Turing Patterns

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During his tragically short life that began 100 years ago, Alan Turing wrote only one paper about chemistry. Published two years before his untimely death, it is arguably one of the most influential ever to have come from an outsider to the field.

Since he was essentially a pure mathematician, it is surprising that Turing had anything to say about chemistry at all – there is no record that he ever so much as handled a test tube. But one never knew what to expect from Turing, whose curiosity wandered far and who was able to find connections between what seemed the most disparate of problems and ideas.

Turing showed how chemical reactions can create patterns. Mix the reacting ingredients, and they separate into quasi-ordered patches of different composition, despite the free diffusion of the reagents. Turing's theory of chemical pattern formation, now vindicated experimentally, looks like the best candidate for explaining a variety of problems in biological development, from the spontaneous differentiation of some tissues to the formation of pigmented markings and the patterns of leaves on plant stems.

Turing-type mixtures and related systems are now being explored as the basis of a kind of chemical computer – a pleasing symmetry, for computation lay at the heart of Turing's more famous work. And his spontaneous patterning scheme is relevant beyond chemistry, having been invoked to explain for example how sand ripples form, how ants dispose of dead bodies, how termites build their nests, and why crime seems often to be focused in 'hotspots'.

Can Alan Turing's chemical pattern formation explain the stripes of the zebra?

Code breaker

Born in London in 1912, Alan Turing is popularly known mostly for three things. First, he helped the Allies win the Second World War through his cryptographic activities at Bletchley Park, in particular by cracking the Enigma code used by the Germany navy. “I won’t say that what Turing did made us win the war”, one of his Bletchley colleagues said later, “but I daresay we might have lost it without him.”

Second, Turing proposed a test for determining whether machines can think. It involves a human interrogator who poses questions to the machine and to a human foil, and seeks to identify which is which. If there is no discernible difference in the responses, we have no logical reason to deny that the machine is thinking. This idea, used in the opening sequence of the 1982 movie *Blade Runner* to flush out non-human ‘replicants’, was one of Turing’s pioneering contributions to the field that became known as artificial intelligence.

Third, Turing committed suicide by biting into an apple laced with cyanide. Prosecuted in 1952 for homosexual activity, he was commanded to undertake a course of ‘corrective’ hormone therapy. Active homosexuality was still a crime at that time, and as he had engaged in confidential war work, Turing was particularly worrying to the authorities because of his perceived vulnerability to blackmail. Although Turing is said to have borne this sentence with “amused fortitude”, the shame and the physical effects of the hormone seem to have driven him to take his life in 1954. This shabby and barbaric treatment of a war hero leaves an indelible stain on the British legal system.

Turing’s work on computation laid some of the foundations of the discipline, and connected what seemed essentially to be an engineering issue to some of the deepest questions in mathematics. By establishing that some mathematical problems are ‘uncomputable’ – no computer will ever solve them in a finite time – he linked the notion of a digital computer to Kurt Gödel’s incompleteness problem, which stated that there will be propositions in any mathematical system the truth of which cannot be formally decidable within that system.

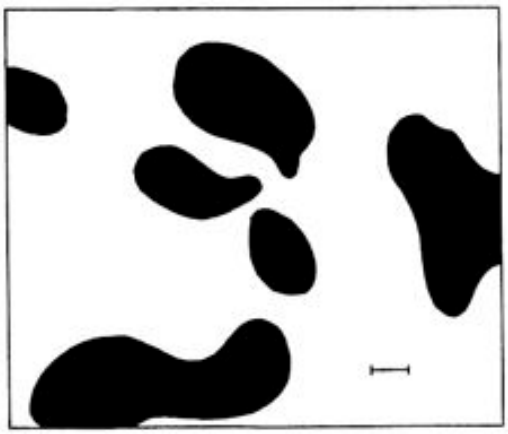
Breaking symmetry

It was essentially a mathematical question that led Turing to think about chemical patterns. He wondered how a ball of identical cells in the early stages of an embryo’s growth can develop into an organism with different features, so that some cells become limbs, some eyes, and so forth. The appearance of an organism’s body plan is called morphogenesis, and it is – or seemed to be – an example of spontaneous symmetry-breaking: from uniform to differentiated.

Turing proposed that the embryo becomes patterned into regions with different anatomical fates by chemical substances called morphogens (literally ‘shape-formers’), which diffuse through cells and tissues. He imagined them as catalysts that react to produce other reagents, some of which will ultimately govern the destiny of cells. Turing was deliberately vague about what the morphogens are – they could be hormones, perhaps, or genes. (It wasn’t yet clear, a year or so before Watson and Crick’s seminal paper on the structure of DNA, what genes

were or how they were encoded in the chromosomes.) The key point is that the morphogens diffuse and react with one another: his scheme is what is now known as a reaction-diffusion system.

Turing presented a mathematical analysis of how, under certain conditions, the interacting morphogens could give rise to 'blobs' of different chemical composition as they drift through a uniform system. He even sketched a two-dimensional "dappled pattern" which he calculated his scheme might produce. He did this by "manual computation", then the only way to crunch the numbers.



Turing's sketch of the patterns his scheme might generate in two dimensions. From Turing (1952).

Turing saw that his scheme had broader ramifications for biology, for example to account for dappled animal markings. In particular, he hoped it might furnish an explanation for phyllotaxis: the arrangement of leaves and florets in plants. This, as Turing knew, was also a fundamentally mathematical question. It had long been noticed that the florets of a sunflower and the leaflets of pinecones are organized in two groups of spirals rotating in opposite directions. The numbers of distinct spirals in each group are related to the Fibonacci series (1, 1, 2, 3, 5, 8, 13…), which had been known at least since the Middle Ages. For flower heads and pinecones, the numbers of spirals in the two groups always correspond to two successive numbers in the series. Turing occasionally discussed this baffling fact with his colleagues at Bletchley Park. He would surely have developed his theory in this direction, had he not died: he began drafting a paper in phyllotaxis between 1952 and 1954, and lectured on the subject at Cambridge.

It later transpired that Turing's mechanism isn't necessary for symmetry-breaking of a fertilized egg. Instead, the symmetry is disrupted from the outset by maternal proteins diffusing from one side of the embryo. Yet as chemist Patrick De Kepper of the University of Bordeaux points out, the real triumph of Turing's paper was to show that "no vitalist principle is required for biological development – ordinary physical and chemical laws could do the job."

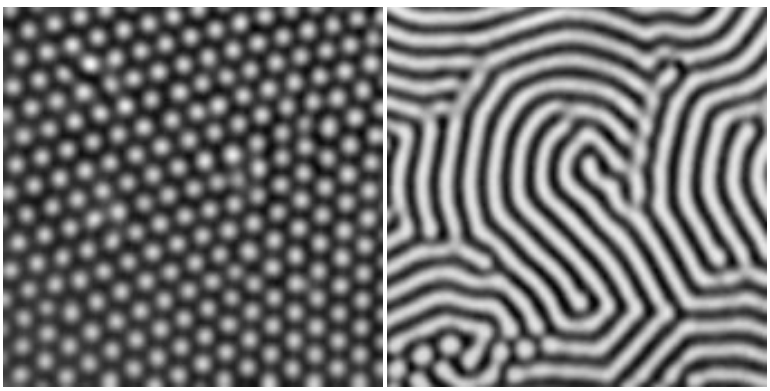
It wasn't obvious from the dense mathematical discussion in Turing's paper what the key ingredients of his process were. That was made apparent in 1972 by developmental biologists Hans Meinhardt and Alfred Gierer at the Max Planck Institute for Virus Research in Tübingen, Germany. They devised their theory of biological pattern formation without knowing about Turing's work, and became

aware of it only when a referee of their paper pointed it out. Meinhardt and Gierer showed that stationary chemical patterns can result from two interacting ingredients – equivalent to Turing's morphogens – if they have specific characteristics. One is an 'activator', which is autocatalytic and so introduces positive feedback. The other is an 'inhibitor', which suppresses the autocatalysis of the activator. Crucially, they must have different rates of diffusion, the inhibitor being faster. In effect, this means that the activator's self-amplification is corralled into local patches, while the inhibitor prevents another such patch from growing too close by. When they consulted Turing's work, the researchers found that his equations describe just this situation.

"In his paper there's no mention of an activator-inhibitor scheme", says Meinhardt. "It's fair to say that he didn't see that local self-enhancement and long-range inhibition is the decisive condition." In fact, Turing's own reaction scheme wasn't even chemically realistic, for example because it allowed for negative concentrations of the reagents. All the same, Meinhardt adds, it seems that Turing later suspected something like inhibition was involved.

"The basic principle we discovered helped tremendously to understand Turing's paper", he says. "It also made it more straightforward for us to understand more complex patterning systems in biology." Philip Maini, a specialist on pattern formation in biology, agrees: "I don't believe much had happened between the 1952 paper and 1972, when Meinhardt really got stuck into the problem."

Computer calculations of the activator-inhibitor scheme revealed that there are two generic types: spots and stripes. In both cases, the pattern features are all of roughly the same size and distance apart. In theory a Turing pattern can be a perfectly ordered lattice of spots or array of stripes, but in practice random defects interrupt this perfection, producing a quasi-regular pattern. Straight away you can see why Turing's theory looked like a good candidate for explaining the zebra's stripes and the leopard's spots.



The generic patterns of an activator-inhibitor scheme. Images: courtesy of Jacques Boissonade & Patrick De Kepper University of Bordeaux.

Making waves

In one of those coincidences that crop up so often in science, experimental evidence for the spatial patterning that a combination of reaction and diffusion might generate was being discovered at the very time that Turing was laying

down the theory. In the Soviet Union during the 1950s, the biochemist Boris Belousov devised a cocktail of reagents as a simplified analogue of the process of glycolysis, and found that the mixture oscillated back and forth between two states. Since they seemed to violate the second law of thermodynamics, Belousov's results were dismissed, and he was barely able to publish them. But in the 1960s they were explored by Anatoly Zhabotinsky as a graduate student in Moscow, who found a variation of Belousov's mixture that would switch back and forth between red and blue. When he discussed these findings at an international conference in Prague in 1967, chemists in the West were intrigued and began to figure out what was going on.

The oscillations were another consequence of a fine balance between rates of reaction and diffusion in an autocatalytic process. The Belousov-Zhabotinsky reaction switches between two branches involving different reaction intermediates, each of which can exhaust itself by runaway autocatalytic feedback and thereby create the conditions for the other branch to take over. Left to its own devices, the oscillations eventually die out as the intermediates capitulate to the final products – which is why there's no real threat to thermodynamic laws. But if the reaction is carried out in an unmixed solution, the switch doesn't happen everywhere at once but propagates as a regular series of pulsed chemical waves, creating striking patterns.

Patterns in the Belousov-Zhabotinsky reaction. Image by Stephen Morris, University of Toronto.

"The wave-like spread is comparable with the spread of an infection or of a forest fire", explains Meinhardt. Essential to the pulsed activity of the waves is the fact that once a wavefront has passed through, a region enters a 'refractory' period during which it can't support another wavelike excitation – in the forest-fire analogy, this is the time taken for trees to regrow.

These chemical travelling waves are different from Turing's stationary patterns, but the general principles of reaction and diffusion are the same. What differs are the relative rates by which the ingredients diffuse. The connections between the two systems first began to emerge in the late 1960s from the work of Russian-born Belgian chemist Ilya Prigogine and his coworkers at the University of

Brussels. Reaction-diffusion patterns, which Prigogine referred to as 'dissipative structures' because they are sustained by dissipation of energy in a non-equilibrium process, formed a central component of the work on non-equilibrium thermodynamics that earned Prigogine a Nobel Prize in chemistry in 1977.

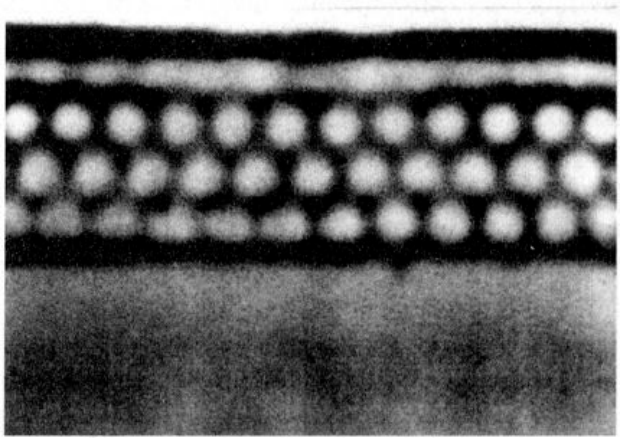
It's often forgotten that Turing himself recognized that under certain conditions systems of three morphogens could produce travelling chemical waves in his scheme. Meinhardt has shown that an activator-inhibitor scheme with a third morphogen that creates short-ranged but long-lasting inhibition can reproduce the kinds of complex patterns seen on some mollusc shells, which are in effect frozen traces of two-dimensional travelling waves on the rim of the growing shell.

Patterns on seashells and their analogues in theoretical activator-inhibitor systems. From Meinhardt (2009).

Slow motion

In 1971 physical chemist Adolphe Pacault, then director of the Centre de Recherche Paul Pascal in Pessac, a suburb of Bordeaux, visited Prigogine in Brussels and became captivated by the pattern-forming systems studied there. He hired Patrick De Kepper to work on these systems at Pessac, where De Kepper and colleagues found a way to hold an oscillating chemical reaction a controlled distance away from thermodynamic equilibrium, using a so-called continuous stirred tank reactor to allow a constant throughflow of reactants.

"In the 70s many groups working on oscillatory chemical reactions were dreaming of producing Turing patterns", De Kepper explains. But although some occasionally claimed success, the spatial patterns always turned out to be something else. Yet in 1990 De Kepper stumbled upon them almost by accident. He was investigating an oscillating reaction called the chlorite-iodide-malonic-acid (CIMA) reaction, which shares some ingredients with the BZ reaction, and in 1985 he and his colleagues began to look for sustained patterns in this mixture. They didn't expect to find actual Turing structures – but when they saw a band of spots appear in a strip of gel into which the reagents had diffused from opposite sides, they recognized them for what they were because De Kepper's colleague Jacques Boissonade had already predicted a genuine Turing structure of this kind in computer calculations in 1988. The discovery "was very exciting", says Maini. "By that stage, I think research on the subject was beginning to wane, but this gave it a great boost."



The first Turing patterns in a laboratory chemical system. From Castets *et al*. (1990).

The reason why these patterns appeared in the CIMA reaction was explained a year later by Irving Epstein and István Lengyel at Brandeis University in Waltham, Massachusetts. The autocatalytic positive feedback in the CIMA reaction is mainly controlled by iodide ions. But the reaction uses starch as a colour indicator for iodine formed in the reaction, and iodide can become bound to iodine and starch, forming a large complex that diffuses slowly through the gel. This slowing down of the diffusion of the 'activator' is what brings the reaction dynamics into the regime for Turing patterns to form. De Kepper happily admits that this turned out to be a stroke of pure luck. But as his compatriot Louis Pasteur famously averred, such good fortune tends to be transformed into discovery only if the mind is prepared for it. Thanks to Boissonade's calculations, says De Kepper, "we were ready to understand what we saw".

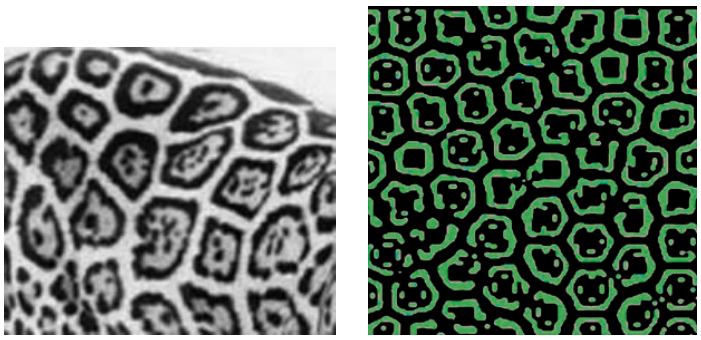
Making chemical Turing structures, as well as other related stationary patterns, has now been refined to a well developed art. In 2009 De Kepper and his colleagues Judit Horváth and István Szalai described a general method for creating Turing structures in oscillatory reaction-diffusion systems, which again involves complexing agents that bind to the positive-feedback agents and slow them down. This has led to the discovery of several new pattern-forming systems, some Turing-like and others not.

In the wild

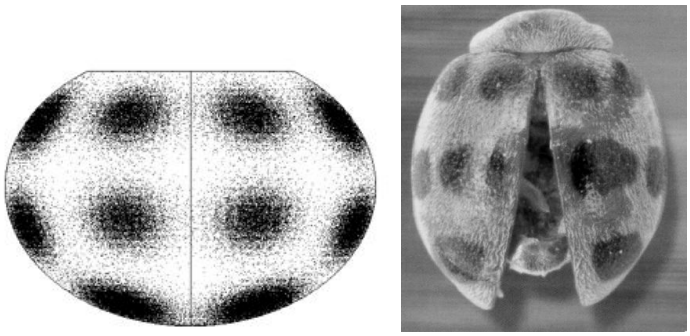
Growing Turing's spots and stripes in the lab is one thing – but are they really found in biology? In the 1980s, Meinhardt and mathematical biologist James Murray at the University of Washington in Seattle worked independently to show that Turing's theory offered a plausible explanation for a wide range of animal pigment patterns, from zebras to giraffes to seashells. The idea here is that the morphogens turn on or off genetic pathways that stimulate the production of pigments – in mammal skins, the pigment melanin, which generates colours from tawny to black.

More recently, Philip Maini and his colleagues have shown that two coupled activator-inhibitor processes can produce the broken ring markings characteristic of jaguars, while Maini's collaborator Sy-Sang Liaw at the National

Chung-Hsing University in Taiwan has demonstrated that a Turing scheme implemented on the curved shells of ladybirds can produce patterns looking very such like those seen in nature.

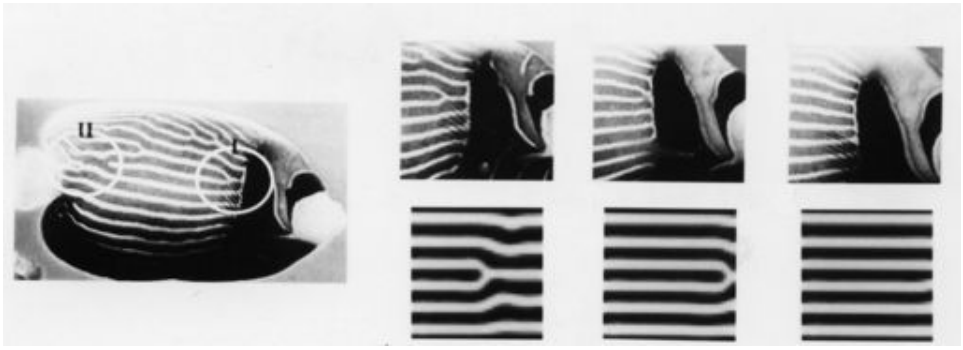


The 'rosette' spots of a jaguar, and analogous patterns produced by two coupled activator-inhibitor processes. Images: courtesy of Philip Maini, University of Oxford.



The markings on ladybirds (here *Epilachna crassimala*) may be the result of a Turing-type mechanism confined to the insect's roughly hemispherical shell. Images: courtesy of Sy-Sang Liaw, National Chung-Hsing University, Taiwan.

One of the most persuasive examples of a potential Turing pattern in animal markings was described by Shigeru Kondo and Rihito Asai at Kyoto University in Japan in 1995. They looked at the stripes of the angelfish, which are unusual in that they continue to grow and develop as the fish grow, rather than just being laid down during embryogenesis and then getting blown up like markings on a balloon. Kondo and Asai showed that the detailed changes in these patterns, such as a characteristic 'unzipping' of two merging stripes, is perfectly mimicked by a Turing model.



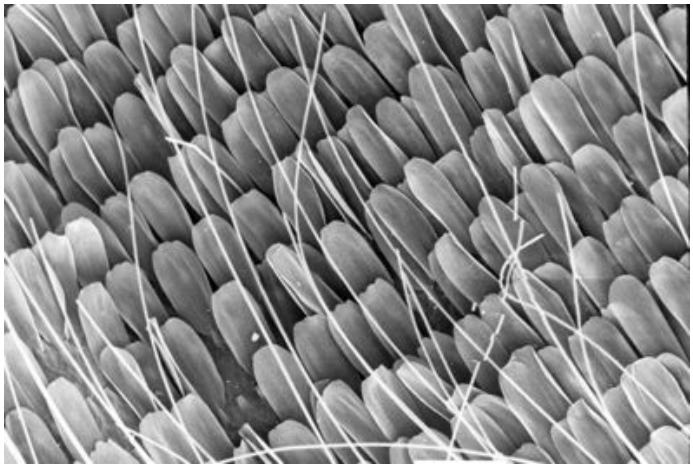
The evolution of stripes on an angelfish (top) is mimicked by changes in a theoretical Turing scheme (bottom). Images: from Kondo & Asai (1995).

However, none of this represents clinching proof that animal pigment patterns are indeed Turing patterns. To show that, one would have to identify the morphogens involved. Although no one has succeeded in doing so for animal markings, there are other types of biological pattern for which we do seem to be closing in on the likely biochemical agents underlying the process.

When you get goose bumps, you can see that your hair follicles are arranged with roughly even separation, resembling Turing's spots. In 2006 Thomas Schlake and his coworkers at the University of Freiburg in Germany found evidence that, at least in mice, these follicles are positioned by a process of activation and inhibition. They proposed that a protein called Wnt is the activator of follicle formation, while a class of proteins known generically as Dkk acts as inhibitors of Wnt. Schlake and colleagues showed that genetic mutant mice that produce Dkk proteins in abnormally high amounts develop follicle patterns that match those predicted theoretically from Turing-style activator-inhibitor models of the diffusion and interaction of Wnt and Dkk.

Meinhardt agrees that there is an activator and an inhibitor at work here, but disagrees about their identity. "The inhibitor has to be produced at the same place as the activator", he says. "Dkk does not satisfy this condition." He thinks that Wnt proteins play both roles at different times. As first secreted from cells, they are self-activating. But they eventually become bound to cell-membrane lipids and get reprocessed into inhibitors with a longer range. Dkk, he thinks, merely modifies this inhibition as a secondary effect. He believes that the diverse family of Wnt-type proteins can produce a range of different patterning mechanisms, and has recently argued that one such explains the formation of tentacles around the cylindrical gastric column of the hydra, a very primitive freshwater animal. This process was mentioned explicitly by Turing himself as an example of biological symmetry-breaking that his mechanism might account for.

Something analogous to the patterning of hair follicles may apply to the arrangement of feathers in birds and the scales of lizards and butterfly wings. As cells in the embryonic chick begin to differentiate to form the specialized structures and tissues that make feathers, they send out diffusing molecular signals to the cells nearby that suppress such differentiation, ensuring that two such patches do not develop close together. So there is local activation in the sense that cells can become spontaneously committed to differentiation into feather-making apparatus, and longer-ranged inhibition of the same thing happening in the vicinity.



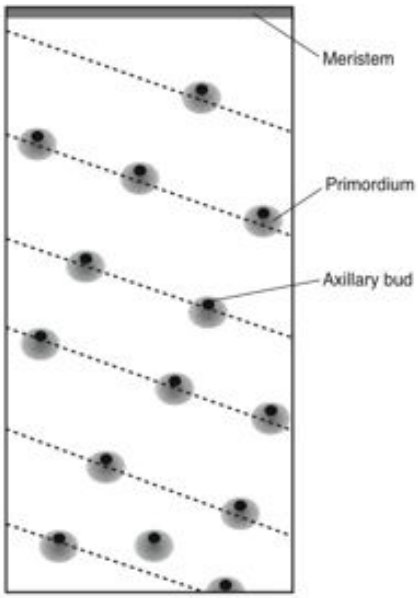
The regular positioning of butterfly wing scales might depend on a Turing-type process.

Meinhardt has collaborated with ornithologist Richard Prum at Yale to show that the periodic barbs of bird feathers can be explained if the protein product of a gene called *Sonic hedgehog* (Shh) – a common patterning gene in many species – behaves as an activator while the bone morphogenic protein 2 is an inhibitor. Through the interaction of these components, the uniform epithelium of the developing feather bud becomes divided into a series of stripe-like ridges that prefigure its break-up into distinct barbs. And very recently, developmental biologist Jeremy Green of King's College London, with collaborators that include Shigeru Kondo, has shown that the regularly spaced ridges of the mammalian palette seem to be arranged by a Turing-type reaction-diffusion mechanism involving the proteins Shh and fibroblast growth factor as the inhibitor and activator respectively.

Blossoming theory

Was Turing right to suspect that phyllotaxis can be explained this way too? In the 1930s it was found that applying the plant hormone auxin to the stems of lupins initiated the growth of new leaf buds. Could auxin be an activator of budding? That possibility was confirmed in 2003 when a team of European biologists showed that phyllotaxis is regulated by proteins that ferry auxin through the outer 'skin' of the stem up towards its apex. They found that existing leaf buds (primordia) soak up auxin and thus act as sinks, inhibiting the formation of any new buds nearby.

But why the Fibonacci spirals? Hans Meinhardt and his colleague André Koch showed in the 1994 that a combination of two activator-inhibitor processes could generate these. They found that the primordia became positioned along a cylindrical stem in a (2,3) spiral phyllotaxis pattern. Whether the model can generate higher-order Fibonacci spirals is not clear, however, and nor is there yet any evidence that such a double activator-inhibitor process really operates in plants.

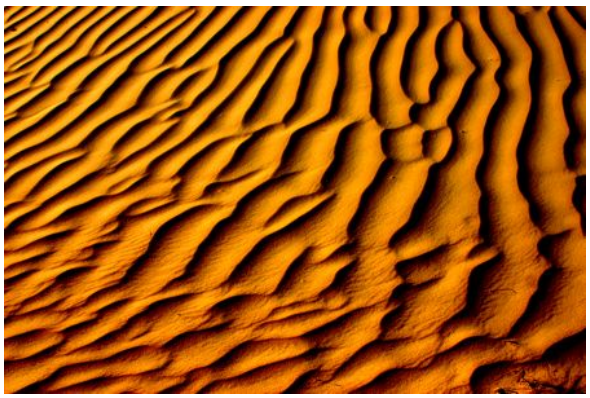


Spiral phyllotaxis on a plant stem, shown here as a rolled-out cylinder, is generated in the activator-inhibitor model of Meinhardt and Koch. Image: redrawn after Koch & Meinhardt & Koch (1994).

Meinhardt says that there's still much to be done in understanding how activation and inhibition governs phyllotaxis. "It is clear that auxin is involved in almost everything, but it is not clear how", he says. "Auxin accumulation, pumped up a concentration gradient by proteins, is self-enhancing, while its consequent depletion from the source regions is presumably the long-ranging inhibition. But how all cells pump in the same direction is, at least for me, still a mystery".

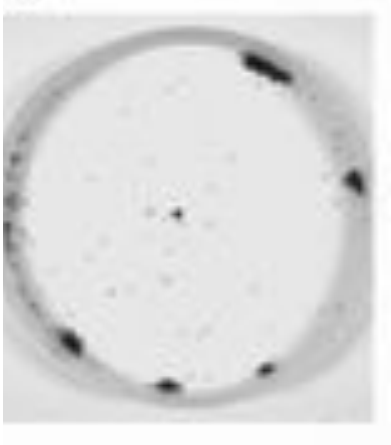
Sand, cemeteries and crime

The generic Turing stripes resemble ripple patterns in wind-blown sand. This may be no coincidence: Meinhardt suggests that, at root, the formation of these sand patterns is akin to an activator-inhibitor system. The mounds and ridges of sand are formed by deposition of wind-blown grains. As a ridge gets bigger, it enhances its own growth by capturing more sand from the air. But in doing so, it acts as a sink, removing sand from the wind and suppressing the formation of other ripples nearby. The balance between these two processes establishes a roughly constant mean distance between ripples.



It's no coincidence that sand ripples resemble the zebra's stripes…

The feedbacks involved in replication, competition and predation might set up Turing-type patterns in animal communities. Spanish physicist Ricard Solé and his coworkers José Vilar and Miguel Rubí think that this might account for the patchiness of zooplankton in the sea and the phytoplankton on which they graze. Meanwhile, Guy Theraulaz of the Université Paul Sabatier in Toulouse and his coworkers have found Turing structures in another animal community: ants. Mediterranean *Messor sancta* ants collect the bodies of expired colony members and arrange them in piles, which Theraulaz and colleagues showed to be analogous to Turing patterns. Although the ants constantly pick up and redistribute the corpses (producing a kind of diffusion), the pattern itself stays fixed. There is activation involved, because ants are more likely to drop a body on a pile as the pile gets larger. And there is long-ranged inhibition, because the region surrounding a big pile gets swept clear of bodies, making it less likely for a new one to be started there. The French researchers suspect that mechanisms like this might underlie many other aspects of habitat formation and grouping, such as nest construction, in higher organisms.



'Ant cemeteries', here arranged in one dimension around the edge of a Petri dish, are a kind of Turing pattern. Image: from Theraulaz *et al*. (2002).

Perhaps even human communities, orchestrated by social feedbacks on behaviour and movement, organize themselves into Turing patterns. That's the implication of a theory developed by mathematician Martin Short at the University of California at Los Angeles and his colleagues to explain the well attested phenomenon of crime hotspots: districts in which the crime rate is anomalously high. They imagine criminal offenders as predators who seek 'prey' (victims) while both move around (diffuse) in the available space. The 'reaction' – predation of criminals on victims – can be potentially suppressed by an inhibiting agency such as a security measure or a police force.

Short's mathematical model produces two types of hotspots due to the competition of activation and inhibition. The first are merely aggregates of individual crimes with overlapping spheres of influence. But the second type of hotspot is caused more directly by positive feedback: crime induces more crime. The first sort of hotspot can be eradicated completely by a sufficiently strong inhibiting influence: that is, by locally concentrated policing. But the second sort is harder to eliminate: focused inhibition may merely cause the hotspots to move or mutate, breaking up into smaller spots or rings in the close vicinity. If this

picture is an accurate reflection of the world, it suggests that not all hotspots will yield to the same style of policing, but that different strategies might be needed in different situations.

It's hard to guess where Turing's patterns might turn up next. The idea that he hatched 60 years ago, as it were literally from a fertilized egg, has proved astonishingly fertile, since it turns out to be one of nature's universal pattern-forming strategies.

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