another example of valid diagnoses being stretched to account for phenomena that may or may not be related. Although classical cases of autism are unmistakable, the concept is being stretched to the point of describing normal variants. Moreover, other conditions can produce cognitive impairments similar to those seen in the autism spectrum. There is also no gold standard for separating Asperger's syndrome from social anxiety and high levels of introversion.

The overdiagnosis of autism spectrum disorders could well be a fad, in which even people outside of medicine are describing almost everyone with eccentricity and social awkwardness as falling within its boundaries. Yet without biological markers, how can we be sure? The diagnosis of autism has become imperial, crossing the boundaries of normality and conquering them.

Children can be quite different in temperament, so trait profiles vary because each can be adaptive under the right circumstances (Beck and Freeman, 2002). Thus, although extravert and impulsive children can have problems in modern urban society, in another setting they may react quickly in a more positive way—some might grow up, for example, to become soldiers or police officers. Similarly, introverted and socially awkward children can use their traits in positive ways—for example, by going into computer science. But if every nerdy child is diagnosed with an autism spectrum disorder, many will be unnecessarily stigmatized.

To counter stigmatization, it has been suggested that autism should be seen as "neurodiversity" (Silberman, 2015). But this concept fails to address the problem of clinical overdiagnosis. Once again, concept creep has affected the definition of autism, leading to a worrisome fad.

Post-Traumatic Stress Disorder (PTSD)

PTSD has been an attractive diagnosis. This is mainly because, unlike most other categories in the DSM manual, it suggests an etiology. It is also popular because it is less stigmatic and it validates a sense of victimization (Grinker, 2021). It has been said that PTSD is the only psychiatric diagnosis that people actually *want* to have, mainly because it puts the blame on other people and not on themselves.

PTSD diagnosis in DSM-5 requires a history of exposure to life-threatening trauma. Yet most people who are exposed to traumatic events never develop PTSD. Thus trauma is not the only cause of the syndrome. (Horwitz, 2018; Paris, 2023).

The Epidemiological Catchment Area Study, based on the DSM-IV definition, found a community prevalence of 7.8% for PTSD (Kessler et al., 2005a). The DSM-5 definition

of PTSD is not radically different (Pai et al., 2017). But PTSD is not common after adverse life events that do not involve the threat of serious injury. Concept creep has been a problem in previous editions of the DSM, as the definition of a stressor was extended to witnessing traumatic events, or only hearing about them. This was a serious mistake, which led directly to overdiagnosis (McNally, 2011).

Clinicians may not always know that most exposure to trauma does not lead to PTSD, or that the vast majority of people are resilient to adversities of all kinds. Most exposures carry a risk of 5–10%, and even the most pathogenic life event (rape) leads to PTSD in only 20% of those exposed.

The explanation lies in temperamental vulnerability. People who develop PTSD are high in neuroticism—that is, they have stronger emotional reactions to adverse experiences (McFarlane, 1989; Breslau et al., 1991). PTSD is another example of how the etiology of most mental disorders cannot be understood in terms of life experiences alone, but emerges from gene–environment interactions (Paris, 2023). Thus patients with PTSD react to trauma in a unique way, and—like most mental disorders—PTSD requires a biopsychosocial model.

Why is this issue not widely understood? One reason is that we now live in a kind of "culture of trauma," associated with high levels of sympathy for victims and a progressive political ideology. Clinicians tend to move too quickly from a history of traumatic exposure to a diagnosis of PTSD—a good example of availability bias. They do not always take the time to determine whether the characteristic symptoms of the disorder (hyperarousal, intrusive memories, and avoidance of stimuli that re-evoke the response) are present.

Psychiatrists and family doctors may fall in step with popular fads that affect the way ordinary people talk about life and its vicissitudes. Everyone who is moody can be seen as "bipolar," everyone who fails to pay attention can be seen as having ADHD, and anyone who has an adverse reaction to life events must have PTSD. Again, a stressor alone is not enough, and the specific symptoms of PTSD have to be present. Moreover, in practice, clinical cases can overlap with depression (Bodkin et al., 2007).

The point is that a history of trauma, no matter how remote in the patient's life history, need not be specifically associated with a clinical problem, or converted into a diagnosis. That is fallacious and faddish thinking. The diagnosis of PTSD is useful when applied cautiously, but becomes a fad when misapplied. It oversimplifies clinical reality by pretending, but ultimately failing, to provide an etiological explanation. The effect of this diagnostic fad is to slot patients into a diagnosis that may not explain symptoms, and can lead to the wrong treatment—either the wrong psychotherapy or unnecessary psychopharmacological interventions.

In summary, the diagnostic fad for PTSD says as much about our culture as it does about the effects of trauma on the mind. The current emphasis on trauma in psychiatry and clinical psychology is problematic. This is not to say that trauma has no importance in the risk for mental illness. There is a grain of truth, as clearly adverse events are bad for us, but the way it is being used by clinicians is a fad and a fallacy. Many disorders, even including psychoses, are correlated with histories of traumatic events in childhood or adulthood (Paris, 2023). But that does not make trauma the primary cause of these forms of psychopathology—even in PTSD.

Several issues need to be understood in order to put the problem into perspective. Crucially, traumatic events are usually accompanied by other psychosocial stressors.

Thus abused children grow up in highly dysfunctional families (Assink et al., 2019). BPD is associated with severe childhood trauma in up to a third of cases, but, as shown by a recent meta-analysis, the most consistent risk factor is not abuse, but emotional neglect (Porter et al., 2020).

One way of looking at the problem is to distinguish between PTSD and personality disorder, which sometimes overlap. What ICD-11 calls "complex PTSD" (CPTSD), in which multiple exposures to trauma have broad and cumulative effects on development, significantly overlaps with BPD. My reading of the literature is that childhood trauma aggravates the risk for PTSD, making it much more chronic (Paris, 2020b). But allowing the concept creep of PTSD overshadows much more complex patterns, both genetic and environmental, that lead to PTSD. This is the kind of mistake that occurs when a diagnostic fad spreads.

We are up against what the sociologist Frank Furedi has called a *culture of trauma* (Furedi, 2003). Clinicians may see their patients as victims of past adverse experiences, and promote PTSD and/or CPTSD as a way of advocating for them. But a diagnostic practice in which personality is downplayed or ignored can do a major disservice to patients. Since the foundational research of Linehan (1993), we understand that the origins of BPD are "biosocial," and that they depend on interactions between hereditary risks and dysfunctional families. Yet, as I shall discuss in Chapter 6, faddish "trauma-focused" therapies are being offered to people whose problems lie primarily in problematic intimate relationships.

Why We Have Difficulty Avoiding Diagnostic Fads and Fallacies

Psychiatrists are faced with complex symptoms that are difficult to understand and difficult to classify in a meaningful way. It is tempting to fall back on diagnostic fads that make complexity simple. For each of the faddish diagnoses described in this chapter, a therapy can be attached. Unfortunately, many of these treatments are unproven, ineffective, or harmful.

On the whole, the more severe the mental disorder, the better the system works. There can be little doubt about diagnosis when a patient presents with a classical picture of schizophrenia, melancholia, or bipolar disorder. Psychiatrists today spend more time than ever on these patients. The main problems with the DSM system concern the common mental disorders, conditions that are often difficult to classify. Many of the patients who are seen by psychiatrists do not fit neatly into any category.

The danger, as pointed out by Batstra and Frances (2012), is that diagnostic inflation identifies people with normal problems as mentally ill. In the milder ranges, they might be better described as distressed than as disordered. We are shoehorning people who feel sad into depression, people who are moody into bipolarity, and people who have trouble focusing into ADHD. The overdiagnosis of mental disorders does a disservice both to patients and to psychiatry as a whole.

Diagnosis and Epidemiology

Epidemiology, the study of factors that govern the frequency of disease in the community, can be the first step in understanding why people fall ill. Clinical samples are unrepresentative of what illnesses look like in the community. In contrast, community surveys tell one how frequent disorders are, and examine their correlates in a wider range of cases.

In psychiatric epidemiology, many large and expensive studies have been conducted over the last 25 years, particularly in the USA. Research began with the Epidemiological Catchment Area Study (Robins and Regier, 1991), was expanded in the National Comorbidity Surveys (Kessler et al., 2005a, 2005b), and has been augmented by large-scale studies of substance abuse (Grant et al., 2004). Yet many of the conclusions that have been drawn by these studies remain doubtful. Some of the methodological problems include an almost complete reliance on self-report, the use of cross-sectional assessments without longitudinal follow-up, and the assignment of interviews to partially trained research assistants (Newson et al., 2011). But the most worrying issue is that so much of psychiatric epidemiology has depended on diagnosis as defined by our current classification systems. No one knows whether diagnoses, such as those derived from the DSM or the ICD, are as valid as medical conditions.

When research has moved beyond these diagnoses, it has assessed the frequency of symptoms considered to be subclinical manifestations (e.g., Merikangas et al., 2007). In the absence of any gold standard for determining the boundary between normality and pathology, this approach leads to a consistent inflation in estimates of prevalence.

Grant reviewers have to read a large number of submissions, and easily get tired. That is why researchers learn to write the first paragraph of their applications to grab the reader's attention. The most common strategy used by grant writers is to support the importance of research by describing the prevalence of the condition under study and its impact on public health. For example, if the grant concerns alcoholism, reviewers can be told that this condition affects 10% of all men (Robins and Regier, 1991). If the research concerns major depression, reviewers can be informed that surveys find that 13% of all men and 21% of all women will meet the criteria at some point during their lifetime (Kessler et al., 1993). The question is whether these numbers are credible. Many people drink too much, almost everyone gets depressed from time to time, and most individuals recover on their own. High-prevalence disorders of this kind are not easy to separate from life's problems.

Depression is particularly ubiquitous. If one accepts the definitions in diagnostic manuals, the high prevalence found in surveys is actually an underestimation. The reason for this is that reported numbers depend on recollection of past episodes, which can be forgotten. In a prospective longitudinal study (Moffitt et al., 2010), the rates of common mental disorders were twice as high as in community surveys, and major depression attained a lifetime prevalence of 41%. This kind of research also suggests that almost half of the general population will meet the criteria for a mental disorder at some point during their lifetime (Moffitt et al., 2010).

This finding could be considered either alarming or reassuring. On the one hand, mental disorders, or at least the symptoms of disorder that the DSM cobbles together into categories, are part of the human condition. On the other hand, if 50% of us go through life without suffering from a mental disorder, that is more than can be said about physical illness. One might argue that if 50% of the population never experience mental disorder, this is in fact good news. Perhaps doubts about the prevalence of mental illness only reflect the continuing stigma attached to any psychiatric diagnosis.

But what if the mental disorders studied by epidemiologists describe life problems that are fuzzily defined and overidentified as diagnostic entities? There is little doubt that severe and persistent mental disorders qualify as medical illnesses, and one might add to them severe forms of substance abuse and personality disorder. But most of the others

are disorders or syndromes, not diseases in the same sense as tuberculosis or epilepsy, even if they describe problems that clinicians see. The best examples are milder forms of anxiety (Horwitz and Wakefield, 2012) and mild to moderate depression (Horwitz and Wakefield, 2007), both of which are associated with distress. Although such feelings are often a reason for consulting a mental health professional, there is a qualitative difference between unhappiness and mental illness.

Psychiatrists do not have a way to define the concept of mental disorder, or to separate it from the vicissitudes of the human condition (Frances and Widiger, 2012). Everyone (with the exception of those who espouse radical antipsychiatry) agrees that schizophrenia and bipolar disorder are diseases in the sense that medicine has always recognized. But do milder disorders qualify for the same status?

Let us reconsider the diagnosis of major depression. It requires only 2 weeks of symptoms, often following exposure to psychosocial stressors. Most people recover from these episodes without treatment, and mild depression may be better understood as a reaction to circumstance than as an illness (Horwitz and Wakefield, 2007). Most of these depressions remit spontaneously after a few weeks (Patten, 2008). That is because they are temporary reactions to circumstance, even if they easily meet current criteria. The bar for major depression is so low (five out of nine symptoms over only 2 weeks in the DSM system) that anyone who is acutely or chronically unhappy can receive a diagnosis.

I am not saying that these patients should be turned away because they are not sick enough. Although most psychiatrists concentrate on more serious cases, unhappy people also need to be evaluated. But they do not necessarily require the same kind of specialized help that is provided for patients with severe illnesses. Physicians sometimes manage the common cold, but "normal" conditions are distinguished from serious illnesses such as pneumonia. The tendency to see all psychopathology as lying on a continuum is one source of the trouble. If there is no boundary between normality and pathology, *everyone* could have a mental disorder, making the whole concept meaningless.

These distinctions are also important because diagnostic categories are rather heterogeneous, and treatment indications can be different for different patients. In depression, mild cases differ greatly from severe ones, and in anxiety disorders, clinicians need to distinguish normal reactions to life stressors from disabling dysfunction such as recurrent panic attacks (Horwitz and Wakefield, 2012).

Psychiatric diagnosis has become imperial in its scope. Patients with normal or near-normal problems can receive both diagnosis and treatment. This may not be a new problem, but it is getting worse. High rates of symptoms in the population have been found in all studies based on the DSM system, including the Epidemiological Catchment Area Study (Robins and Regier, 1991) and the National Comorbidity Surveys (Kessler et al., 1994, 2005a).

Depression, anxiety, and alcoholism are examples of high-prevalence disorders, but many of the same problems arise when less common disorders are broadly defined as part of a spectrum. Some epidemiologists have claimed a very high rate for bipolar disorders, the prevalence of which has dramatically increased from 1% to 4% (Merikangas et al., 2007). ADHD, which cannot be reliably separated from other behavioral disorders, has been estimated to affect 10% of all boys in school (Barkley, 2014).

This level of diagnostic inflation is a fad based on a fallacy. Thus community surveys are used to support the claim that disorders are much more prevalent than was

previously thought. The implication is that psychiatrists have been missing something important, and that they need to lower their diagnostic bar.

In every case, it is simply *assumed* that the diagnoses based on criteria or prototypes listed in the manual are valid. But that cannot be taken for granted. In a previous era, when researchers took classification less seriously, the preferred method was to measure symptoms or levels of functioning, instead of illness categories. Classic research studies from up to 60 years ago, such as the Midtown Manhattan Study (Srole, 1980) and the Stirling County Study (Leighton et al., 1963), both of which were conducted before the publication of DSM-III, measured distress or dysfunction and ignored diagnosis. Even so, the same problems emerged, in that at least a third of the general population scored as mentally ill in some way. At the time, many were shocked at this claim. Today few would even blink at it.

Some researchers conducting surveys that identify problems caused by subclinical symptoms have expressed deep concern that so few of these individuals seek therapy. This may merely show that participants in community surveys are wiser than epidemiologists. Nobody has ever shown that drugs are effective for simple moodiness (Patten and Paris, 2008). This normal variation does not require medical intervention. Moreover, increased emotional reactivity can be adaptive under some circumstances (Beck and Freeman, 2002).

Psychiatrists have more than enough to do without looking for more business. (Those of us who work for the government are always swamped by demand.) With support from public and/or private insurance, mental health practitioners will almost always have more patients than they can manage. In any case, psychiatrists are highly trained specialists who should be focusing their efforts on the sickest patients. Little will be accomplished if screening programs based on epidemiological surveys bring large numbers of patients with mild symptoms into the busy world of primary care. In the end, people are wiser than we give them credit for. They are perfectly right not to go to psychiatrists or other mental health professionals for minor problems. Nor should epidemiology be used to encourage even more people to take psychotropic medication. Nobody ever said life had to be happy.

What Is a Mental Disorder?

Epidemiological research measures the frequency of psychological symptoms. But everyone experiences periods of anxiety and depression, as well as periods of moodiness, inattentiveness, or use of substances. This raises the question of whether there is any valid way of separating psychopathology from normality (McNally, 2011; Kagan, 2012).

These issues have been debated for at least 100 years. Kraepelin (1921), who worked in mental hospitals, wanted psychiatry to focus on severely ill patients whose symptoms would not overlap with normality. On the other hand, Freud (1916/1958), who conducted an office practice, took the view that everyone was a little ill, and that differences between patients and non-patients were a matter of degree. Ironically, this concept, which was overthrown by DSM-III and the neo-Kraepelinian movement, is now being revived by the neuroscience model.

If subclinical symptoms of mental disorders are points on a smooth continuum between disorder and normality, mental illness would blend into normal trait variation. This view was shared by the editors of DSM-5 (Regier et al., 2011) and by researchers at

the National Institute of Mental Health (Insel et al., 2010). At one end of the continuum, symptoms would be sufficiently severe that few would contest that an illness is present. At the other end would be people who have only transient distress, and who almost everyone would agree do not have a disorder. The problem lies with the group in the middle, who may have "subclinical" symptoms. Some epidemiologists have argued that subclinical symptoms are, on a statistical basis, risk factors for serious disorders (Kessler et al., 2003). Yet most people with minor symptoms either recover or continue to have subclinical distress. Classifying them as ill could lead to an enormous number of false-positive diagnoses.

To be fair, every academic discipline is ambitious to expand its boundaries. That goes some way toward explaining why some psychiatrists see everyone (particularly celebrities) as disordered. Meanwhile, the boundaries of diagnosis grow broader with time, with each edition of the DSM manual thicker than the previous one. But most disorders, as defined by current criteria, are syndromes (i.e., collections of symptoms), not true diseases. Admittedly, patients come with a wide range of problems that psychiatrists are asked to deal with, so we need to have a way of classifying them. But that does not justify making diagnoses that require the skills of highly trained specialists to help people to deal with the normal vicissitudes of life.

Finally, one might ask whether psychiatric diagnoses are culturally relative (Grinker, 2021). Severe mental disorders such as schizophrenia and bipolar I disorder seem to be universal, and common mental disorders can also be observed in all parts of the world (Kessler et al., 2009). But some conditions are highly cultural—for example, one does not see eating disorders in countries where people are starving.

In summary, mental disorders have a high prevalence because it is impossible to separate psychopathology from life and its vicissitudes. Current diagnoses are provisional formulations that may or may not stand the test of time. As long as diagnosis is based on an imprecise classification system, many research findings in psychiatry will have to be taken with a grain of salt.

Chapter 5

Psychopharmacology Fads

Triumph and Tragedy in Psychopharmacology

The successful treatment of mental illness with drugs began in the 1950s, and has been a landmark in the history of psychiatry. There can be little doubt that antipsychotic drugs are among the most effective drugs in all of medicine. As an undergraduate student in 1958, I had the good fortune to see it happen. I joined a group that spent several weekends visiting a nearby mental hospital with 4,000 beds. The psychiatrists there were just beginning to use antipsychotics. The condition of their patients was rather desperate. Yet a few years later, by the time I entered medical school, effective treatment for psychoses was widely available. Although relapses were still a problem, and full recovery rare, patients no longer spent months or years languishing in hospitals. Medications, even if they do not cure psychotic illnesses, can control them.

Other major breakthroughs in pharmacotherapy followed. Antidepressants (mainly tricyclics) were introduced in the late 1950s, and were effective in patients with severe depression. (This advance also changed electroconvulsive therapy from a standard treatment to a back-up option.) By 1970, lithium had become available for the management and prevention of manic episodes. I saw bipolar patients who had been in hospital up to 25 times recover and return to a normal life. This was an inspiring time to train in psychiatry.

Like most psychiatrists of my generation, I was deeply impressed by the efficacy of drugs, and had no hesitation in prescribing them. Moreover, psychopharmacology was a scientifically based domain, with theories built on neuroscience research, and practice was rooted in randomized clinical trials. Yet decades later, I find myself a critic of this field, at least as currently practiced.

I am puzzled, and sometimes appalled, by the way drugs are now being prescribed, and find myself resisting a tidal wave of overprescription. Although today's agents are not much better than what was available 50 years ago, they are given to almost every patient who comes to a physician with mental symptoms.

To understand these problems better, I wrote a book (Paris, 2010) about what the research literature in psychopharmacology actually shows. I was surprised to discover how large is the gap between what we know and what practitioners do. Many practices that are taken for granted, and supported by government agencies and/or treatment guidelines, turn out to be backed up by only two or three studies (some of which are problematic on close inspection). The power of hope drives the prescription of drugs, or multiple drugs, for almost every problem known to psychiatry.

I have learned that a discipline based on empirical data is not necessarily immune to fads and fallacies. The source of the problem is that practitioners want results.

Psychiatrists and family physicians who treat patients with mental disorders may not always accept the limitations of their tools. With the encouragement of academic physicians who have promoted the routine use of these agents, the hammer has found its nail.

Some of the problems of psychopharmacology derive from our inadequate diagnostic system. Mental illnesses cannot yet be scientifically classified, and we do not quite know what we are treating. Even so, physicians cannot resist using drugs to get rid of symptoms. This leads to faddish treatments that have invaded mainstream psychiatry. Given that most disorders listed in DSM-5 and ICD-11 are really syndromes, it is not surprising that treatment is not necessarily specific to a diagnosis. Psychiatric drugs target symptoms, but the psychopathology behind these clinical features is not well understood. The result is that drugs are being used for problems for which they were not designed.

How Psychopharmacology is Practiced

Today's psychiatry looks much more like a medical specialty than it did when I was in training. Since most practitioners agree that we should apply findings from neuroscience, psychopharmacology has become the dominant force in modern psychiatry. This paradigm shift was driven by the wish of psychiatrists to practice in the same way as other physicians—that is, to make diagnoses and write prescriptions. "Just talking" to patients is now, at least for many, seen as old-fashioned and not that effective. There has been a consistent decline in the use of psychotherapy, and it continues to this day (Tadmon and Olfson, 2022).

There are also practical reasons for this trend. When psychiatrists are paid on a fee-for-service basis, they can make much more money by seeing four patients within an hour instead of just one. Moreover, not all academic centers provide trainees with adequate experience in psychotherapy, or in the management of psychosocial problems.

The American psychiatrist Daniel Carlat published an eloquent book examining the problems associated with the way that biological psychiatry is being practiced (Carlat, 2010). Carlat, who had formerly worked for the pharmaceutical industry, described a dispiriting picture, with most patients seen for 10–15 minutes, and asked about current symptoms, after which their medications could be "adjusted." The DSM manual was used to classify their problems, and the psychiatrist might not know much about the patient's current life situation. This is a travesty of how mental health services should be provided.

Although psychiatrists report that they do talk with their patients (Mojtabai and Olfson, 2010), this may not correspond to any recognizable form of evidence-based psychotherapy. The prescription pad comes out well before time has been taken to find out what is going on in the patient's life. Finally, when symptoms are the focus of every clinical encounter, prescription rates are bound to go up.

The Power of the Placebo

Even the most commonly used drugs are not as consistently effective as many physicians think. It is hard to know whether what we prescribe is actually working. Patients get better, but is that a case of cause and effect? People can improve for other reasons—their life situation may change, or their symptoms may spontaneously remit. Yet psychiatrists,

and their patients, sometimes want to explain everything on the basis of the last prescription. That is why medications may be endlessly "adjusted" to no avail.

We tend not to see the obvious explanation. The elephant in the room is the placebo effect. Physicians are taught about placebos, but do not always understand their power (Benedetti, 2008). By and large, the sickest and most acutely ill patients show the weakest placebo response. That turns out to be the case for depression—placebos have little effect on the most severe cases (Kirsch et al., 2008). In contrast, placebo effects are stronger in chronic illnesses, in conditions with an intermittent course, and in conditions that overlap with normality. Mild to moderate episodes of low mood (from which so many people suffer from time to time) show placebo effects that can be as high as 40%. This is uncomfortably close to the figure of about 50% that we get for the effects of prescribed antidepressant drugs. Also, placebo responses tend to be more temporary than drug effects, leading to the otherwise mysterious scenario in which drugs stop working after a few months. Some of my colleagues tell me that they still prescribe even when drugs act as placebos, but I prefer not to do that.

What concerns me is that industry pressure, supported by overly optimistic opinion leaders in academia, has convinced physicians (and their patients) that finding the right cocktail is only a matter of trial and error. I am concerned that clinicians are irresistibly drawn to seeing every change in mood in the light of the latest prescription. Once again, *post hoc, ergo propter hoc.*

None of these problems mean that antidepressants do not work. They do, especially in severe depression, and they are also effective for panic attacks (Cipriani et al., 2018). But many patients stay on these agents for years despite a lack of any clear benefit, because they fear the results of stopping them.

Moreover, the pharmaceutical industry has been more closely involved with psychiatry than with other specialties, and it promotes the wider use of antidepressants. The reason for this is that almost everybody has periods when they are anxious or depressed. Thus, since major depression is a high-prevalence condition, the market for treatment could be almost unlimited. Tricyclics were never that popular, given their side-effect profile and the high risk of taking a fatal overdose. But they gave way to selective serotonin reuptake inhibitors (SSRIs), which are much better tolerated, rarely involved in fatal overdoses, and seem (thus far) to carry little risk when taken for years. This may be another reason why the diagnosis of major depression has become more common.

I remember beginning to prescribe fluoxetine for depression when it became available in the 1980s. At first, many patients came in feeling so much better, even a week later, that it seemed miraculous. What I forgot was that interviewing depressed patients, while taking a careful history and showing empathy for their distress, can be therapeutic in its own right. Many depressed patients feel better after a thorough consultation without taking any drug at all. That is why the National Institute for Health and Care Excellence (2022) guidelines suggest that in most cases physicians should withhold pharmacotherapy until the patient has been seen more than once.

Another issue concerns how long patients should stay on antidepressants before they stop taking them. Research does support maintenance treatment in relapsing cases (Geddes et al., 2003). But this does not mean that every patient needs long-term maintenance treatment. Another puzzle is that some patients describe a temporary benefit which disappears after a few weeks or months, sometimes called "Prozac poop-out." Some experts (e.g., Fava, 2003) have suggested that this phenomenon reflects a

long-term tolerance to the drug. It seems equally likely that in such cases the initial response was a placebo effect, which could only be expected to decline with time.

Context has a powerful influence on the placebo effect (Benedetti, 2008). A patient who has already read about a drug, or who has (in the USA) been exposed to direct-to-consumer advertising, may have positive expectations even before they consult a physician. If the clinician is enthusiastic about the drug, these effects will be even stronger. But the effects are no guarantee that the benefits will last.

Placebo effects may be particularly common when drugs are new. It has been wryly suggested that physicians should prescribe the latest agents before they stop working.

Problems of Overprescription

The current trend for an almost exclusive reliance on drugs in psychiatry is supposedly based on the application of neuroscience research to clinical practice. This is something of an illusion, but we do have effective drugs, especially if we use them properly.

The most serious mental disorders (psychoses and melancholic depression) unquestionably require medication. However, in the management of common mental disorders (anxiety and depression), drugs are only one option, and research shows that many patients in clinical practice do not respond to them (Rush, 2007). A good deal of data on treatment for depression (often comorbid with anxiety) supports either psychological treatment or a combination of pharmacotherapy and psychotherapy (Cuijpers et al., 2020). Unfortunately, that is not necessarily what happens in practice. Instead, symptoms tend to be treated "aggressively" with multiple pharmacological agents, and little time is spent talking about the context of the patient's life.

Thus practice has swung wildly—from talking without a purpose to not talking at all. Again, this is what Eisenberg (1986) described as a shift from brainlessness to mindlessness. Contemporary psychiatrists are not necessarily interested in conducting formal psychotherapy, and even those who have received training in such interventions may not use these skills. Talking therapy requires time and commitment. In North America, the insurance system is organized in a way that encourages psychiatrists to offer drug treatment only.

The result is that psychiatrists leave talking therapy to clinical psychologists, who are unfortunately not usually well insured. Even where psychotherapy is covered by insurance in principle, psychologists are limited in number. Finally, faddishness continues to affect the practice of psychotherapy. These problems, some of which are rooted in pseudoscience, have been well documented (Lilienfeld et al., 2015; Arkowitz and Lilienfeld, 2017), and will be discussed in Chapter 6.

A deeper concern is that physicians who mainly prescribe may forget how to listen. Psychiatric drugs are effective when used for the right indications, but not when applied to problems for which they lack a clear-cut evidence base. For example, antidepressants are essential for severe depression, but can be unimpressive in mild to moderate cases of depression and anxiety, and are sometimes not much better than placebo (Nelson et al., 2008). This is not to say that patients do not respond, at least to some extent. But the well-known Sequenced Treatment Alternatives to Relieve Depression (STAR*D) study found that less than 50% of patients benefit from a first trial of medication, and that switching to another antidepressant and/or adding an antipsychotic still leaves a significant number of non-responders (Rush, 2007).

A more recent large-scale randomized clinical trial of patients in the US Veterans Affairs system (Mohamed et al., 2017) investigated whether the most popular methods of augmentation increase efficacy. They examined bupropion (a non-SSRI antidepressant) and aripiprazole (an antipsychotic with fewer side effects). The results showed that although both drugs helped some patients, the effects on outcome were modest, and were not necessarily cost-effective. This is not to say that augmenting drugs is never useful— the evidence shows that it can be for some patients. But I object to a practice in which depressed patients are *routinely* given multiple drugs, and stay on them even when these agents do not produce a clear change in symptoms. Finally, I have to take issue with those who claim that combining drugs covers all bases for chemical imbalances, when there is no evidence that such imbalances actually occur in major depression.

One might think that some patients who do not respond well to drugs would be offered psychotherapy. But when patients do not remit with antidepressants, clinicians tend to press on with more prescriptions. It should be noted that augmentation involves prescribing agents originally designed for other symptoms, such as antipsychotics, and that this practice can lead to polypharmacy associated with problematic side effects. Switching to another agent has more evidence behind it (Rush, 2007), but the evidence does not support the value of proceeding after a second trial. Yet I often see patients who have been tried on four or five different agents. These practices remind me of the way psychoanalysts used to recommend more therapy when their standard therapy failed, while stubbornly refusing to consider alternatives to their paradigm. In both cases the result can be an impasse that locks patients into endless treatment.

The Role of the Pharmaceutical Industry

I know quite a few colleagues who always prefer the latest drugs on the market over the most established ones. I take the opposite view, favoring drugs that have been around for many years, and whose therapeutic effects (and side effects) are now well known. Old drugs are often just as good as—if not better than—new ones. They are also much cheaper. But the wish to be up to date influences physicians to use newer, more expensive agents (Bolton et al., 2012).

One would think that inexpensive drugs would be a boon to mental health practice. But in practice the prescription of any drug goes down sharply the day after it becomes generic (Avorn, 2004). This observation demonstrates how strongly practice is influenced by pharmaceutical marketing. Physicians, impressed by the latest option, or curious to try it out, may not want to continue using yesterday's drug. This explains why the most recent antidepressants are more widely prescribed than the ones that clinicians have been using for years.

Drug companies take advantage of these attitudes to "the latest thing," and also try to extend their patents for as long as they can. But even when patents eventually expire, and the drug becomes generic, the pharmaceutical industry has a few tricks up its sleeve (Goldacre, 2013). A favorite is to market an "extended-release" form of the same drug, which can then be sold at the old price. Some physicians, and many patients, are fooled by this ploy. A capsule that releases a dose slowly over the course of the day might be useful in a few clinical situations. Yet, by and large, there is little or no evidence that extended-release formulations make any difference for most psychiatric drugs, almost all of which can be taken once a day with no loss in effectiveness. It is usually better to take

medications at bedtime, when patients are most likely to remember to do so, and when sedative effects become an advantage.

Drug companies spend most of their budgets on programs to persuade physicians to prescribe products that remain under patent. Their multi-billion profits show how effective these strategies are. The simplest way to market a drug is by advertising it. But although glossy ads in medical journals promote half-truths, their scientific limitations have not made them disappear. This is because many journals would have to shut down in the absence of advertising. About 30 years ago, the *Journal of the American Medical Association (JAMA)* learned the hard way about the power of the pharmaceutical industry. After the journal published articles criticizing the information provided in drug ads, several large companies stopped advertising in it altogether, nearly making *JAMA* bankrupt, and forcing the editorial board to resign en masse (Angell, 2000).

It remains to be seen whether journals will experience the same problem as they all go online. Even the best journals still contain advertisements. And we are now seeing enormous growth of "predatory journals" which make money by charging large fees for publication without rigorous peer review. This system avoids dependence on drug companies, but encourages the publication of substandard research.

Industry uses highly effective strategies to influence physicians. The main one is a small army of pharmaceutical representatives, hired for their charm and good looks. Small gifts such as calendars and pens are common, and some of my colleagues accept invitations to expensive dinners associated with "drug talks" at the company's expense. But personal contact is more important. Representatives drop by physicians' offices and encourage them to try the latest drugs. Often, free samples are provided to get things started. That is why I keep seeing indigent patients coming for consultation who have already been prescribed the most recently developed and expensive agents. When the samples run out, they cannot afford to pay for these medications.

Some have claimed that pharmaceutical companies should be *less* strictly regulated, since industry needs to invest in developing new drugs (Goldberg, 2010). But that is not how drug companies spend their profits. Developing a new agent can cost many millions or even billions of dollars, and investments can be completely lost if the drug fails to deliver. The high risk involved in drug development is the reason why the pharmaceutical industry prefers to market the drugs it already has, or to develop variants of existing agents. Although some completely new drugs for cancer have been developed in recent years, in psychiatry the majority of new agents are "copycat" products that closely resemble drugs which have long been on the market. (A recent example is vortioxetine, an SSRI antidepressant that has never been directly compared with the many other agents already on the market, but is being widely prescribed by physicians in my community.)

Psychiatry is in need of better drugs, but does not need "me-too" drugs. There have been no dramatic improvements in the efficacy of pharmaceuticals for mental disorders over the last 50 years, and the main advantage of the newer drugs lies in their less severe side-effect profiles. To a great extent, one can do well for one's patients if one knows how to use the older drugs.

Most of the great advances in psychopharmacology occurred decades ago. Psychiatrists could practice quite effectively using only agents that were available in the 1970s. Atypical antipsychotics are no more effective than typicals (Lewis and Lieberman, 2008). Second-generation antidepressants are no more effective than

tricyclics, and even the newer ones have few advantages in terms of efficacy (Gartlehner et al., 2008; Cipriani et al., 2009). Anticonvulsant mood stabilizers are no better than (and usually inferior to) lithium (Geddes et al., 2010). The main advantage of the newer generation of drugs is a somewhat more favorable side-effect profile.

If psychiatrists could practice perfectly well with a 1975 pharmacopeia, the glory days of psychopharmacology may be behind us. We all know that antidepressants which work faster and more consistently would be a great advance. But nobody knows how to develop them, and industry is not seriously trying to do so.

Another way in which Big Pharma markets new drugs and persuades physicians to adopt them is by paying for the services of prominent academics, often called "key opinion leaders." This does not mean that these experts are in charge of clinical trials, as these studies are run by industry using their own protocols. Instead, companies pay university-based experts large sums of money for giving "drug talks." These lectures are infomercials masquerading as "continuing medical education" (Carlat, 2010). Sometimes just mentioning a new drug can have an impact on practice. Another tactic in industry-sponsored drug talks is not to openly praise the sponsor's product—that would be too obvious—but to say something critical about the sponsor's competitors.

Thus if you are an academic researcher with an excellent reputation, you do not have to make do on your salary, but can earn millions from "consultant fees" and paid speaking tours. Top academics may even be paid to attend "consultations" that take place at luxury resorts. I am ashamed to acknowledge that of all the fields in medicine, it is psychiatrists who take the most money from industry. That could be because psychiatric drugs are used so widely that this specialty is more likely to make profits for industry (Angell, 2004).

Interestingly, hardly any academic who earns a large income from industry thinks that he or she is doing anything wrong. It is easy to fool yourself that you are just providing information, and that your relationship with drug companies is a partnership that benefits patients. I used to think this way, too, and although I was never the kind of "key opinion leader" whom industry would want to seduce, I had to realize that my uncritical attitude was part of the problem and not part of the solution. Carlat (2010) has eloquently described from his own experience how easy it is to be corrupted by industry money. He eventually started a report on the Internet that critically appraises all the latest developments in psychopharmacology.

Antipsychotic Fads

Antipsychotic drugs are highly effective for the purpose for which they were developed—the management of psychotic symptoms. But we need better agents to relieve the negative symptoms of schizophrenia that remain after acute treatment. Sadly, the need for a different class of drugs has not always been seen as a priority.

Antipsychotics are now being used for a wide variety of indications in non-psychotic patients, despite having serious side effects. The prescription of atypical antipsychotics as augmenting agents for major depression without melancholia is now popular. Research shows that this option can be effective in patients with melancholia (Parker, 2005), but is only weakly supported in mild to moderate depression (Nelson et al., 2008). Although the Food and Drug Administration (FDA) in the USA has approved indications for some of these agents, their decisions are based on a minimum requirement. One only needs to

report two positive clinical trials—which, needless to say, have been conducted by the manufacturers.

Of even greater concern, atypical antipsychotics are now being used for some of the indications that were traditionally reserved for benzodiazepines. They are being widely prescribed for the management of anxiety and insomnia (Comer et al., 2011). This practice is not entirely new. When I was in training, there was a heavy advertising campaign in journals for "Etrafon-D," which is a combination of the tricyclic amitriptyline and the typical antipsychotic perphenazine. Today, adding atypical antipsychotics to a cocktail reflects a perception that they are safe. However, several of these agents have many long-term side effects, particularly a "metabolic syndrome" that resembles diabetes.

How did antipsychotic drugs, which were originally developed to treat schizophrenia and mania, became the "new Valium" of clinical practice? Quetiapine is being widely prescribed for anxiety and depression, and since it is sedating, it offers a way to treat insomnia. Aripiprazole, which has fewer side effects, is popular as an adjunct for antidepressants. Typical antipsychotics were not used in this way, as they had more serious side effects, including an irreversible neurological syndrome called tardive dyskinesia. Although atypical antipsychotics do not have as many short-term side effects, they can lead to some serious long-term problems (Maher et al., 2011; Depping et al., 2010). Therefore these agents should only be prescribed for depression when absolutely necessary, and whenever possible we should use agents with lower levels of side effects.

Unfortunately, this is not what happening. Antipsychotics are being commonly offered to a wide range of patients (Mojtabai and Olfson, 2011), and their use for anxiety symptoms is an off-label practice that has not been supported by clinical trials (Comer et al., 2011). By and large, adding an antipsychotic to treatment for patients who fail to respond to antidepressants does not necessarily produce a better outcome (Fullerton et al., 2011), except in melancholic depression (Hilton et al., 2007). Clinicians who prefer augmentation might be advised to consider psychosocial interventions, if and when access to talking therapy is feasible.

Antipsychotics should also be prescribed with caution in dementia, as they have been shown to increase mortality (Lee et al., 2004; Schneider et al., 2006). The problem is that some demented patients are agitated, and even when psychiatrists know the risk involved, they may still feel that they lack a better alternative.

The overprescribing of antipsychotics is a fad that demonstrates the power of industry marketing over clinical science. I have been trained to respect these drugs, which means that I recognize their power but fear them. I am stunned by how few physicians think twice about writing these prescriptions. And high doses can be almost as amazing. It is no longer surprising for me to receive depressed patients for consultations who are on regimes usually reserved for the treatment of psychosis. Of all the psychiatric fads current today, the overprescribing of antipsychotics could be the most harmful. At least psychoanalysis only wasted people's time. Prescribing drugs for every patient whom psychiatrists see may be a threat to public health.

Antidepressant Fads

We have used antidepressants for decades, but tricyclics have been almost entirely replaced by safer agents, particularly SSRIs. Yet, in the last two decades, a controversy

has emerged about the real-world efficacy of these antidepressants. There has been concern about publication bias, whether the pharmaceutical industry has had too much influence on clinical trials, and whether newer drugs only do better because of an expectancy effect. But the larger question is how many depressed patients get better. Consultants constantly see patients who have failed several trials of SSRIs, although it is possible that those who clearly benefited are never sent for consultation.

Some studies, supported by meta-analyses (Kirsch et al., 2008; Fournier et al., 2010), have suggested that antidepressants are not much more effective than placebo for mild to moderate depression, and that initial severity is the strongest predictor of response (Khan et al., 2011). Thus sicker patients respond better than those with mild symptoms, who may be more likely to show a placebo response.

A more recent, larger, and more comprehensive meta-analysis, which made use of unpublished data and applied more advanced statistics, was published in *The Lancet* (Cipriani et al., 2018. It showed that antidepressants are generally superior to placebo. Perhaps we can close the book on claims that antidepressants are nothing but expensive placebos. However, the margin between drug and placebo is not that large (about 50% compared with 40%).

In 2008, findings about strong placebo effects in depression (Kirsch et al., 2008) were discussed in almost every newspaper in the world. They made Irving Kirsch, the psychology professor who conducted the meta-analysis, briefly famous (he was interviewed on the American TV program *60 Minutes*). His data seemed to contradict years of clinical trials, but took into account unpublished negative findings (which researchers supported by drug companies never publish). Unfortunately, Kirsch fell victim to his own unexpected renown, publishing a book which claimed that antidepressant therapy is a "myth" (Kirsch, 2009). But that is not what his own data showed—drugs were clearly better than placebo when depression was severe. More accurately, we should say that even if they do not always work, antidepressants are often worth trying.

However, it does not follow that drugs should be the only treatment for depression, or that patients should be tried on every agent on the market until something works. The evidence actually suggests that psychotherapy leads to more durable effects (Cuijpers et al., 2020). Moreover, antidepressants have more reliable outcomes in anxiety disorders (Gomez et al., 2018). This suggests that these drugs are misnamed, and should not be thought of as specific to a single constellation of symptoms. (Even though the term "neurosis" is no longer used in psychiatry, I would suggest that these agents be called "antineurotics," given the underlying trait of neuroticism in internalizing disorders.)

Although only about 50% of patients who are treated for depression with antidepressants have a full remission, we do not know how to identify them in advance (Thase, 2011). And when SSRIs are prescribed for people who are unhappy rather than clinically depressed, or for patients who have other diagnoses (e.g., personality disorders) that explain low mood, they may not work at all (Paris, 2020b).

Psychiatrists sometimes think that other physicians have more effective treatment methods. They should be less envious. Although effect sizes (which measure change in standard deviation units) are modest in psychiatry, those for most drugs in medical practice are also fairly modest (Seumuller et al., 2012; Leucht et al., 2012). We need not idealize the rest of medicine just because psychiatrists remain in the dark about etiology.

What should clinicians do when drugs do not work? This is an increasingly common scenario as antidepressants are routinely prescribed for less than convincing reasons, or

are re-prescribed for years. (This explains why 11% of Americans are taking them.) It should not be sufficient for patients to merely report unhappiness to be offered a drug.

As recommended in the UK by the National Institute for Health and Care Excellence (2022), for less severe cases of depression, psychiatrists should consider watchful waiting or non-pharmacological evidence-based alternatives, such as psychotherapy. Even if the current climate of opinion focuses on trying a different drug ("switching") or adding another drug ("augmenting"), these procedures do not work consistently. As shown by large-scale effectiveness studies such as STAR*D (Valenstein, 2006), about 50% of depressed patients remain symptomatic after antidepressant therapy. Moreover, the evidence base for augmentation and switching is much weaker than most clinicians think (Rush, 2007). STAR*D showed that some patients who fail to respond to the first drug benefit from a course of a second drug, but after that a law of diminishing returns sets in. Finally, since there is little evidence that any one of these agents is better than any of the others (Cipriani et al., 2009), the choice of drug is largely a matter of guesswork.

I consider the routine use of antidepressants in primary care and specialty clinics to be unfortunate. I often consult on patients who have been tried on four or five antidepressants, sometimes in combination, "augmented" by antipsychotics (and/or mood stabilizers). This is polypharmacy, based on the idea that any patient can be helped if the physician can just find the right cocktail of drugs for them.

One generally unrecognized reason for the uncertain effectiveness of antidepressants derives from the fact that "major depression" is a heterogeneous syndrome (Parker, 2005). Most of the patients who are sent from primary care for consultation will not have classic features of melancholia, and many might be better described as unhappy. (These patients may meet the diagnostic criteria for the less researched category of "persistent depressive disorder.") Life problems do not necessarily respond to drug therapy alone, and their low mood needs to be addressed in other ways. For example, a patient who is chronically unemployed and socially isolated should not be expected to gain dramatic benefits from psychopharmacology. Often, all that can be expected is a better night's sleep, and even that can come at the price of side effects.

Surveys show that psychiatrists prescribe drugs on a very large scale (Mojtabai and Olfson, 2011). One reason for this is that the definition of major depression has undergone concept creep. Horwitz and Wakefield (2007) have called this "the loss of sadness." This diagnostic inflation helps to explain why 11% of all Americans over the age of 12 years are currently taking an antidepressant (Pratt et al., 2011), including almost 25% of all women aged 40–59 years. This pattern of overprescription is rooted in overdiagnosis. It also fails to take into account the evidence that psychotherapy is at least as effective a treatment as antidepressants for most cases that clinicians see. This kind of practice could be one of the greater psychiatric fads of our time.

Yet antidepressants are effective when used for the right indications. The more closely the clinical picture approximates to melancholia, the better these agents work. Some patients with severe depression need augmentation with antipsychotics or lithium (Parker, 2005). In contrast, outcomes are unimpressive in patients who only fall within an overly broad definition of major depression, and antidepressants are much less effective in people who are unhappy about adverse life circumstances.

Surveys have also shown that antidepressants are often prescribed in the absence of a diagnosis of depression (Mojtabai and Olfson, 2011). It is true that they are effective for anxiety symptoms such as panic attacks. But these agents are often given in the context of

a life crisis. Then, when the patient gets better, which might also have happened with no prescription, their improvement is attributed to pharmacotherapy (yet another example of *post hoc, ergo propter hoc*). Patients may be kept on these drugs for years because of fear of a relapse. Some depressed patients are themselves so afraid of relapsing that they report symptoms after missing a single dose.

In my work as a consultant for physicians working in primary care, most of the patients I see are already on antidepressants (unless they refuse to take them). The cases who are sent to a specialist are those who fail to respond, sometimes to multiple agents. That is why about half of my consultations concern what is misleadingly called "treatment resistance" (misleading because one should not speak of resistance when one does not know what one is treating, or whether the treatment offered is the right one).

I understand why antidepressants are overprescribed by busy practitioners. Moreover, since the response is so variable from one patient to another, if there is little else to offer the patient, and if antidepressants have not been tried (or have only been given in subtherapeutic doses), it may be worth prescribing them. But I advise consultees not to be overly aggressive. If more than one agent has already been tried, I do not routinely suggest going to another, since effectiveness studies suggest that a law of diminishing returns kicks in after two tries (Rush et al., 2006). I also rarely recommend pursuing augmentation strategies. In summary, one can keep trying a new drug or a new combination of drugs, but at some point one may have to stop chasing the illusion that one can find the right "cocktail." I advise my consultees to rethink clinical problems and to ask *why* their patients feel sad, and I often suggest referrals to psychotherapy.

The vast majority of depressed patients are experiencing life adversities that give them reasons for being unhappy. For example, on a recent (and not atypical) day, I was asked to consult on medication regimes for a woman whose husband had unexpectedly abandoned her after a long marriage, a woman whose husband was dying of cancer, and a man whose brother had fired him from the family business. As far as I could tell, none of these circumstances had been examined in any detail by the referring physician before multiple drugs were prescribed. Not surprisingly, all of these patients were described as "treatment resistant," despite multiple psychopharmacological interventions and a failure to refer for psychotherapy.

I am not saying that circumstances provide a complete explanation for mood symptoms. Depression, like all diagnoses in psychiatry, has a significant heritable component (Jang and Choi, 2020). By and large, clinical depression is an exaggerated response to stressors that do not consistently cause illness in everyone. Yet, in the past, psychiatrists emphasized psychosocial factors to the point of being "brainless" (Eisenberg, 2004). However, if one treats depression after a major life adversity as a purely medical problem, one should not be surprised when this strategy proves insufficient. Unfortunately, the climate of modern psychiatry leads physicians to ask the wrong questions. Instead of reframing symptoms on a human level, they only want to know whether they need to be prescribing a different drug.

Mood Stabilizer Fads

Patients today may be overdiagnosed with bipolar spectrum disorders (Paris, 2012a). The reason for this is concept creep, in which it is easy to see symptoms as falling within

a spectrum. These conclusions lead to pharmacological treatment that would be useful if the diagnosis had been correct.

Bipolarity requires documentation of either full mania or a clear episode of hypomania. The faddish aspect arises when patients who have mood instability for other reasons are seen as lying within a "bipolar spectrum." This concept inevitably leads to the overprescription of mood stabilizers. If one assumes that mood swings are *pathognomonic* of bipolarity, then anyone who is moody—adult, adolescent, or child—can be diagnosed in this way. Although many more patients are being given anticonvulsant mood stabilizers or lithium, research does not support this expansion of drug treatment (Patten and Paris, 2008).

The main problem lies in the distinction between a mood disorder and a personality disorder. Most patients with personality disorders have unstable mood, and will meet the criteria for major depression at some point (Paris, 2020b). Given the low bar (five symptoms in 2 weeks), we should not be surprised that depression itself is overdiagnosed. But since mood instability tends to be seen by clinicians as bipolarity, many of my patients with borderline personality disorder (BPD) have been diagnosed with bipolar II disorder at some point, and treated accordingly.

Neither the guidelines of the National Institute for Health and Care Excellence (Kendall et al., 2009) nor the Cochrane reports (Lieb et al., 2010) support the use of mood-stabilizing drugs in BPD. These agents are misnamed, because they do not have specific effects on mood stability. A large-scale study in the UK found that lamotrigine was not clinically effective for patients with BPD (Crawford et al., 2018). But if one never diagnoses that disorder, and if one thinks that patients fall within a bipolar spectrum, one will treat them with these drugs. Some physicians seem irresistibly attracted to this fad. Anticonvulsant drugs are also sedatives, so some patients feel a little calmer on these agents, but these effects are in no way specific.

It is important to distinguish between the mood instability that is seen in BPD and that seen in hypomania (Paris, 2012a). Mood shifts in BPD, unlike those in hypomania, are environmentally responsive and rapid, changing by the hour rather than by the week. They are almost always brought on by interpersonal conflict. And these symptoms do not respond to treatment with mood stabilizers.

However, some patients with personality disorders can become attached to their medications, and have been taught to believe that they have a disorder with a specific pharmacological remedy. If they knew that bipolar disorders have a much more serious prognosis than personality disorders, they might feel differently.

Personality disorders are a difficult clinical problem, and I feel a responsibility to advocate on behalf of these patients. They were mistreated when I was young, and they still can be. The difference is that instead of being offered five years of psychoanalysis which they cannot afford, they may be prescribed five drugs which they do not need. That is not much of an improvement.

Stimulant Fads

Chapter 4 discussed the overdiagnosis of attention-deficit hyperactivity disorder (ADHD), which so often leads to unnecessary and ineffective treatment with stimulants. We lack a gold standard for making this diagnosis, and have to rely on uncertain data to support it. DSM-5 requires a childhood onset, but patients may report symptoms in

childhood in the absence of data showing that this was indeed the case. The results of psychological testing procedures are not specific. Crucially, given that most patients these days who are diagnosed with ADHD fit better within the inattentive subtype, we need to keep in mind that a lack of focus can have many other causes.

ADHD is a real syndrome, not just a social construct. Yet it is likely that attention problems were never an issue before children went to school and before most adults had desk jobs. Yet in the same way as many people need eyeglasses for reading, some may need medication to cope with the attentional demands of the modern world.

Although ADHD is sometimes identified using neuropsychological testing, the results are non-specific for this diagnosis. One such measure is the Continuous Performance Test (CPT), a deliberately boring task designed to measure sustained attention, on which patients with ADHD show abnormal results. However, although these tests separate cases from normal controls, they fail to differentiate ADHD from other disruptive behavioral disorders (McKee, 2008).

A "rough-and-ready" measure that is frequently used for identifying ADHD is the Conners' Rating Scales–Revised (CRS-R; Conners et al., 1998). This is a checklist that parents and teachers can fill out, and it describes the clinical features of ADHD. The items differ little from the DSM-5 criteria, so the CRS-R is in no way an independent "gold standard" for diagnosis.

All of these issues are confounded by the fact that stimulants make *everyone* more focused, so that doing better when taking them does not confirm that the diagnosis of ADHD is justified. The question is whether we really know who should take medication and who does not need it. This dilemma concerns children, who are the traditional recipients of stimulant treatment, but it now also concerns adults, for whom prescriptions have rapidly increased (Piper et al., 2018).

Let us start with childhood presentations. A large research literature supports the usefulness of stimulants for children with clear-cut ADHD (Barkley, 2014). However, not every case responds to these drugs. Although other options (d-amphetamine and modafinil) have been tried, as well as non-stimulants (atomoxetine, tricyclic antidepressants, bupropion, and guanfacine), about 30% of children with ADHD are resistant to any drug (Owens et al., 2003; Cumyn et al., 2009). And although longer-acting stimulants have been widely used, there is little evidence that they lead to a more robust response (Faraone and Glatt, 2010).

In the Multimodal Treatment Study of Children with ADHD, a large multi-centered trial in which children were offered methylphenidate with or without multimodal psychosocial interventions, this drug was the most effective choice in the short term, outperforming psychosocial treatment alone (Abikoff et al., 2004). However, a report from this group gives a less clear verdict about the long-term value of stimulant treatment (Swanson et al., 2017).

It follows that there should be more than one option for the treatment of problems with attention. Although most US physicians would immediately choose stimulants, the National Institute for Health and Care Excellence (2018) guidelines take a different view and prefer conservative management (i.e., watchful waiting and increasing environmental structure), relying on medication only when more conservative strategies fail. That procedure might help to separate the more typical cases from the atypical ones who may not have the same condition. We do not know exactly what ADHD is, or what its boundaries are. If too many children and adults are receiving drugs that they do not need, we should not assume that the data support a routine prescription of stimulants.

I have reviewed the problem of overdiagnosis of adult ADHD in Chapter 4. There is no doubt that some children with ADHD continue to have symptoms as adults (Weiss and Hechtman, 1993). However, adults are now receiving stimulants in much larger numbers than in the past (Piper et al., 2018). Most often, physicians prescribe these drugs for people who complain of a lack of focus. And since they can work in people without ADHD, some adults do benefit from them, even if this might be considered a form of "cosmetic psychopharmacology." But if there is another diagnosis that explains inattention, I take patients off stimulants. What we do not know is whether the long-term use of these agents carries a significant medical risk. This is the danger that lurks in pharmacological fads.

The Cult of Medication Adjustment

This book has focused on some of the most problematic aspects of current psychiatric practice—an over-reliance on DSM diagnosis, the use of checkups rather than therapy sessions, and a tendency to carry out medication "adjustments" of doubtful validity. When patients see a physician for a checkup they are asked about their current symptoms. If they are feeling better or about the same, the current prescription will be renewed. But if they do not feel better, the regime is likely to be changed, either by an increased dose or by introducing a new agent. These procedures treat mental disorder as if it were a chemical imbalance that is susceptible to titration. Such procedures make sense in the management of diabetes, but they are not an evidence-based treatment for mental disorders.

When physicians start patients on new medications, they often have to make adjustments. Generally, one increases the dose until a clear response is apparent. If side effects are problematic, the dose may be continued or decreased. In severe cases, one can indeed add a second medication for augmentation. A second drug can also be prescribed to target other symptoms (e.g., insomnia). But once a response is obtained, there are few reasons to change the prescription. Patients with depression often stay on the same treatment for 6 months to a year, after which one can try stopping it. This means that some patients can eventually be taken off drugs, but those who relapse and have further episodes may have to be managed on the same regime for years to come (Hansen et al., 2008). Unfortunately, many physicians are reluctant to stop antidepressants, so they never find out whether maintenance therapy was in fact required. Also, since many patients show only partial responses, frequent changes in medication are common. Some patients actually become attached to the idea of having their medications adjusted, as this makes them feel attended to and cared for.

Medication adjustments are based on the assumption that changes in symptoms are a reflection of the instability of brain chemistry, and that a chemical imbalance can be corrected by changing a drug cocktail. But there are many other reasons why people may feel better or worse at any particular time. Obviously, life circumstances can and do change. But you will never find out if all you do is ask about symptoms.

Second, practitioners are susceptible to the cognitive error *post hoc, ergo propter hoc.* The tendency to explain everything that happens to patients on the basis of the last treatment intervention is not unique to psychiatry, but it manages to confuse both physicians and patients. Clinicians often fail to recognize placebo effects, and relapse may be due to life circumstances rather than to fluctuations in neurotransmitters.

The cult of medication adjustment reflects the theoretical orientation of psychiatry and the training of its practitioners. When leaders in the field focus almost exclusively on neural networks and neurochemistry, one can hardly expect practitioners to see things differently. A new breed of psychiatrist has emerged—a specialist who is knowledgeable about psychopharmacology, but at a loss when asked to talk to people about their lives.

Prescriptions Without End

The 11% of the adult population who are now taking antidepressants reflect a 400% increase over the last two decades (Pratt et al., 2011). Some patients remain on these drugs for life. Although it is well established that depression can be chronic, and that some patients may need maintenance treatment (Geddes et al., 2020), it is much less clear who requires maintenance and who does not. Maintenance therapy is most likely to be needed for patients with recurrent and severe unipolar depression, but it is not always necessary for all patients who have met the criteria for major depression at some point in their lifetime. Fortunately, the long-term side effects of antidepressants appear to be fairly benign. Yet that is just what makes it tempting for physicians and patients to take the path of least resistance and prescribe for the long term. And if non-psychotic patients are on maintenance treatment with antipsychotics, the practice is more worrisome.

Patients may be prescribed multiple drugs as much due to frustration as to an evidence-based therapeutic plan. Polypharmacy, in which patients take four or five drugs for years, develops gradually. When patients do not respond to the initial therapy (an antidepressant or an atypical antipsychotic), they may be given a drug from another major group, and this "augmentation" procedure tends to continue, even when the revised regime does not help much.

Psychiatrists often prescribe drugs "off label," with or without encouragement from pharmaceutical representatives. This is part of a larger trend toward *most* patients being managed with polypharmacy (Mojtabai and Olfson, 2010). One of the more striking features of modern practice is that no matter what the diagnosis may be, patients can receive a cocktail that includes at least one antidepressant, an antipsychotic, a mood stabilizer, and a benzodiazepine.

Notably, polypharmacy at this level has never undergone clinical trials, so is not really scientific. One sometimes hears multiple prescriptions defended on the grounds that mental disorders are complex problems that require complex solutions (Ghaemi, 2002). But in the absence of research, these practices may simply reflect a fad.

The prescription of drugs should be cautious and supported by systematic clinical trials. Moreover, these trials need to be fair, and not "fixed" to produce a predetermined outcome (Healy, 2012). New drugs are regulated by government bodies, and are only supposed to be approved when empirical data are sufficient. But that is the ideal. Drugs are allowed on the market on the basis of two or three studies, which do not provide enough data to justify a meta-analysis (Brooke et al., 2005; Paris, 2010). And in practice, physicians can do much as they please.

In summary, psychopharmacology—which should be evidence based, and often is—can sometimes reflect fashion more than science. This need not happen. A more conservative and more effective practice would involve prescribing drugs more sparingly, with more specific targets and goals.

Chapter 6

Psychotherapy Fads

I was trained at a time when psychotherapy was a central focus for psychiatry in North America. It now plays a much more marginal role in the profession. Most therapy today is offered by other mental health professionals, particularly clinical psychologists. Moreover, not every patient who sees a psychiatrist will be referred for this kind of treatment.

Fifteen years ago, a survey found that although some psychiatrists reported that they carried out psychotherapy (Mojtabai and Olfson, 2008), there had been a steady decline. This trend has continued, with the most recent survey showing that 50% of practitioners do not offer any form of psychological treatment (Tadmon and Olfson, 2022). And what is reported as therapy may only consist of supportive chats, rather than systematic or evidence-based interventions. Ironically, psychiatrists receive more consistent training in psychotherapy than do most clinical psychologists and social workers (Weissman et al., 2006).

Yet the demand for therapy is increasing and greatly exceeds the supply. The problem is lack of availability and affordability. This treatment tends to be poorly insured (as in the USA) and/or limited in access (as in the UK and Canada). Talking therapy requires expensive human resources.

Psychotherapy has a strong evidence base, just as good as most psychopharmacological interventions (Markham et al., 2021). Unfortunately, this treatment domain has suffered from more than its fair share of fads and fallacies. Extreme methods that are not based on evidence have been used in the treatment of adults (Lilienfeld et al., 2015), as well as in that of children and adolescents (Hupp, 2019). Some of these ideas are rooted in pseudoscience, but one can also see problems with current methods that lie closer to the mainstream.

For many decades, psychoanalysis (or psychodynamic psychotherapy) was a favored method in American psychiatry (Hale, 1995). That dominance has now greatly declined. Psychoanalytic methods have little support in research, take up too much of clinicians' time, and are difficult to access due to their high cost. Today, cognitive–behavioral therapy (CBT) is the most favored method, and this is the model used by most clinical psychologists. Yet CBT is still too expensive for many patients.

From the creation of psychotherapy in the late nineteenth century, many professionals have been puzzled as to how "just talking" can help people with mental disorders. Patients may ask the same question. Yet in common mental disorders (depression and anxiety), talking therapies give drugs a good run for their money (Cuijpers et al., 2020). There is also a large body of evidence that supports the value of psychotherapy in more complex conditions, such as personality disorders (Paris, 2020b).

Over four decades ago a landmark meta-analysis concluded that "psychotherapy benefits people of all ages as reliably as school educates them, medicine cures them, or business turns a profit" (Smith et al., 1980, p. 10). But medicine does not always cure, business does not necessarily make a profit, and psychotherapy does not always work. Even so, talking therapies work often enough to be an important option, particularly when pharmacological interventions are not effective.

One of the main problems with the current practice of psychotherapy is that it tends to last much longer than is supported by research. When its goals are unclear, treatment can go on and on, sometimes interminably. To be fair, this only happens when the cost of extended therapy is not a problem, and in fact most courses of therapy in practice tend to be brief (Markham et al., 2021). Yet I was taught to see patients once or twice a week over several years, and I used to practice that way. (I could do so because I work in Canada, where the work of psychiatrists, unlike that of psychologists, is fully insured by the government.) Eventually I realized that this kind of treatment is neither efficacious nor cost-effective. It can continue forever, in endless circles of "cyclotherapy."

In middle age, I took a 2-year sabbatical from the active practice of psychotherapy, and spent time reading the research literature. What I learned was that most of what I had been taught was wrong. Research supports brief and focused therapies that abjure focusing on the past and that concentrate on current problems. These findings are nicely summarized in a standard handbook of research (Markham et al., 2021).

I have long thought that psychiatrists, who have been trained at great expense to become medical specialists, should not be treating the "worried well," but should focus on the sickest patients. Some of these people are suitable for talking therapy, but have pathology that psychologists prefer not to manage.

That is why I have spent most of my career treating chronically suicidal patients. Most of them can be diagnosed with borderline personality disorder (BPD), and this group, however challenging, does best with specialized therapies (Linehan, 1993; Paris, 2020b). As I began to use more targeted methods, I became more satisfied with the results, and returned to practice with renewed enthusiasm. I then went on to open clinics that treat BPD patients in a time-limited frame, using a stepped care model, which our research group has found to be an effective strategy for most patients (Paris, 2017; Laporte et al., 2018). Even patients who need more time (a year or so) do not need to remain in therapy indefinitely.

What can one say about psychoanalytic therapy today? Sigmund Freud can be considered the main founder of most current methods of psychotherapy—even CBT draws on some of his ideas. Moreover, an emphasis on active listening and empathy remains a crucial element of talking therapy. However, Freud was also the source of many of the problems that continue to bedevil the field (Paris, 2005, 2019).

Psychoanalysis had all the features of a fad. It sprang from one person's mind, almost entirely unsupported by evidence, and at first the method spread like wildfire. Today very few patients are willing to lie on a couch four days a week for years. But even once-weekly psychoanalytic therapy has no evidence base if it continues for years. The one format of this approach that is well supported by research is brief psychodynamic therapy (Leichsenring et al., 2004; Abbass et al., 2014), usually offered once a week for a few months.

Since Freud's time, faddish methods and fallacious reasoning have affected all other forms of psychotherapy, each claiming to be something new. Therapists who promote

these methods are keen to define them as unique, and to give them specific names (often a three-letter acronym that is easy to remember). There are now hundreds of brands of psychotherapy.

Yet research shows that most psychotherapies work in much the same way and yield fairly similar results (Wampold, 2001). Psychological treatment is not a specific technical intervention, but a healing relationship. Its most essential elements—attentive and open-ended listening, empathy, and the provision of an explanatory narrative—can be found in virtually every method (Frank and Frank, 1991). These are the "common factors" that research has shown best predict a positive outcome. They do not guarantee a good result, but therapy does not work well when these factors are absent. This does not mean that psychotherapy is little more than "tea and sympathy." Its elements are measurable, and researchers can assess its outcome in reliable and valid ways. In other words, psychotherapy has entered the world of evidence-based practice.

Psychotherapy Research

Psychotherapy needs to be based on research in much the same way as psychopharmacology. It should be rooted in science, not in authority or rhetoric. Clinicians should never accept anecdotes as proof of the value of any method. We should expect talking therapies to be tested systematically for efficacy. Treatment should not be offered to patients unless it is supported by clinical trials. Outcomes can and should be held to a high standard.

Psychotherapy is not a benign procedure. It sometimes does harm, and therefore needs to be regulated in the same way as drugs. But no government agency is ever asked to approve any form of talking therapy, so oversight of what goes on in practice is variable or absent. Up until recently, almost anyone could claim to be a therapist or a "counselor" without being challenged. Even when a Master's or a doctoral degree is required, nobody checks up on the product delivered to patients.

Unfortunately, psychological treatment sometimes consists of little more than sympathetic listening. That is a necessary but not sufficient condition for good therapy. Patients have the right to expect that they are receiving evidence-based interventions.

Moreover, psychotherapy can cost a great deal of money, something that most of the patients whom I see lack. In the UK, a program called "Improving Access to Psychological Therapies" (IAPT; Clark et al., 2018) offers CBT within the National Health Service (NHS). This is a major move forward toward access to evidence-based therapy for common mental disorders. However, treatment for more complex conditions (e.g., personality disorders, eating disorders, substance use disorders) remains limited. And in North America, if one wants psychotherapy, one needs to be financially comfortable, have the support of a wealthy family, or benefit from generous insurance coverage.

Let us have a look at what research tells us about efficacy. The first clinical trials of psychotherapy were conducted about 60 years ago, around the same time as evidence-based practice was being applied to medicine. Within a few years the research literature became voluminous, leading to the publication of a *Handbook of Psychotherapy and Behavior Change* (Bergin and Garfield, 1978), which summarized thousands of studies. This standard text has been regularly updated, and has now reached its seventh edition (Barkham et al., 2021).

What this research shows is that psychotherapy is useful for a wide variety of psychological problems, that talking therapy is equivalent to medication in its effectiveness for common mental disorders, and that its effects are more stable over time than are those of antidepressant drugs (Cuijpers et al., 2020).

The most crucial finding of research is that most psychotherapies produce fairly similar results (Wampold, 2007). These data do not support the existence of the hundreds of methods that are now available. As has been known for decades (Frank and Frank, 1991), patients benefit from all kinds of psychological treatment if they are motivated, work well with the therapist, can talk freely about their feelings, and are ready to explore practical alternatives to deal with their current problems.

The proliferation of so many therapies with different names is a marketing phenomenon that is contrary to scientific data. There is little or no evidence for the superiority of any one approach, or even for a matching of symptoms and methods. This conclusion even applies to therapy for BPD, in which different methods tend to yield similar results (Cristea et al., 2017; Keefe et al., 2021).

This lack of specificity is reminiscent of false claims for the superiority of one antidepressant over another. The failure to find differences between methods in head-to-head comparisons has been called a "dodo bird effect" (Wampold, 2001), with reference to a scene in *Alice in Wonderland* in which the bird informs participants in a race that "all have won and all shall have prizes." If psychotherapy works in much the same way in all effective methods, it follows that therapists should focus more on the most effective common factors, and less on specific interventions.

Does the importance of common factors mean that psychotherapy works, at least in part, as a placebo? Yes and no. Yes, in the sense that any intervention that promotes hope also promotes healing. But no, in the sense that well-structured treatments are more effective than watchful waiting, monitoring, or chatting. There is also good evidence that some therapists are much more effective than others (Wampold, 2007).

What distinguishes formal psychotherapy from low-cost interventions by non-professionals? "Befriending"—that is, the provision of a sympathetic ear by unpaid volunteers—can be effective for mild depression (Mead et al., 2010). But that kind of case is not typical in practice. By and large, the more severe the problem, the more patients need something more specific than support.

As a consultant, I see many people who have tried psychotherapy but failed to benefit from it. That makes me wonder about the quality of the treatment they received. Quite a few describe a course in which the therapist listened sympathetically, reviewing the details of the patient's week, but offered little feedback or specific skills to promote change. This could be a distorted view of what actually happened. But although support may be enough for transient or mild symptoms, it is insufficient for chronic and severe problems. This approach to therapy can create an endless cycle that leads nowhere.

Even in the best hands, psychotherapy can still fail. Outcome depends at least partly on the characteristics of the patient (Barkham et al., 2021). By and large, patients with better functioning at baseline get more out of therapy. This could be seen as a kind of "Matthew effect," in which the rich get richer and the poor get poorer. However, given that therapy depends more on content than on brand name, skill also plays a role in outcome.

Most therapies can be ended within a few months. But when the treatment does not work, it needs a rethink. All too often, therapists try to do too much, which leads them to

continue treatment for too long. This can lead to endless therapy—assuming that the patient or the insurer can afford it. Moreover, there is no research evidence that long-term therapy is efficacious. Some researchers have claimed evidential support for the efficacy of open-ended psychoanalytic therapy (Leichsenring and Rabung, 2008). However, the evidence supporting these conclusions is weak. Their meta-analyses were based on studies with small samples and small effect sizes, describing treatment for a heterogeneous range of problems.

Keep in mind that research does not support the use of *any* therapy for more than a year. Notably, almost all of the research supporting psychological treatment has examined short-term interventions lasting a few months. The mean length of effective therapy is about 20 sessions (Norcross and Goldfried, 2005), or around 6 months. Having a time limit tends to mobilize both the patient and the therapist, so one should plan for that length of treatment for most patients. (The door can still be left open for future courses of treatment.) The problem of interminability is not specific to psychoanalysis—it can affect therapy of every theoretical persuasion.

It is true that longer therapies have not been well researched, mainly due to lack of funding. That may be the case, but we cannot practice on the basis of what *might* be found in clinical trials. Even if some patients require more time, few studies have followed patients for that long. In the absence of solid evidence to support open-ended practice, therapy can become little more than an extended form of befriending.

For common mental disorders (anxiety and depression), most patients can be offered a choice between medication and a brief course of therapy. Some will prefer to talk, whereas others will only want to take a pill. Some patients may choose both options. Yet, in practice, patients are not always offered this choice. Some physicians have their prescription pads open from the early stage of an interview.

Even if therapy is brief, it cannot be guaranteed that sufficient human resources will be available for the large population of patients who are seeking treatment, and who can benefit from psychotherapy. Moreover, psychiatrists need to work in collaboration with psychologists and other mental professionals to make access a realizable goal. This could be one reason why working in a multidisciplinary clinic is superior to a solo office practice.

The best answer to the access problem may lie in a principle called *stepped care* (Bower and Gilbody, 2005). This concept involves assigning patients with more easily treatable symptoms to primary care, offering secondary care through consultation and brief interventions, and reserving more extended tertiary care for severe disorders and complex clinical problems. Common mental disorders do not necessarily require medical management, although patients can benefit from a psychiatric opinion. But some consultant psychiatrists are only interested in suggesting drugs that have not yet been tried. If they would take a broader view of what works for most patients, they would make appropriate referrals for psychotherapy to other mental health professionals, rather than offering additional medications based on misleading pharmacological algorithms.

Evidence-Based Psychotherapies

All psychotherapies are more or less equal, but some are more equal than others. We need to separate methods that are evidence based from those that are not.

There is still a place in practice for brief psychodynamic therapies, which have a comparable evidence base to CBT. The latter still has the strongest evidence base of any

form of psychotherapy (Beck, 2008), and was specifically designed to last for a few months. Thus CBT is the best known (and most requested) method. Even so, it cannot claim to be uniquely effective. Decades ago, head-to-head comparisons with other methods failed to show any superiority in depression (Elkin et al., 1989), and that verdict has not changed with further research. The same can be said about any of the derivatives of CBT that have been used for more severe disorders. Thus dialectical behavior therapy for BPD is not necessarily better than other well-structured treatments (McMain et al., 2009). Where CBT therapists have been most creative is in developing toolkits for almost every symptom seen in psychiatric practice.

CBT capitalizes on the common factors that make talking therapy effective. This is probably why it is usually better than placebo or "treatment as usual" (i.e., the unstructured and messy reality of clinical practice). But it is similar to other evidence-based methods, such as interpersonal therapy (IPT; Klerman and Weissman, 1993) and acceptance and commitment therapy (ACT; Hayes et al., 2006), and these variants yield similar results. Severe personality disorders may need methods that are even more specific (Linehan, 1993; Paris, 2020b). It is because pharmacological interventions are not very effective for BPD that I remain active as a psychotherapist.

Cognitive interventions can also add to the pharmacological management of the severe mental disorders that psychiatrists focus on. CBT therapists have developed interventions to reduce the effects of delusions in schizophrenia (Turkington et al., 2006), and similar methods have been applied to patients with bipolar disorder (Jones, 2004). If a unified and evidence-based form of psychotherapy emerges in the future, it will probably look a lot like CBT. In the meantime, rest assured that psychotherapy is evidence based, efficacious, and effective. Integrative psychotherapy would probably be as effective as brand-name alternatives (Norcross and Goldfried, 2005), but needs to be made more accessible.

The Proliferation of Psychotherapies and the Prospect of Integration

Among the hundreds of "brands" of psychotherapy that now exist, only a few have undergone systematic clinical testing. The existence of so many options suggests that the history of the field could be a story of one fad after another. Why can therapists not agree on one overarching method and stick to it?

The reason is that psychotherapy is a crowded and competitive market. There is real money to be made by developing a new approach with a new name. That is why every brand is presented as unique, even though almost all of them are variants on a few basic themes. For marketing purposes, three-letter acronyms are memorable, and some give an impression of scientific support for their efficacy. (Some wags have suggested that having four or five letters could yield even better results.)

The proliferation of therapies is also a reflection of the way that psychological treatments are marketed to mental health professionals. Marketing works because psychotherapists are poorly trained in science (Dawes, 1994). Freud, who was indifferent to research, established a tradition that encouraged speculation based on clinical observations, not on data. Anecdotes were treated as conclusive evidence. (As one witticism points out, the plural of anecdote is not data.) Most therapists who followed the age of

psychoanalysis did much the same thing, writing books that were based on clinical experience, not on clinical trials.

Practitioners of psychotherapy may not read the scientific literature on their craft or pay much attention to what the data show. Many clinicians have a doctoral degree, but few make much use of their research training. This is particularly likely for therapists working outside of academic centers (or the NHS in the UK). Unlike physicians who prescribe drugs, psychologists are not visited in their offices by industry representatives who encourage them to adopt a particular brand of psychotherapy. Clinicians can read about a new method, or hear about it, and may want to try it out. The doubt that characterizes the scientific method does not fit well with the enthusiasm that tends to drive clinical practice.

The "guru" tradition in psychotherapy means that its ideas tend to be associated more with a person than with data. Therapists with an idea can create a niche through articles, books, and workshops, and make a financial profit. I know of some experts who spend much time traveling, and who expect a first-class ticket, a hefty fee, and a book-signing opportunity. Like key opinion leaders in psychopharmacology, they fall victim to the appeal of money and fame. Moreover, the popularity of psychotherapy gurus, along with apostles and acolytes to spread their ideas, has affected every single method, including CBT. Although CBT is the therapy most committed to clinical trials, it does not always work, which can lead some of its practitioners to go on seeing patients for years, just like psychoanalysts.

Psychotherapy is a complex procedure that no one can fully master. A gap also remains between what evidence-based treatments can do and what patients need—many problems have never been addressed by empirical research. But practitioners who belong to a psychotherapy culture are looking for answers, not questions.

The best hope for the field of psychotherapy is to give up on proliferation and promote integration. Eventually there should be only one form of psychotherapy, which makes use of the best ideas from all sources. The movement for "psychotherapy integration" (Norcross and Goldfried, 2005) has been around for a long time, but in a competitive environment it has not gained sufficient traction.

Psychotherapy Fads: Benign and Malignant

Psychotherapy fads can be benign, especially when they are little more than variants of already available and evidence-based methods. Or they can be malignant, introducing procedures that have not been tested and that can cause harm.

Let us consider an example. Eye movement desensitization and reprocessing (EMDR; Shapiro, 1995) is a benign fad, in that it is as good as (but no better than) existing methods for the treatment of PTSD (Seidler and Wagner, 2006). An American psychologist, Francine Shapiro (Shapiro, 1995), developed the method and skillfully marketed it. (As discussed in Chapter 3, EMDR has also been associated with a diagnostic fad, namely the overdiagnosis of PTSD, creating a demand for more treatment methods.)

It has long been known that cognitive reprocessing of traumatic events helps patients with PTSD (Bisson et al., 2007). In EMDR, patients are asked to make eye movements while remembering traumatic experiences. This is one of several methods based on the theory that trauma is predictably pathogenic. EMDR has gained support from clinical

trials, and is superior to placebo or waiting-list comparisons, but does not yield better results than other well-structured treatments (Chen et al., 2015). In a session that I watched, the therapist waved a wand back and forth in front of the patient. Yet the "magic wand" that EMDR therapists use to guide eye movements may be no more specific than the methods that were used centuries ago by Mesmer to induce hypnosis (McNally, 1999). There is reason to believe that eye movements are a necessary element of EMDR (Seidler and Wagner, 2006).

EMDR has become a business, producing a product with brand recognition. It helps some people, but its wide adoption is a fad. This story is typical of psychotherapy. Standard methods work often enough. There is no need to get excited about the latest therapy or the most recent acronym.

Malignant fads are different. These methods do not draw on common factors but depend on drama—patients screaming with rage, or past events emerging into consciousness as in a movie. The best example is the fad for recovered memories, associated with a tendency to attribute almost all forms of psychopathology to childhood trauma.

Recovered Memories: A Malignant Psychotherapy Fad

The recovery of "repressed" memories as a goal of therapy dates back to Freud, but this idea became more prominent in the 1990s, and for a time there was a real vogue for it (McHugh, 2008). Building on Freud's idea that adult problems are rooted in childhood experiences, and that memories of these events can be repressed. the movement proposed to exorcise these devils. Therapists often used hypnosis, a highly suggestive technique that can convince people that false memories are true. Although such methods are rarely used now, they have not entirely died out, and are a classic example of the danger of fads.

The theory of recovered memories has been promoted through books written by two psychiatrists working in Boston, namely Judith Herman and Bessel van der Kolk (Herman, 1992; van der Kolk, 2016). Herman's book has been cited almost 25,000 times, and van der Kolk's book has been at (or near) number one on the *New York Times* paperback bestseller list for several years.

Clearly, there is something attractive about the idea that childhood trauma consistently affects people for the rest of their life, and that some of the mysteries of mental illness can be explained as remnants of a forgotten past. This scenario has been the basis of a good deal of fiction and cinema for decades.

The trauma narrative is also a return to the early work of Sigmund Freud. Thus Herman (1992, p. 5) claimed that "the ordinary response to atrocities is to banish them from consciousness." But this view is entirely mistaken, and not in any way supported by evidence. In fact the very opposite is the case—people who suffer from PTSD cannot stop remembering what happened to them (McNally, 2003). PTSD is not characterized by amnesia, but by "hypermnesia" (excessive memory), in which painful memories return to consciousness as "flashbacks," even when people desperately want to forget them.

Another influential concept developed by Herman (1992) is the diagnosis of "complex PTSD" (CPTSD), a disorder that purports to describe outcomes of multiple traumatic events with cumulative effects on personality development. It is true that multiple adversities are worse than single ones. Even so, the CPTSD construct is vague, and can be used to describe almost any kind of unhappy childhood (Paris, 2023).

Nonetheless, it has been adopted as a new diagnosis in the eleventh edition of the International Classification of Diseases (ICD-11; World Health Organization, 2018). CPTSD is not listed in DSM-5-TR, which only recognizes PTSD as a separate category.

The construct of CPTSD expands the concept of trauma, which is already much too broad (Paris, 2022, 2023). It basically describes the symptoms of BPD and attributes them to childhood trauma. Once again, researchers who pursue specific domains of psychopathology have a tendency to see them everywhere and expand their definitions, reflecting a process of "concept creep" (Haslam, 2016).

The effects of childhood experience on development have been a subject of my own research, mainly on BPD (Paris, 2020a). Trauma in childhood is a risk factor for many forms of adult psychopathology (Paris, 2022). But it needs to be defined more precisely, and to be seen in the wider context of other adversities. These include not only dramatic events, such as childhood sexual abuse, but also more subtle factors, particularly emotional neglect and invalidation. In fact, emotional neglect is the most consistent childhood adversity that one finds in BPD (Porter et al., 2020). The biosocial theory of Linehan (1993) describes a temperamental variant (emotion dysregulation) that is exaggerated by the absence of validation from significant others. This is one of the most important and clinically relevant theories for the emergence of severe personality disorders (Paris, 2020b).

Neglect is subtle and undramatic, in marked contrast to the drama of trauma. My impression is that clinicians who consider themselves "traumatologists" see everything negative in life as traumatic or "abusive," and this makes them lose sight of the big picture. An excessive focus on one issue out of many is characteristic of fads.

Herman proposed that even when patients do not remember having been subjected to child abuse, if they have symptoms that could be accounted for by such experiences, memories of these events must have been repressed. This led to the strategy of attempting to recover and process such memories, which ended up being the basis of a very dangerous fad.

Therapies based on a theory of recovered memories are based on Freud, but have taken a new form in our own times. The concept of repressed and recovered memories of abuse or trauma was popularized in a book, *The Courage to Heal*, written by two teachers with no mental health training, which went on to sell over a million copies (Bass and Davis, 1988). These authors proposed that not remembering trauma is actually *proof* that memories of such events must have been repressed. But the assumption that data which run contrary to theory actually prove the opposite is a feature of conspiracy theories. That scenario was originally observed by Festinger et al. (1956) in his study of a failed cult.

Research on memory shows that it is wrong to think that all life events are fully recorded in the brain. The idea that the mind works like a video recorder is also mistaken. In fact we normally forget most things that happen to us, and memories of the past can change with time, being revised each time we access them (Schacter, 1996). Thus memories of childhood are almost never completely accurate, but are reconstructions that reflect what has happened since (McNally, 2003). Moreover, there is no good evidence that abused children repress memories of traumatic events. They may protect themselves by putting those events out of mind or by not seeing them as traumatic, but they almost always remember what happened. That is a key feature of PTSD.

Memory is a complex system, with separate mechanisms for names, behavior, recent life events, and childhood experiences (Schacter, 1996). Events associated with strong

emotion are not remembered more accurately, and research on eyewitness testimony (which is surprisingly inaccurate) have had an important influence on the legal system. The use of hypnosis as a means of recovering memories only makes recall less accurate (Lilienfeld et al., 2015). In fact, memories obtained by hypnosis are much more likely to be false. Even without hypnotic methods, it is not difficult to implant highly detailed, but false, memories of past events that are later regarded as true (Loftus and Davis, 2006).

Thus no objective method, short of corroborating data, can determine whether a memory is true or false. And it is surprisingly easy to create memories that are entirely false but which are reported with enormous conviction. Moreover, false memories are common (Loftus and Davis, 2006). So why did the idea that patients can recover memories gain popularity among therapists, patients, and the educated public? It was largely because remembering a traumatic experience—a phenomenon dear to the heart of cinema and fiction—is dramatic. Moreover, some of these methods can create "multiple personalities," a dubious theory which claims that trauma can shatter the self into a number of pieces. The consequences of these ideas about recovering memories also extended beyond the consulting room, as some people began to imagine that day-care centers were hotbeds of child abuse. This is not to deny that childhood trauma is important, or that memories of experiences of such trauma are not necessarily false. Yet childhood trauma is almost always remembered by people who have suffered from it. (Intrusive memories are a key clinical feature of PTSD.)

The recovered memory movement led to a faddish type of therapy that sometimes broke up families when claims about repressed trauma involved incest, and it often led to lengthy and pointless procedures (McHugh, 2008). In cases where children were prompted to accuse day workers of abuse, innocent people were sent to prison.

The effects on clinical work were limited by the fact that only people with certain personality types would agree to take part in this kind of treatment. Most patients would walk out on therapists who rapidly come to the conclusion, usually based on symptoms alone, that they are victims of forgotten child abuse. Memories that are "uncovered" in therapy are almost certain to be factually untrue. But for those who badly need a narrative, false memories can become their truth.

Recovered memories have sometimes been associated with a feminist perspective, which tends to focus on how women are abused by men. (Although that is often the case, it does not explain much about psychopathology.) For some, the idea that there was a perpetrator seems to be validating. It creates a narrative that becomes part of their identity. But we can support the political and social goals of feminism without believing in fads about memory.

One of the triumphs of feminism is that most women today have their own career. Yet with families commonly having two working parents, or a single parent, we live in a cultural context in which people worry about what can happen to children if they fall into the wrong hands. As a result, parents are more protective of their children than in the past (Lukianoff and Haidt, 2019). Nonetheless, child maltreatment of all kinds has decreased dramatically over the last few decades, as parents and other guardians have become aware of the problem (Finkelhor et al., 2005).

The diagnosis of "dissociative identity disorder" (DID), a condition purported to have been caused by childhood trauma, was another consequence of the recovered memory movement (Paris, 2012b). A few rare cases that fitted this description (previously known as multiple personality disorder) had been described over the last 100 years.

However, more recent research has shown that memories of childhood trauma tend to appear after being strongly suggested by clinicians (McHugh, 2008). Some patients tend to strive to please their therapists, and may wish to be unusual and fascinating. It is doubtful whether anyone ever had these experiences prior to seeing a therapist or reading about them.

Cases of DID remained rare for many decades. This was the case even after the publication of a best-selling book (later turned into a movie), *The Three Faces of Eve* (Thigpen and Cleckley, 1992), which claimed to describe such a case. The story of the most famous of all these patients, Shirley Mason, was told in another best-selling book, *Sybil* (Schreiber, 1973). The author described how Mason, who is called "Sybil" in the book, suffered terrible child abuse that made her separate off parts of herself as "alters."

In the end, the Sybil story turned out to be a fabrication (Nathan, 2011). Careful investigation revealed that Mason had a relatively normal childhood. It seems likely that she agreed to play along with the idea of multiple personalities to please her psychiatrist, Cornelia Wilbur. And when she was seeing a substitute psychiatrist, Herbert Spiegel, Mason did not discuss her problems in that way. It should also be pointed out that Mason and Wilbur developed a personal relationship, even taking holidays together, and both benefited financially from the publication of Schreiber's book.

Unfortunately, DID has been listed in the DSM manuals for over 40 years. This happened in part because a Stanford University psychiatrist, David Spiegel, was assigned the portfolio. Spiegel is a long-time advocate for DID, and has chaired committees on this diagnosis for several editions of the manual. The continued presence of DID in the DSM manual provides unjustified validation for true believers. The presence of dissociative disorders in a separate section of a standard manual also means that every textbook has to include a chapter on them. It takes a certain amount of grit to resist the appeal of the bizarre and the fantastic. (This was exactly what Kraepelin had to say about Freud.)

By and large, DID is only diagnosed by members of the fringe group that supports doing so, and dissociative disorders are very rarely seen in clinical practice or in outpatient clinics (Paris, 2012b). Unlike other mental disorders, they almost always appear in people who are in therapy with clinicians who believe in the concept and persuade patients to support that view. The best one can hope for is that these clinical symptoms, and the mistaken ideas behind them, will disappear once their advocates are no longer around.

The recovered memory "movement" will go down in the history of clinical psychology and psychiatry as one of their greatest scandals, creating a cult that sometimes threatened to bring the practice of psychotherapy into disrepute. Even so, trauma remains a highly emotional issue. We live in a society that is deeply concerned about the protection of children, and about the potential for harm to be inflicted on the innocent. The worldwide scandal about child abuse in the Catholic Church is a prominent example. Ironically, we now know much more about child maltreatment and its relationship to long-term sequelae (Paris, 2022). But research should help us to think about these risks in a broader context, which goes beyond the simple idea of trauma.

In conclusion, dissociative disorders are rare—so rare that they may not even exist. Some of the symptoms are real enough. Depersonalization and derealization are common symptoms in anxiety disorders, mood disorders, and personality disorders, and in a few cases can present as a separate syndrome (Simeon and Abugel, 2006). But the concept of DID reflects an interaction between enthusiastic therapists who promoted

dissociation, and patients who enjoyed the attention that their condition brought them (Piper and Merskey, 2004).

The fad for dissociative disorders did great damage to patients and families, led to expensive and arduous treatment methods (years of hospital-based psychotherapy), and was often associated with false memories of child abuse. The diagnostic fad reached a peak in the 1990s, but has subsequently declined. Yet it lasted long enough to do real damage. In a social climate where parents worry about the quality of care for their children, it became dangerous to work in a day-care center, and a few employees went to prison on false charges. The fad was most prominent in the USA, and although it had less impact in the UK, treatment guidelines for these (fictional) disorders have been considered by the National Institute for Health and Care Excellence.

When psychotherapy turns into a belief system that resembles a religious cult, it has the potential to be malignant. Fringe movements and cults have borrowed some of their ideas from psychoanalysis. Scientology, founded by the science fiction writer L. Ron Hubbard, began as a quasi-therapeutic method called "dianetics" that claimed to recover memories from intrauterine life (Gardner, 1957), and later turned into a formal religion that was a dangerous cult (Kent, 2001). Although science was never any part of Scientology, its ideas emerged within a culture of armchair speculation, and of therapies in which almost anything is possible and anything goes.

The Self-Esteem Fad

Let us consider one more psychotherapy fad based on a bad theory. Psychotherapy has long been susceptible to fuzzy concepts and buzzwords. In recent years, one of the most pervasive—albeit empty—fads has been the idea that therapy should promote "self-esteem."

People like to feel good about themselves, but they have to do something to deserve that feeling. The cult of self-esteem is a quick-fix fad, promoted by media personalities and infecting the practice of psychotherapy (Furedi, 2003). I cannot count the number of patients who attribute their troubles to a deficit of this elusive faculty.

What the idea fails to take into account is that self-esteem is a result of achievement—you should value yourself if you have contributed something to the world. If you are alone and just collect a welfare check, you are perfectly right to have low self-esteem. People who have inflated self-esteem are rightly described as narcissistic. Research also suggests that criminals have unusually high levels of this trait (Baumeister et al., 1996).

Twenge (2006) describes the cult of self-esteem as a cultural fad derived from psychotherapy that has come to broadly influence parenting styles. The belief that having a good opinion of oneself is a necessary condition for success in life gets things backwards. The rest of us are, if not too narcissistic, more self-critical. This is a good thing in small doses. Children need tactful criticism as much as they need praise. Therapists who promote unscientific ideas about self-esteem are doing a disservice to their clients.

In the modern age, people value individuality. If they seek therapy, they may be attracted to self-exploration, promulgated as a positive value. We live in a culture where people can actually be proud to be "in therapy," proclaiming the value of searching for meaning within their own psyche. Unfortunately, the original purpose of psychotherapy, which was to relieve symptoms, can be lost. Some patients become therapy addicts,

spending many hours looking for answers within their psyche, instead of within the world they live in.

Perhaps one need not complain too vigorously, but psychotherapy has come dangerously close to becoming a secular religion, and patients may embark on it as a spiritual journey (Epstein, 2006). As long as no public money supports such ventures, taxpayers are not being cheated. Yet there is still a price to be paid. Some talented psychiatrists have turned away from the care of severely mentally ill patients, for which they have been given unique and costly training by society. One can sympathize with a wish to focus one's career on the treatment of people whose problems are milder and more tractable. But that does not make doing so an ethical choice.

The good news is that psychiatrists now spend most of their time doing what they are trained for—caring for patients with severe mental illness. The bad news is that the division of the mental health professions into biological and psychological camps undermines the prescription of psychotherapy. And among those therapists who do offer talking therapy, the absence of an integrative perspective can discourage them from taking science into account. The continuing divisions within the mental health professions weaken their influence and fail to give psychotherapy its due.

Chapter 7

Prevention Fads

Is Primary Prevention Possible?

Vaccines have saved more lives than antibiotics. In view of this, it has been suggested that psychiatrists should redirect more of their energies to prevention, so as to nip pathology in the bud (Bhui and Dinos, 2011). If only that were possible!

How do you prevent something that you do not understand? I have emphasized throughout this book that the causes of severe mental disorders remain largely obscure. The biological factors in psychopathology are very important, but do not in any way correspond to the diagnostic categories that psychiatrists use. Moreover, although psychosocial risk factors are also important, they do not reliably predict psychopathological outcomes, and they also lack specific relationships to categories of disorder.

A hope for prevention has recently been raised by genomics. Insel (2009), among others, has suggested that whole-genome scans could be used to identify vulnerability markers that could support a "personalized" psychiatry. However, almost 20 years after the introduction of these methods, there is no evidence whatsoever to support use of the genome to guide treatment.

This is part of a long trend in psychiatry toward harnessing the latest technological advances. One example is repetitive transcranial magnetic stimulation (rTMS) for treatment-resistant depression. Yet, after 20 years of research, the precise indications for this treatment remain uncertain (Mahli et al., 2021). It has also been suggested that machine learning and smart wearable devices could be used to overcome barriers to recovery from mental disorders (Perna et al., 2018). But that is another area in which judgment must be withheld until more convincing data become available.

The reader will have noticed that these models of prevention are rooted in biology, and are not biopsychosocial. But the same problem arises with proposals to prevent mental illness by changing the environment. These ideas date back 60 years to the heyday of the community psychiatry movement. The idea was to prevent exposure to psychological adversities, largely through education, and/or to use crisis intervention to bring about the early resolution of problems. But is this possible? Life is full of negative events of all kinds. Even if you think that families are at least partly to blame for some forms of mental disorder, you cannot regulate parenting. Some clinical trials have seemed to support the use of parental education to reduce the incidence of behavioral disorders in children (Hutchings et al., 2007). Yet it would be premature to generalize from these samples to broader populations.

Fifty years ago, when psychiatrists *thought* they knew the answers to these questions, prevention programs were focused on early interventions in families (Caplan, 1964). But these proposals were never implemented. Applying them would have been very

expensive, and such programs should not be funded without strong evidence that they would work. Mental health professionals have played a useful role in promoting public education. But they probably do not know much more than the average person about how to raise children. Despite all our efforts, rates of mental illness today are as high as or higher than they were in the past (Kessler et al., 2005b; Patel and Prince, 2010).

When I was studying psychiatry in the 1960s, one influential idea was that society is to blame for mental illness. This view is still popular in some quarters, and accords with a Marxist political perspective, in which capitalism is seen to be the cause of all the ills of society. It supports more radical ideas about developing social interventions that could reduce the prevalence of mental disorders (Paykel and Jenkins, 1994).

There is indeed evidence that social stressors (e.g., poverty, exclusion) are risk factors for many forms of psychopathology (Scheid and Brown, 2010; U'Ren, 2011). These findings are particularly important for global mental health (Patel and Prince, 2010). But clinicians are not politicians, and we do not really know how to make society better. Psychiatrists are hardly the best people to make radical changes in the way that most of us live.

Social psychiatry has been noted for the passion of its advocates. But the field runs the risk of crossing from medicine to politics, and has not always been committed to a cautious, evidence-based perspective. Research in transcultural and social psychiatry has shown that social disadvantage makes psychopathology more likely (Bhugra and Bhui, 2018). But is not clear how such findings can be used to support interventions. Psychiatrists have their hands full looking after people with severe mental illness. They might be advised to stay away from pontification and avoid prescribing for society as a whole.

Even so, social activism is far from dead. Hopes for utopian change always tend to be the province of the young, and are particularly prominent among university students. Moreover, most academics, especially in the humanities and social sciences, hold left-wing political views (Haidt, 2012), and the same may be true of mental health professionals. Right-wing students are more likely to go into business.

To put social psychiatry into perspective, social risks have to be considered—in the same way as genes or child maltreatment—as one of many interacting risk factors within a biopsychosocial model. Unfortunately, social activists tend to see patients as victims responding to oppression and society's intolerance of difference. Those who believe this can be dismissive of the vast amount of evidence for genetic risks for mental illness. Such ideas form the basis of a still active antipsychiatry movement (Whitaker, 2001).

Some mental health professionals share this "critical" perspective, and it has been promoted in the UK by academics such as Joanna Moncrieff (Moncrieff, 2008) and Richard Bentall (Bentall, 2010). I agree with many of Moncrieff and Bentall's critiques of the limitations of biological treatments. But they are much too one-sided in their criticism, and they fail to understand the implications of a biopsychosocial model. Although a psychiatry based entirely on neuroscience is overly reductive, the same can also be true of social psychiatry.

In the end, there is no evidence that making everyone wealthier or more comfortable would reduce the prevalence of severe mental illness. Some people who are living in poverty still find meaning in life and even describe themselves as happy. Mental disorders affect rich and poor, and are part and parcel of the human condition. Psychopathology can be traced back to every period of history, and is found in every society (Shorter, 1997).

For all of these reasons, the idea that future physicians will eventually be able to predict risk for psychopathology in individuals remains a pious hope. Much as in the discipline of history, the pathways to mental disorder are highly contingent. At this point in time, most forms of primary prevention are not evidence based. To prevent mental illness before it starts, we need to understand its etiology. Therefore we shall have to be patient.

Secondary Prevention

Secondary prevention describes a good deal of what psychiatrists do. It refers to early identification of illness, particularly in childhood or adolescence, allowing interventions to prevent its progress. I shall now examine the extent to which research supports such a program for prevention.

One example concerns the early roots of antisocial behavior. These patterns tend to begin in childhood, and if apparent prior to the age of 18, the clinical picture is called conduct disorder. In fact, as documented by Moffitt et al. (2001), there are two patterns of antisocial behavior. The first pattern starts as early as the preschool years, and is more likely to continue into adulthood, whereas the second pattern has adolescent onset, and tends to remit by the end of that period. Both conduct disorder and adult antisocial behavior are among the clinical phenomena found in behavioral genetic studies to be heritable. However, unlike other diagnoses, they are partly shaped by shared environment, suggesting that dysfunctional families may play a role, but they also have a component derived from non-shared environment, pointing to a role for the larger social environment (Black and Kolla, 2022).

To prevent antisocial behaviors, we need to identify their early precursors. The Canadian psychologist Richard Tremblay devoted his career to this problem. His research team produced some of the largest and most important longitudinal studies of children at risk followed into adulthood, and I was fortunate enough to collaborate with him on a few of them. Yet at the end of the day, Tremblay (2010) concluded that antisocial behavior cannot, on the basis of current knowledge, be prevented consistently. Existing programs have limited results and may not be widely applicable.

This problem has been nicely summarized by Glenn and McCauley (2019, p. 103):

> Youth who develop antisocial behavior at early ages are at greater risk for criminal behavior in adulthood than those with later occurring antisocial behavior. Several interventions have been developed to prevent the development of behavior problems in youth who are showing early signs of antisocial behavior. However, these programs are often complex and expensive and the average effects are often modest. Modest effects may be due to the fact that the programs are not uniformly effective—they do not work equally well for all children This produces several challenges for interventionists. First, it is critical to determine which youth are most in need of interventions. For example, in the absence of intervention, some youth may naturally "age out" of behavior problems whereas others may develop even more serious behavioral problems. These two groups may appear similar in behavioral symptoms at a single time point, but may develop very different trajectories. If we could determine which youth are on a trajectory for persistent behavior problems and target them with more intensive intervention programs, we would greatly improve our ability to reap the most benefit from limited intervention resources.

These authors supported the use of genetics and other biomarkers to identify high-risk groups. But to what extent do current methods allow us to do this? There was once

excitement about a finding that antisocial behavior is more likely to emerge from an interaction between a genetic variant and maltreatment (Caspi et al., 2002). But those results were only statistical correlations, and they are not consistent enough to be used to guide prevention (Fergusson et al., 2011). Moreover, we now know that single genetic variants are not strongly linked to psychopathology, and that we need to examine the whole genome. At this point, the concept of preventing antisocial behavior requires the Scottish verdict of "not proven."

Another example of secondary prevention is the currently influential movement to identify adolescents at risk for psychosis, so that they can be treated early in the course of the illness (McGorry et al., 2008). In psychiatry, many if not most of the major disorders begin in adolescence, with precursors that can be identified before a characteristic clinical picture develops.

When DSM-5 was being prepared, a controversy centered on the concept of "risk psychosis," or what DSM-5-TR calls "attenuated psychosis syndrome." Patients who eventually develop first-episode psychosis often have prodromal symptoms, and it has been suggested that treating them before more severe symptoms emerge might prevent sequelae (Addington et al., 2008).

In Australia, a national program has been put in place for the early identification and treatment of such cases. However, two-thirds of those with attenuated symptoms do not convert to psychosis, leading to a large number of false positives (Cannon et al., 2008). Thus many people with prodromal features could be treated unnecessarily. This example also highlights some of the pitfalls of secondary prevention.

Some evidence suggests that early interventions can lead to a better prognosis, as well as a lower death rate (Anderson et al., 2018). However, a recent large-scale umbrella review of multiple meta-analyses failed to confirm any consistent effect on the emergence of full-blown psychosis (Fusar-Poli et al., 2019a). Given these contradictory findings, we need much more data before firm conclusions can be reached.

Another problem is that even if interventions are effective, they may not be cost-effective (Aceituno et al., 2019). These programs certainly provide much more intensive follow-up for young people with schizophrenia than has been usual, but these resources are also expensive. It is possible that we could do as well with any approach that provided a closer follow-up, or that similar programs could be offered to chronic patients. In short, the early psychosis movement should be regarded as promising, but it remains unclear whether it provides a basis for shifting scarce resources.

Problems of Suicide Prevention

Of all the clinical dilemmas that psychiatrists face, the most troubling one concerns patients who die by suicide. Most mental health clinicians will lose some patients in this way. It would be great if we knew how to predict such outcomes. If we were able to determine which patients are most at risk, we could use that information to prevent fatalities.

Suicide is also one of the leading causes of death in both young and old age groups. About 800,000 people worldwide die by suicide each year, and the rates in countries where statistical data are most accurate range from 10/100,000 to 14/100,000 (Ritchie et al., 2022). This is a major public health problem, but it is also an emotional issue, leading to a great deal of controversy about the best approaches to prevention.

In the UK, the Royal College of Psychiatrists (2020) has published a useful set of clinical guidelines for the management of suicidal patients. They offer much sensible advice, such as the need for being empathic in the face of suicidality, remaining available, and offering safety plans. I recommend following these guidelines while keeping their limitations in mind. First, they apply to patients who are *already* in treatment, not those who fail to seek help (and who are more at risk for a fatal outcome). Clinical guidelines are not necessarily applicable to the population at large. Second, these guidelines do not tell us *which* patients in treatment are most at risk for death by suicide. Thus, although we are probably able to prevent some suicides in practice, preventive measures are limited by a very large number of false positives (Turecki and Brent, 2016).

The research literature helps to explain why we remain uncertain about the feasibility of prevention. In a classic paper, Beautrais (2001) noted that there are two partially overlapping populations at risk for suicide. One group consists largely of males, who tend to die on the first attempt, use more lethal means (e.g., firearms, hanging), and do not necessarily contact the mental health system. The other group consists largely of females, most of whom are seen clinically for attempting or threatening suicide. These patients actively seek treatment, and many of them have personality disorders (Paris, 2020b). The first group lies beyond our grasp, as they do not usually seek professional help. It follows that we need a different strategy for suicide prevention—one that focuses on the population at large.

In fact the most convincing evidence that suicide is subject to primary prevention comes from a population-based strategy (Kapur and Goldney, 2019). This involves using interventions such as limiting access to fatal means (Paris, 2021). As shown by a recent systematic review, these are the interventions that have the best evidence for primary prevention of death by suicide (Altavini et al., 2022). The main measures include restricting the availability of guns and poisonous substances, preventing access to unprotected bridges, or even simply reducing the pack sizes for medications that are prescribed. A famous example of the effectiveness of such measures was the lowering of fatality rates that occurred when the composition of natural gas provided to homes in the UK was changed in order to reduce the amount of toxic fumes (Clarke and Lester, 1989). If society wants to stop people from killing themselves, it should focus on this kind of intervention. Strict gun control would be a good place to start. But, as we know, politics stands in the way of such reforms.

Fortunately, we are often able to help patients in the second group, and a large body of research shows that they can benefit from treatment (O'Connor and Nock, 2014). Long-term follow-up of suicidal patients presenting in emergency settings shows that only about 2–3% of them will eventually die by suicide (Hawton and Zahl-Weatherall, 2003). The other 97–98% are open to treatment. Although we do not know the size of the impact of good clinical care, it is very likely to make a difference. But those of us who regularly work with suicidal patients need to be prepared to lose some patients.

The problem concerns assessing suicide risk accurately in clinical settings. Psychiatrists are taught to assess risk, but since most of the patients they see will not die by suicide, there are a vast number of false positives. We lack an evidence-based procedure to predict this outcome with any precision.

The reason for the problem is that death is a rare outcome by comparison with suicidal attempts, threats, and ideation, which are common in both clinical and

community populations. About 50% of the population will have suicidal thoughts at some time during their life, and about 5% will make a suicidal attempt (Kessler et al., 2005c).

Decades ago, two large-scale studies (Pokorny, 1983; Goldstein et al., 1991) attempted to predict suicide in large populations of patients who were followed for years after admission to hospital for suicidality. Both studies found that although algorithms based on risk factors found some factors to be statistically associated with fatality, they failed to predict any individual death by suicide. The problem is that one cannot predict a rare outcome from a set of risk factors that are much more common.

Patients who have made repeated attempts are more at risk for a fatal outcome, but the vast majority do not die by suicide (Zahl and Hawton, 2004). This is another example of the problems in establishing causality in psychiatry. Thus, even when risks that can be identified have a statistical relationship to outcome, most people with the risk will not have the outcome, and most people with the outcome will not have the risk factors.

Despite a dramatic increase in the number of mental health professionals and in the prescription of antidepressants, suicide rates—although somewhat variable over time—have not shown dramatic changes over the long term. This is true for developed and undeveloped countries alike. Suicide rates go up and down, and we can only guess why this is so. Nor has there been any reduction over several decades in the frequency of ideation, gestures, or attempts (Kessler et al., 2005c). In fact there has never been a time or a society in which suicide or suicidality was absent.

The good news is that for the patients whom psychiatrists see, a large amount of data shows that managing suicidality can be effective. In a review, Turecki and Brent (2016, p. 29) concluded that "Psychotherapeutic, pharmacological, or neuromodulatory treatments of mental disorders can often prevent suicidal behaviour; additionally, regular follow-up of people who attempt suicide by mental health services is key to prevent future suicidal behaviour." I share these views, but keep in mind that they apply to treatment-seeking people with suicidality, and that clinical work inevitably involves active treatment of false-positive cases. We do not have the evidence to show which patients would have risked a fatal outcome without our intervention.

These considerations also raise the question of whether hospitalizing patients who are thinking of suicide is an effective intervention. There is a lack of evidence to justify doing so, but no one would even consider performing a randomized trial to resolve these issues (Paris, 2021). Again, the problem is that suicidal ideation is much too common to be a useful "gateway" measure. In a wide-ranging review, two experts in the field concluded that no current method of suicide risk assessment has sufficient sensitivity and specificity to be useful in practice (Berman and Silverman, 2014).

Having spent most of my career providing psychotherapy to suicidal patients, I believe that we can help most of them (Paris, 2020b). There is also evidence that certain pharmacological interventions make death by suicide less likely. The data supporting antidepressant therapy as a preventative are not impressive (Nischal et al., 2012). The most convincing findings come from studies of bipolar disorder (Cipriani et al., 2005), which show that patients who take lithium have a lower rate of completed suicide than those who do not. Moreover, schizophrenic patients who are prescribed clozapine (but not any other antipsychotic) have a reduced rate of suicide (Meltzer and Okayli, 1995).

Nothing I have said should be taken to suggest that we should abandon efforts to treat suicidal patients. This has been the main subject of my career. We just have to accept that these outcomes are not predictable. Psychiatrists will lose some patients to suicide, but they should not be blamed for fatalities. And physicians as a whole have to accept that some of their patients will die despite their best efforts.

The Future of Prevention

When we learn more about the causes of mental disorders, we will have a better idea of how to prevent them. It is true that smallpox vaccination worked before anyone knew about viruses or immunology. But that was a lucky break. Most advances in prevention require precise knowledge of etiology and pathogenesis. And when an outcome is rare, it may not be cost-effective to prevent every case.

Moreover, an understanding of etiology cannot always be directly applied to treatment or prevention. A good example is cystic fibrosis, in which a specific genetic flaw is well known, but in which therapy still remains dependent on physiotherapy or lung transplants. Consider the large-scale resources devoted in recent years to developing vaccines for COVID-19 and malaria. Better research on suicidal fatalities would also be costly. Yet given that suicide is near the top of the list of conditions associated with an early death, the expense of investigating the pathways to death by suicide is well justified.

Chapter 8

Antidotes to Fads and Fallacies

Research Is Not Immune

The antidote to fads and fallacies is evidence-based practice. In other words, we should—as much as is possible—practice psychiatry and clinical psychology on the basis of what scientific data tell us. This requires maintaining a state of doubt about how to treat patients until the evidence can be evaluated and supported by meta-analyses and Cochrane reports.

As I have reminded the reader throughout this book, many of the most important questions about the causes and treatment of mental disorders remain unanswered. We need to accept that we are studying extremely complex phenomena that cannot be reduced to simple or linear pathways. Moreover, we may find ourselves asking the wrong questions. Practitioners should keep up with the research literature to ensure that their clinical decisions are neither faddish nor fallacious. But they should also remember that the current research consensus may not be immutable, but it can change.

As a former editor of a psychiatric journal, an associate editor of a specialized journal, and an active peer reviewer of scientific articles, I like to think of myself as an educated consumer of research. But recent developments in this world have opened my eyes to problems of greater scope than I had ever realized. Some reflect fads and fallacies that apply specifically to the world of research.

This territory has been nicely covered in a book by the Scottish psychologist Stuart Ritchie, in which he sets out the reasons why psychology, medicine, and other sciences have been subject to a *replication crisis* (Ritchie, 2020). Some years ago, John Ioannidis wrote a much quoted article, published in *PLOS Medicine*, entitled "Why most published research findings are false" (Ioannidis, 2005). His point was that unless results are consistently replicated by multiple research groups, they cannot be trusted. He showed that some of the most quoted findings in the medical literature, even including many pertaining to cancer research, have either not been replicated by other research, or have never been subject to replication. This problem affects most scientific disciplines (Baker, 2016). But the same sense of doubt has come to particularly afflict psychological research, in which many much-quoted findings have not been replicated (Witkowski, 2019).

Science is a method that encourages researchers to carry out studies that test a hypothesis. But its procedures sometimes leave room for doubt about conclusions. For example, consider the most common method of testing statistical significance, measuring how well the findings in the sample under study match those of a larger population—that is, a p-value of less than 0.05 is too low a bar. Experts have long recommended either that it should be changed to $p < 0.01$ or $p < 0.005$, or that the results should be evaluated

using an *effect size*, which measures the strength of the findings in terms of standard deviations (Cohen, 1994).

The main problem in mental health research is that samples are often too small, which makes it much more likely that results will not be replicated. It can be easy to recruit enough people to get a *p*-value from univariate analyses. But researchers are now often expected to consider multiple variables and multiple outcomes, and to conduct regression analyses that require much larger samples. Of course, larger samples require more money. And as every researcher knows from bitter experience, funds are never easy to access.

Thus it is not surprising that researchers try to get around this, particularly if they have a small grant or none at all. As a reviewer, I am not impressed when an underpowered study describes itself as a "pilot." It is usually obvious that the authors have no serious intention of carrying out the same procedure in a larger group. Another problem with research is "p-hacking" (Ritchie, 2020). If you do not find anything significant in your results, you look for a way of seeing if *anything* reaches the 0.05 level, even if the study was designed to test a different hypothesis. This form of cheating could only be controlled by publishing a plan of analysis in advance, and following it,

Progress in identifying valid research results also requires changes in how the system rewards scientists (Ritchie, 2020). For example, journals do not often publish negative findings or failures of replication. In a notable example, a well-known psychologist published a paper claiming to prove the existence of extra-sensory perception (Bem, 2011). But the same journal declined to publish another paper that failed to replicate these results—it later found a home in another widely read journal, *PLoS ONE* (Ritchie et al., 2012).

Moreover, research that fails to find anything that reaches significance may never even be submitted for publication—an outcome that has been called the "file-drawer problem." In this way, publication bias gives undeserved weight to positive findings. At the same time, this undermines the value of meta-analyses that combine findings from multiple sites, which are skewed by file drawers containing unpublished negative data.

I encountered an example of this problem in my research on the long-term outcome of borderline personality disorder (BPD). Our team had published the findings of a 15-year follow-up (Paris et al., 1987), and showed, as did several other research reports, that most patients improve remarkably with time. But a colleague at another site in Canada, who had been in charge of an inpatient ward where patients received residential treatment for about 2 years, never published his own data on a 10-year follow-up, which found much less improvement. This happened despite the fact that the program had received a grant from their province to carry out such a study. I assume that this colleague did not want the world to know that his program had failed to yield the dramatic changes which he had thought were possible.

Finally, "statistical significance" is itself a misnomer. In a complex world, findings can reach the $p < 0.05$ level and still only account for a small percentage of the variance. This is why the very term "significance" can be misleading—it is not the same as being either clinically significant or scientifically significant. (A better term might be something neutral, such as "estimate of probability.")

In view of all these problems, I advise my students to always doubt single studies, and wait for a meta-analysis before they apply any finding to clinical practice. Meta-analyses are a kind of gold standard—when many studies are combined and a clear finding

emerges, the limitations of each may cancel one another out and the results will be more replicable.

The years I served as a journal editor also taught me about what to believe or not to believe about research. We did not accept articles based entirely on opinion, and routinely turned down submissions that lacked a control group or drew conclusions from small samples. Although some of our readers may have found our standards too high, raising the bar was our way of encouraging the practice of psychiatry on the basis of evidence.

Evidence-Based Psychiatry

Evidence-based medicine (EBM; Evidence-Based Medicine Working Group, 1992) has now been a movement for over 30 years, and has greatly influenced the way in which medicine is practiced. It has been closely associated with two academic institutions (Oxford University in the UK, and McMaster University in Canada). The originator of the concept of EBM was a pioneering British physician, Archie Cochrane, whose name lives on in a series of authoritative (and famously skeptical) "Cochrane reports" that assess the efficacy of medical treatments. Those of us who follow Cochrane reports continue to be surprised at just how little medical science knows for sure.

EBM is based on a simple but powerful idea. An editorial in the *British Medical Journal* (Sackett et al., 1996) described it as follows: "the conscientious, explicit and judicious use of current best evidence in making decisions about the care of individual patients. The practice of EBM means integrating individual clinical expertise with the best available external clinical evidence from systematic research."

These principles can be applied to mental health practice. In a major cultural shift, evidence-based psychiatry has become a force to be reckoned with (Geddes and Carney, 2001). Its influence is apparent in the changed content of all psychiatric journals.

As was described in Chapter 1, when I took the trouble to review the contents of several major psychiatric journals published 60–70 years ago, I found that top journals were replete with case reports, case series, and unsupported clinical opinions. Such articles are now rarely (if ever) published. Instead, the pages of medical journals are filled with quantitative studies that test hypotheses, as well as systematic reviews and/or meta-analyses. It is no longer sufficient to have clinical experience. One has to back up one's ideas with empirical data.

The gold standard in research is the randomized controlled trial (RCT), designed to measure the efficacy of a treatment—that is, whether it is superior to placebo or to an alternative intervention. These observations can be supplemented by effectiveness studies that examine how well the treatment works in the messy reality of clinical practice.

Largely because of EBM, psychiatrists are now expected to follow clinical practice guidelines, such as those published by the National Institute for Health and Care Excellence (NICE) and the American Psychiatric Association (APA), and Cochrane reports. These organizations publish systematic reviews with conclusions based on the best existing empirical evidence for treatment effectiveness. Although guidelines can sometimes be wrong, it is better to have them than not to have them. At the very least they provide a brake that discourages faddish treatments. By and large, they represent the state of the art. But given that often there are not enough data to allow firm conclusions, clinical practice guidelines should be viewed as a work in progress.

Although some have criticized the application of EBM to psychiatry, certain limitations are inevitable. Westen (2006) and Ghaemi (2009b) have highlighted problems with the generalizability of RCTs. Since these studies require patients to sign up for them, they tend to attract unrepresentative samples (i.e., patients with less severe disorders, lower levels of comorbidity, and higher compliance). Some individuals participate only after answering an advertisement and being paid to take part. These limitations make it difficult to generalize, even in the best studies, from RCTs to practice. That is another reason why I advise my trainees never to rely on single studies, but to wait for replications and meta-analyses. As long as generalizability is limited, clinicians should never change the way they treat patients on the basis of a single study.

Meta-analyses can be flawed if they depend on data drawn from unrepresentative RCTs. Also, given the judgment calls required to decide what studies should be entered in meta-analyses, it is not uncommon for different analyses to yield different answers to the same question. But that is the nature of science. Conclusions depend on what is accepted as the best evidence, and that can change over time.

This is also why most clinical practice guidelines lack high-quality data to support their recommendations. When experts are forced to say *something*, they may say more than they should. Although the NICE guidelines can usually be counted on to be cautious, the American Psychiatric Association guidelines have tended to overstep the evidence. This problem can emerge whenever conclusions are assigned to experts with a bias. Fortunately, Cochrane reports are famous for their conservatism.

Some years ago, the Cochrane Collaboration asked me to review a report on the pharmacological treatment of BPD. I found it unworthy of the Cochrane imprint, since it made several recommendations based on single trials involving small samples. The final version that eventually appeared on their website was revised and took my critique into account (Stoffers et al., 2010). But I was shocked when, soon after I had written my review, a précis of the original version was published in the *British Journal of Psychiatry (BJP)* (Lieb et al., 2010), *prior* to review by the Cochrane Collaboration. I wrote to the editors, who admitted their mistake in failing to set and enforce an embargo on that version, and they assured me that they would be more careful in the future. In fact this debacle occurred because Peter Tyrer, the then editor of the *BJP* and a noted expert on personality disorders, had to be kept out of the loop because of his involvement in writing the NICE guidelines. Thus. even though a toned-down version of the original paper was published on the Cochrane website, it was the *BJP* paper that often got cited. That practice could do harm by misleading clinicians into prescribing drugs when the evidence base is extremely weak.

The lesson is that one has to remain critical, even of guidelines written by academic leaders. Some of their recommendations reflect strong biases, supported by cherry-picking the literature. One would like to believe that experts are dispassionate, but most of them have strongly held points of view. One cannot always trust researchers who have invested years in their own research to be fair to opposing ideas.

Although the basic principles of EBM remain valid, it can take many decades to collect the data that clinicians need to make evidence-based decisions. Moreover, there are few data that can be used to guide most of the questions faced by physicians. Some psychotherapists dislike EBM because it does not tell one how to talk to people, or how to be empathic and caring. But evidence-based practice is about outcomes, not communication. It need not be associated with mindless empiricism, or lead to algorithmic

diagnoses followed by routine prescriptions of drugs or psychotherapy. You cannot blame the parlous state of modern psychiatry on science, which actually supports spending more time with patients. The problem runs much deeper than that.

For the last 45 years I have run an EBM-based journal club seminar for psychiatric residents. I ask trainees to bring in clinical questions arising from their work, and to explore the literature for answers. Almost without exception, we find that research is insufficient to allow any conclusion to be reached, and that many clinical principles which everyone knows to be "true" have never been empirically tested. Some trainees find this exercise discouraging, since they want answers to help them practice, and may view Cochrane reports as depressingly conservative. But I find its cautious, measured perspective a breath of fresh air.

The rise of EBM reflects a change in the authority of physicians and their relationship to patients. Patients are no longer passive consumers of services. Many, quite rightly, expect to be informed about their treatment, and to be a part of any decision-making process. Some even arrive with printouts downloaded from the Internet. This makes clinical practice a little more challenging. Physicians used to be expected to know the answer to any and all questions. Psychiatrists have been particularly guilty of speaking from a position of authority, even on issues well outside their area of expertise. Now physicians can sit down with their patients in front of a computer and they can look up the answers together. Patients can also be encouraged to go home and look up their diagnosis on a reputable website. Medical practice has become much more of a collaboration.

In summary, the practice of EBM promotes doubt, not certainty. That is a good thing, mainly because doctors know much less than they think they do. A few years ago I was explaining EBM to a humanities professor. Looking at me with dismay, he asked, "Do you mean to say that doctors haven't *always* been practicing on the basis of evidence?" The public has an absolute right to expect physicians to base clinical decisions on scientific data.

Yet EBM still remains more of an ideal than a reality. Many decisions in practice must still be based on clinical experience. For example, decisions as basic as whether or not to hospitalize a patient are almost always made without data. But the percentage of such decisions may shrink gradually over time.

The Need for Patience

Fads and fallacies are poisons for which evidence-based psychiatry is the antidote. Yet psychiatrists still need to be critical about the quality of published evidence. They can easily be fooled by a single study, most of which are never replicated (Ioannidis, 2005). Agencies that approve drugs, such as the Food and Drug Administration (FDA) in the USA, set the bar much too low, allowing drugs to be put on the market after only two clinical trials. That is why I never prescribe any drug that is new on the market. I stick with the older ones until more evidence is available. It is also why established psychotherapies are usually better choices than newly developed methods. To change one's practice, one needs a large number of trials in clinically representative samples, sufficient for meta-analysis and pointing in the same direction. It is better to keep to tried-and-true methods until the weight of evidence supports something better. Following these principles leads to a much more conservative approach to the practice of psychiatry. That is just as it should be.

If even Cochrane reports can get things wrong, how can busy clinicians hope to make informed decisions? Moreover, clinical trials—however rigorous they may be—are not conducted in real-world samples. Although they can point the way, they should not be considered the last word on clinical practice. That is why efficacy studies have to be supplemented by effectiveness studies, in which researchers do not select patients, or enroll a control group, but make systematic observations on what happens naturalistically in a real-world clinical setting.

I have mentioned in Chapter 5 the findings of one of the most famous and influential effectiveness studies in the history of psychiatry, namely the Sequenced Treatment Alternatives to Relieve Depression (STAR*D) study, which drew on a large and representative sample from clinics around the USA (Valenstein, 2006; Rush, 2007). The most important findings were as follows: (1) major depression can be chronic, severe, and associated with substantial comorbidity; (2) many patients drop out of treatment; (3) the initial response to antidepressants is often slow; (4) remission is less frequent than partial improvement; (5) some, but not most, patients do better if switched to a different antidepressant; (6) a minority of patients benefit from other drugs used for augmentation; (7) a subsample benefits from adding cognitive therapy. Welcome to the real world of clinical practice—and leave the artificial world of clinical trials behind!

But what did this massive and expensive project actually tell us about how to treat depression? Those who already favor aggressive therapy took heart from the results, but the effects of augmentation and switching were not in fact that impressive. And although two-thirds of patients eventually recovered, many took a year to do so. Yet we know that depression has a natural course, and that many individuals recover within a year, even without treatment, as often happened prior to the era of modern psychopharmacology (Shorter, 2009). Last but not least, without a control group we cannot know if any of the medications worked, or whether they only acted as placebos. Thus, although STAR*D added to our knowledge, it raised as many questions as it provided answers. It seems to show that we are not as effective as we think we are in treating depression. If it is hard to interpret multi-million-dollar projects sponsored by the National Institute of Mental Health, it is even more difficult to draw conclusions from the smaller studies that appear in journals.

Patience is hard for students. They are learning a new set of skills, and they want answers. I may not gain popularity for doing so, but I tell them not to believe what their teachers say until they have checked out the data themselves. I also tell them to doubt what they are told by expert lecturers with an axe to grind. Finally, I suggest that they need to be particularly dubious about what they read in textbooks, which are often written with superficial and unjustifiable certainty. I remind my students that medical science only slowly increases knowledge, and that this process can take decades.

That being said, the limitations of EBM reflect an early stage in its development. We are talking about a method that is only a few decades old, and that has only gradually come to be a widely accepted paradigm for the practice of medicine. It is still the best way to remain doubtful about what we read.

Evidence-Based Consultation

The work environment of psychiatrists influences how they think. These days, practitioners are somewhat less likely to work in offices outside institutions, isolated from their

protective umbrella. That model was suitable for the exclusive practice of psychotherapy, but makes little sense for the management of severely or chronically ill patients, who need services from a professional team that provides skills which medical specialists lack. Today, psychiatrists are less likely to provide direct care to patients with conditions of mild to moderate severity, who can be just as effectively managed by others. But we still have an important role to play as consultants to those who do treat these populations. This is not to say that psychiatrists have stopped treating patients, but that they now tend to concentrate on the sickest individuals, who require the skills of a specialist.

The growth of primary care allows psychiatrists to pass on their expertise to a much larger community of providers. Often, family physicians provide medication while psychologists provide therapy, and psychiatrists back them up. Psychiatrists no longer practice much psychotherapy (Tadmon and Olfson, 2022), and most of this work is carried out by clinical psychologists. Yet, increasingly, patients are benefiting from treatment by multidisciplinary teams. Psychiatrists can support these teams by consulting on the most difficult cases. In many ways this is a central role for our profession.

To make consultation evidence based, one has to resist the temptation to recommend something new for every patient who has not responded to standard therapy. If a patient has not yet been offered a fair trial of any medication, and if there is a reasonable indication for making the attempt, one can recommend pharmacotherapy. But in many of the cases that come to consultants, several agents have already been tried without result. We can often provide consultees and their patients with a better service by introducing a different perspective.

Once patients are on pharmacological treatment, they sometimes stay on it indefinitely—much like interminable psychotherapies. Physicians and patients are afraid to stop drugs for fear of a relapse. Since some of the agents that we use have long-term side effects, if the patient is not psychotic I may recommend that they be stopped. However, people get used to sedatives, and after years of use many cannot sleep without them. Therefore I am reluctant to give advice that may not be followed. The long-term use of antidepressants is much less dangerous, but most clinical guidelines recommend that they be given for 6 months, and then tapered to see whether there is a problem with stopping. Maintenance therapy is generally reserved for patients who have had multiple episodes of depression. But that is not necessarily what happens in practice. Once an antidepressant has been prescribed, it may be renewed indefinitely unless the patient insists on getting off the drug. And the same problems emerge with the long-term use of mood stabilizers (in patients who are not bipolar) and in relation to the long-term use of stimulants (in patients whose ADHD diagnosis is doubtful).

My consultations often suggest approaching treatment from a psychosocial perspective. However, following this advice may require resources that are not readily available, particularly if a multidisciplinary team is not in place. In cases in which nothing further can be done, I offer a reassuring (and factually correct) message by saying that all bases have been covered, and that there is little to do but follow patients who suffer from a chronic illness. This advice may disappoint those who receive it, but they can also reassure consultees that they are not missing anything. And when conclusions are cautious, recommendations are more realistic.

EBM may be more useful for preventing bad practices than for proving good practices effective. Once one understands these problems, one is less likely to be enticed by fads. Journal articles are written in that way. They never speak of "proof," but say "the

evidence suggests ...." If correlations are interpreted as causation, peer reviewers will bring them up short. Unfortunately, critical views of this nature are uncommon in clinical settings where hope sometimes trumps experience. Scientific progress in medicine is gradual, but physicians are not always able to avoid becoming carried away by enthusiasm.

I consider myself a "born-again" follower of EBM. Its principles inspired me and helped me to emerge from the inevitable funks that can affect the practice of medicine over several decades. EBM has taught me to be conservative and patient both in the way that I manage patients, and in the recommendations that I give to other professionals. It has taught me to accept the limitations of my craft, and to value what I *can* do for patients despite these limitations. It has made me a different kind of teacher, even if some trainees would prefer a little less criticism of the literature, and more encouragement to try "the latest thing." EBM has also informed my own research. But practitioners should keep in mind that it might be another 100 years before some of the most important questions are resolved. Patience and caution are called for. Moreover, we already have the tools to help most of the patients we see, even if we cannot always provide a cure.

Chapter 9

Overview

In the 60 years since I was a medical student, psychiatry has moved forward. This book has not intended in any way to deny that progress has been made.

Although research based on a biopsychosocial model is just beginning, this approach is already gradually illuminating the causes of mental illness. We now prescribe drugs with fewer side effects, and use more practical forms of psychotherapy. We have treatments that have been shown to be both efficacious (i.e., they are supported by clinical trials) and effective (i.e., they work in the real world of practice). Our record for treatment of patients is as good as that of internal medicine (Leucht et al., 2012).

Yet even when not enough is known, our discipline can fall victim to hubris. Psychiatry has sometimes searched for premature closure and easy answers. The twenty-first century began with great expectations for breakthroughs in both theory and practice. Following "the decade of the brain" in the 1990s, we prepared for a brave new world of specific biomarkers and bedside functional magnetic resonance imaging. One of the more recent hopes, although unsupported by research so far, was for personalized treatment based on genome analysis (Perna et al., 2018). None of these things have come to pass, and one can only be skeptical about the view that they lie just over the horizon.

Psychiatry still faces a long journey, and few of us will live to see it arrive at its destination. In the meantime, the current standard of mental health care offers grounds for both hope and concern. On the plus side, psychiatrists do a good job of caring for patients with severe illnesses. For the psychoses, clinical guidelines are largely based on data, and are widely followed. But as soon as one moves into the realm of common mental disorders, or of conditions that are known to be responsive to psychotherapy, it ceases to be certain that patients will be offered evidence-based treatment. This situation leaves mental health practice open to fads and fallacies.

A Critical Portrait of Modern Mental Health Services

Fewer patients today go directly to specialists. Those with common mental disorders usually pass through a gateway of primary care. But mental health treatment offered in that setting, with the support of consultations by specialists, is shaped by the culture of contemporary psychiatry. Unfortunately, psychiatric consultants tend to support what can be called "aggressive" treatment when standard therapies fail. If a patient has not responded to a drug, most will recommend that another drug is tried. This can be a reasonable decision. But it is not reasonable to try four or five.

These practices are based on the belief that mental disorders are caused by chemical imbalances, and that one only has to find the right drug or combination of drugs in order to manage "treatment-resistant" cases. There is limited evidence to support such an approach, given that not all depressed patients respond to antidepressants. There is also little reason to prescribe the latest agents on the market, which are generally no better than the older ones, and have less well-known side-effect profiles. (The National Health Service in the UK mandates the prescription of generic drugs, and Canadian provinces do not routinely insure newer and more expensive alternatives to standard therapy.)

The other serious problem afflicting modern psychiatry is that practitioners may spend insufficient time obtaining a life history to evaluate past and present psychosocial stressors and personality traits. Psychiatrists are being trained to ask screening questions so that they do not miss any important symptoms, particularly those that define DSM diagnostic criteria. Although there is nothing wrong with such procedures, they take up time that could be used more profitably. Our current training for specialists does not properly prepare practitioners to understand the context in which symptoms develop. This requires more than the 15 minutes that are devoted to a "medication check" in many clinics.

Psychiatry was right to move psychotherapy out of a central role in the treatment of severe mental illness. This shift allows us to concentrate on the sickest patients, and to transfer many forms of care to our colleagues in clinical psychology. Yet this trend has worsened the split between biological and psychosocial psychiatry.

Our profession has not seriously lobbied for better access to psychotherapy. There will never be enough psychiatrists to do this kind of work. Thus we need to forge a closer partnership with psychologists, who offer most of the therapy that patients receive. There are also social workers and occupational therapists who are well trained in psychotherapy.

The main obstacle to moving in this direction is lack of funding. In the USA, one is unlikely to receive high-quality therapy unless one has good insurance. In Canada, health care does not cover psychologist sessions in private clinics or offices. In the UK, cognitive–behavioral therapy (CBT) is now more widely available, but specialized therapies are still difficult to access.

Behind the lack of mental health services lies a deeper problem. Despite the fact that mental illness ranks high in terms of the global burden of diseases and in terms of impact on functioning, it is seriously underfunded worldwide. Ultimately this reflects the stigma of mental illness, and the hope of those who control public funds that it will just go away if it is ignored for long enough. (People are afraid of psychiatry because they do not want to be associated with the stigma of madness or despair.)

Moreover, the quality of psychotherapy is not well monitored. Although all practitioners are encouraged to follow the literature and offer evidence-based treatment, they tend to fall short even if they attend the required number of yearly professional conferences. This book therefore challenges the complacency that afflicts current mental health practice as a whole. The professionals who provide these services do not always follow clinical guidelines, and many of them have allowed themselves to be oversold on the use of inconsistently effective treatment methods.

We also need to provide better consultations to physicians working in primary care, who write most of the prescriptions for mental disorders. Those of us who do this work

need to apply a biopsychosocial model to the understanding of therapeutic options, and when indicated to promote referrals to psychological forms of treatment.

Another recommendation would be to move psychiatry from its more grandiose hopes to the art of the possible. A good analogy here is the management of chronic pain (Cohen et al., 2021). Like depression and anxiety, chronic pain is ubiquitous, but those who suffer from it want relief. Whereas short-term interventions for chronic pain can be successful, long-term management tends to be problematic. This led to the widespread prescription of opioids, which carry a real risk of addiction. This analogy shows how physicians can feel impelled to do *something* for their suffering patients. Clinicians may find it difficult to accept that they are doing the best they can, and to accept their therapeutic limitations. When practice is driven by what physicians *want* to believe, it is more open to fads.

The practice of psychiatry, in both specialized and primary care, is shaped by what physicians hear from experts and opinion leaders. (Only a minority regularly read professional journals.) And what clinicians often hear from experts is that symptoms are almost always manageable, and that treatment resistance reflects a failure to prescribe the right drugs. We are not told the truth—that we spend much of our time treating chronic illnesses, and we are doing the best we can.

Finally, in a climate of limited funding, guidelines and protocols are being used more to contain costs than to be cost-effective. The embracing of a "can-do" ideology, reinforced by publicity from the pharmaceutical industry, encourages physicians to misinterpret temporary improvements after prescribing a drug as a "response," and fail to recognize the ubiquity of placebo effects.

I have seen some of my most intelligent and idealistic colleagues and former students fall victim to fads and fallacies. Their intentions are good, but most, even those who work in teaching hospitals, do not live in an academic culture. Few clinicians are guided by clinical trials, and some of them misinterpret studies if they support doing what they want to do anyway.

Psychiatrists need to be healthy skeptics, and to follow the literature in critical detail. They do not need to be influenced by medical culture, and jump on bandwagons. Many of my colleagues want to try out something new, even if it is more expensive and no more efficacious than older agents.

As an educator, I would like to believe that the next generation of psychiatrists, trained in academic centers, will embrace an evidence-based perspective. But most students follow what they are taught by their supervisors, including an excessive reliance on diagnostic manuals and an overly aggressive use of psychopharmacology. The leaders of psychiatry today have great influence, but most are researchers who strongly favor the use of biological tools. Students may also go to conferences where they are told about "the latest thing" in drug therapy. One of my brightest trainees surprised me by stating that he had never prescribed a generic drug, because his teachers always recommended the newest agents on the market.

A few years ago I was chatting with an American professor who informed me that he routinely sends treatment-resistant patients to psychopharmacological consultants for treatment. When I asked him why he trusted their opinion, his answer was that "These are experienced people who know what they are doing." Unfortunately, relying on the reputation of "experts" is not evidence-based practice. I felt sadly reminded of the

undeserved adulation of psychoanalysts, who were at one time considered to be founts of wisdom.

There is a large gap between theory and practice in psychiatry. Those who are in love with theory miss the point that clinical care is ultimately judged on the basis of outcomes and changes in those whom it serves. We need to embrace a more skeptical point of view. A recent article by a leading American researcher (Nemeroff, 2020) was refreshing in this respect, as it acknowledged how little we really know about depression, which has long been considered the "bread and butter" of psychiatry. We need to combat unjustified hubris and to accept uncertainty.

The Ideology of Psychiatry

Psychiatry has a history of going to extremes. Decades ago, it promoted fantastic psychological theories. Then, craving the prestige of other specialties, it fell in love with neuroscience. By and large, medicine tends to attract people who prefer action to reflection. Students who go into psychiatry have to face great skepticism, from both peers and teachers, about their choice of career. One of my trainees was told in medical school not to waste his time on psychiatry, given the certainty that all mental illnesses would soon be accounted for by neuroscience and treated with drugs. Moreover, some leaders in our own field (e.g., Insel and Quirion, 2005) have made the same argument, calling for the abolition of psychiatry as a separate discipline and its absorption into neurology. The result is that students who nonetheless train to become psychiatrists have something to prove. But this may also make them reluctant to accept the limitations of their craft.

I began this chapter by applauding the progress we have made in recent decades. Yet, to review the problems, we are working with a diagnostic system that is at best provisional, and that limits the validity of epidemiological data. Moreover, treatments based on current diagnoses, whether pharmacological or psychological, are not consistently effective. Finally, we have little idea of how to prevent mental illness. Even so, psychiatrists are doing the best they can with limited knowledge. We just need to be humble. A hundred years from now, our successors will view our field in much the same way as we see the psychiatry of the early twentieth century. We can only hope to be viewed with compassion.

The best hope for a more rational practice is to change the culture of psychiatry by acknowledging these limitations and depending more on evidence. Most psychiatrists spend little time reading, although they are required to attend scientific conferences. But what they are exposed to at these meetings is at best expert opinion, sometimes influenced by the pharmaceutical industry. If the culture of academic psychiatry did a better job of communicating what we know and what we do not know, the word could eventually get around that clinical practice needs to be evidence based.

I remember being excited by the challenge of joining a field in which so much remains to be discovered, and I still feel that way. With that in mind, our students can be encouraged to view their choice of medical specialty with pride.

Beyond Fads and Fallacies

I have written a critical book, but my stance is deliberate. I love psychiatry, but I want it to be better. I would like to raise awareness of the questions for which it currently lacks

clear answers. And given my commitment to science and evidence-based practice, I feel the need to promote doubt. This point of view has not always made me popular.

Nonetheless, psychiatrists still do much good for their patients, and their services remain in high demand, because they have the skills to manage some of the most troubling and terrifying illnesses in all of medicine. Even the medical colleagues who dismiss our discipline come to us if they, or a member of their family, are suffering from a mental disorder.

We are all susceptible to fads, at least from time to time. Most of us are also susceptible to the fallacious thinking that leads to incorrect conclusions. To avoid making these mistakes, I suggest ten general principles to follow:

1 Do not jump to conclusions without good evidence.
2 Be comfortable with how much you do not know.
3 Accept that you are treating disorders whose causes you do not fully understand.
4 Keep in mind that all diagnoses are provisional, and do not consider DSM or ICD to be a "bible."
5 Read the literature, especially high-quality reviews such as Cochrane reports and NICE guidelines.
6 As much as possible, practice on the basis of evidence.
7 If you hear an expert give a talk, remain skeptical, no matter how charismatic and convincing the presentation.
8 If a treatment claims to be appropriate for too wide a range of patients, be suspicious.
9 Do not believe anything you are told by a pharmaceutical representative.
10 Remember that medical science moves slowly, and that well-established treatments may be better than new ones.

I acknowledge that following all of these principles is easier said than done. But no one ever said that practicing medicine was simple. We owe it to our patients to do better.

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