A Revised and Expanded Edition of *A New Science of Life*

MORPHIC RESONANCE

THE NATURE OF FORMATIVE CAUSATION

"Rupert Sheldrake's contributions will be recognized one day on the same level as those of Newton and Darwin."
DEEPAK CHOPRA

RUPERT SHELDRAKE

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To Dom Bede Griffiths, O.S.B.

MORPHIC RESONANCE

"Rupert Sheldrake is one of the most innovative and visionary scientists of our times. Rupert will be both vilified and praised for his theory of morphic resonance. Whatever your personal opinion of his work, he will not be ignored. In my opinion, his contributions will be recognized one day on the same level as those of Newton and Darwin."

DEEPAK CHOPRA, *AUTHOR OF REINVENTING THE BODY, RESURRECTING THE SOUL*

"*Morphic Resonance* presents a revolutionary information-field understanding of the nature and evolution of life. Acquaintance with it is an essential part of new-paradigm scientific literacy."

ERVIN LASZLO, AUTHOR OF *SCIENCE AND THE AKASHIC FIELD*

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PREFACE TO THE 2009 EDITION

This book is about the hypothesis of formative causation, which proposes that nature is habitual. All animals and plants draw upon and contribute to a collective memory of their species. Crystals and molecules also follow the habits of their kind. Cosmic evolution involves an interplay of habit and creativity.

This hypothesis is radically different from the conventional assumption that nature is governed by eternal laws. But I believe that the idea of the habits of nature will have to be considered sooner or later, whether we like it or not, because modern cosmology has undermined the traditional assumptions on which science was based.

Until the 1960s, most physicists took it for granted that the universe was eternal, governed by changeless laws and made up of a constant amount of matter and energy. This idea of the Laws of Nature has been fundamental in modern science ever since the scientific revolution of the seventeenth century, and is rooted in the Pythagorean and Platonic philosophies of ancient Greece. The patriarch of modern science, Sir Francis Bacon, asserted in 1620 that the Laws of Nature were "eternal and immutable"[1] and science's founding fathers, including Kepler, Galileo, Descartes, and Newton, saw them as immaterial mathematical ideas in the mind of God. The Laws of Nature were eternal because they participated in God's eternal nature, and like God transcended time and space. They were enforced by God's omnipotence.

When the entire universe was believed to be eternal, made up of a constant amount of matter and energy, eternal laws presented no problems. In the nineteenth and early twentieth centuries, most physicists believed that all fundamental aspects of physics were fixed forever— the total amount of matter, energy, and electric charge was always the same, according to the laws of conservation of mass, energy, and electric charge.

Only the second law of thermodynamics sounded a different note. The total amount of entropy would increase until the entire universe froze up forever—a state epitomized in 1852 by William Thomson, later Lord Kelvin, as "a state of universal rest and death."[2] But although heat death would ensue when entropy reached a maximum, the frozen universe would still endure forever and so would the Laws of Nature.

Everything changed with the great revolution in cosmology in the 1960s, when the big bang theory became the new orthodoxy. Ever since, most cosmologists have believed that the universe began about 15 billion years ago. When everything first appeared from nowhere—there was no space and time before the cosmos—it was less than the size of the head of a pin and immensely dense and hot. The cosmos has been expanding and cooling ever since. All atoms, molecules, stars, galaxies, crystals, planets, and forms of life have come into being in time. They have evolutionary histories. The universe now looks like a vast developing organism, not like an eternal machine slowly running out of steam.

The big bang theory was first proposed in 1927, as the theory of the "primeval atom," by Father Georges Lemaître, a Roman Catholic priest and cosmologist. He suggested that the universe began with an initial "creation-like event," which he described as "the Cosmic Egg exploding at the moment of the creation."[3] His theory, which predicted the expansion of the universe, encountered much skepticism, but evidence for an initial "creation-like event" eventually became too persuasive to be ignored. One of this theory's opponents, the astronomer Fred Hoyle, disparagingly called it the big bang theory, and Hoyle's name has stuck.

Although cosmology is now evolutionary, old habits of thought die hard. Most scientists take eternal Laws of Nature for granted—not because they have thought about them in the context of the Big Bang, but because they haven't.

If the Laws of Nature are Pythagorean mathematical truths, or Platonic Ideas, or ideas in the mind of God, they transcend time and space. They would necessarily be present when the universe was born: the Laws do not come into being or pass away; they transcend space and time.

Clearly, this is a philosophical or theological doctrine rather than a scientific hypothesis. It could not possibly be tested experimentally before

there was a universe to test it in.

To avoid the doctrine of transcendent laws, we could suppose that the Laws of Nature came into being at the very moment of the big bang. This theory avoids an explicit Platonic philosophy or theology, but it creates new problems. As Terence McKenna observed, “Modern science is based on the principle: ‘Give us one free miracle and we’ll explain the rest.’ The one free miracle is the appearance of all the mass and energy in the universe and all the laws that govern it in a single instant from nothing.”[4]

The sudden appearance of all the Laws of Nature is as untestable as Platonic metaphysics or theology. Why should we assume that all the Laws of Nature were already present at the instant of the Big Bang, like a cosmic Napoleonic code? Perhaps some of them, such as those that govern protein crystals, or brains, came into being when protein crystals or brains first arose. The preexistence of these laws cannot possibly be tested before the emergence of the phenomena they govern.

Besides all these problems, as soon as we think about the Laws of Nature, we cannot help seeing that this concept is anthropocentric. Only human beings have laws, and not even all humans. Only civilized societies have laws; traditional societies have customs. Applying the concept of law to the universe involves the metaphor of God as a kind of universal emperor whose writ runs everywhere and always. This assumption was readily accepted by the founding fathers of modern science, who believed in a mathematically minded, omnipotent God. But the Laws of Nature now float in a metaphysical void.

Evolutionary cosmology makes eternal Laws of Nature yet more problematical. Perhaps the laws of nature are not all fixed forever, but evolve along with nature. New laws may arise as phenomena become more complex. And as soon as we admit this possibility, we realize that the metaphorical source of the Laws of Nature, namely human laws, are not in fact eternal but evolve along with society. The laws of the United States, or Kenya, or Bhutan are not the same today as they were one hundred years ago, or even twenty years ago. They are continually changed and updated. But there is no parallel in nature for monarchs or parliaments or congresses. The legal metaphor is incoherent.[5]

I suggest a new possibility. The regularities of nature are not imposed on nature from a transcendent realm, but evolve within the universe. What happens depends on what has happened before. Memory is inherent in nature. It is transmitted by a process called morphic resonance, and works through fields called morphic fields.

In this book, I discuss the hypothesis of formative causation primarily in the context of biology and chemistry. In my book *The Presence of the Past*[6] I extend this discussion to psychological and cultural evolution.

This new edition

The first edition of this book was published in 1981. It proved controversial, as described below. In the second edition (1985) I summarized these controversies, along with the results of some early experimental tests of the hypothesis. Much has happened since. In this new edition, I have revised and updated the book throughout. I summarize the results of research so far in appendix A, where I discuss ten new tests. Appendix B consists of a dialogue with the physicist David Bohm in which we explored connections between formative causation and quantum physics.

The remarkable developments in biology over the last quarter of a century have made the limitations of the conventional mechanistic approach more obvious, and have increased the plausibility of the hypothesis of formative causation.

How mechanistic biology has revealed its own limitations

In the 1980s, the mechanistic theory of life seemed set for ultimate triumph. The neo-Darwinian theory of evolution had eliminated God from nature, and life itself was about to be explained in terms of physics and chemistry, with no need for any mysterious fields or factors. Many scientists believed that molecular biology was on the verge of revealing the secrets of life through an understanding of the genetic code and the control of protein synthesis. Meanwhile, brain-scanning techniques were about to unveil the mechanistic workings of the mind. The Decade of the Brain, inaugurated in 1990 by President George Bush Sr., led to further acceleration in the growth

of the neurosciences, and stimulated yet more optimism about the power of brain-scanning to probe our innermost being.[7]

Meanwhile, an enthusiasm for artificial intelligence led to the expectation that a new generation of computers would soon be able to rival, or even exceed, the mental abilities of human beings. If intelligence, and even consciousness itself, could be programmed into machines, then the final mysteries would be solved. Life and mind would be fully explicable in terms of molecular and neural machinery. Reductionism would be vindicated. All those who thought that minds involved something beyond the reach of mechanistic science would be refuted forever. But this has not happened.

It is hard to recall the atmosphere of exhilaration in the 1980s as new techniques enabled genes to be cloned and the sequence of “letters” in the “genetic code” to be discovered. This seemed like biology’s crowning moment: the instructions of life itself were finally laid bare, opening up the possibility for biologists to modify plants and animals genetically, and to grow richer than they could ever have imagined. There was a continuous stream of new discoveries; almost every week newspaper headlines reported some new “breakthrough”: “Scientists find genes to combat cancer,” “Gene therapy offers hope to victims of arthritis,” “Scientists find secret of ageing,” and so on.

The new genetics seemed so promising that soon the entire spectrum of biological researchers was busy applying its techniques to each specialty. Their remarkable progress led to a vast, ambitious vision: to spell out the full complement of genes in the human genome. As Walter Gilbert of Harvard University put it, “The search for this ‘Holy Grail’ of who we are has now reached its culminating phase. The ultimate goal is the acquisition of all the details of our genome.” The Human Genome Project was formally launched in 1990 with a projected budget of $3 billion.

The Human Genome Project was a deliberate attempt to bring “Big Science” to biology, which had previously been more like a cottage industry. Physicists were used to huge budgets, partly as a result of the Cold War: there was enormous expenditure on missiles and hydrogen bombs, Star Wars, multibillion-dollar particle accelerators, the space program, and the Hubble Space Telescope. For years, ambitious biologists suffered from physics envy. They dreamed of the days when biology would also have

high-profile, high-prestige, multibillion-dollar projects. The Human Genome Project was the answer.

At the same time, a tide of market speculation in the 1990s led to a boom in biotechnology, reaching a peak in 2000. In addition to the official Human Genome Project, there was a private genome project carried out by Celera Genomics, headed by Craig Venter. The company's plan was to patent hundreds of human genes and own the commercial rights to them. Its market value, like that of many other biotechnology companies, rocketed to dizzying heights in the early months of 2000.

Ironically, the rivalry between the publicly funded Human Genome Project and Celera Genomics led to a bursting of the biotechnology bubble before the sequencing of the genome had even been completed. In March 2000 the leaders of the public genome project publicized the fact that all their information would be freely available to everyone. This led to a statement by President Clinton on 14 March 2000: "Our genome, the book in which all human life is written, belongs to every member of the human race . . . We must ensure that the profits of the human genome research are measured not in dollars, but in the betterment of human life."[8] The press reported that the president planned to restrict genomic patents, and the stock markets reacted dramatically. In Venter's words, there was a "sickening slump." Within two days, Celera's valuation lost $6 billion, and the market in biotechnology shares collapsed by a staggering $500 billion.[9]

In response to this crisis, a day after his speech President Clinton issued a correction saying that his statement had not been intended to have any effect on the patentability of genes or the biotechnology industry. But the damage was done. The stock market valuations never recovered. And although many human genes were subsequently patented, very few proved profitable to the companies that owned them.[10]

On 26 June 2000, President Clinton and the British prime minister, Tony Blair, together with Craig Venter and Francis Collins, the head of the official genome project, announced the publication of the first draft of the human genome. At a press conference in the White House, President Clinton said, "We are here today to celebrate the completion of the first survey of the entire human genome. Without a doubt this is the most important, most wondrous map ever produced by mankind."

This astonishing achievement has indeed transformed our view of ourselves, but not in the way that was anticipated. The first surprise was that there were so few genes. Rather than the predicted 100,000 or more, the final tally of about 25,000 was very puzzling, and all the more so when compared with the genomes of other animals much simpler than ourselves. There are about 17,000 genes in a fruit fly and about 26,000 in a sea urchin. Many species of plants have far more genes than we do—rice has about 38,000, for example.

In 2001, the director of the chimpanzee genome project, Svante Paabo, anticipated that when the sequencing of the ape's genome was completed, it would be possible to identify "the profoundly interesting genetic prerequisites that make us different from other animals." When the complete chimpanzee sequence was published four years later, his interpretation was more muted: "We cannot see in this why we are so different from chimpanzees."[11]

In the wake of the Human Genome Project, the mood has changed dramatically. The old assumption that life would be understood if molecular biologists knew the "program" of an organism is giving way to a realization that there is a huge gap between gene sequences and the way living organisms grow and behave. The present book sketches out a means of bridging that gap.

Meanwhile, the optimism of stock market investors has suffered a further series of blows. After the biotech bubble burst in 2000, many companies that were part of the biotechnology boom of the 1990s either went out of business or were taken over by pharmaceutical or chemical corporations. Several years later the economic outcomes were still disappointing. An article in the *Wall Street Journal* in 2004 was entitled "Biotech's Dismal Bottom Line: More than $40 Billion in Losses."[12] It went on to say, "Biotechnology . . . may yet turn into an engine for economic growth and cure deadly diseases. But it's hard to argue that it's a good investment. Not only has the biotech industry yielded negative financial returns for decades, it generally digs its hole deeper every year."

Despite its disappointing business record, this vast investment in molecular biology and biotechnology has had wide-ranging effects on the practice of biology, if only by creating so many jobs. The enormous

demand for graduates in molecular biology and for people with doctorates in this subject has transformed the teaching of biology. The molecular approach now predominates in universities and secondary schools. Meanwhile, leading scientific journals such as *Nature* are replete with glossy full-page advertisements for gene-sequencing machines, protein-analysis systems, and equipment for cloning cells.

Precisely because there has been such a strong emphasis on the molecular approach, its limitations are becoming increasingly apparent. The sequencing of the genomes of ever more species of animals and plants, together with the determination of the structures of thousands of proteins, is causing molecular biologists to drown in their own data. There is practically no limit to how many more genomes they could sequence or proteins they could analyze. Molecular biologists now rely on computer specialists in the rapidly growing field of bioinformatics to store and try to make sense of this unprecedented quantity of information, sometimes called the "data avalanche."[13] But in spite of all this information, the way in which developing organisms take up their forms and inherit their instincts remains mysterious.

The evolution of development

In the 1980s, there was great excitement when a family of genes called homeobox genes was discovered in fruit flies. Homeobox genes determine where limbs and other body segments will form in a developing embryo or larva; they seem to control the pattern in which different parts of the body develop. Mutations in these genes can lead to the growth of extra, nonfunctional body parts.[14] At first sight, they appeared to provide the basis for a molecular explanation of morphogenesis, the coming into being of specific forms: here were the key switches. At the molecular level, homeobox genes act as templates for proteins that "switch on" cascades of other genes.

However, research on other species soon revealed that these molecular control systems are very similar in widely different animals. Homeobox genes are almost identical in flies, reptiles, mice, and humans. Although they play a role in the determination of the body plan, they cannot themselves explain the shape of the organisms. Since the genes are so

similar in fruit flies and in us, they cannot explain the differences between flies and humans.

It was shocking to find that the diversity of body plans across many different animal groups was not reflected in diversity at the level of the genes. As two leading developmental molecular biologists have commented, “Where we most expect to find variation, we find conservation, a lack of change.”[15]

This study of genes involved in the regulation of development is part of a growing field called evolutionary developmental biology, or “evo-devo” for short. Once again, the triumphs of molecular biology have shown that morphogenesis itself continues to elude a molecular explanation, but seems to depend on fields. That is why the idea of morphogenetic fields, discussed in this book, is more relevant than ever.

Epigenetics

Throughout the twentieth century, one of the strongest taboos in biology was against the inheritance of acquired characteristics, sometimes called Lamarckian inheritance, after the pioneering evolutionary biologist Jean-Baptiste Lamarck (1744–1829). Lamarck proposed that adaptations by plants and animals could be passed on to their offspring. In this respect, Charles Darwin was a convinced Lamarckian. He believed that habits acquired by individual animals could be inherited, and played an important part in evolution: “We need not . . . doubt that under nature new races and new species would become adapted to widely different climates, by variation, aided by habit, and regulated by natural selection.”[16] In this sense, the inheritance of habits by morphic resonance is in good accordance with Darwinism, as opposed to neo-Darwinism. Darwin provided many examples of the inheritance of acquired characters in his book *The Variation of Animals and Plants Under Domestication*, and also proposed a theory to explain it, the theory of pangenesis.

Modern neo-Darwinism was established in the 1940s, and firmly rejected the Lamarckian aspect of Darwin’s theory. Neo-Darwinians asserted that genes were passed on without modification from parents to offspring, apart from rare random mutations. Any kind of Lamarckian modification of the genes was impossible. By contrast, in the Soviet Union under Stalin, the

inheritance of acquired characteristics became official doctrine under Trofim Lysenko. The debate degenerated into polemics and denunciations, and in the West the taboo against the inheritance of acquired characteristics was reinforced.

In his rejection of Lamarckism, Richard Dawkins, the leading modern exponent of neo-Darwinism, is clear about his feelings: “To be painfully honest, I can think of few things that would more devastate my world view than a demonstrated need to return to the theory of evolution that is traditionally attributed to Lamarck.”[17]

Evidence in favor of the inheritance of acquired characteristics continued to accumulate throughout the twentieth century, but was generally ignored. However, soon after the turn of the millennium, the taboo began to lose its power with a growing recognition of a new form of inheritance, called epigenetic inheritance. The prefix *epi* means “over and above.” Epigenetic inheritance does not involve changes in the genes themselves, but rather changes in gene expression. Characteristics acquired by parents can indeed be passed on to their offspring. For example, water fleas of the genus *Daphnia* develop large protective spines when predators are around; their offspring also have these spines, even when not exposed to predators.[18]

Several molecular mechanisms of epigenetic inheritance have been identified. Changes in the configuration of the chromatin—the DNA-protein complex that makes up the structure of chromosomes—can be passed on from cell to daughter cell. Some such changes can also be passed on through eggs and sperm, and thus become hereditary. Another kind of epigenetic change, sometimes called genomic imprinting, involves the methylation of DNA molecules. There is a heritable chemical change in the DNA itself, but the underlying genes remain the same.

Epigenetic inheritance also occurs in humans. Even the effects of famines and diseases can echo down the generations. The Human Epigenome Project was launched in 2003, and is helping to coordinate research in this rapidly growing field of inquiry.[19]

Morphic resonance provides another means by which the inheritance of acquired characteristics can occur. Its effects can be distinguished experimentally from other forms of epigenetic inheritance, as discussed in chapter 7 and appendix A.

Morphogenetic and morphic fields

In this book I discuss morphogenetic fields, the organizing fields of molecules, crystals, cells, tissues, and indeed all biological systems. I also discuss the organizing fields of animal behavior and of social groups. Whereas morphogenetic fields influence form, *behavioral fields* influence behavior. The organizing fields of social groups, such as flocks of birds, schools of fish, and colonies of termites, are called *social fields*. All these kinds of fields are *morphic fields*. All morphic fields have an inherent memory given by morphic resonance. Morphogenetic fields, the organizing fields of morphogenesis, are one kind of the larger category of morphic fields, rather like a species within a genus. In *The Presence of the Past*,[20] I explore the wider nature of morphic fields in their behavioral, social, and cultural contexts, and their implications for the understanding of animal and human memory. I also suggest that our own memories depend on morphic resonance rather than on material memory traces stored in our brains.

The relationship of morphic fields to modern physics

One of the paradoxes of twentieth-century science was that quantum theory ushered in a revolutionary change of perspective in physics revealing the limits of a reductionistic approach, while biology moved in the opposite direction, away from holistic approaches to an extreme reductionism. As the German quantum physicist Hans-Peter Dürr expressed it:

> The original emphasis on the whole in consideration of living things, their shapes and Gestalts, has been replaced by a fragmenting, functionalist description, in which, for an explanation of the sequences of events, the focus is on the substances, matter, and its building blocks, the molecules and their interactions. The surprising thing about this development from holism and even vitalism to molecular biology is that it is occurring some decades after—and not before— a profound change in just the opposite direction took place at the foundations of natural science, in microphysics, during the first third of the century that has recently ended. There, fundamental limitations of the fragmenting, reductionist way of looking at things had become

> apparent. Divisible substance revealed in a strange way holistic aspects.[21]

Many biologists are still trying to reduce the phenomena of life and mind to the mechanistic physics of the nineteenth century, but physics has moved on. And quantum physics provides a far more promising context for morphic fields than anything in classical physics. Morphic fields must in some way interact directly or indirectly with electromagnetic and quantum fields, imposing patterns on their otherwise indeterminate activities. But exactly how this interaction occurs remains unclear. One possible starting point is the idea of the implicate order, proposed by the quantum physicist David Bohm.

> In the enfolded or implicate order, space and time are no longer the dominant factors determining the relationships of dependence or independence of different elements. Rather, an entirely different sort of basic connection of elements is possible, from which our ordinary notions of space and time, along with those of separately existent material particles, are abstracted as forms derived from the deeper order. These ordinary notions in fact appear in what is called the "explicate" or "unfolded" order, which is a special and distinguished form contained within the general totality of all the implicate orders.[22]

The implicate order involves a kind of memory that is expressed through quantum fields, and is compatible in general terms with the ideas put forward in this book. A discussion between David Bohm and myself about morphic resonance and the implicate order is reprinted in appendix B of this book. Hans-Peter Dürr has also discussed how "processes of quantum physics might in principle contain a fruitful potential for an explanation of Sheldrake's morphic fields."[23]

Another way in which morphic resonance and morphic fields might be related to modern physics is through extra dimensions of space-time. Although our commonsense thinking is confined to three dimensions of space and one of time, as in Newtonian physics, physics has moved on by adding further dimensions. In the theory of General Relativity, first put forward in 1915, Einstein treated space-time as four-dimensional. In the

1920s, in the Kaluza-Klein theory, space-time was extended to five dimensions in an attempt to find a unified theory for gravitational and electromagnetic fields. Modern hopes of unifying the known fields of physics, including the strong and weak nuclear forces, are mainly centered on superstring theory, with ten dimensions, or M-theory (short for master theory) with eleven.[24]

The value of superstring and M-theory is disputed, but their very existence shows that extra dimensions are no longer the preserve of esoteric speculations; they are mainstream in modern physics.[25] But what do these extra dimensions do, and what difference do they make? Some physicists propose that they include "information fields" that could help to explain the phenomena of life and mind.[26]

Another possible point of connection between morphic fields and modern physics is through the quantum vacuum field. According to standard quantum theory, all electrical and magnetic forces are mediated by virtual photons that appear from the quantum vacuum field and then disappear into it again. Thus all molecules within living organisms, all cell membranes, all nerve impulses, and indeed all electromagnetic and chemical processes depend on virtual photons appearing and disappearing within the all-pervading vacuum field of nature. Could morphic fields interact with regular physical and chemical processes through the vacuum field? Some theoreticians speculate that they can and do.[27]

Theories of these kinds may help to relate morphic fields and morphic resonance to the physics of the future. But at present no one knows how the phenomena of morphogenesis are related to physics, whether conventional or unconventional.

Experimental tests

The experimental tests for morphic resonance proposed in the first edition of this book were primarily in the realms of chemistry and biology. However, the greatest interest they stimulated was in the realm of human psychology. According to the hypothesis of morphic resonance, human beings draw upon a collective memory: something learned by people in one place should subsequently become easier for others to learn all over the world.

In 1982, the British magazine *New Scientist* ran a competition for ideas about testing this hypothesis. All the winning ideas were for psychological experiments. At the same time, an American think tank, the Tarrytown Group of New York, offered a $10,000 prize for the best test of this hypothesis. Again the winning entries were in the realm of psychology, and provided evidence that supported the morphic resonance hypothesis. These results were summarized in my book *The Presence of the Past*.

In appendix A, I summarize the results of more-recent morphic resonance research, and propose a range of new tests for morphic resonance in physics, chemistry, biology, psychology, and computer sciences.

A new way of doing science

Since the 1990s, much of my own experimental research has been concerned with the role that morphic fields play in social behavior in animals and people. My studies on unexplained aspects of animal and human behavior are summarized in my books *Seven Experiments That Could Change the World* (1994), *Dogs That Know When Their Owners Are Coming Home* (1999), and *The Sense of Being Stared At* (2003). These investigations were concerned primarily with the *spatial* aspects of morphic fields, rather than with morphic resonance, which gives these fields their temporal or historical aspect.

This research is radical in two senses: it proposes not only a new kind of scientific thinking, but also a new way of *doing* science. This is the main theme of *Seven Experiments That Could Change the World*. Many of the experiments to test for morphic fields are simple and inexpensive. They show that science need no longer be the monopoly of a scientific priesthood. Research at the frontiers of science is open to participation by students and by nonprofessionals.

Already thousands of nonprofessionals have contributed to this research through supplying case histories; through taking part in tests with their animals, such as dogs, cats, horses, and parrots; and through carrying out experiments with their families and friends, or with fellow students in schools, colleges, and universities. There have been dozens of student projects on topics related to morphic fields, including several that have won prizes in science fairs. Much of this research is summarized in *Dogs That*

Know When Their Owners Are Coming Home and *The Sense of Being Stared At*.

Meanwhile, any reader who would like to take part in my current experiments can do so through the Online Experiments Portal on my website, www.sheldrake.org. Some of these experiments are Internet based; others take place through mobile telephones. These tests work well as homework assignments in schools and colleges. They are fun to do, they illustrate the principles of statistics and controlled experimentation, and they make a valuable contribution to research in progress.

In the past, some of the most innovative scientific research was carried out by amateurs. Charles Darwin, for example, never held an institutional post. He worked independently at his home in Kent studying barnacles, keeping pigeons, and doing experiments in the garden with his children. He was just one of many independent researchers who, not reliant on grants or constrained by the conservative pressures of anonymous peer review, did highly original work. Today that kind of freedom is almost nonexistent. From the latter part of the nineteenth century onward, science has become increasingly professionalized. After the Second World War there was a vast expansion of institutional research. There are now only a handful of independent scientists, the best known being James Lovelock, the leading proponent of the Gaia hypothesis.

Nevertheless, the conditions for widespread participation in science have become more favorable than ever. There are hundreds of thousands of people all over the world who have had scientific training. Computing power, once the monopoly of large organizations, is widely available. The Internet gives access to information undreamed of in past decades, and provides an unprecedented means of communication. There are more people with leisure time than ever before. Every year thousands of students do scientific research projects as part of their training, and some would welcome the chance to be real pioneers. And many informal networks and associations already provide models for self-organizing communities of researchers, working both within and outside scientific institutions.

As in its most creative periods, science can once again be nourished from the grass roots up. Research can grow from a personal interest in the nature of nature, an interest that originally impels many people into scientific careers but is often smothered by the demands of institutional life.

Fortunately, an interest in nature burns as strongly, if not more strongly, in many people who are not professional scientists.

I believe that not only in relation to controversial frontier areas of research, but also in more conventional areas, science needs democratizing. It has always been elitist and undemocratic, whether in monarchies, communist states, or liberal democracies. But it is currently becoming more hierarchical, not less so, and this trend needs remedying.

Today, the kinds of research that can happen are determined by science funding committees, not the human imagination. What is more, the power in those committees is increasingly concentrated in the hands of politically adept older scientists, government officials, and representatives of big business. Young graduates on short-term contracts constitute a growing scientific underclass. In the United States, the proportion of biomedical grants awarded to investigators under thirty-five plummeted from 23 percent in 1980 to 4 percent in 2003. This is bad news. As science becomes more and more about climbing corporate career ladders and less and less about soaring journeys of the mind, so the public distrust of scientists and their work seems to grow.

In 2000, a government-sponsored survey in Britain on public attitudes to science revealed that most people believed that “science is driven by business—at the end of the day it’s all about money.” Over three-quarters of those surveyed agreed, “It is important to have some scientists who are not linked to business.” More than two-thirds thought, “Scientists should listen more to what ordinary people think.” Worried about this public alienation, in 2003 the British government said it wanted to engage the wider public in “a dialogue between science, policy makers, and the public.” In official circles, the fashion shifted from a “deficit” model of the public understanding of science—which sees simple factual education as the key—to an “engagement” model of science and society.

Public participation would involve more than setting up committees of nonscientists to advise the existing funding bodies. In 2003 in *New Scientist*[28] and in 2004 in *Nature*,[29] I proposed a more radical possibility, namely to set aside a small proportion of the public science budget, say 1 percent, for research proposed by laypeople.

What questions would be of public interest? Why not ask? Organizations such as charities, schools, local authorities, trade unions, environmental groups, and gardening associations could be invited to make suggestions. Within each organization, the very possibility of proposing research would probably trigger far-ranging discussions, and would lead to a sense of involvement in many sections of the population.

To avoid the 1 percent fund being taken over by the science establishment, it would need to be administered by a board largely composed of nonscientists, as in many research charities. Funding would be restricted to areas not already covered by the other 99 percent of the public science budget. This system could be treated as an experiment, and tried out for, say, five years. If it had no useful effects, it could be discontinued. If it led to productive research, greater public trust in science, and increased interest among students, the percentage allocated to this fund could be increased. I believe this new venture would make science more attractive to young people, stimulate interest in scientific thinking and hypothesis-testing, and help break down the depressing alienation many people feel from science.

Controversies

When *A New Science of Life* was first published in Britain, in 1981, there was a widespread discussion about the idea of morphogenetic fields and morphic resonance. After three months, a now notorious editorial appeared on the front page of *Nature*. Under the title “A book for burning?” the editor condemned my proposals in an extraordinary attack.

> Even bad books should not be burned; works such as *Mein Kampf* have become historical documents for those concerned with the pathology of politics. But what is to be made of Dr. Rupert Sheldrake’s book *A New Science of Life*? This infuriating tract has been widely hailed by newspapers and popular science magazines as the “answer” to materialistic science, and it is now well on the way to becoming a point of reference for the motley crew of creationists, anti-reductionists, neo-Lamarckians and the rest. The author, by training a biochemist and by demonstration a knowledgeable man, is, however,

> misguided. His book is the best candidate for burning there has been for many years.[30]

The editor did not advance any reasoned arguments against the hypothesis I proposed. Instead, he put his hope in the future advances of molecular biology.

> Sheldrake's argument takes off from his catalogue of the ways in which the molecular biologists, no doubt the shock-troops of the reductionists, have so far been unable to calculate the phenotype of the single organism from a knowledge of its genotype. But so what? Have not the past 20 years shown clearly enough that molecular explanations of most biological phenomena are, contrary to some earlier expectations, possible and powerful?

The editor, the late Sir John Maddox, also dismissed my proposals for experiments as "impractical in the sense that no self-respecting grant-making agency will take the proposal seriously."

This editorial was followed by correspondence in *Nature*, continuing for months, in which many scientists objected to the intemperate tone of this attack and supported the need for radical thinking about the unsolved problems of science.[31] One of the letters was from the quantum physicist Brian Josephson, a Nobel laureate.

> The rapid advances in molecular biology to which you refer do not mean very much. If one is on a journey, rapid progress on the way implies neither that one is close to one's destination, nor that the destination will be reached at all by continuing to follow the same road. By referring to "self-respecting grant-making agencies" you show a concern not for scientific validity but for respectability. The fundamental weakness is a failure to admit even the possibility that genuine physical facts may exist which lie outside the scope of current scientific descriptions. Indeed a new kind of understanding of nature is now emerging, with concepts like implicate order and subject-dependent reality (and now, perhaps formative causation). These developments have not yet penetrated to the leading journals. One can

> only hope that the editors will soon cease to obstruct this avenue of progress.[32]

In 1994, BBC television interviewed John Maddox about his outburst. He was unrepentant, saying, “Sheldrake is putting forward magic instead of science, and that can be condemned in exactly the language that the Pope used to condemn Galileo, and for the same reason. It is heresy.”[33] Perhaps he was unaware that two years earlier, on 15 July 1992, Pope John Paul II formally declared that the church had erred in condemning Galileo.

In the German-speaking countries, there were many articles and discussions of this hypothesis by scientists, philosophers, psychologists, and others. Some of their varied reactions were brought together in a book published in German in 1997 entitled *Rupert Sheldrake in der Diskussion*.[34]

In the 1980s and 1990s many people within the scientific community, like the editor of *Nature*, were confident that more research on gene sequences and molecular mechanisms would reveal almost all we need to know about life, explaining the mysteries of biological form, instinctive behavior, learning, and even consciousness itself. Several leading scientists believed that science was nearing its ultimate culmination; all the important discoveries had already been made. This mood was summed up in 1996 in John Horgan’s bestselling book *The End of Science: Facing the Limits of Knowledge in the Twilight of the Scientific Age*. As Horgan expressed it:

> If one believes in science, one must accept the possibility—even the probability—that the great era of scientific discovery is over. By science I mean not applied science, but science at its purest and grandest, the primordial human quest to understand the universe and our place in it. Further research may yield no more great revelations or revolutions, but only incremental, diminishing returns.[35]

Fortunately, science has not come to an end despite the sequencing of the human genome, the avalanche of data in molecular biology, the boom in brain scanning, the speculations of superstring theorists, and the discovery that more than 90 percent of the universe is made up of dark matter and dark energy, whose nature is literally obscure.

The unsolved problems of biology summarized in chapter 1 were unsolved in 1981, and they are still unsolved today. The questions discussed in this book remain completely open. The debate continues; and by reading this book, you can be part of it.

INTRODUCTION

At present, the orthodox approach to biology is given by the mechanistic theory of life: living organisms are regarded as physico-chemical machines, and all the phenomena of life are considered to be explicable in principle in terms of physics and chemistry.[1] This mechanistic paradigm[2] is by no means new; it has been predominant for well over a century. The main reason most biologists continue to adhere to it is that it works: it provides a framework of thought within which questions about the physico-chemical mechanisms of life processes can be asked and answered.

The fact that this approach has resulted in spectacular successes such as the “cracking of the genetic code” is a strong argument in its favor. Nevertheless, critics have put forward what seem to be good reasons for doubting that all the phenomena of life, including human behavior, can ever be explained entirely mechanistically.[3] But even if the mechanistic approach were admitted to be severely limited not only in practice but in principle, it could not simply be abandoned; at present it is almost the only approach available to experimental biology, and will undoubtedly continue to be followed until there is some positive alternative.

Any new theory capable of extending or going beyond the mechanistic theory will have to do more than assert that life involves qualities or factors at present unrecognized by the physical sciences: it will have to say what sorts of things these qualities or factors are, how they work, and what relationship they have to known physical and chemical processes.

The simplest way in which the mechanistic theory could be modified would be to suppose that the phenomena of life depend on a new type of causal factor, unknown to the physical sciences, which interacts with physico-chemical processes within living organisms. Several versions of this vitalist theory were proposed in the early twentieth century,[4] but none succeeded in making predictions that could be tested, or suggested new

kinds of experiments. If, to quote Sir Karl Popper, “the criterion of the scientific status of a theory is its falsifiability, or refutability, or testability,”[5] vitalism failed to qualify.

However, the organismic or holistic philosophy of nature provides a context for a more radical revision of the mechanistic theory. This philosophy denies that everything in the universe can be explained from the bottom up, as it were, in terms of the properties of subatomic particles, or atoms, or even molecules. Rather, it recognizes the existence of hierarchically organized systems that, at each level of complexity, possess properties that cannot be fully understood in terms of the properties exhibited by their parts in isolation from each other; at each level the whole is more than the sum of its parts. These wholes can be thought of as organisms, using this term in a deliberately wide sense to include not only animals and plants, organs, tissues, and cells, but also crystals, molecules, atoms, and subatomic particles. In effect this philosophy proposes a change from the paradigm of the machine to the paradigm of the organism in the biological *and* in the physical sciences. In Alfred North Whitehead’s well-known phrase: “Biology is the study of the larger organisms, whereas physics is the study of the smaller organisms.”[6]

Various versions of this organismic philosophy have been advocated by many writers, including biologists, since the 1920s.[7] But if organicism is to have more than a superficial influence on the natural sciences, it must be able to give rise to testable predictions.[8]

The most important organismic concept put forward so far is that of *morphogenetic fields*.[9] These fields are supposed to help account for, or describe, the coming-into-being of the characteristic forms of embryos and other developing systems. The trouble is that this concept has been used ambiguously. The term itself seems to imply the existence of a new type of physical field that plays a role in the development of form. But some organismic theoreticians deny that they are suggesting the existence of any new type of field, entity, or factor at present unrecognized by physics;[10] rather, they are providing a new way of *talking about* complex physico-chemical systems.[11] This approach seems unlikely to lead very far. The concept of morphogenetic fields can be of practical scientific value only if it leads to testable predictions that differ from those of the conventional

mechanistic theory. And such predictions cannot be made unless morphogenetic fields are considered to have measurable effects.

The hypothesis put forward in this book is based on the idea that morphogenetic fields do indeed have measurable physical effects. It proposes that specific morphogenetic fields are responsible for the characteristic form and organization of systems at all levels of complexity, not only in the realm of biology, but also in the realms of chemistry and physics. These fields order the systems with which they are associated by affecting events that, from an energetic point of view, appear to be indeterminate or probabilistic; they impose patterned restrictions on the energetically possible outcomes of physical processes.

If morphogenetic fields are responsible for the organization and form of material systems, they must themselves have characteristic structures. So where do these field structures come from? They are derived from the morphogenetic fields associated with previous similar systems: the morphogenetic fields of all past systems become *present* to any subsequent similar system; the structures of past systems affect subsequent similar systems by a cumulative influence that acts across both space and time.

According to this hypothesis, systems are organized in the way they are because similar systems were organized that way in the past. For example, the molecules of a complex organic chemical crystallize in a characteristic pattern because the same substance crystallized that way before; a plant takes up the form characteristic of its species because past members of the species took up that form; and an animal acts instinctively in a particular manner because similar animals behaved like that previously.

The hypothesis is concerned with the *repetition* of forms and patterns of organization; the question of the *origin* of these forms and patterns lies outside its scope. This question can be answered in several different ways, but all of them seem to be equally compatible with the suggested means of repetition.[12]

A number of testable predictions, which differ strikingly from those of the conventional mechanistic theory, can be deduced from this hypothesis. A single example will suffice: If an animal, say a rat, learns to carry out a new pattern of behavior, there will be a tendency for any subsequent similar rat (of the same breed, reared under similar conditions, etc.) to learn more

quickly to carry out the same pattern of behavior. The larger the number of rats that learn to perform the task, the easier it should be for any subsequent similar rat to learn it. Thus, for instance, if thousands of rats were trained to perform a new task in a laboratory in London, similar rats should learn to carry out the same task more quickly in laboratories everywhere else. If the speed of learning of rats in another laboratory, say in New York, were to be measured before and after the rats in London were trained, the rats tested on the second occasion should learn more quickly than those tested on the first. The effect should take place in the absence of any known type of physical connection or communication between the two laboratories.

Such a prediction may seem so improbable as to be absurd. Yet, remarkably enough, there is already evidence from laboratory studies of rats that the predicted effect actually occurs.[13]

This hypothesis, called the hypothesis of formative causation, leads to an interpretation of many physical and biological phenomena that is radically different from that of existing theories, and enables a number of well-known problems to be seen in a new light. In this book, it is sketched out in a preliminary form, some of its implications are discussed, and various ways in which it could be tested are suggested.

1

THE UNSOLVED PROBLEMS OF BIOLOGY

1.1 The background of success

In the world of science, the predominant theory of life is mechanistic. Living organisms are machines. They have no souls or mysterious vital principles; they can be fully explained in terms of physics and chemistry. This is not a new idea: it dates back to the philosopher René Descartes (1596–1650). In 1867, T. H. Huxley summed it up as follows:

> Zoological physiology is the doctrine of the functions or actions of animals. It regards animal bodies as machines impelled by various forces and performing a certain amount of work that can be expressed in terms of the ordinary forces of nature. The final object of physiology is to deduce the facts of morphology on the one hand, and those of ecology on the other, from the laws of the molecular forces of matter.[1]

The subsequent developments of physiology, biochemistry, biophysics, genetics, and molecular biology are all foreshadowed in these ideas. In many respects these sciences have been brilliantly successful, none more so than molecular biology. The discovery of the structure of DNA, the "cracking of the genetic code," the elucidation of the mechanism of protein synthesis, and the sequencing of the human genome seem impressive

confirmations of the validity of this approach. The most articulate advocates of the mechanistic theory are molecular biologists. Their accounts usually begin with a brief dismissal of the vitalist and organismic theories. These are defined as survivals of "primitive" beliefs that are bound to retreat further and further as mechanistic biology advances. They then proceed along the following lines:[2]

> The chemical nature of the genetic material, DNA, is now known and so is the genetic code by which it codes for the sequence of amino acids in proteins. The mechanism of protein synthesis is understood in considerable detail. The structure of many proteins has now been worked out. All enzymes are proteins, and enzymes catalyze the complex chains and cycles of biochemical reactions that constitute the metabolism of an organism. Metabolism is controlled by biochemical feedback; several mechanisms are known by which the rates of enzymic activity can be regulated. Proteins and nucleic acids aggregate spontaneously to form structures such as viruses and ribosomes. Given the range of properties of proteins, plus the properties of other physicochemical systems such as lipid membranes, plus complex systems of physicochemical interaction, the properties of living cells can, in principle, be fully explained.
>
> The key to the problems of differentiation and development, about which very little is known, is the understanding of the control of protein synthesis. The way in which the synthesis of certain metabolic enzymes and other proteins is controlled is understood in detail in the bacterium *Escherichia coli*. The control of protein synthesis takes place by more complicated mechanisms in higher organisms, but we now know more about them than ever before. In due course, differentiation and development should be explicable in terms of series of chemically operated "switches," which "switch on" or "switch off " genes or groups of genes. Major systems of switches are already known, such as the homeobox genes.[3]
>
> The way in which the parts of living organisms are adapted to the functions of the whole, and the apparent purposiveness of the structure and behavior of living organisms, depends on random genetic mutations followed by natural selection: those genes that increase the ability of an organism to survive and reproduce will be selected for;

harmful mutations will be eliminated. Thus the neo-Darwinian theory of evolution can account for purposiveness; it is totally unnecessary to suppose that any mysterious “vital factors” are involved.

More and more is known about the functioning of the central nervous system, and the advances of biochemistry, biophysics, electrophysiology and brain scanning are already helping us to explain what we speak of as the mind in terms of physical and chemical mechanisms in the brain. Computer modelling enables us to see the mind as software operating through the hardware of the brain. Dreams of creating artificial intelligence, and even consciousness itself, within machines may soon come closer to reality.[4]

Thus living organisms are, in principle, fully explicable in terms of physics and chemistry. Our limited understanding of the mechanisms of development and of the central nervous system is due to the enormous complexity of the problems; but now, armed with the powerful new concepts of molecular biology and with the aid of computer models, these subjects can be tackled in a way not previously possible.

In the light of past successes, optimism that all the problems of biology can ultimately be solved mechanistically is understandable. But a realistic opinion about the prospects for mechanistic explanation must depend on more than an act of faith; it can be formed only after a consideration of the outstanding problems of biology, and how they might be solved.

1.2 The problems of morphogenesis

Biological morphogenesis can be defined as the “coming-into-being of characteristic and specific form in living organisms.”[5] The first problem is precisely that form comes into being: new structures appear, such as eyes and flowers, which cannot be explained in terms of structures already present in the egg. There are no miniature eyes in an eagle’s eggs, or miniature flowers in foxglove seeds.

The second problem is that many developing systems can regulate; in other words, if a part of a developing system is removed (or if an additional part is added), the system continues to develop in such a way that a more or

less normal structure is produced. The classical demonstration of this phenomenon was in Hans Driesch's experiments on sea-urchin embryos. When one of the cells of a very young embryo at the two-celled stage was killed, the remaining cell gave rise not to half a sea urchin but to a small but complete sea urchin. Similarly, small but complete organisms developed after the destruction of any one, two, or three cells of embryos at the four-celled stage. Conversely, the fusion of two young sea-urchin embryos resulted in the development of one giant sea urchin.[6]

Regulation occurs in all developing organisms, in animals and plants. In animals, as development proceeds, this capacity is often lost as the fate of different regions of the embryo becomes determined, as in limbs and livers. But even when determination occurs at an early stage, as in insect embryos, regulation still occurs after damage to the egg (see figure 1).

Results of this type show that developing plants and animals proceed toward a morphological goal. They have some property that specifies this goal and enables them to reach it, even if parts of the system are removed and the normal course of development is disturbed.

The third problem is regeneration. Organisms replace or restore damaged structures. Many plants have almost unlimited regenerative abilities. If the trunk, branches, and twigs of a willow tree are cut up into hundreds of pieces, all can grow into new trees. Some animals also regenerate from parts. A flatworm, for example, can be cut up into several pieces that all grow into new worms.

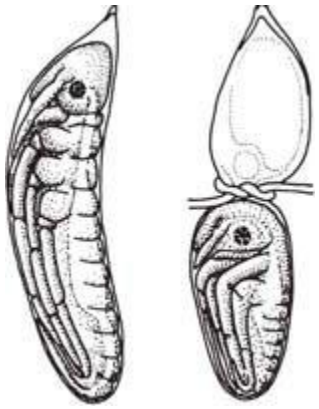

Figure 1. An example of regulation. On the left is a normal embryo of the dragonfly Platycnemis pennipes*. On the right is a small but complete embryo formed from the posterior half of an egg ligated around the middle soon after laying. (After Weiss, 1939)*

Some vertebrates show striking powers of regeneration. If the lens is surgically removed from a newt's eye, a new lens regenerates from the edge of the iris (figure 2); in normal embryonic development, the lens is formed in a very different way, from the skin. The German biologist Gustav Wolff studied this type of regeneration in the 1890s. He deliberately chose a kind of mutilation that would not have occurred accidentally in nature; there would therefore have been no natural selection for this regenerative process.[7]

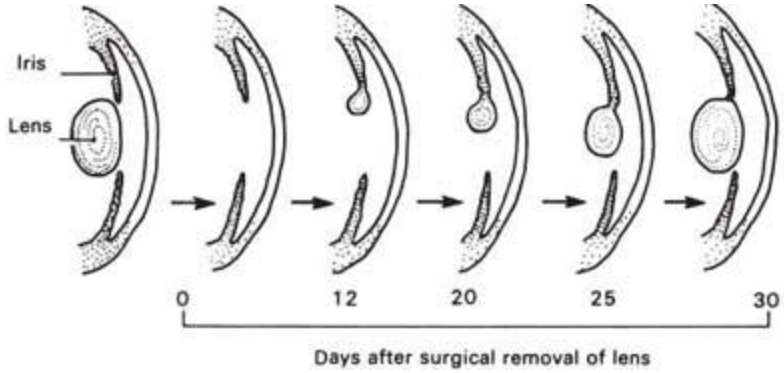

Figure 2. Regeneration of a lens from the margin of the iris in a newt's eye after the surgical removal of the original lens. (Cf. Needham, 1942)

The fourth problem is posed by the simple fact of reproduction: a detached part of the parent becomes a new organism; a part becomes a whole.

The only way in which these phenomena can be understood is in terms of causes that are somehow more than the sum of the parts, and which determine the goals of the processes of development.

Vitalists ascribe these properties to *vital factors*, organicists to *systems properties* or *morphogenetic fields*, and mechanists to *genetic programs*.

The concept of genetic programs is based on an analogy with computer programs. The metaphor implies that the fertilized egg contains a preformed program that somehow coordinates the organism's development. But the genetic program must involve something more than the chemical structure of DNA, because identical copies of DNA are passed on to all cells; if all cells were programmed identically, they could not develop differently. So

what exactly is it? In response to this question, the idea can only disintegrate into vague suggestions about physico-chemical interactions structured in time and space; the problem is merely restated.[8]

There is a further problem with the program metaphor. A computer program is put into a computer by an intelligent conscious being, the computer programmer. It is intelligently designed in order to achieve a computational goal. Insofar as the genetic program is analogous to computer software, it implies the existence of a purposive mind that plays the role of the programmer.

Mechanists reject the idea that developing organisms are under the control of a vital factor that guides them to their morphological goals. But insofar as mechanistic explanations depend on teleological concepts such as genetic programs or genetic instructions, goal-directedness can be explained only because it has already been smuggled in. Indeed the properties attributed to genetic programs are remarkably similar to those with which vitalists endowed their hypothetical vital factors; ironically, the genetic program seems to be very like a vital factor in a mechanistic guise.[9]

In Richard Dawkins's concept of the "selfish gene," the genes themselves have come to life. They are like little people: they are as ruthless and competitive as "successful Chicago gangsters"; they have powers to "mold matter," to "create form," to "choose," and even "aspire to immortality."[10] Dawkins's rhetoric is vitalistic. His selfish genes are miniaturized vital factors.

Nevertheless, the fact that biological morphogenesis cannot be explained in a rigorously mechanistic manner at present does not prove that it never will be. The prospects for arriving at such an explanation in the future are considered in the next chapter.

1.3 Behavior

If the problems of morphogenesis are dauntingly difficult, those of behavior are even more so. First, instinct. Consider, for example, how spiders are able to spin webs without learning from other spiders.[11] Or consider the behavior of European cuckoos. The young are hatched and reared by birds of other species, and never see their parents. Toward the end of the summer,

the adult cuckoos migrate to their other home in southern Africa. About a month later, the young cuckoos congregate together and they also migrate to southern Africa, where they join their elders.[12] They instinctively migrate and know when to migrate; they instinctively recognize other young cuckoos and congregate together; and they instinctively know in which direction they should fly and how to find their ancestral habitats in southern Africa, after flying unaccompanied over the Straits of Gibraltar and the Sahara Desert.

Second, there is the problem posed by the goal-directedness of animal behavior. Even if an animal is prevented from reaching its goal in one way, it may get there by another. For example, a dog after amputation of a leg learns how to walk on three legs rather than four. Another dog after brain damage gradually recovers most of its previous abilities. A third dog has obstacles put in its path. But all three dogs can go from one place to another place they want to get to in spite of disturbances to their limbs, central nervous systems, and environments.

Third, there is the problem of intelligent behavior; new patterns of behavior appear that cannot be explained entirely in terms of preceding causes. Animals can be creative.

An enormous gulf of ignorance lies between these phenomena and the sciences of molecular biology, biochemistry, genetics, and neurophysiology.

How can the migratory behavior of young cuckoos ultimately be explained in terms of DNA, protein synthesis, and molecular cell biology? Obviously a satisfactory explanation would require more than a demonstration that appropriate genes containing appropriate base-sequences in DNA were necessary for this behavior, or that the behavior of cuckoos depends on electrical impulses in nerves; it would require some understanding of the connections between specific sequences of bases in DNA, the birds' nervous system, and the migratory behavior. At present, these connections can be provided only by the same elusive entities that "explain" all the phenomena of morphogenesis: genetic programs, vital factors, system properties, or morphogenetic fields.

In any case, an understanding of behavior presupposes an understanding of morphogenesis. Even if all the behavior of a relatively simple animal, say a nematode worm, could be understood in detail in terms of the

“wiring” and physiology of its nervous system, there would still be the problem of how the nervous system was wired so precisely in the first place.

1.4 Evolution

Long before Mendelian genetics was thought of, plant and animal breeders developed many varieties of cultivated plants and domesticated animals, like Damascene roses and Pharaoh hounds. Selective breeding was the basis of their success. Charles Darwin argued persuasively that a comparable development of races and varieties occurred in the wild under the influence of natural rather than artificial selection.

Darwin also believed that habits acquired by plants and animals could be inherited.[13] The neo-Darwinian theory of evolution agrees about the importance of natural selection but rejects the inheritance of habits, and tries instead to explain all evolutionary innovation in terms of random genetic mutations, which is why it is neo-Darwinian rather than Darwinian.

Everyone agrees that mutation and natural selection can lead to the formation of varieties or subspecies. But there is no general agreement among evolutionary biologists that gradual microevolution within a species can account for the origin of species themselves, or genera, families, and higher taxonomic divisions. One school of thought holds that all large-scale evolution, or macroevolution, can indeed be explained in terms of long-continued processes of microevolution;[14] the other school denies this, and postulates that major jumps occur suddenly in the course of evolution.[15] But while opinions differ as to the relative importance of many small mutations or a few large ones, there is general agreement that mutations are random, and that evolution can be explained by a combination of random mutation and natural selection.

This theory is inevitably speculative. The evidence for evolution is open to a variety of interpretations. Opponents of neo-Darwinism can argue that evolutionary innovations are not entirely explicable in terms of chance events, but are due to the activity of a creative principle unrecognized by mechanistic science. Moreover, the selection pressures arising from the behavior and properties of living organisms may themselves depend on inner organizing factors that are essentially non-mechanistic.

Thus the problem of evolution cannot be solved conclusively. Organismic theories necessarily involve an extrapolation of organismic ideas, just as the neo-Darwinian theory involves an extrapolation of mechanistic ideas.

1.5 The origin of life

This problem of the origin of life is just as insoluble as that of evolution, for the same reasons. What happened in the distant past can never be known for certain; there will probably always be a plethora of speculations. Scenarios for life's origin include its spontaneous appearance in a primeval broth on Earth; the infection of the Earth by microorganisms deliberately sent on a space ship by intelligent beings on a planet in another solar system;[16] and the evolution of life on comets containing organic materials derived from interstellar dust.[17]

Even if the conditions under which life originated were known, this information would shed no light on the nature of life. Assuming it could be demonstrated, for example, that the first living organisms arose from nonliving chemical aggregates or "hypercycles" of chemical processes[18] in a primeval broth, this would not prove that they were entirely mechanistic. Organicists would argue that new organismic properties emerged in the first living system precisely when it came to life. The same arguments would apply even if living organisms were to be synthesized artificially from chemicals in a test tube.

1.6 Minds

The mechanistic theory postulates that all the phenomena of life, including human behavior, can in principle be explained in terms of physics. It is a form of materialism or physicalism, the theory that only material or physical things exist; they are the only reality. Materialism is opposed to the more commonsense view that minds affect bodies, and are capable of interacting with them.[19]

Materialism runs into logical problems from the outset: attempting to explain mental activity in terms of physical science is circular, because science itself depends on mental activity.[20] This problem became apparent within modern physics in connection with the role of the observer in

processes of physical measurement; the principles of physics "cannot even be formulated without referring (though in some versions only implicitly) to the impressions—and thus to the minds—of the observers" (Bernard D'Espagnat).[21] Since physics presupposes the minds of observers, these minds cannot be explained in terms of physics.[22]

Among materialist philosophers of mind, the most extreme stance is called eliminative materialism. This philosophy claims that beliefs and feelings have no coherent definition and play no part in the scientific understanding of the brain. The neuroscience of the future will have no need for outmoded concepts like beliefs and feelings; they will join previously discarded concepts like phlogiston and vital forces. Minds will be explained completely in terms of the objectively measurable activity of the nervous system.[23]

Another materialist approach to the problem of consciousness is to admit that it exists while denying it does anything. This view is called epiphenomenalism, the claim that "mental events are caused by physical events in the brain although mental events themselves do not cause anything."[24] As the philosopher Alex Hyslop has put it, "The case for epiphenomenalism is the case for materialism, together with the case against materialism. The case for materialism is the argument from science, from a triumphant, or at least steadily triumphing science. The case against materialism is that there are features of our conscious experience that are not accounted for by science."[25]

In psychology, the science of the mind, there are different schools of thought about the relationship between mind and body. The most extreme materialist solution is to deny the reality of the mind and to assume that only the body is real. This was the approach of the Behaviorist school, which dominated academic psychology for much of the twentieth century. Behaviorists confined their attention to objectively observable behavior and ignored the existence of consciousness.[26] But behaviorism was not a testable scientific hypothesis; it was a methodology.[27] It is now out of fashion within academic psychology, and has largely been replaced by cognitive psychology.

Like behaviorism, cognitive psychology rejects introspection, but it admits the existence of internal mental states, such as belief, desires, and

motivations. Its dominant metaphor is the computer. Mental activity is thought of as “information processing.” But the limitations of the computer metaphor are becoming increasingly apparent, not least through a new recognition of the role of the emotions[28] and an acknowledgment that minds are embodied and actively related to the environment.[29]

In the 1990s, the philosopher David Chalmers made a distinction between what he called the “easy problems” of consciousness, like finding neural correlates of sensation—for example, which parts of the brain become active during the visual perception of moving objects—and the “hard problem.” The hard problem is, “Why does awareness of sensory information exist at all?” There is a radical distinction between the biology of the brain and mental experience, which includes the experience of qualities, such as red. (Philosophers of mind call these subjective experiences “qualia.”) Chalmers argues that to take consciousness seriously, it is necessary to go beyond a strict materialist framework.[30]

Unlike the materialist psychologies that predominate within academic institutions, other schools of psychology accept subjective experience as their starting point, but also recognize that not all mental activity is conscious: many aspects of behavior and subjective experience depend on the subconscious or unconscious mind. The unconscious mind may also have properties that defy mechanistic explanation. For example, in Carl Jung’s development of this concept, the unconscious is not confined to individual minds, but provides a common substratum shared by all human minds, the collective unconscious.

> In addition to our immediate consciousness, which is of a thoroughly personal nature and which we believe to be the only empirical psyche (even if we tack on the personal unconscious as an appendix) there exists a second psychic system of a collective, universal, and impersonal nature which is identical in all individuals. This collective unconscious does not develop individually but is inherited. It consists of pre-existent forms, the archetypes, which can only become conscious secondarily and which give definite form to certain psychic contents.[31]

Jung tried to explain the inheritance of the collective unconscious physically by suggesting that the archetypal forms were “present in the germplasm.”[32] But it is doubtful that anything with the properties of the archetypal forms could be inherited chemically in the structure of DNA, or in any other physical or chemical structure in sperm or egg cells. Indeed the idea of the collective unconscious makes little sense in terms of current mechanistic biology, whatever its merits as a psychological theory might be.

However, there is no a priori reason why psychological theories should be confined within the framework of the mechanistic theory. Mental phenomena need not necessarily depend on the known laws of physics, but may depend on principles as yet unrecognized by science.

1.7 Parapsychology

In all traditional societies, stories are told of men and women with seemingly miraculous powers, and such powers are acknowledged by all religions. In many parts of the world, various psychic abilities are cultivated within systems such as shamanism, sorcery, tantric yoga, and spiritualism. And even within modern Western society, there are persistent reports of unexplained phenomena, such as telepathy, clairvoyance, precognition, memories of past lives, hauntings, poltergeists, psychokinesis, and so on. Surveys show that the most common kind of telepathy occurs in connection with technology, namely telephone telepathy, whereby people think of someone for no apparent reason who calls soon afterward.[33]

Although dogmatic skeptics dismiss all this evidence out of hand,[34] the possibility that at least some of these events actually occur is an open question. It can be answered only after an examination of the evidence.

The scientific study of allegedly psychic phenomena has now been going on for more than a century. Investigators in this field of psychic research have discovered some cases of fraud, and found that some apparently paranormal events can in fact be explained by normal causes. But there remains a large body of evidence that seems to defy explanation in terms of any known physical principles.[35] Numerous experiments designed to test for so-called extrasensory perception have yielded positive results with

odds against chance coincidence of thousands, millions, or even billions to one.[36]

Insofar as these phenomena cannot be explained in terms of the known laws of physics and chemistry, from the mechanistic point of view they ought not to occur.[37] But if they do, then there are two possible approaches. The first is to suppose that they depend on nonphysical causal factors or connecting principles.[38] The second is to start from the assumption that they depend on laws of physics as yet unknown, or on extensions of quantum theory,[39] for example by postulating that mental states play a role in determining the outcomes of probabilistic processes of physical change.[40]

1.8 Conclusions

This brief consideration of the outstanding problems of biology does not offer much hope that they can all be solved by an exclusively mechanistic approach. In the case of morphogenesis and animal behavior, the question is open. The problems of evolution and the origin of life are insoluble per se and cannot help to decide between the mechanistic and other possible theories of life. The mechanistic theory runs into serious philosophical difficulties in connection with the problem of the limits of physical explanation; in relation to psychology, it leads to seemingly insoluble problems; and it is in conflict with the apparent evidence for parapsychological phenomena.

The prospects for improved versions of mechanistic, vitalist, and organismic theories are discussed in the following chapter. Morphogenesis is the starting point.

2

THREE THEORIES OF MORPHOGENESIS

2.1 Descriptive and experimental research

The description of development can be carried out in many ways: the external form of the developing animal or plant can be drawn, photographed, or filmed, providing a series of pictures of its changing morphology; its internal structure, including its microscopic anatomy, can be described at successive stages (see figure 3); changes in physical quantities such as weight, volume, and rate of oxygen consumption can be measured; and changes in the chemical composition of the system as a whole and of regions within it can be analyzed.

The progressive improvement of techniques permits such descriptions to be made in ever greater detail; for example, with the electron microscope the processes of cellular differentiation can be studied at a far higher resolution than with the light microscope, enabling many new structures to be seen; the sensitive analytical methods of modern molecular biology enable changes in concentrations of specific molecules, including proteins and nucleic acids, to be measured in very small samples of tissue; by means of radioactive isotopes or fluorescent antibodies, chemical structures can be “labeled” and “traced” as a system develops; and techniques for inducing genetic changes in some of the cells of embryos enable their genetically “marked” descendants to be identified and their fate to be “mapped.”

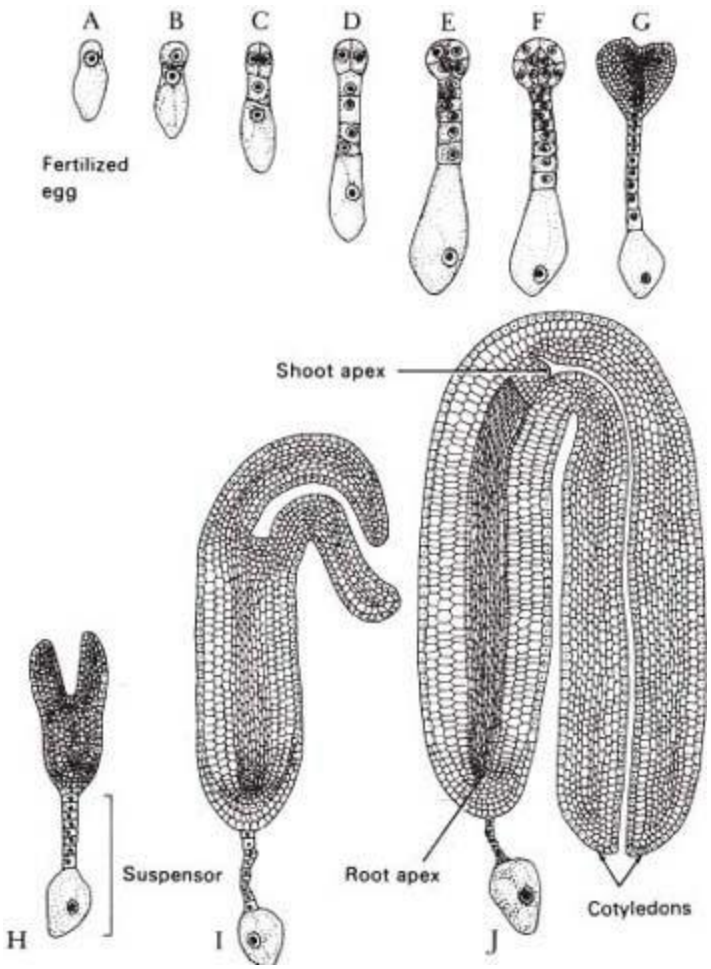

Figure 3. Stages in the development of the embryo of the shepherd's purse plant, Capsella bursa-pastoris. *(After Maheshwari, 1950)*

The majority of research in cell and developmental biology is concerned with providing factual descriptions by means of such techniques; these descriptions are then classified and compared in order to establish how different sorts of changes are correlated within a given system, and in what ways different systems resemble each other. These purely descriptive results cannot in themselves lead to an understanding of the causes of development, although they may suggest hypotheses.[1] The latter can then be investigated by means of experimental perturbations of development: for example, the environment can be changed; physical or chemical stimuli can be applied to specific places on or in a system; parts of the system can be removed and their development studied in isolation; the reaction of the system to the removal of parts can be observed; and the effects of combining different parts can be studied by grafts and transplantations.

The major problems thrown up by this type of research have been summarized in section 1.2: biological development involves an increase in complexity of form and organization that cannot be explained in terms of the unfolding of a preformed but invisible structure; many developing systems can regulate, i.e., produce a more or less normal structure if part of the system is destroyed or removed at a sufficiently early stage; many systems can regenerate or replace missing parts; and in vegetative and sexual reproduction, new organisms are formed from detached parts of parent organisms. One further important generalization is that in developing systems the destiny of cells and tissues is determined by their position within the system.

Mechanistic, vitalist, and organismic theories all start from this established body of facts, about which there is general agreement, but they differ radically in their interpretations.

2.2 Mechanism

The modern mechanistic theory of morphogenesis ascribes a role of prime importance to DNA, for four main reasons. First, many cases of hereditary differences between animals or plants of a given species have been found to depend on genes, which can be “mapped” and located at particular places on particular chromosomes. Second, the chemical basis of genes is known to be DNA and their specificity is known to depend on the sequence of purine and pyrimidine bases in the DNA. Third, it is known how DNA is able to act as the chemical basis of heredity: on the one hand, it serves as a template for its own replication, owing to the specificity of the pairing of the bases in its two complementary strands; on the other hand, it serves as the template for the sequence of amino acids in proteins. It does not play the latter role directly; one of its strands is first “transcribed” to give a single-stranded molecule of “messenger” RNA from which, in the process of protein synthesis, the sequence of bases is “read off ” three at a time. Different triplets of bases specify different amino acids, and thus the genetic code is “translated” into a sequence of amino acids, which are linked together to give characteristic polypeptide chains, which then fold up to give proteins. Finally, the characteristics of a cell depend on its proteins: its metabolism and its capacities for chemical synthesis on enzymes, some of

its structures on structural proteins, and the surface properties that enable it to be “recognized” by other cells on special proteins on its surface.

Within the mechanistic framework of thought, the central problem of development and morphogenesis is seen as the control of protein synthesis. In bacteria, specific chemicals called inducers can cause specific regions of the DNA to be transcribed into messenger RNA, on which template specific proteins are then made. The classic example is the induction of the enzyme β-galactosidase by lactose in *Escherichia coli*. The “switching on” of the gene takes place through a complicated system involving a repressor protein that blocks transcription by combining with a specific region of the DNA; its tendency to do so is greatly reduced in the presence of the chemical inducer. By a comparable process, specific chemical repressors can “switch off ” genes.

In animals and plants a range of developmental genes have now been identified that are concerned with the regulation of the overall body plan and the number, identity, and pattern of body parts. These genes are usually called the “genetic toolkit.” The most surprising discovery of developmental biology in the 1990s was that these tool-kit genes are remarkably similar, indeed almost identical, in widely different organisms. For example, the “homeobox” family of genes that affect the patterning of the body axis in fruit flies, mice, and humans are very similar, and yet the body forms of these organisms are obviously very different. As the molecular biologist Sean B. Carroll and his colleagues have put it, “The conservation of the genetic toolkit provokes many developmental and evolutionary questions. How do such different structures as the insect compound eye and the vertebrate lens-type eye develop when their formation is controlled by such similar, even functionally interchangeable genes?”[2]

This convergence of developmental and evolutionary biology has created a new field called evolutionary developmental biology (“evodevo” for short).

Most tool-kit genes code for proteins that affect the activity of other genes involved in the developmental process, and are part of “signaling pathways.” Some of them code for receptor proteins on cell surfaces that bind to specific molecules that act as signals.

In the early days of molecular biology, there seemed to be a simple, straightforward picture: one gene was transcribed into one messenger RNA molecule, which coded for one protein. But the picture has grown more complicated. Messenger RNA can be made up of pieces transcribed from different regions of the DNA, and subsequently joined together in a specific way. Moreover, the synthesis of proteins is also controlled at the "translational level"; protein synthesis can be "switched" on and off by a variety of factors even in the presence of appropriate messenger RNA.

The different proteins made by different types of cells thus depend on the way in which protein synthesis is controlled. The only way in which this can be understood mechanistically is in terms of physical and chemical influences on the cells; patterns of differentiation must therefore depend on physical and chemical patterns within the tissue. These are concentration gradients of specific chemicals called morpho-gens; these include diffusion-reaction systems with chemical feedback, electrical gradients, electrical or chemical oscillations, mechanical contacts between cells, or various other factors or combinations of different factors. The cells must then respond to these differences in characteristic ways. One way of thinking about this problem is to regard these physical or chemical factors as providing "positional information" that the cells then "interpret" in accordance with their genetic program by "switching on" the synthesis of particular proteins.[3]

These various aspects of the central problem of the control of protein synthesis are at present under active investigation. Most biologists hope that the solution of this problem will provide, or at least lead toward, an explanation of morphogenesis in purely mechanistic terms.

In order to assess whether such a mechanistic explanation is likely, or even possible, a number of difficulties need to be considered one by one.

(1) The explanatory role of DNA and the synthesis of specific proteins are severely restricted in their scope by the fact that both the DNA and the proteins of different species may be very similar. For example, in a detailed comparison of human and chimpanzee proteins, many are identical and others differ only slightly: "Amino acid sequencing, immunological and electrophoretic methods yield concordant estimates of genetic resemblance. These approaches all indicate that the average human polypeptide is more than 99 percent identical to its chimpanzee counterpart."[4] Comparisons of

the so-called non-repeated DNA sequences (i.e., those parts believed to be of genetic significance) show that the overall difference between the DNA sequences of humans and chimpanzees is only 1.1 percent. Now that both genomes have been sequenced, even more detailed comparisons are possible, but as Svante Paabo, the director of the chimpanzee genome project, commented, “We cannot see in this why we are so different from chimpanzees.”[5]

Comparisons between closely related species in the genus *Drosophila* have revealed *larger* differences among these species of fruit fly than between humans and chimpanzees. Different species of mice are also more dissimilar than humans and chimpanzees, leading to the conclusion that “the contrasts between organismal and molecular evolution indicate that the two processes are to a large extent independent of each other.”[6] If genes and proteins do not explain the differences between chimpanzees and us, then what does?

However, leaving aside all these problems, assume for the purpose of argument that the hereditary differences between chimpanzees and humans will indeed be explained in terms of small changes in protein structure, or small numbers of different proteins, or genetic changes that affect the control of protein synthesis (perhaps depending to some extent on differences of arrangement of DNA within the chromosomes), or combinations of these factors.

(2)Within the same organism, different patterns of development take place while the DNA remains the same. Consider, for example, your arms and legs: both contain identical cell types (muscle cells, connective tissue cells, etc.) with identical proteins and identical DNA. So the differences between the arms and the legs cannot be ascribed to DNA per se; they must be ascribed to pattern-determining factors that act differently in developing arms and legs. They also give rise to mirror-image patterns in right and left arms and legs. The precision of arrangement of the tissues—for example, the joining of tendons to the right parts of the bones—shows that these patterns are established in detail and with precision. The mechanistic theory of life means that these factors must be regarded as physical or chemical, but their nature is unknown.

(3) Even if physical or chemical factors affecting the growth of an arm, the formation of an eye, or the development of an apple are identified, this

raises the question of how these factors are themselves patterned in the first place. This problem can be illustrated by considering two cases in which chemical morphogens have actually been isolated and chemically identified.

First, in the cellular slime molds, free-living amoeboid cells aggregate together under certain conditions to form a “slug” that, after moving around for some time, grows up into the air and differentiates into a stalk bearing a spore-mass (figure 4). The aggregation of these cells depends on a relatively simple chemical, cyclic AMP (adenosine 3',5'-monophosphate). But in the composite organism, although the distribution of cyclic AMP is related to the pattern of differentiation, “it is not clear whether the cyclic AMP pattern is a cause or consequence of prestalk–prespore differentiation.” Moreover, even if it does play a causal role in differentiation, it cannot itself account for the pattern in which it is distributed, nor for the fact that this pattern varies from species to species: some other factors must be responsible for its patterned distribution. There is a wide variety of opinion on the possible nature of these factors.[7]

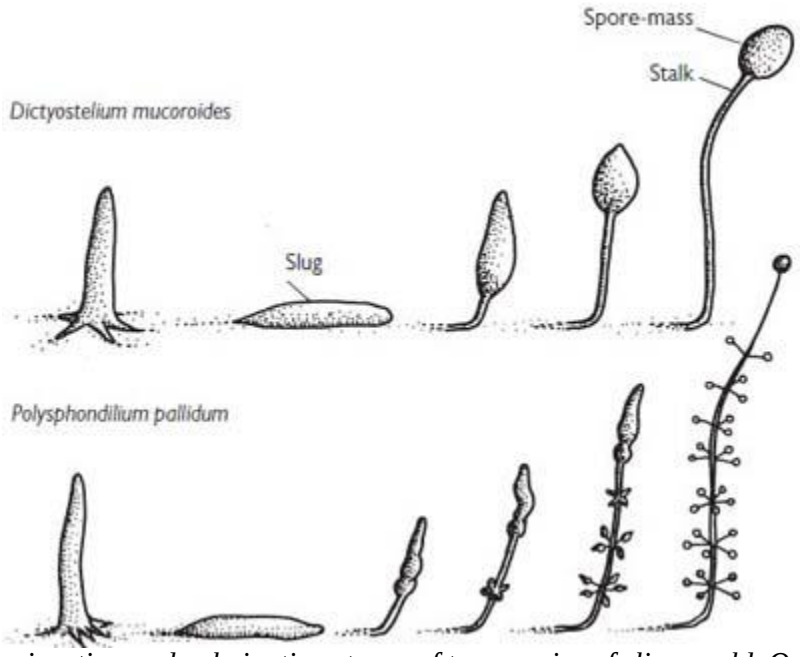

Figure 4. The migration and culmination stages of two species of slime mold. On the left are the newly developed composite organisms, formed by the aggregation of numerous free-living amoeboid cells. These migrate as “slugs” and then grow upward, differentiating into stalks bearing spore bodies. (After Bonner, 1958)

Second, in higher plants the hormone auxin (indole-3-yl-acetic acid) is known to play a role in the control of vascular differentiation, as in the formation of wood (xylem) cells. But then what controls the production and distribution of auxin? The answer seems to be: vascular differentiation itself. Auxin is probably released by differentiating xylem cells as a by-product of protein breakdown occurring as the cells commit suicide. Thus the system is circular: auxin helps to maintain patterns of differentiation, but it does not explain how they are established to start with.[8]

However, assume for the purpose of argument that it might be possible to identify preexisting physical factors that set up the pattern of distribution of auxin, or cyclic AMP, or other chemical morphogens. Assume also that the ways in which these controlling factors are themselves controlled can be identified, in a series going right back to the fertilized egg or spore from which the organism grew.

Now there is the problem of regulation: If part of the system is removed, this complicated series of physico-chemical patterns must be disrupted. But somehow the remaining parts of the embryo manage to change their usual course of development and produce a more or less normal adult.

This problem is generally agreed to be extremely difficult; it is far from being understood even in outline. Supporters of the mechanistic theory hope that it can be solved through much painstaking effort and mathematical modeling. Let us assume once more that a mechanistic solution can be achieved.

(4) The next problem is how this “positional information” brings about its effects. The simplest possibility is that the “positional information” is specified by a concentration gradient of a specific morphogen, and cells exposed to more than a certain concentration synthesize one set of proteins while cells exposed to concentrations below this threshold synthesize another set of proteins.

Again, assume that mechanisms by which “positional information” can be “interpreted” can be identified and described in detail.[9] Now, at the end of a chain of highly optimistic assumptions, we reach the situation in which different cells arranged in a suitable pattern make different proteins.

So far, there has been a set of one-to-one relationships: a gene is “switched on” by a specific stimulus; the DNA is transcribed into RNA; and the RNA is translated into a particular sequence of amino acids, a polypeptide chain. But now this simple causal sequence comes to an end. How do the polypeptide chains fold up into the characteristic three-dimensional structures of proteins? How do the proteins give the cells their characteristic structures? How do cells aggregate together to give tissues of characteristic structures? And so on. These are the problems of morphogenesis proper: the synthesis of specific polypeptide chains provides the basis for the metabolic machinery and the structural materials on which morphogenesis depends. The polypeptide chains and the proteins into which they fold up are undoubtedly necessary for morphogenesis; but what actually determines the patterns and structures into which the proteins, cells, and tissues combine? Mechanists assume that all this can be explained in terms of self-assembly. Morphogenesis takes place spontaneously, given the right proteins in the right places at the right times and in the right sequence. This is rather like saying that a house can build itself spontaneously as long as the right building materials are delivered to the building site at the right times. At this crucial stage, biology effectively abdicates, and the problem of morphogenesis is left to spontaneous physical and chemical processes. In the building-site analogy, the activity of the builders and a plan to which they are working are needed in addition to the starting materials. By contrast, morphogenesis must depend on spontaneous physical processes driven by spontaneous energy flows, and the systems must be self-organizing. But how?

Protein folding is a good example. Polypeptide chains fold up spontaneously, given the right conditions, into proteins of characteristic three-dimensional structure. Some proteins can be made to unfold and then, by changing the conditions, fold up again in test tubes. Hence protein folding does not depend on any mysterious property of living cells.

Protein subunits can aggregate together under test-tube conditions to form structures normally produced inside living cells: for example, subunits of the protein tubulin join together into long, rodlike structures called microtubules.[10] Yet-more-complex structures, such as ribosomes, are formed by the spontaneous aggregation of various protein and RNA

components. Lipid molecules can come together in test tubes to form membranelike structures.

Insofar as these structures undergo spontaneous self-assembly, they resemble crystals; many of them can indeed be regarded as crystalline or quasi-crystalline. So in principle they pose no more, or no less, of a problem than normal crystallization; the same sorts of physical process can be assumed to be at work.

Nevertheless, by no means all morphogenetic processes can be regarded as types of crystallization. They must involve a number of other physical factors; for example, the forces of surface tension must influence the shapes taken up by membranes. And then some of the patterns may arise from statistically random fluctuations; simple examples of the appearance of "order through fluctuations" have been studied from the point of view of irreversible or nonequilibrium thermodynamics in inorganic systems.[11] Some patterning processes can be modeled mathematically in complex systems theory.[12]

But the mechanistic theory does not merely suggest that physical processes play a part in morphogenesis; it asserts that morphogenesis is entirely explicable in terms of physics. What does this mean? If everything observable is *defined* as being physically explicable in principle just because it happens, then it must be so by definition. But this does not necessarily mean it can be explained in terms of the *known* laws of physics.

In relation to biological morphogenesis, a complete explanation would be achieved if a biologist supplied with the entire genome sequence of an organism, and a detailed description of the physical and chemical state of the fertilized egg and of the environment in which it developed, could *predict* in terms of the fundamental laws of physics (e.g., quantum field theory, the equations of electromagnetism, the second law of thermodynamics, etc.) first, the three-dimensional structure of all the proteins the organism would make; second, the enzymic and other properties of these proteins; third, the organism's entire metabolism; fourth, the nature and consequences of all the types of positional information that would arise during its development; fifth, the structure of its cells, tissues, and organs and the form of the organism as a whole; and finally, in the case of an animal, its instinctive behavior.

If all these predictions could be made successfully, and if, in addition, the course of processes of regulation and regeneration could also be predicted a priori, this would indeed be a conclusive demonstration that living organisms are fully explicable in terms of the known laws of physics. As T. H. Huxley put it in 1867, “The final object of physiology is to deduce the facts of morphology on the one hand, and those of ecology on the other, from the laws of the molecular forces of matter”[13] (section 1.1). But, of course, this could not be done in the nineteenth century, nor can it be done today, despite all the discoveries of molecular biology and the hundreds of billions of dollars that have been spent on it. So there is no way of demonstrating that such an explanation is possible. It might not be.

Thus when the mechanistic theory states that all the phenomena of morphogenesis are capable in principle of being explained in terms of the known laws of physics, it might well be wrong: so little is understood at present that there seem to be no good grounds for a firm belief in the adequacy of the known laws to explain all the phenomena. But at any rate this is a testable theory; it would be refuted by the discovery of a new law of physics. If on the other hand the mechanistic theory states that living organisms obey both known and unknown laws of nature, then it would be irrefutable; it would simply be a general statement of faith in the possibility of explanation. It would not be opposed to organicism and vitalism; it would include them.

In practice, the mechanistic theory of life is not treated as a rigorously defined, refutable scientific theory; rather, it serves to provide a justification for the conservative method of working within the established framework of thought provided by existing physics and chemistry. Although it is usually understood to mean that living organisms are in principle fully explicable in terms of the known laws of physics, if a new law of physics were to be discovered, the mechanistic theory could easily be modified to include it. Whether this modified theory of life were to be called mechanistic or not would be a matter of definition.

When so little is understood about the phenomena of morphogenesis and behavior, the possibility cannot be ruled out that at least some of them depend on a causal factor or factors as yet unrecognized by physics. In the mechanistic approach, this question is simply put aside. Nevertheless, it remains entirely open.

2.3 Vitalism

Vitalism asserts that the phenomena of life cannot be fully understood in terms of physical laws derived only from the study of inanimate systems, but that an additional causal factor is at work in living organisms. A typical statement of a nineteenth-century vitalist position was made by the chemist Justus von Liebig in 1844. He argued that although chemists could already produce all sorts of organic substances, and would in future produce many more, chemistry would never be in a position to create an eye or a leaf. Besides the recognized causes of heat, chemical affinity, and the formative force of cohesion and crystallization, "in living bodies there is added yet a fourth cause that dominates the force of cohesion and combines the elements in new forms so that they gain new qualities—forms and qualities that do not appear except in the organism."[14]

Ideas of this type, although widely held, were too vague to provide an effective alternative to the mechanistic theory. It was only at the beginning of the twentieth century that neovitalist theories were worked out in some detail. In relation to morphogenesis, the most important was that of the embryologist Hans Driesch.

Driesch did not deny that many features of living organisms could be understood in physico-chemical terms. He was well aware of the findings of physiology and biochemistry, and of the potential for future discovery: There are many specific chemical compounds present in the organism, belonging to the different classes of the chemical system, and partly known in their constitution, partly unknown. But those that are not yet known will probably be known some day in the near future, and certainly there is no theoretical impossibility about discovering the constitution of albumen (protein) and how to "make" it.[15]

He knew that enzymes ("ferments") catalyze biochemical reactions and could do so in test tubes: "There is no objection to our regarding almost all metabolic processes inside the organism as due to the intervention of ferments or catalytic materials, and the only difference between inorganic and organic ferments is the very complicated character of the latter and the very high degree of their specification."[16] He knew that Mendelian genes are material entities located in the chromosomes, and that they are probably

chemical compounds of specific structure.[17] He thought that many aspects of metabolic regulation and physiological adaptation could be understood along physico-chemical lines[18] and that there were in general "many processes in the organism . . . which go on teleologically or purposefully on a fixed machine-like basis."[19] His opinions on these subjects have been confirmed by the subsequent advances of physiology, biochemistry, and molecular biology. Obviously, Driesch was unable to anticipate the details of these discoveries, but he regarded them as possible and in no way incompatible with vitalism.

In relation to morphogenesis, he considered that "it must be granted that a machine, as we understand the word, might very well be the motive force of organogenesis in general, if only normal, that is to say, if only undisturbed development existed, and if taking away parts of our system led to fragmental development."[20] But, in fact, in many embryonic systems the removal of part of the embryo is followed by a process of regulation, whereby the remaining tissues reorganize themselves and go on to produce an adult organism of more or less normal form.

The mechanistic theory has to attempt to account for development in terms of complex physical or chemical interactions among the parts of the embryo. Driesch argued that the fact of regulation made any such machinelike system inconceivable, because the system was able to remain a whole and produce a typical final result, whereas no complex three-dimensional machinelike system could remain a whole after the arbitrary removal of parts.

This argument is open to the objection that it is, or will be at some time in the future, invalidated by advances in technology. But at least it does not seem to have been refuted so far. For example, although some computerized systems can respond appropriately to certain types of functional disturbance, they do so on the basis of a fixed structure. They cannot regenerate their own physical structure; for example, if parts of the computer are destroyed at random, they cannot be regenerated by the machine itself, nor can the system go on functioning normally after the arbitrary removal of parts. The other item of modern technology that might seem relevant is the hologram, from which pieces can be removed but which can still give rise to a complete three-dimensional image. But,

significantly, the hologram is not a machine: it is an interference pattern in a field.

Driesch believed that the facts of regulation, regeneration, and reproduction showed that there was something about living organisms that remained a whole, even though parts of the organism could be removed; it acted on the physical system but was not itself part of it. He called this nonphysical causal factor *entelechy*. He postulated that entelechy organizes and controls physico-chemical processes during morphogenesis. The genes were responsible for providing the material *means* of morphogenesis—the chemical substances to be ordered—but the ordering itself was brought about by entelechy.

Clearly morphogenesis could be *affected* by genetic changes that changed the means of morphogenesis, but this would not prove that it could be *explained* simply in terms of genes and the chemicals to which they gave rise.

Similarly, the nervous system provided the means for the actions of an animal, but entelechy organized the activity of the brain, using it as an instrument, as a pianist plays on a piano. Again, behavior can be affected by damage to the brain, just as the music played by the pianist is affected by damage to the piano; but this proves only that the brain is a necessary means for behavior, as the piano is a necessary means for the pianist.

Entelechy is a Greek word whose derivation (*en-telos*) indicates something that bears its end or goal in itself; it "contains" the goal toward which a system under its control is directed. Thus if a normal pathway of development is disturbed, the system can reach the same goal in a different way. Driesch considered that development and behavior were under the control of a hierarchy of entelechies, which were all ultimately derived from, and subordinated to, the overall entelechy of the organism.[21] As in any hierarchical system, such as an army, mistakes are possible and entelechies might behave "stupidly," as they do in cases of super-regeneration, when a superfluous organ is produced.[22] But such stupidities do not disprove the existence of entelechy any more than military errors disprove that soldiers are intelligent beings.

Driesch described entelechy as an "intensive manifoldness," a non-spatial causal factor that nevertheless acted into space. He emphasized that it was a

natural (as opposed to a metaphysical or mystical) factor that acted on physico-chemical processes. It was not a form of energy, and its action did not contradict the second law of thermodynamics or the law of conservation of energy. Then how did it work?

Driesch was writing during the era of classical physics, when it was generally thought that all physical processes were fully deterministic, in principle completely predictable in terms of energy, momentum, etc. But he considered that physical processes could not be fully determinate, because otherwise the nonenergetic entelechy could not act upon them. He therefore concluded that, at least in living organisms, microphysical processes were not fully determined by physical causality, although, on average, physico-chemical changes obeyed statistical laws. He suggested that entelechy acted by affecting the detailed *timing* of microphysical processes, by suspending them and releasing them from suspension whenever required for its purposes:

> This faculty of a temporary suspension of inorganic becoming is to be regarded as the most essential ontological characteristic of entelechy . . . Entelechy, according to our view, is quite unable to remove any kind of "obstacle" to happening . . . for such a removal would require energy, and entelechy is non-energetic. We only admit that entelechy may set free into actuality what it has *itself* prevented from actuality, what it has suspended hitherto.[23]

Although this bold proposal of a physical indeterminism within living organisms was completely unacceptable from the point of view of deterministic classical physics, it seems much less outrageous in the light of quantum theory. Some twenty years after Driesch's speculations about indeterminism within living organisms, Heisenberg deduced the uncertainty principle, and it soon became clear that positions and timings of microphysical events could be predicted only in terms of probabilities. By 1928, the physicist Sir Arthur Eddington was able to speculate that the mind influences the body by affecting the configuration of quantum events within the brain through a causal influence on the probability of their occurrence. "Unless it belies its name, probability can be modified in ways which ordinary physical entities would not admit of."[24] Comparable ideas were

proposed by the neurophysiologist Sir John Eccles, who summarized his suggestion as follows:

> The neurophysiological hypothesis is that the "will" modifies the temporal activity of the neuronal network by exerting spatio-temporal "fields of influence" that become effective through this unique detector function of the active cerebral cortex. It will be noted that the "will" or "mind influence" has itself some spatio-temporal patterned character in order to allow it this operative effectiveness.[25]

A number of similar proposals have been put forward by physicists and by parapsychologists[26] (section 1.7).

A vitalist theory of morphogenesis can be summarized as follows: The genome specifies all the possible proteins that the organism can make. But the organization of the cells, tissues, and organs, and the coordination of the development of the organism as a whole, is determined by entelechy. The latter is inherited nonmaterially from past members of the same species; it is not a type of matter or energy, although it acts upon the physico-chemical systems of the organism under its control. This action is possible because entelechy acts by influencing probabilistic processes.

This theory is by no means vacuous, and could probably be tested experimentally, but it seems fundamentally unsatisfactory simply because it is vitalistic. Entelechy is essentially nonphysical by definition; even though it could, *ex hypothesi*, act on material systems by providing a set of variables that from the point of view of quantum theory are hidden, this would still be an action of unlike on unlike. The physical world and the nonphysical entelechy could never be explained or understood in terms of each other.

This dualism, inherent in all vitalist theories, seems particularly arbitrary in the light of the discovery of the self-assembly of structures as complex as ribosomes and viruses, indicating a difference of degree, and not of kind, from crystallization. Although the self-organization of living organisms as a whole is more complex than that of ribosomes or viruses, there is sufficient similarity to suggest that here again is a difference of degree. This, at any rate, is what both mechanists and organicists prefer to think.

Possibly a vitalist theory would have to be accepted if no other satisfactory explanation of the phenomena of life were conceivable. In the early part of the twentieth century, when vitalism seemed to be the only alternative to the mechanistic theory, it gained considerable support in spite of its essential dualism. But the organismic theory incorporates many aspects of vitalism within a larger perspective, and effectively supersedes it.

2.4 Organicism

Organismic theories of morphogenesis have developed under a variety of influences: some from philosophical systems, especially those of Alfred North Whitehead and Jan Christian Smuts; some from modern physics, in particular from the field concept; others from Gestalt psychology, itself strongly influenced by the concept of physical fields; and some from the vitalism of Driesch.[27] These theories deal with the same problems that Driesch claimed were insoluble in mechanistic terms—regulation, regeneration, and reproduction—but whereas Driesch proposed the nonphysical entelechy to account for the properties of wholeness and directiveness exhibited by developing organisms, organicists proposed morphogenetic (or embryonic, or developmental) *fields*.

This idea was put forward independently by Alexander Gurwitsch in Russia in 1922,[28] Hans Spemann in Germany in 1924, and Paul Weiss in Austria in 1926.[29] All were leading developmental biologists, and Spemann received the Nobel Prize in 1935 for his work on embryology. However, apart from stating that morphogenetic fields played an important role in the control of morphogenesis, none of them specified how these fields worked. The field terminology was soon taken up by other developmental biologists, but it remained ill-defined, although it served to suggest analogies between properties of living organisms and inorganic electromagnetic systems. For example, if an iron magnet is cut into two parts, two whole magnets are produced, each with its own magnetic field. If two magnets are brought together in the right orientation, they form a single magnet with a unified magnetic field. Similarly, the morphogenetic field was supposed to account for the “wholeness” of detached parts of organisms that were capable of growing into new organisms, and for the ability of parts of organisms to form a unified whole when brought together.

The British biologist Conrad Hal Waddington suggested an extension of the idea of the morphogenetic field to take into account the temporal aspect of development. He called this new concept the *chreode* (from the Greek *chre,* it is necessary, and *hodos,* route or path) and illustrated it by means of a simple three-dimensional “epigenetic landscape” (figure 5).[30]

In this model, the path followed by the ball as it rolls downward corresponds to the developmental history of a particular part of an egg. As embryology proceeds, a branching series of alternative paths are represented by the valleys. These correspond to the pathways of development of the different types of organ, tissue, and cell. In the organism these are quite distinct; for example, the kidney and liver have definite structures and do not grade into each other through a series of intermediate forms. Development is *canalized* toward definite end points. Genetic changes or environmental perturbations may push the course of development (represented by the pathway followed by the ball) away from the valley bottom up the neighboring hillside, but unless it is pushed above the threshold into another valley, the process of development will find its way back. It will not return to the point from which it started, but to some later position on the canalized pathway of change. This represents regulation.

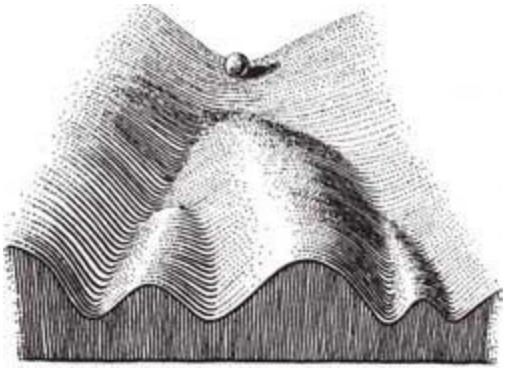

Figure 5. Part of an “epigenetic landscape,” illustrating the concept of the chreode as a canalized pathway of change. (From Waddington, 1957. Reproduced by courtesy of George Allen & Unwin, Ltd.)

The concept of the chreode is very similar to that of the morphogenetic field, but it makes explicit the dimension of time that is only implicit within the latter.

Both these concepts were taken further by the mathematician René Thom as part of a comprehensive attempt to create a mathematical theory embracing morphogenesis, behavior, and language.[31] His main concern was to find an appropriate mathematical formalism for these problems, which had so far resisted mathematical treatment. The final objective was to produce mathematical models that corresponded as closely as possible to developmental processes. These models would be topological, qualitative rather than quantitative, and would not depend on any particular scheme of causal explanation.

> One essential feature of our use of local models is that it implies nothing about the "ultimate nature of reality"; even if this is ever revealed by analysis complicated beyond description, only a part of its manifestation, the so-called observables, are finally relevant to the macroscopic description of the system. The phase space of our dynamical model is defined using only these observables and without reference to any more or less chaotic underlying structures.[32]

Thom's mathematical models were dynamic in the modern mathematical sense of the word.[33] Dynamic systems move toward *attractors,* and Thom explicitly connected his models with Waddington's idea of chreodes, with ends or goals toward which systems develop.[34]

The problem with this approach is that it is essentially descriptive; it does little to *explain* morphogenesis. This is indeed the case with all existing organismic theories of morphogenesis. Compare, for example, Thom's attractors and Waddington's chreodes with Driesch's entelechy. They all include the idea that development is guided or canalized in space and time by something that cannot itself be regarded as confined to a particular place and time; all see this as somehow including within itself the end or goal of the developmental process, and thereby provide a way of thinking about regulation and regeneration. The main difference is that Driesch tried to say how this process might actually work, whereas Waddington and Thom did not. The concept of the chreode was therefore less open to attack because it remained so vague.[35] In fact, Waddington regarded the concepts of chreodes and morphogenetic fields as "essentially a descriptive

convenience."[36] Like a number of other organicists, he denied that he was suggesting the operation of anything other than known physical causes.[37]

However, not all organicists make this denial; some leave the question open. This explicitly noncommittal attitude is illustrated by the following discussion of the morphogenetic field by the developmental biologist Brian Goodwin.

> One aspect of the field is that electrical forces can affect it. Other developing and regenerating organisms have also been found to have interesting and significant electrical field patterns, but I would not wish to suggest that the morphogenetic field is essentially electrical. Chemical substances also affect polarity and other spatial aspects of developing organisms; and again I would not wish to draw the conclusion that the morphogenetic field is essentially chemical or biochemical in nature. My belief is that its investigation should proceed on the assumption that it could be any, or all, or none of these things; but that, despite agnosticism regarding its material nature, it plays a primary role in the developmental process.[38]

The openness of this concept makes it the most promising starting point for a detailed organismic theory of morphogenesis. But clearly, if morphogenetic fields are considered to be fully explicable in terms of known physical principles, they represent nothing but an ambiguous terminology superimposed upon a sophisticated version of the mechanistic theory. Only if they are assumed to play a causal role, at present unrecognized by physics, can a testable theory be developed. This possibility is discussed in the following chapters.

Exploring the nature of morphogenetic fields takes on a new urgency in the light of modern evolutionary developmental biology. With the rise of molecular biology from the 1960s to the 1990s, the concept of morphogenetic fields was eclipsed in favor of genes. But as the limitations of the molecular approach became increasingly apparent, fields reemerged as a central concept for the understanding of development. The formation of entire structures such as wings and antennae in fruit flies can be "switched on" or "switched off" by mutations in "toolbox" genes. The morphogenetic

field behaves as a whole and the genetic switches are now referred to as "field selector genes."[39] Development is "modular."[40]

In an evolutionary context, morphogenetic fields take on an even more important explanatory role. As the developmental biologist Scott Gilbert and his colleagues have argued:

> Homologous developmental pathways . . . are seen in numerous embryonic processes, and they are seen occurring in discrete regions, the morphogenetic fields. These fields (which exemplify the modular nature of developing embryos) are proposed to mediate between genotype and phenotype. Just as the cell (and not its genome) functions as the unit of organic structure and function, so the morphogenetic field (and not the genes or the cells) is seen as a major unit of ontogeny [development] whose changes bring about changes in evolution.[41]

So what are these fields and how do they work? These questions cannot be answered without looking at the bigger question of the causation of form.

3

■

THE CAUSES OF FORM

3.1 The problem of form

It is not immediately obvious that form presents any problem at all. The world around us is full of forms; we recognize them in every act of perception. But we easily forget that there is a vast gulf between this aspect of our experience, which we simply take for granted, and the quantitative factors with which physics concerns itself: mass, momentum, energy, temperature, pressure, electric charge, etc.[1]

The relationships between the quantitative factors of physics can be expressed mathematically, and physical changes can be described by means of equations. The construction of these equations is possible because fundamental physical quantities are conserved according to the Principles of Conservation of Mass and Energy, Momentum, Electric Charge, etc.: the total amount of mass and energy, momentum, electric charge, etc. before a given physical change equals the total amount afterward. But form does not enter into these equations: it is not a vector or scalar quantity, nor is it conserved. If a bunch of flowers is thrown into a furnace and reduced to ashes, the total amount of matter and energy remains the same, but the form of the flowers simply disappears.

Physical quantities can be measured with instruments to a high degree of accuracy. But forms cannot be measured on a quantitative scale, nor do they need to be, even by scientists. In this context, the word *form* is taken to include not only the shape of the outer surface or boundary of a system, but

also its internal structure. A botanist does not measure the difference between two species on the dial of an instrument; nor does an entomologist recognize butterflies by means of a machine; nor an anatomist bones; nor a histologist cells. All these forms are recognized directly. Then specimens of plants are preserved in herbaria, butterflies and bones in cabinets, and cells on microscope slides. As forms they are simply themselves; they cannot be reduced to anything else.

The description and classification of forms is the primary concern of many branches of science; even in a physical science such as chemistry, a major objective is the elucidation of the forms of molecules, represented diagrammatically in two-dimensional "structural formulae" or in three-dimensional models of the "ball and stick" type.

The forms of all but the simplest systems can be represented only visually, whether by photographs, drawings, diagrams, or models. They cannot be represented mathematically. Even the most advanced topological methods are not yet sufficiently developed to be capable of providing a mathematical formula for, say, a giraffe or an oak tree.[2]

If the mere description of any but the simplest static forms presents a mathematical problem of appalling complexity, the description of change of form—of morphogenesis—is even more difficult. This is the subject of René Thom's "catastrophe theory," which classifies and describes in general terms the possible types of change of form, or "catastrophe." He applies this theory to the problems of morphogenesis by constructing mathematical models in which the end or goal of a morphogenetic process, the final form, is represented by an attractor within a morphogenetic field. He postulates that every object, or physical form, can be represented by such an attractor and that all morphogenesis "can be described by the disappearance of the attractors representing the initial forms, and their replacement by capture by the attractors representing the final forms."[3]

In order to develop topological models that correspond to particular morphogenetic processes, formulae are found by a combination of trial and error and inspired guesswork. If a mathematical expression gives too many solutions, restrictions have to be introduced into it; and if a function is too restricted, a more generalized function is used instead. By methods such as these, Thom hoped that it would eventually be possible to develop topological expressions, which correspond in detail to actual morphogenetic

processes. But even so, these models would probably not enable quantitative predictions to be made. Their main value might lie in drawing attention to formal analogies between different types of morphogenesis.[4]

At first sight, the mathematical formalism of Information Theory may seem preferable to this topological approach. But in fact Information Theory is severely limited in its scope. It was originally developed by telephone engineers in connection with the transmission of messages from a source, through a channel, to a receiver; it was primarily concerned with the question of how the characteristics of a channel influence the amount of information that can be transmitted in a given time. One of the basic results is that in a closed system, no more information can be transmitted to the receiver than was contained in the source, although the form of the information can be changed, for example from the dots and dashes of Morse code to words. The information content of an event is defined not by what has happened, but only with respect to what might have happened instead. For this purpose binary symbols are usually used, and then the information content of a pattern is determined by finding out how many yes or no decisions are needed to specify which particular class of a pattern out of a known number of classes has occurred.

In biology this theory has some relevance to the quantitative study of the transmission of impulses by nerve fibers; to a lesser extent it has a bearing on the transmission of a sequence of bases in the DNA of parents to the DNA of their offspring, although even in such a simple case as this it can be seriously misleading, because in living organisms things happen that do not occur in telephone wires: genes mutate, parts of chromosomes undergo inversions, translocations, etc. But Information Theory is not relevant to biological morphogenesis: it applies only to the transmission of information within closed systems, and it cannot allow for an increase in the content of information during this process.[5] Developing organisms are not closed systems, and as they develop the complexity of form and organization increases. Although biologists often speak of “genetic information” and “positional information” as if these terms have some well-defined meaning, this is an illusion: they borrow only the jargon of Information Theory and leave its rigor behind.

However, even if impressively detailed mathematical models of morphogenetic processes could be made by whatever method, and even if

they gave rise to predictions that agreed with experimental evidence, there would still be the question of what these models correspond to. Indeed the same question is raised by the correspondence between mathematical models and empirical observations in any branch of science.

One answer is provided by mathematical mysticism of the Pythagorean type: the universe is dependent upon a fundamental mathematical order that somehow gives rise to all empirical phenomena; this transcendent order is revealed and becomes comprehensible only through the methods of mathematics. Although this attitude is rarely advocated explicitly, it has a strong influence within modern science, and can often be found, more or less thinly disguised, among mathematicians and physicists.[6]

Alternatively, the correspondence can be explained by the tendency of the mind to seek and find order in experience: the ordered structures of mathematics, creations of the human mind, are superimposed onto experience, and those that do not fit are discarded; thus by a process resembling natural selection, those mathematical formulae that fit best are retained. In this view, scientific activity is concerned only with the development and empirical testing of mathematical models of more or less isolated and definable aspects of the world; it cannot lead to any fundamental understanding of reality.

However, in relation to the problem of form, there is a different approach that requires neither an acceptance of Pythagorean mysticism nor the abandoning of the possibility of explanation. If the forms of things are to be understood, they need not be explained in terms of *numbers,* but in terms of more fundamental *forms*. Plato considered that the forms in the world of sense-experience were like imperfect reflections of transcendent, archetypal Forms or Ideas. But this doctrine, strongly influenced by the mysticism of the Pythagoreans, failed to explain how the eternal Forms were related to the changing world of phenomena. Aristotle believed this problem could be overcome by regarding the forms of things as immanent, rather than transcendent: specific forms were inherent in the souls of living beings and actually *caused* them to take up their characteristic forms.

In Driesch’s system, which was explicitly based on that of Aristotle, the specific forms of living organisms were caused by a nonenergetic agency, entelechy. The morphogenetic fields and chreodes of organismic biologists

play a similar role in guiding morphogenetic processes toward specific final forms. But their nature has so far remained obscure.

This obscurity may be due, in part, to the Platonic tendency of much organismic thought,[7] most clearly apparent in Whitehead's system of philosophy. Whitehead postulated that all actual events involved what he called Eternal Objects; the latter collectively made up the realm of possibility, and included all possible forms; indeed, they strongly resembled Platonic Forms.[8] But clearly, a metaphysical notion of morphogenetic fields as aspects of Platonic Forms or Eternal Objects would be of little value to experimental science. Only if they are regarded as physical entities that have physical effects can they help to provide a scientific understanding of morphogenesis.

The organismic philosophy embraces both biology and physics; hence, if morphogenetic fields are assumed to play a causal role in biological morphogenesis, they should also play a causal role in the morphogenesis of simpler systems such as crystals and molecules. Such fields are not recognized in the existing theories of physics. Therefore it is important to consider to what extent these existing theories are capable of explaining the morphogenesis of purely chemical systems. If they are able to provide an adequate explanation, then the idea of morphogenetic fields is unnecessary; but if they are not, the way lies open for a new hypothesis of the causation of form through morphogenetic fields in both biological and nonbiological systems.

3.2 Form and energy

In Newtonian physics, all causation was seen in terms of energy, the principle of movement and change. All moving things have energy— the kinetic energy of moving bodies, thermal vibration and electromagnetic radiation—and this energy can cause other things to move. Static things may also have energy—potential energy—due to their tendency to move; they are static only because they are restrained by forces that oppose this tendency.

Gravitational attraction was thought to depend on a force that acted at a distance causing bodies to move, or giving them a tendency to move, a

potential energy. However, no reason could be given for the existence of this attractive force itself.

Gravitational as well as electromagnetic effects are now explained in terms of *fields*. Whereas Newtonian forces were supposed to arise in some unexplained way from material bodies and to spread out from them into space, in modern physics the fields are primary: they underlie both material bodies and the space between them.

This picture is complicated by the fact that there are several different types of field. First, the gravitational field, which in Einstein's General Theory of Relativity is equated with space-time, and is curved in the presence of matter. Second, the electromagnetic field, within which electrical charges are localized, and through which electromagnetic radiations propagate as vibrational disturbances. According to quantum theory, these disturbances are particle-like photons associated with discrete quanta of energy. Third, in the quantum field theory of matter, subatomic particles are quanta of excitation of matter fields. Each kind of particle has its own field: a proton is a quantum of the proton-antiproton field, an electron a quantum of the electron-positron field, and so on.

In these theories, physical phenomena are explained by a combination of fields and of energy, not in terms of energy alone. Thus although energy can be regarded as the cause of change, the *ordering* of change depends on the spatial structure of the fields. These structures have physical effects, but they are not in themselves a type of energy; they act as "geometrical" or spatial causes. The radical difference between this idea and the notion of exclusively energetic causation is illustrated in the contrast between Newton's and Einstein's theories of gravitation: according to Newton, the Moon moves around the Earth because it is pulled toward it by an attractive force; according to Einstein, it does so because the very space in which it moves is curved.

The modern understanding of the structure of chemical systems depends on the concepts of quantum mechanics and of electromagnetism; gravitational effects are very small by comparison and can be ignored. The possible ways in which the atoms can combine together are given by the Schrödinger equation of quantum mechanics, which enables the orbitals of electrons to be calculated in terms of probabilities; in the quantum field theory of matter these orbitals can be regarded as structures within the

electron-positron field. But since electrons and atomic nuclei are electrically charged, they are also associated with spatial patterns within the electromagnetic field, and hence with potential energies. Not all the possible spatial arrangements of a given number of atoms have the same potential energy, and only the arrangement with the lowest potential energy will be stable, for reasons indicated in figure 6. If a system is in a state that has a higher energy than possible alternative states, any small displacement (for example, due to thermal agitation) will cause it to move into another state (A). If it is in a state with a lower energy than possible alternatives, after small displacements it will return to this state, which is consequently stable (B). A system may also exist temporarily in a state that is not the most stable so long as it is not displaced above the level of a "barrier" (C); when this happens, it will move into a stabler, lower-energy state.

These energetic considerations determine which is the most stable state of a chemical structure, but they do not account for its spatial characteristics, which in figure 6 are represented by the slopes down which the ball rolls, and that act as barriers confining it. These depend on spatial patterns given by the fields of matter and electromagnetism.

According to the second law of thermodynamics, spontaneous processes within a closed system tend toward a state of equilibrium; as they do so, initial differences in temperature, pressure, etc., between different parts of the system tend to disappear. In technical language, the entropy of a closed macroscopic system either stays the same or increases.

The significance of this law is often exaggerated in popular accounts; in particular, the term *entropy* is treated as if it was synonymous with *disorder.* Then the increasing complexity of organization that occurs in the evolution and development of living organisms appears to contradict the principle of increasing entropy. This confusion arises from a misunderstanding of the limitations of classical thermodynamics. First, it applies only to closed systems, whereas living organisms are open systems, exchanging matter and energy with their environment. Second, it deals only with the interrelations between heat and other forms of energy: it is relevant to the energetic factors that affect chemical and biological structures, but does not account for the existence of these structures in the first place. And third, the technical definition of entropy bears little relation to any nontechnical conception of disorder; in particular, it is not concerned with the type of

order inherent in the specific structures of chemical and biological systems. According to the third law of thermodynamics, at absolute zero the entropies of all pure crystalline solids are zero. They are perfectly “ordered” from a thermodynamic point of view because there is no disorder due to thermal agitation. But all are equally ordered: there is no difference in entropy between a simple salt crystal and a crystal of a complex macromolecule such as hemoglobin. It follows that the greater structural complexity of the latter is not measurable in terms of entropy.

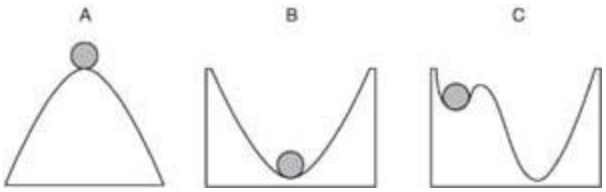

Figure 6. A diagrammatic representation of unstable (A), *stable* (B), *and partially stable* (C) *states.*

The contrast between “order” in the sense of chemical or biological structure and thermodynamic “order” owing to inequalities of temperature, etc., in a large system containing countless atoms and molecules is illustrated by the process of crystallization. If a solution of a salt is placed in a dish inside a cold enclosure, the salt crystallizes as the solution cools. Initially, its constituent ions are redistributed at random within the solution, but as crystallization takes place, they become ordered with great regularity within the crystals, and the crystals themselves develop into macroscopically symmetrical structures. From a morphological point of view, there has been a considerable increase in order; but from a thermodynamic point of view, there has been a decrease in “order,” an increase in entropy, owing to the equalization of temperature between the solution and its surroundings, and to the release of heat during the process of crystallization.

Similarly, when an animal embryo grows and develops, there is an increase in entropy of the thermodynamic system consisting of the embryo and the environment from which it takes its food and to which it releases heat and excretory products. The second law of thermodynamics serves to emphasize this dependence of living organisms on external sources of energy, but it does nothing to explain their specific forms.

In the most general terms, form and energy bear an inverse relationship to each other: energy is the principle of change, but a form or structure can

exist only as long as it has a certain stability and resistance to change. This opposition is clearly apparent in the relationship between the states of matter and temperature. Under sufficiently cool conditions, substances exist in crystalline forms in which the arrangements of the molecules show a high degree of regularity and order. As the temperature is raised, at a certain point the thermal energy causes the crystalline form to disintegrate; the solid melts. In the liquid state, the molecules arrange themselves in transient patterns that continually shift and change. The forces between the molecules create a surface tension that imparts simple forms to the liquid as a whole, as in spherical drops. With a further rise in temperature, the liquid vaporizes; in the gaseous state, the molecules are isolated and behave more or less independently of each other. At higher temperatures still, the molecules themselves disintegrate into atoms, and at yet higher temperatures, even the atoms break up to give a mixed gas of electrons and atomic nuclei— a plasma.

When this sequence is reversed, more-complex and -organized structures appear as the temperature is reduced, the most stable ones first and the least stable ones last. As a plasma cools, electrons congregate around atomic nuclei in their appropriate orbitals. At lower temperatures, atoms come together into molecules. Then as the gas condenses into droplets, supramolecular forces come into play. Finally, when the liquid crystallizes, a high degree of supra-molecular order is established.

These forms appear spontaneously. They cannot be explained in terms of external energy, except negatively in the sense that they can come into being and persist only below a certain temperature. They can be explained in terms of internal energy only to the extent that out of all the possible structural arrangements, only the one with the lowest potential energy will be stable; this is therefore the structure that will spontaneously tend to be taken up.

3.3 The structures of crystals

Quantum mechanics can describe in detail the electronic orbitals and the energy states of the simplest of all chemical systems, the hydrogen atom. With more complicated atoms and simple molecules, its methods are no longer so precise; the complexity of the calculations becomes formidable.

For complex molecules and crystals, detailed calculations are impossible. The structures of the molecules and the atomic arrangements within crystals can be found out empirically by chemical and crystallographic methods; these structures may indeed be more or less predictable by chemists and crystallographers on the basis of empirical laws. But this is a very different matter from providing a fundamental explanation of chemical structures by means of the Schrödinger wave equation.

It is important to realize this severe limitation of quantum mechanics. Certainly it helps to provide a qualitative or semi-quantitative understanding of chemical bonds and of certain aspects of crystals, such as the difference between insulators and electrical conductors. But it does not enable the forms and properties of even simple molecules and crystals to be predicted from first principles. The situation is even worse with regard to the liquid state, of which there is still no satisfactory quantitative account. And it is illusory to imagine that quantum mechanics in any detailed or rigorous way explains the forms and properties of the very complex molecules and macromolecular aggregates studied by biochemists and molecular biologists, not to mention the vastly greater complexity of form and properties of even the simplest living cell.

So widespread is the assumption that chemistry provides a firm foundation for the mechanistic understanding of life that it is perhaps necessary to emphasize on what very slender foundations of physical theory chemistry itself rests. In the words of Linus Pauling:

> We may believe the theoretical physicist who tells us that all the properties of substances should be calculable by known methods— the solution of the Schrödinger equation. In fact, however, we have seen that during the 30 years since the Schrödinger equation was discovered only a few accurate non-empirical quantum-mechanical calculations of the properties of substances in which the chemist is interested have been made. The chemist must still rely upon experiment for most of his information about the properties of substances.[9]

In the fifty years since this passage was published, there have been important improvements in approximate methods of calculation available to quantum chemists, as well as huge advances in computing power. It is now

possible to compute some of the chemical properties of simple molecules like carbon monoxide (CO), and with more approximate methods several quantitative properties of molecules like methane (CH4) and ammonia (NH3).[10] But it is still true that chemists must rely on empirical observations, rather than calculation, for most of their information on the properties and structures of molecules.

Nevertheless, it may be argued that the detailed calculations could be carried out in principle. But even assuming, for the purpose of argument, that these calculations could indeed be performed, it cannot be known in advance that they will be *correct,* that is to say agree with empirical observations. So at present there is no evidence for the conventional assumption that complex chemical molecules and biological structures can be fully explained in terms of existing physical theory.

The reasons for the difficulty, if not impossibility, of predicting the form of a complex chemical structure on the basis of the properties of its constituent atoms can be understood more clearly by means of a simple illustration. Consider elementary building blocks that can be added to each other one at a time either endways or sideways (see figure 7). With two building blocks there are $2^2 = 4$ possible combinations; with three, $2^3 = 8$; with four, $2^4 = 16$; with five $2^5 = 32$; with ten, $2^{10} = 1{,}024$ with twenty, $2^{20} = 1{,}048{,}576$; with 30, $2^{30} = 1{,}073{,}741{,}824$; and so on. The number of possibilities soon becomes enormous.

In a chemical system, the different possible arrangements of atoms have different potential energies owing to the electrical and other interactions between them; the system will spontaneously tend to take up the structure with the minimum potential energy. In a simple system with only a few possible structures, one may have a distinctly lower energy than the others; in figure 8A this is represented by the minimum at the bottom of the “potential well”; other, less stable possibilities are represented by local minima on the side of the “well.” In systems of increasing complexity, the number of possible structures increases (figure 8B, C, D); as it does so, the chance of there being a unique minimum-energy structure diminishes.

In the situation represented by figure 8D, several different structures are equally stable from an energetic point of view. If the system was found to take up any of these possible structures at random, or if it oscillated

between them, then there would be no problem. But if it invariably took up only one of these structures, this would indicate that some factor other than energy somehow determined that this particular structure was realized rather than the other possibilities. No such factor is at present recognized by physics.

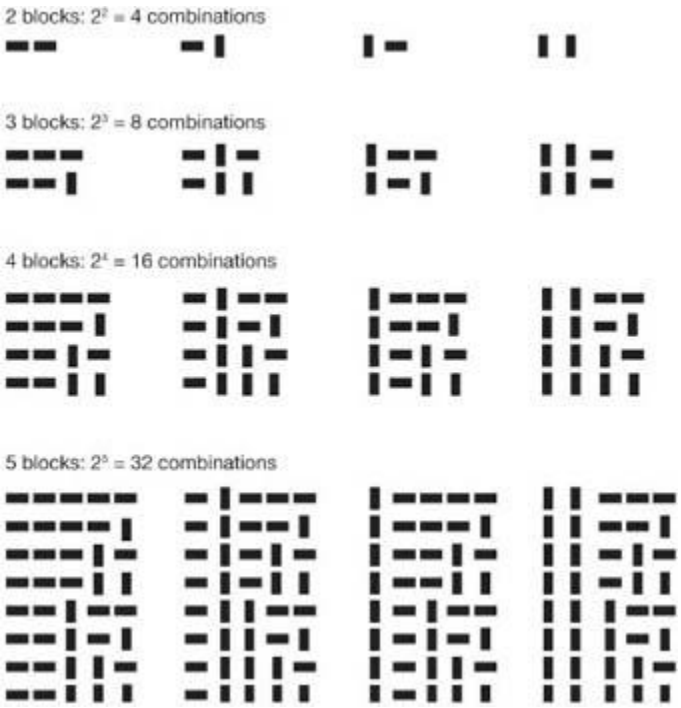

Figure 7. Possible combination of different numbers of building blocks capable of being joined together either endways or sideways.

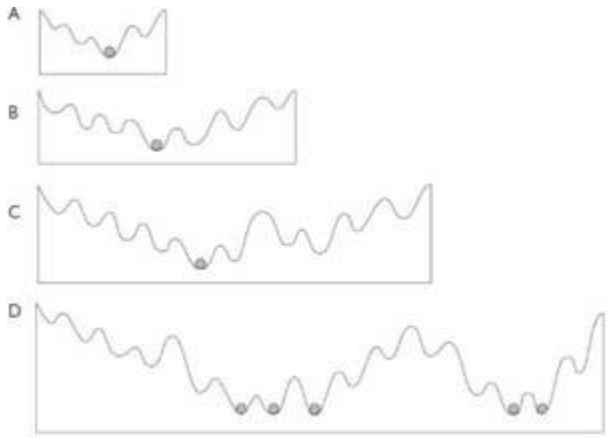

Figure 8. A diagrammatic representation of the possible structures of systems of increasing complexity. In A *there is a unique minimum-energy structure, but in* D *several different possible structures are equally stable.*

Although chemists, crystallographers, and molecular biologists cannot carry out the detailed calculations necessary to predict the minimum-energy structure or structures of a system a priori, they are able to use various approximate methods in combination with empirical data on the structures of similar substances. In general, these calculations do not permit unique structures to be predicted (except for the simplest of systems), but only a range of possible structures with more or less equal minimum energies. Thus, these approximate results support the idea that energetic considerations are insufficient to account for the unique structure of a complex chemical system. But this conclusion can always be avoided by reasserting that the unique stable structure must have a lower energy than any other possible structure. This assertion could never be falsified because in practice only approximate methods of calculation can be used; the unique structure actually realized could therefore always be attributed to subtle energetic effects that eluded calculation. The following discussion of Pauling's illustrates the situation with regard to the structure of inorganic crystals.

> Simple ionic substances such as the alkali halogenides have little choice of structure; and a very few relatively stable ionic arrangements corresponding to the formula $M^+ X^-$ exist, and the various factors that influence the stability of the crystal are pitted against one another, with no one factor necessarily finding clear expression in the decision between the sodium chloride and the caesium chloride arrangements. For a complex substance, such as mica, $KAl_3Si_3O_{10}(OH)_2$, or zunyite, $Al1_3Si_5O_{20}(OH)_{18}C1$, on the other hand, many conceivable structures differing only slightly in nature and stability can be suggested, and it might be expected that the most stable of these possible structures, the one actually assumed by the substance, will reflect in its various features the different factors that are of significance in determining the structure of ionic crystals. It has been found possible to formulate a set of rules about the stability of complex ionic crystals . . . These rules were obtained in part by induction from the structures known in 1928, and in part by deduction from the equations of crystal energy. They are not rigorous in their derivation nor universal in their application, but they have been found useful as a criterion for the probable correctness of reported structures for complex crystals and an aid to X-ray

> investigation of crystals by making possible the suggestion of reasonable structures for experimental test.[11]

As John Maddox, the late editor of *Nature*, expressed it in 1988: “One of the continuing scandals in the physical sciences is that it remains impossible to predict the structure of even the simplest crystalline solids from their chemical composition.”[12]

There have been enormous advances in computing power since 1988, but it is still the case that most predictions of crystal structure rely on knowing the structures of similar substances. With the use of various approximations, better a priori predictions are now possible, but they still run into the inevitable problem of multiple minimum-energy structures. A review published in 2004 summarized the situation as follows: “The main problem seems to be not so much a matter of generating stable crystal structures but rather of selecting one or more possible structures from very many almost equienergetic candidates. For example, even for a simple molecule such as benzene, with only one known crystal structure at normal pressure, calculations yield at least 30 possible crystal structures.”[13]

A series of Crystal Structure Prediction Workshops, organized by the Cambridge Crystallographic Data Centre in 1999, 2001, 2004, and 2007, were designed to evaluate present methods of calculation. Competing teams were given the molecular formulae of several simple organic chemicals and asked to predict their crystal structures “blind.” The actual structures of the crystals were known to the organizers, but were unpublished. In 2004, the results “were not marked by spectacular success.” But “many groups found the experimental structure somewhere in their list of possible low-energy structures. Thus, present methods are capable, if not of predicting the experimental structure(s) *a priori*, then at least of providing a set of structures as possible polymorphs.”[14]

In the 2007 contest there was a major advance: one team correctly predicted the structure of all four test molecules. They used a two-stage process to select the three most probable minimum-energy structures, one of which was the right one. But the molecules involved were very small, with between eight and thirty-three atoms.[15]

3.4 The structures of proteins

The range of possible structures becomes enormous with large molecules, especially proteins. Even a small protein like insulin contains nearly eight hundred atoms, and the largest have hundreds of thousands of atoms. The polypeptide chains twist, turn, and fold into complicated three-dimensional forms (figure 9). Under conditions in which a given type of protein molecule is stable, it folds up into a unique structure.

In numerous experimental studies, proteins have been made to unfold to varying degrees by changing their chemical environment; they then fold up again into their normal structure when they are replaced in appropriate conditions. In spite of starting from different initial states and following different "pathways" of folding, they reach the same structural end point.[16]

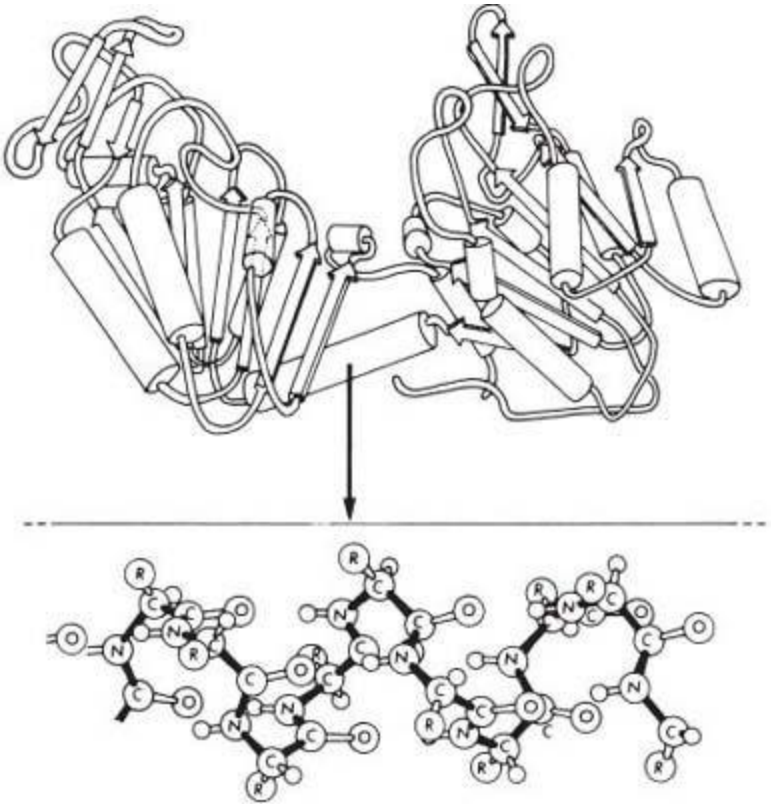

Figure 9. Above: The structure of the enzyme phosphoglycerate kinase, isolated from horse muscle; α-helices are represented by cylinders and β-strands by arrows. Below: The structure of an α-helical region in more detail. (After Banks et al., 1979)

This stable end point is likely to be a minimum-energy structure. But this does not prove that it is the only possible structure with a minimum energy;

there may be many other possible structures with the same minimum energy. Indeed, calculations to predict the three-dimensional structure of proteins, starting from the linear sequence of amino acids coded for by DNA, give far too many solutions. In the literature on protein folding, this is known as the "multiple-minimum problem."[17]

In an ongoing series of workshops on predicting protein structures, held under the aegis of the Lawrence Livermore National Laboratory in California, teams from all over the world try to predict the three-dimensional structure of proteins working blind, as in the Crystal Structure Workshops. These evaluations are called the Critical Assessment of Techniques for Protein Structure Prediction (CASP). By far the most successful predictions are based on a detailed knowledge of similar proteins, known as comparative modeling. The CASP competitions used to include an *ab initio* category, implying that the predictions started from first principles, but for CASP6 in 2004, the name of the category was changed: "This name implies that there is no reliance on known structures in building models. In practice, most of the methods used for such targets do make extensive use of available structural information, both in devising scoring functions to distinguish between correct and incorrect predictions, and in choosing fragments to incorporate in the model. For this reason, the category was renamed as new folds."[18]

Nevertheless, even using knowledge about similar proteins, the multiple-minimum problem will not go away. This was the situation in 2004: "As in crystal structure prediction . . . the problem of selecting the most stable tertiary structure from many almost equienergentic forms is a severe one."[19]

There are persuasive reasons for thinking that the protein itself does not "test" all these minima until it finds the right one.

> If the chain explored all possible configurations at random by rotations about the various single bonds of the structure, it would take too long to reach the native configuration. For example, if the individual residues of an unfolded polypeptide chain can exist in only two states, which is a gross underestimate, then the number of possible randomly generated conformations is 10^{45} for a chain of 150 amino acid residues (although, of course, most of these would probably be sterically impossible ones). If each conformation could be explored with a

> frequency of a molecular rotation (10^{12} sec^{-1}), which is an overestimate, it would take approximately 10^{26} years to examine all possible conformations. Since the synthesis and folding of a protein chain such as that of ribonuclease or lysozyme can be accomplished in about 2 minutes, it is clear that all conformations are not traversed in the folding process. Instead, it appears to us that, in response to local interactions, the peptide chain is directed along a variety of possible low-energy pathways (relatively small in number), possibly passing through unique intermediate states, toward the conformation of lowest free energy.[20]

But not only may the folding process be "directed" along certain pathways, it may also be directed toward one particular conformation of minimum energy, rather than any other possible conformations with the same minimum energy.

This discussion leads to the general conclusion that the existing theories of physics may well be incapable of explaining the unique structures of complex molecules and crystals; they permit a range of possible minimum-energy structures to be suggested, but there is no evidence that they can account for the fact that one rather than another of these possible structures is realized. It is therefore conceivable that some factor other than energy "selects" between these possibilities and thus determines the specific structure taken up by the system.[21] The hypothesis that will now be developed is based on the idea that this "selection" is brought about by a new type of causation, at present unrecognized by physics, through the agency of morphogenetic fields.

3.5 Formative causation

The hypothesis of formative causation proposes that morphogenetic fields play a causal role in the development and maintenance of the forms of systems at all levels of complexity. This suggested causation of form by morphogenetic fields is called formative causation in order to distinguish it from the energetic type of causation with which physics already deals so thoroughly.[22] Although morphogenetic fields can only bring about their

effects in conjunction with energetic processes, they are not in themselves energetic.

The idea of nonenergetic formative causation is easier to grasp with the help of an architectural analogy. In order to construct a house, bricks and other building materials are necessary; so are the builders who put the materials into place; and so is the architectural plan that determines the form of the house. The same builders doing the same amount of work using the same quantity of building materials would produce a house of different form with a different plan. Thus the plan can be regarded as a cause of the specific form of the house, although of course it is not the only cause: it could never be realized without the building materials and the activity of the builders. Similarly, a specific morphogenetic field is a cause of the specific form taken up by a system, although it cannot act without suitable "building blocks" and without the energy necessary to move them into place.

This analogy is not intended to suggest that the causative role of morphogenetic fields depends on conscious design, but only to emphasize that not all causation need be energetic. The plan of a house is not in itself a type of energy. Instead, it is a kind of information. Even when it is drawn on paper, or finally realized in the form of the house, it does not weigh anything or have any energy of its own. If the paper is burned or the house is demolished, there is no measurable change in the total amount of mass and energy; the plan simply vanishes. Likewise, according to the hypothesis of formative causation, morphogenetic fields are not in themselves energetic; but nevertheless they play a causal role in determining the forms of the systems with which they are associated. If a system were associated with a different morphogenetic field, it would develop differently.[23] This hypothesis is empirically testable in cases where the morphogenetic fields acting on systems can be altered (sections 5.6, 7.4, 7.6, 7.9, 11.2, and 11.4).

Morphogenetic fields can be regarded as analogous to the known fields of physics in that they are capable of ordering physical changes, even though they themselves cannot be observed directly. Gravitational and electromagnetic fields are spatial structures that are invisible, intangible, inaudible, tasteless, and odorless; they are detectable only through their respective gravitational and electromagnetic effects. In order to account for the fact that physical systems influence each other at a distance without any

apparent material connection between them, these hypothetical fields are endowed with the property of traversing empty space, or even actually constituting it. In one sense they are nonmaterial; but in another sense they are aspects of matter because they are known through their effects on material systems. In effect, the scientific definition of matter has simply been widened to take them into account. Similarly, morphogenetic fields are spatial structures detectable only through their morphogenetic effects on material systems; they too can be regarded as aspects of matter if the definition of matter is widened still further to include them.

Although in the preceding sections only the morphogenesis of biological and complex chemical systems has been discussed, the hypothesis of formative causation applies to biological and physical systems at all levels of complexity. Since each kind of system has its own characteristic form, each must have a specific kind of morphogenetic field: thus there must be one kind of morphogenetic field for protons; another for nitrogen atoms; another for water molecules; another for sodium chloride crystals; another for the muscle cells of earthworms; another for the kidneys of sheep; another for elephants; another for beech trees; and so on.

According to the organismic theory, systems or "organisms" are hierarchically organized at all levels of complexity.[24] In the present discussion, these systems will be referred to as morphic units. The adjective *morphic* (from the Greek root *morphe* = form) emphasizes the aspect of structure, and the word *unit* the unity or wholeness of the system. In this sense, chemical and biological systems are composed of hierarchies of morphic units: a crystal, for example, contains molecules, which contain atoms, which contain subatomic particles. Crystals, molecules, atoms, and subatomic particles are morphic units, as are animals and plants, organs, tissues, cells, and organelles. Simple examples of this hierarchical type of organization can be visualized diagrammatically either as a "tree" or as a series of "Chinese boxes" (figure 10).

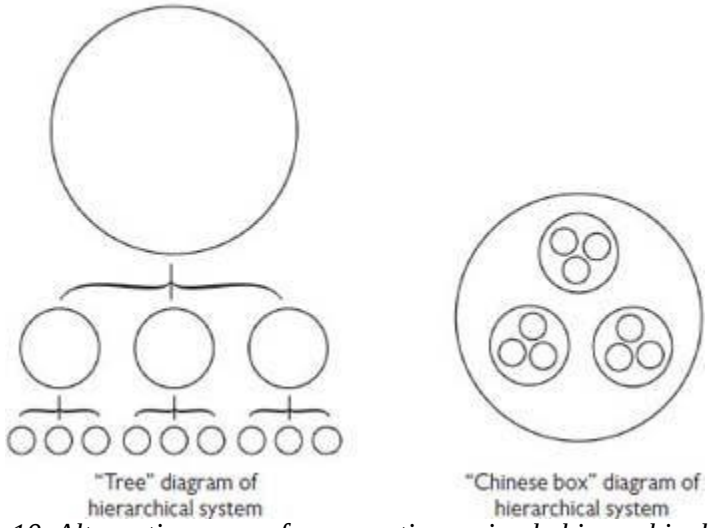

Figure 10. Alternative ways of representing a simple hierarchical system.

A higher-level morphic unit must somehow coordinate the arrangement of the parts, or modules, of which it is composed. It does so through the influence of its morphogenetic field on the morphogenetic fields of lower-level morphic units. Thus morphogenetic fields, like morphic units themselves, are essentially hierarchical in their organization.

The way in which morphogenetic fields might act upon the systems under their influence is discussed in the following chapter; and the question of where they come from and what gives them their specific structure is discussed in chapter 5.

4

■

MORPHOGENETIC FIELDS

4.1 Morphogenetic germs

Morphogenesis does not take place in a vacuum. It can only begin from an already organized system that serves as a *morphogenetic germ*. During morphogenesis a new higher-level morphic unit comes into being around this germ, under the influence of a specific morphogenetic field. So how does this field become associated with the morphogenetic germ to start with?

The answer may be that just as the association of material systems with gravitational fields depends on their mass, and with electromagnetic fields on their electrical charge, so the association of systems with morphogenetic fields depends on their form. Hence a morphogenetic germ becomes surrounded by a particular morphogenetic field because of its characteristic form.

The morphogenetic germ is a part of the system-to-be. Therefore part of the system's morphogenetic field corresponds to it. However, the rest of the field is not yet "occupied" or "filled out"; it contains the *virtual form* of the final system, which is actualized only when all its material parts have taken up their appropriate places. The morphogenetic field is then in coincidence with the actual form of the system.

These processes are represented diagrammatically in figure 11A. The stippled areas indicate the virtual form and the solid lines the actual form of the system. The morphogenetic field can be thought of as a structure

surrounding or embedding the morphogenetic germ and containing the virtual final form; this field then orders events within its range of influence in such a way that the virtual form is actualized.

In the absence of the morphic units that constitute the parts of the final system, this field is undetectable; it reveals itself only through its ordering effects on these parts when they come within its influence. A rough analogy is provided by the “lines of force” in the magnetic field around a magnet; these spatial structures are revealed when particles capable of being magnetized, such as iron filings, are introduced into the vicinity. Nevertheless, the magnetic field exists even when the iron filings are absent; likewise, the morphogenetic field around a morphogenetic germ exists as a spatial structure even though it has not yet been actualized in the final form of the system. However, morphogenetic fields differ radically from electromagnetic fields in that the latter depend on the *actual* state of the system—on the distribution and movement of charged particles—whereas morphogenetic fields correspond to the *potential* state of a developing system and are already present before it takes up its final form.[1]

In figure 11A, there are several intermediate stages between the morphogenetic germ and the final form. The final form could also be reached by a different morphogenetic pathway (figure 11B), but if a particular pathway is usually followed, this can be regarded as a canalized pathway of change, or chreode (cf. figure 5).

If the developing system is damaged by the removal of a part of it, it may still be able to reach the final form (figure 11C). This represents regulation. After the final form is actualized, the continued association between the morphogenetic field and the system whose form corresponds to it will tend to stabilize the latter. Any deviations of the system away from this form will tend to be corrected as the system is attracted back toward it. And if part of the system is removed, the final form will tend to be actualized again (figure 11D). This represents regeneration.

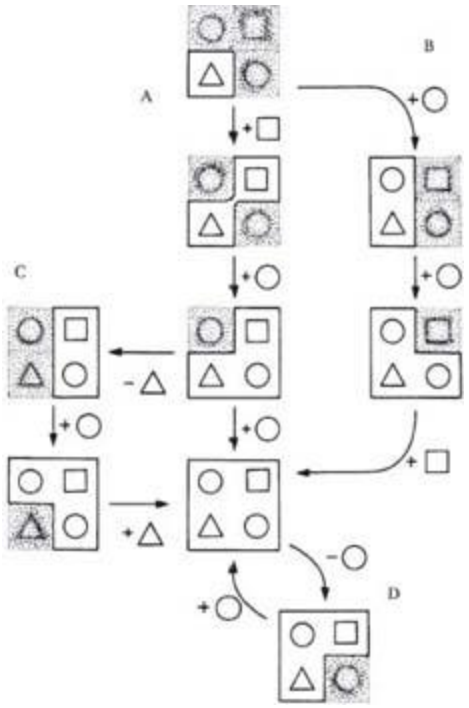

Figure 11. Diagrammatic representation of the development of a system from a morphogenetic germ (triangle) by the normal chreode, A. *An alternative morphogenetic pathway is represented by* B, *regulation by* C, *and regeneration by* D. *The virtual form within the morphogenetic field is indicated by the stippled area.*

The type of morphogenesis shown in figure 11 is essentially *aggregative:* previously separate morphic units come together into a higher-level morphic unit. Another type of morphogenesis is possible when the morphic unit that serves as the morphogenetic germ is already part of a different higher-level morphic unit. The influence of the new morphogenetic field leads to a *transformation* in which the form of the original higher-level morphic unit is replaced by the form of the new one. Most types of chemical morphogenesis are aggregative, whereas biological morphogenesis usually involves a combination of transformative and aggregative processes. Examples are considered in the following sections.

4.2 Chemical morphogenesis

Aggregative morphogeneses occur progressively in inorganic systems as the temperature is reduced: as a plasma cools, subatomic particles aggregate into atoms; at lower temperatures, atoms aggregate into molecules; then molecules condense into liquids; and finally liquids crystallize.

In the plasma state, hydrogen atoms split up into electrons and naked atomic nuclei. The nuclei can be regarded as the morphogenetic germs of atoms; they are associated with the atomic morphogenetic fields, which contain the virtual orbitals of electrons. In one sense these orbitals do not exist, but in another sense they have a reality that is revealed in the cooling plasma as they are actualized by the capture of electrons.

Electrons that have been captured within atomic orbitals may be displaced from them again through the influence of external energy, or by entering a virtual orbital of lower potential energy. In the latter case, they lose a discrete quantum of energy that is radiated as a photon. In atoms with many electrons, each orbital can contain only two electrons (with opposing spins); thus in a cooling plasma, the virtual orbitals with the lowest potential energies fill up with electrons first, then the orbitals with the next lowest energies, and so on until the complete atomic form has been actualized around the morphogenetic germ of the nucleus.

Atoms are in turn the morphogenetic germs of molecules, and small molecules the germs of larger molecules. Chemical reactions involve either the aggregation of atoms and molecules into larger molecules— for example in the formation of polymers—or the fragmentation of molecules into smaller ones, or into atoms and ions, which may then aggregate with others, for example in combustion: under the influence of external energy, molecules fragment into atoms and ions that then combine with those of oxygen to form small, simple molecules like H2O and CO2. These chemical changes involve the actualization of virtual forms associated with the atoms or molecules that act as morphogenetic germs.

The idea that molecules have virtual forms before they are actualized is illustrated by the familiar fact that entirely new compounds can first be “designed” on the basis of empirically determined principles of chemical combination and then actually synthesized by organic chemists. These laboratory syntheses are carried out step by step; in each step a particular molecular form serves as the morphogenetic germ for the next virtual form to be synthesized, ending up with the form of the entirely new molecule.

If it seems rather artificial to think of chemical reactions as morphogenetic processes, it should be remembered that much of the effect of catalysts, both inorganic and organic, depends on their morphology. For example, enzymes, the specific catalysts of the numerous reactions of

biochemistry, provide surfaces, grooves, notches, or basins into which the reacting molecules fit with a specificity that is often compared to that of a lock and key. The catalytic effect of enzymes depends to a large extent on the way in which they hold reactant molecules in the appropriate relative positions for reaction to occur. In free solution, the chance collisions of the molecules occur in all possible orientations, most of which are inappropriate.

The details of chemical morphogeneses are vague, partly because of their great rapidity, partly because the intermediate forms may be highly unstable, and also because the ultimate changes consist of probabilistic quantum jumps of electrons between the orbitals that constitute the chemical bonds. The virtual form of the molecule-to-be is outlined in the morphogenetic field associated with the atomic or molecular morphogenetic germ. When the other atom or molecule approaches in an appropriate orientation, the form of the product molecule is actualized by means of quantum jumps of electrons into orbitals that previously existed only as virtual forms; at the same time, energy is released, usually as thermal motion. The role of the morphogenetic field in this process is, as it were, energetically passive but morphologically active; it creates virtual structures that are then actualized as lower-level morphic units “slot” or “snap” into them, releasing energy as they do so.

Any given type of atom or molecule can take part in many different types of chemical reaction, and it is therefore the potential germ of many different morphogenetic fields. These fields could be thought of as possibilities “hovering” around it. However, the atom or molecule may not take on its role as the germ of a particular morphogenetic field until an appropriate reagent atom or molecule approaches it, perhaps owing to specific electromagnetic or other effects upon it.

The morphogenesis of crystals differs from that of atoms and molecules in that a particular pattern of atomic or molecular arrangement is repeated indefinitely. The morphogenetic germ is provided by this pattern itself. It is well known that the addition of “seeds” or “nuclei” of the appropriate type of crystal greatly accelerates the crystallization of supercooled liquids or supersaturated solutions. In the absence of these seeds or nuclei, morphogenetic germs of the crystal come into being only when the atoms or molecules take up their appropriate relative positions by chance, owing to

thermal agitation. Once the germ is present, the virtual forms of repetitions of the lattice structure given by the morphogenetic field extend outward from the surfaces of the growing crystal. Appropriate free atoms or molecules that approach these surfaces are captured and “slot” into position; again thermal energy is released as they do so.

The seeding or nucleation of supercooled liquids or supersaturated solutions can also be carried out, although less effectively, with small fragments of unrelated substances; for example, chemists often scratch the sides of test tubes to seed solutions with fragments of glass. These fragments provide surfaces that facilitate the appropriate relative positioning of the atoms or molecules that constitute the true morphogenetic germ of the crystal. In their morphogenetic effect, these seeds resemble the catalysts of chemical reactions.

All the types of chemical morphogenesis considered so far are essentially aggregative. Transformations are less common in nonliving systems. But crystals, for example, sometimes undergo transformation into other crystalline forms, as when carbon crystals in the form of graphite are transformed under high temperature and pressure into the diamond form. Molecules can also undergo transformations, as in the folding of proteins and the reversible changes of shape that occur when certain enzymes bind to the molecules whose reaction they catalyze.[2]

The fact that proteins fold up far more rapidly than would be expected if they found their final form by a random search indicates that their folding follows particular pathways, or a limited number of pathways (section 3.4). These “canalized pathways of change” can be regarded as chreodes. For the folding process to begin, according to the ideas developed in section 4.1, above, a morphogenetic germ must be present, and this germ must already have the characteristic three-dimensional structure that it has in the final form of the protein. The existence of such morphogenetic starting points has in fact already been suggested in the literature on protein folding.

> The extreme rapidity of the refolding makes it essential that the process takes place along a limited number of pathways . . . It becomes necessary to postulate the existence of a limited number of allowable initiating events in the folding process. Such events, generally referred to as nucleations, are most likely to occur in parts of the polypeptide

> chain that can participate in conformational equilibria between random and cooperatively stabilized arrangements . . . Furthermore it is important to stress that the amino acid sequences of polypeptide chains designed to be the fabric of protein molecules only make functional sense when they are in the three-dimensional arrangement that characterizes them in the native protein molecule. It seems reasonable to suggest that portions of a protein chain that can serve as nucleation sites for folding will be those that can "flicker" in and out of the conformation that they occupy in the final protein, and that they will form a relatively rigid structure stabilized by a set of cooperative interactions.[3]

Such "nucleation sites" would act as morphogenetic germs through their association with the morphogenetic field of the protein, which would then canalize the pathway of folding toward the characteristic final form.

4.3 Morphogenetic fields as "probability structures"

The orbitals of electrons around an atomic nucleus can be regarded as structures within the morphogenetic field of the atom. These orbitals can be described by solutions of the Schrödinger equation. However, according to quantum mechanics, the precise orbits of electrons cannot be specified, but only the probabilities of finding electrons at particular points; the orbitals are regarded as probability distributions in space. Within the context of the hypothesis of formative causation, this result suggests that just as these structures in the morphogenetic fields of atoms must be thought of as spatial probability distributions, so morphogenetic fields in general are not precisely defined, but are given by probability distributions.[4] It will be assumed that this is in fact the case, and the structures of morphogenetic fields will henceforth be referred to as *probability structures*.[5] An explanation for the probabilistic nature of these fields will be suggested in section 5.4.

The action of the morphogenetic field of a morphic unit on the morphogenetic fields of its parts, which are morphic units at lower levels (section 3.5), can be thought of in terms of the influence of this higher-level probability structure on lower-level probability structures; the higher-level field modifies the probability structures of the lower-level fields.

Consequently, during morphogenesis, the higher-level field modifies the probability of probabilistic events in the lower-level morphic units under its influence.[6]

In the case of free atoms, electronic events take place with the probabilities given by the unmodified probability structures of the atomic morphogenetic fields. But when the atoms come under the influence of the higher-level morphogenetic field of a molecule, these probabilities are modified in such a way that the probability of events leading toward the actualization of the final form is enhanced, while the probability of other events is diminished. Thus the morphogenetic fields of molecules restrict the possible number of atomic configurations that would be expected on the basis of calculations starting from the probability structures of free atoms. And this is what is found: in the case of protein folding, for example, the rapidity of the process indicates that the system does not explore the countless configurations in which the atoms could conceivably be arranged (section 3.4).

Similarly, the morphogenetic fields of crystals restrict the large number of possible arrangements that would be permitted by the probability structures of their constituent molecules; hence one particular pattern of molecular arrangement is taken up as the substance crystallizes, rather than any of the other conceivable structures.

Thus the morphogenetic fields of crystals and molecules are probability structures in the same sense as the electronic orbitals in the morphogenetic fields of atoms are probability structures. This conclusion agrees with the conventional assumption that there is no difference in kind between the description of simple atomic systems by quantum mechanics and a potential quantum mechanical description of more-complex forms. But unlike the hypothesis of formative causation, the conventional theory seeks to explain complex systems from the bottom up, as it were, in terms of the quantum mechanical properties of atoms.

The difference between these two approaches can be seen more clearly in a historical context. Quantum theory itself was primarily elaborated in connection with the properties of simple systems such as hydrogen atoms. As time went on, new fundamental principles were introduced in order to account for empirical observations such as those on the fine structure of the spectra of light emitted by atoms. The original quantum numbers

characterizing the discrete electronic orbitals were supplemented by another set referring to angular momentum, and then yet more referring to “spin.” The latter is considered to be an irreducible property of particles, just as electric charge is, and has its own conservation law. In nuclear particle physics, yet more irreducible factors, such as “strangeness” and “charm,” together with further conservation laws, have been introduced more or less ad hoc in order to account for observations not explicable in terms of the already accepted quantum factors. Moreover, the discovery of large numbers of new subatomic particles has led to the postulation of an ever-increasing number of new kinds of matter field.

When so many new physical principles and physical fields have been introduced in order to account for the properties of atoms and subatomic particles, the conventional assumption that no new physical principles or fields come into play at levels of organization above that of the atom seems remarkably arbitrary. It is in fact little more than a relic of nineteenth-century atomism; now that atoms are no longer regarded as ultimate and indivisible, its original theoretical justification has vanished. From the point of view of the hypothesis of formative causation, although the existing body of quantum theory, developed in connection with the properties of atoms and subatomic particles, sheds much light on the nature of morphogenetic fields, it cannot be extrapolated to describe the morphogenetic fields of more-complex systems. There is no reason why the morphogenetic fields of atoms should be considered to have a privileged position in the order of nature; they are simply the fields of morphic units at one particular level of complexity.

4.4 Probabilistic processes in biological morphogenesis

There are many examples of physical processes whose spatial outcomes are probabilistic. In general, changes involving the breaking of a symmetry or homogeneity are indeterminate; examples occur in the phase transitions between the gaseous and liquid states and between the liquid and solid states. If, for instance, a spherical balloon filled with vapor is cooled below saturation point in the absence of external gradients of temperature and gravity, the liquid will start by condensing on the walls, but the final distribution of the liquid will be unpredictable, and almost never spherically symmetrical.[7] Thermodynamically, the relative amounts of liquid and vapor

can be foreseen, but their spatial distribution cannot. In the crystallization of a substance under uniform conditions, the spatial distribution and the numbers and sizes of the crystals cannot be predicted; in other words, if the same process is to be repeated under identical conditions, each time the spatial outcome differs in detail.

The forms of crystals themselves, although they exhibit a definite symmetry, may be indeterminate; snowflakes, which come in myriad shapes, provide a familiar example.[8]

In the "dissipative structures" of macroscopic physical and chemical systems far from thermodynamic equilibrium, random fluctuations can give rise to spatial patterns, for example convection cells in a heated liquid or colored bands in solutions in which the Zhabotinski reaction is proceeding. The mathematical descriptions of such cases of "order through fluctuations" by the methods of nonequilibrium thermodynamics show striking analogies to phase transitions.[9]

These examples of spatial indeterminism are drawn from quite simple physical and chemical processes. In living cells, the physico-chemical systems are far more complex than any encountered in the inorganic realm, and include many potentially indeterminate phase transitions and nonequilibrium thermodynamical processes. In the protoplasm there are crystalline, liquid, and lipid phases in dynamic interrelation; then there are numerous types of macromolecules that come together into crystalline or quasi-crystalline aggregates; lipid membranes, which as "liquid crystals" hover on the borderline between the liquid and solid states, as do the colloidal sols and gels; electrical potentials across membranes that fluctuate unpredictably; and "compartments," containing different concentrations of inorganic ions and other substances, separated by membranes across which these substances move probabilistically.[10] With such complexity, the number of energetically possible patterns of change must be enormous, and there is thus a vast scope for the operation of morphogenetic fields through the imposition of patterns on these probabilistic processes.

This is not to say that *all* form in living organisms is determined by formative causation. Some patterns may come about through random processes.[11] Others may be fully explicable in terms of minimum-energy configurations: for instance, the spherical shape of free-floating egg cells

(e.g., those of sea urchins) may be fully explicable in terms of the surface tension of the cell membrane. Sir D'Arcy Thompson, in his classic essay *On Growth and Form* (1917), suggested that many aspects of biological morphogenesis could be explained in terms of physical forces: for example, the plane of cell division in terms of surface tension, which would tend to give a minimum surface area. But there are so many exceptions that these simple interpretations have met with very little success.[12] Other explanations are needed, like morphogenetic fields. It should be reemphasized that these fields do not act alone, but together with the energetic and chemical causes studied by biophysicists and biochemists.

One example of the way morphogenetic fields could operate within cells is provided by the positioning of microtubules, tiny rodlike structures formed by the spontaneous aggregation of protein subunits. Microtubules play an important role as microscopic "scaffolds" within both animal and plant cells: they guide and orientate processes such as cell division (the spindle fibers in mitosis and meiosis are made up of microtubules), and the patterned deposition of cell wall material in differentiating plant cells; they also serve as intracellular "skeletons," maintaining particular cellular shapes, as in radiolarians.[13] Now if the spatial distribution of microtubules is responsible for the patterning of many different sorts of process and structure within cells, then what controls the spatial distribution of the microtubules? If other patterns of organization are responsible,[14] the problem is simply pushed back one stage: what controls these patterns of organization themselves? But the problem cannot be pushed back indefinitely, because development involves an increase in spatial diversity and organization that cannot be accounted for in terms of preceding patterns or structures; sooner or later something else has to account for the emergence of the pattern in which the microtubules aggregate.

On the present hypothesis, this pattern is due to the action of specific morphogenetic fields. These fields greatly increase the probability of aggregation of microtubules in appropriate dispositions either directly or indirectly, through the establishment of a preceding pattern of organization. Obviously, the patterning activity of the fields depends on the presence of a supersaturated solution of microtubule subunits within the cell, and on appropriate physical and chemical conditions for their aggregation: these are necessary conditions for the formation of microtubules, but they are not

in themselves sufficient to account for the pattern in which the microtubules appear.

The objection might be raised that the suggested action of formative causation in patterning probabilistic processes within cells is impossible because it would lead to a local violation of the second law of thermodynamics. But this objection is not valid. The second law of thermodynamics refers only to assemblies of very large numbers of particles and not to processes on a microscopic scale. Moreover, it applies only to closed systems: a region of a cell is not a closed system, nor of course are living organisms in general.

In living organisms, as in the chemical realm, morphogenetic fields are hierarchically organized: those of organelles—for example, the cell nucleus, the mitochondria, and chloroplasts—act by ordering physicochemical processes within them; these fields are subject to the higher-level fields of cells; the fields of cells to those of tissues; those of tissues to those of organs; and of organs to the morphogenetic field of the organism as a whole. At each level the fields work by ordering processes that would otherwise be indeterminate. For example, at the cellular level the morphogenetic field orders the crystallization of microtubules and other processes that are necessary for the coordination of cell division. But the planes in which the cells divide may be indeterminate in the absence of a higher-level field: for instance, in plant wound calluses the cells proliferate more or less randomly to produce a chaotic mass.[15]

Within an organized tissue, on the other hand, one of the functions of the tissue's morphogenetic field may be to impose a pattern on the planes of cell division, and thus control the way in which the tissue as a whole grows. Then the development of tissues may itself be inherently indeterminate in many respects, as revealed when they are artificially isolated and grown in tissue culture;[16] under normal conditions this indeterminacy is restricted by the higher-level field of the organ. Indeed at each level in biological systems, as in chemical systems, the morphic units in isolation behave more indeterminately than they do when they are part of a higher-level morphic unit. The higher-level morphogenetic field restricts and patterns their intrinsic indeterminism.

4.5 Morphogenetic germs in biological systems

At the cellular level, the germs for morphogenetic transformations must be lower-level morphic units within the cells: they could be organelles, macromolecular aggregates, cytoplasmic or membranous structures, or the cell nuclei. In many cases nuclei might well play this role. But since so many different types of differentiated cell can be produced in the same organism, if the nuclei are to act as morphogenetic germs, they must be capable of taking on different patterns of organization in the different cell types: the differentiation of a cell must be preceded by a differentiation of its nucleus, owing to changes in its membrane, or in the arrangement of its chromosomes, or in the associations between proteins and nucleic acids within the chromosomes, or in the nucleoli, or in other components. Such changes could be brought about directly or indirectly through the action of the higher-level morphogenetic field of the differentiating tissue. There is indeed considerable evidence that many types of cellular differentiation are preceded by nuclear changes. The suggestion advanced here diverges from the usual interpretation of these changes in regarding their significance as not simply chemical, owing to the production of special types of messenger RNA, but, in addition, as morphogenetic: the modified nuclei might serve as germs with which the specific morphogenetic fields of differentiated cells become associated.[17]

There is at least one process of cellular morphogenesis in which the nucleus cannot be the morphogenetic germ: in nuclear division. It loses its identity as a separate structure when the nuclear membrane breaks down and disappears.[18] The doubled, highly coiled chromosomes become aligned in the equatorial region of the mitotic spindle and a complete set then moves to each spindle pole. Then new nuclear membranes develop around each set of chromosomes to form the daughter nuclei. The morphogenetic germs for these processes must be extra-nuclear structures or organelles, and there must be two of them. In animals the centrioles, barrel-shaped organelles whose walls are made up of microtubules, may appear to be likely candidates for this role, because they are located near the spindle poles of dividing cells, but higher plants have no centrioles. In both cases “microtubule organizing centers” may well be responsible for the development of the spindle poles; the centrioles may be merely “passengers” assured of equal partitioning into daughter cells by association

with these centers.[19] The centrioles serve as organizing centers, or morphogenetic germs, for the development of cilia and flagella, and this may be their primary role.

The development of tissues and organs usually involves both transformative and aggregative changes. In morphogenesis, the morphogenetic germs must be groups of cells that are present at the beginning of the morphogenetic process; they cannot be those specialized cells that appear only after the process has begun. Thus the morphogenetic germs are likely to be relatively unspecialized cells that undergo little change. In higher plants such cells are present, for example, in the apical zones of the meristems or growing points.[20] In shoots, the flowering stimulus transforms the meristems in such a way that they give rise to flowers rather than leaves; the apical zones, suitably modified by the flowering stimulus, are the morphogenetic germs for the formation of flowers. In animal embryos, embryologists have identified many "organizing centers" that play a key role in the development of tissues and organs; one example is the apical ectodermal ridge at the tip of developing limb buds.[21] These "organizing centers" may well be the germs with which the major morphogenetic fields become associated.

Although the presence of morphogenetic germs can be suggested, if not actually identified, in both the chemical and biological realms, much remains obscure, especially the reason for the particular form of each morphogenetic field and for the way in which it becomes associated with its germ. The consideration of these problems in the following chapter leads to a more complete hypothesis of formative causation that, although surprising and unfamiliar, is perhaps less difficult to understand.

5

THE INFLUENCE OF PAST FORMS

5.1 The constancy and repetition of forms

Time after time when atoms come into existence, electrons fill the same orbitals around the nuclei; atoms repeatedly combine to give the same molecular forms; again and again molecules crystallize into the same patterns; seeds of a given species give rise year after year to plants of the same appearance; generation after generation, spiders spin the same types of web. Forms come into being repeatedly, and each time each form is more or less the same as the previous versions. On this fact depends our ability to recognize, identify, and name things.

This constancy and repetition would present no problem if changeless physical laws or principles uniquely determined all forms. This assumption is implicit in the conventional theory of the causation of form. These fundamental physical principles are taken to be temporally prior to the actual forms of things: theoretically, the way in which a newly synthesized chemical will crystallize should be calculable before its crystals appear for the first time; likewise, the effects of a given mutation in the DNA of an animal or plant on the form of the organism should be predictable in advance. But such calculations have never been made; this comfortable assumption is untested, and is in practice untestable.

By contrast, according to the hypothesis of formative causation, the known laws of physics do not uniquely determine the forms of complex chemical and biological systems. These laws permit a range of possibilities between which formative causes select. The repeated association of the

same type of morphogenetic field with a given type of physicochemical system explains the constancy and repetition of forms. But then what determines the particular form of the morphogenetic field?

One possible answer is that morphogenetic fields are eternal. They are simply given, and are not explicable in terms of anything else. Thus even before this planet appeared, there already existed in a latent state the morphogenetic fields of all the chemicals, crystals, animals, and plants that have ever occurred on the Earth, or that will ever come into being in the future.

This answer is essentially Platonic, or even Aristotelian insofar as Aristotle believed in the eternal fixity of specific forms. It differs from the conventional physical theory in that these forms would not be predictable in terms of energetic causation; but it agrees with it in taking for granted that behind all empirical phenomena lie preexisting principles of order.

The other possible answer is radically different. Chemical and biological forms are repeated not because they are determined by changeless laws or eternal forms, but because of a *causal influence from previous similar forms*. This influence would require an action across space *and time* unlike any known type of physical action.

On this view, the unique form taken up by a system would not be physically determined in advance of its first appearance. Nevertheless it would be repeated, because the form of the first system would itself determine the form taken up by subsequent, similar systems. Imagine, for instance, that out of several different possible forms, P, Q, R, S . . . all of which are equally probable from an energetic point of view, a system happens to take up form R on the first occasion. Then on subsequent occasions similar systems will also take up form R because of a trans-spatial and trans-temporal influence from the first such system.

In this case, what determines the form on the first occasion? No scientific answer can be given: the question concerns unique and energetically indeterminate events that, *ex hypothesi*, once they have happened are unrepeatable because they themselves influence all subsequent similar events. Science can deal only with regularities, with things that are repeatable. The initial choice of a particular form could be ascribed to chance, or to creativity inherent in matter, or to a transcendent creative

agency. But there is no way in which these different possibilities could be distinguished from each other by experiment. A decision between them could be made only on metaphysical grounds. This question is discussed in the final chapter of this book, but for present purposes it does not matter which of these possibilities is preferred. The hypothesis of formative causation is concerned only with the *repetition* of forms, and not with the reasons for their appearance in the first place.

This new way of thinking is unfamiliar, and it leads into uncharted territory. But only by exploring it does there seem to be any hope of arriving at a new scientific understanding of form and organization in general, and of living organisms in particular. The alternative to going on would be to return to the starting point; the choice would once again be narrowed to that between an unquestioning faith in future mechanistic explanations and a metaphysical or Platonic organicism.

In the following discussion, it is proposed that this hypothetical trans-spatial and trans-temporal influence passes through morphogenetic fields and is an essential feature of formative causation.

5.2 The general possibility of trans-temporal causal connections

Although the hypothesis of formative causation proposes a new kind of trans-temporal, or diachronic, causal connection that has not so far been recognized by science, the possibility of "action at a distance" in time has already been considered in general terms by several philosophers. There seems to be no a priori reason for excluding it. The philosopher John Mackie, for example, wrote as follows:

> While we are happiest about contiguous cause-effect relations, and find "action at a distance" over either a spatial or temporal gap puzzling, we do not rule it out. Our ordinary concept of causation does not absolutely require contiguity; it is not part of our idea of causation in a way that would make "C caused E over a spatial, or temporal, or both spatial and temporal, gap, without intermediate links" a contradiction in terms.[1]

Moreover, from the point of view of the philosophy of science, there is nothing to prevent the consideration of new kinds of causal connection. As the philosopher of science Mary Hesse observed:

> Scientific theory in general does not presuppose any particular mode of causal connection between events, but only that it is possible to find laws and hypotheses, expressed in terms of some model, which satisfy the criteria of intelligibility, confirmation, and falsifiability. The mode of causal connection in each case is shown by the model, and changes with fundamental changes of model.[2]

However, although the new kind of causal connection proposed in the hypothesis of formative causation seems to be possible in principle, the plausibility of this hypothesis can be assessed only after predictions deduced from it have been tested empirically.

5.3 Morphic resonance

The idea of a process whereby the forms of previous systems influence the morphogenesis of subsequent similar systems is difficult to express in terms of existing concepts. The only way to proceed is by means of analogy.

The physical analogy that seems most appropriate is that of *resonance*. Energetic resonance occurs when an alternating force acting on a system coincides with its natural frequency of vibration. Examples include the "sympathetic" vibration of stretched strings in response to appropriate sound waves; the tuning of radio sets to the frequency of radio waves given out by transmitters; the absorption of light waves of particular frequencies by atoms and molecules, resulting in their characteristic absorption spectra; and the response of electrons and atomic nuclei in the presence of magnetic fields to electromagnetic radiation in Electronic Spin Resonance and Nuclear Magnetic Resonance. Common to all these types of resonance is the principle of selectivity: out of a mixture of vibrations, however complicated, the systems respond only to particular frequencies.

A resonant effect of form upon form across space and time would resemble energetic resonance in its selectivity, but it could not be accounted for in terms of any of the known types of resonance, nor would it involve a

transmission of energy. In order to distinguish it from energetic resonance, this process will be called *morphic resonance*.

Morphic resonance is analogous to energetic resonance in a further respect: it takes place between vibrating systems. Atoms, molecules, crystals, organelles, cells, tissues, organs, and organisms are all made up of parts in ceaseless oscillation, and all have their own characteristic patterns of vibration and internal rhythm; the morphic units are dynamic, not static.[3] But whereas energetic resonance depends only on the specificity of response to particular frequencies, to "one-dimensional" stimuli,[4] morphic resonance depends on three-dimensional patterns of vibration. By morphic resonance the form of a system, including its characteristic internal structure and vibrational frequencies, becomes *present* to a subsequent system with a similar form; the spatio-temporal pattern of the former *superimposes* itself on the latter.

Morphic resonance takes place through morphogenetic fields and indeed gives rise to their characteristic structures. Not only does a specific morphogenetic field influence the form of a system (as discussed in the previous chapter), but also the form of this system influences the morphogenetic field and through it becomes present to subsequent similar systems.

5.4 The influence of the past

Morphic resonance is nonenergetic, and morphogenetic fields themselves are neither a type of mass nor energy. Therefore there seems to be no a priori reason why it should obey the laws that have been found to apply to the movement of bodies, particles, and waves. In particular, it need not be attenuated by either spatial or temporal separation between similar systems; it could be just as effective over 10,000 miles as over an inch, and over a century as over an hour.

The assumption that morphic resonance is not attenuated by time and space will be adopted as a provisional working hypothesis, on the ground of simplicity. It will also be assumed on the ground of simplicity that morphic resonance takes place only from the *past*, that only morphic units that have already actually existed are able to exert a morphic influence in the present. The notion that *future* systems, which do not yet exist, might be able to

exert a causal influence “backward” in time may perhaps be logically conceivable;[5] but only if there were persuasive empirical evidence for a physical influence from future morphic units would it become necessary to take this possibility seriously.[6]

However, assuming that morphic resonance occurs only from past morphic units and that it is not attenuated by the lapse of time or by distance, how might it take place? The process can be visualized with the help of several different metaphors. The morphic influence of a past system might become present to a subsequent similar system by passing “beyond” space-time and then “reentering” wherever and whenever a similar pattern of vibration appeared. Or it might be connected through other “dimensions.” Or it might go through a space-time “tunnel” to emerge unchanged in the presence of a subsequent similar system. Or the morphic influence of past systems might simply be present everywhere. However, these different ways of thinking about morphic resonance would probably not be distinguishable from each other experimentally. All would have the same consequence: the forms of past systems would automatically become present to subsequent similar systems. Morphic resonance would lead to the reinforcement of similarity.

An immediate implication of this hypothesis is that a given system could be influenced by *all* past systems with a similar form and pattern of vibration. *Ex hypothesi*, the influence of these past systems is not attenuated by temporal or spatial separation. Nevertheless, the ability of past systems to influence subsequent systems could be weakened or exhausted by action; they could have only a limited potential influence that is expended in morphic resonance. This possibility is discussed in section 5.5 below. But first consider the possibility that their potential action is not reduced in this way, with the consequence that the forms of all past systems influence all subsequent similar systems (figure 12). This postulate has several important consequences:

(1) The first system with a given form influences the second such system, and then both the first and the second influence the third, and so on cumulatively. In this process the direct influence of a given system on any subsequent system is progressively diluted as time goes on; although its

absolute effect does not diminish, its *relative* effect declines as the total number of similar past systems increase (figure 12).

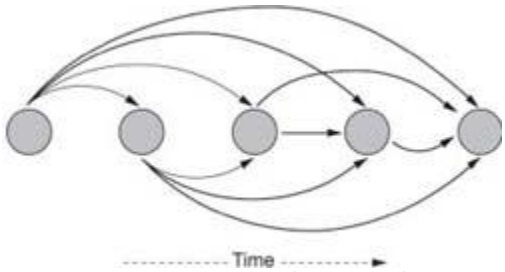

Figure 12. Diagram illustrating the cumulative influence of past systems on subsequent similar systems by morphic resonance.

(2) The forms of even the simplest chemical morphic units are variable: subatomic particles are in ceaseless vibratory motion, and atoms and molecules are subject to deformation by mechanical collision and by electrical and magnetic fields. Biological morphic units are still more variable; even if cells and organisms have the same genetic constitution and develop under the same conditions, they are not identical in every respect. By morphic resonance, the forms of all similar past systems become present to a subsequent system of similar form. Even assuming that differences in absolute size are adjusted for (see section 6.3, below), many of these forms will differ from each other in detail. Hence they will not coincide with each other exactly when they are superimposed by morphic resonance. The result will be a process of *automatic averaging* whereby those features that most past systems have in common will be reinforced. However, this "average" form will not be sharply defined within the morphogenetic field, but surrounded by a "blur" owing to the effect of less common variants. This process can be visualized more easily by analogy with "composite photographs," made by superimposing the photographic images of different individuals. As a result of this superimposition, the common features are reinforced; but because of the differences among the individual images, the "average" photographs are not sharply defined (see figures 13 and 14).

(3) The automatic averaging of past forms will result in a spatial probability distribution within the morphogenetic field, or, in other words, a *probability structure* (section 4.3). The probability structure of a morphogenetic field determines the probable state of a given system under its influence in accordance with the *actual* states of all similar systems in

the past; the most probable form the system will take up is that which has occurred most frequently already.

(4) In the early stages of a form's history, the morphogenetic field will be relatively ill-defined and significantly influenced by individual variants. But as time goes on, the cumulative influence of countless previous systems will confer an ever-increasing stability on the field; the more probable the average type becomes, the more likely that it will be repeated in the future. To put it in a different way: At first the basin of attraction of the morphogenetic field will be relatively shallow, but it will become progressively deeper as the number of systems contributing to morphic resonance increases. Or to use yet another metaphor, through repetition the form will get into a rut, and the more often it is repeated, the deeper will this rut become.

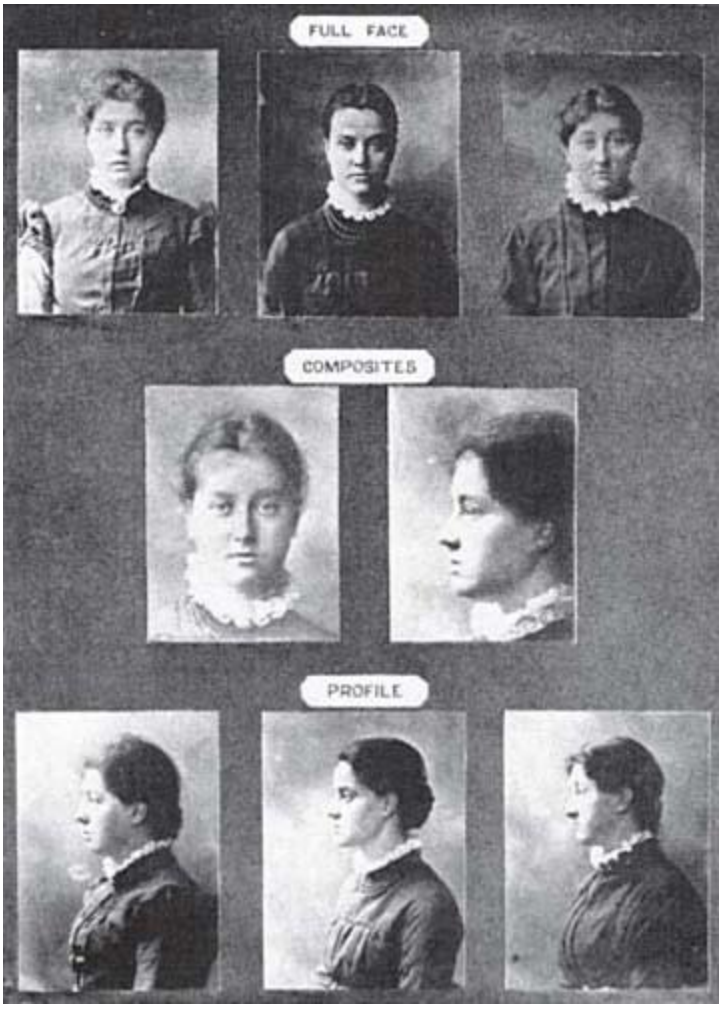

Figure 13. Photographic portraits of three sisters in full face and profile with the corresponding composites. These pictures are by Francis Galton, who invented the technique of composite photography over a century ago. (From Pearson, 1924. Reproduced by courtesy of Cambridge University Press)

Figure 14. Above: Composite photographs of officers and men of the Royal Engineers by Francis Galton. (From Pearson, 1924. Reproduced by courtesy of Cambridge University Press) Below: Composite photographs of thirty female and forty-five male members of the staff of the John Innes Institute, Norwich, U.K. (Reproduced by courtesy of the John Innes Institute)

(5) The amount of influence a given system has on subsequent similar systems seems likely to depend on the length of time it survives: one that continues to exist for a year may have more effect than one that disintegrates after a second. Thus the automatic averaging may be "weighted" in favor of long-lasting previous forms.

(6) At the beginning of a morphogenetic process, the morphogenetic germ comes into morphic resonance with similar past systems that are part of higher-level morphic units: it thus becomes associated with the morphogenetic field of the higher-level morphic unit (section 4.1). Let the morphogenetic germ be represented by morphic unit F and the final form toward which the system is attracted by D-E-F-G-H. Let the intermediate stages in the morphogenesis be as shown in figure 15. Now not only will the morphogenetic germ and the intermediate stages enter into morphic resonance with the final form of previous similar systems, but the intermediate stages will also enter into morphic resonance with similar

intermediate stages E-F, D-E-F, etc., in previous similar morphogeneses. Thus these stages will be stabilized by morphic resonance, resulting in a chreode.

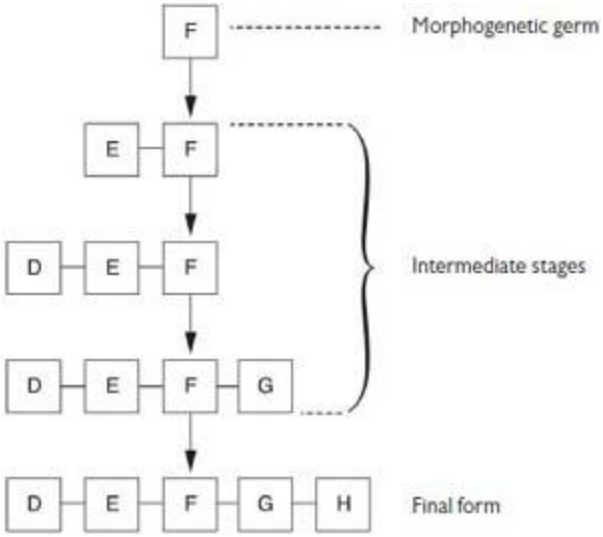

Figure 15. Diagram representing stages in the aggregative morphogenesis of the morphic unit D-E-F-G-H from the morphogenetic germ F.

The more frequently this particular pathway of morphogenesis is followed, the more will this chreode be reinforced. In terms of the "epigenetic landscape" model (figure 5), the valley of the chreode will be deepened the more often development passes along it.

5.5 Implications of an attenuated morphic resonance

The discussion in the preceding section was based on the assumption that the morphic influence of a given system is not exhausted in its action on subsequent similar systems, although its *relative* effect is diluted as the number of similar systems increases. The alternative possibility that this influence is somehow exhausted will now be considered. If such exhaustion takes place, only if the rate of exhaustion were very fast would it be detectable. Consider first the extreme case, in which the influence of a system is expended by morphic resonance with only one subsequent system. If the number of similar systems increases with time, then most of them will not be influenced by morphic resonance from previous similar systems (see figure 16A). They will consequently be free to take up

different forms by “chance” or “creativity”; the forms of these systems may therefore be very variable.

Next consider the case in which each system can influence two subsequent systems. In the situation represented in figure 16B, most but not all of the subsequent forms would be stabilized by morphic resonance. If each system influenced three subsequent systems, all would be stabilized; an instability of form would appear only if the number of subsequent similar systems increased more rapidly still, as in a population explosion. And if each system influenced many subsequent systems, this low but finite rate of exhaustion of morphic influence would be practically undetectable.

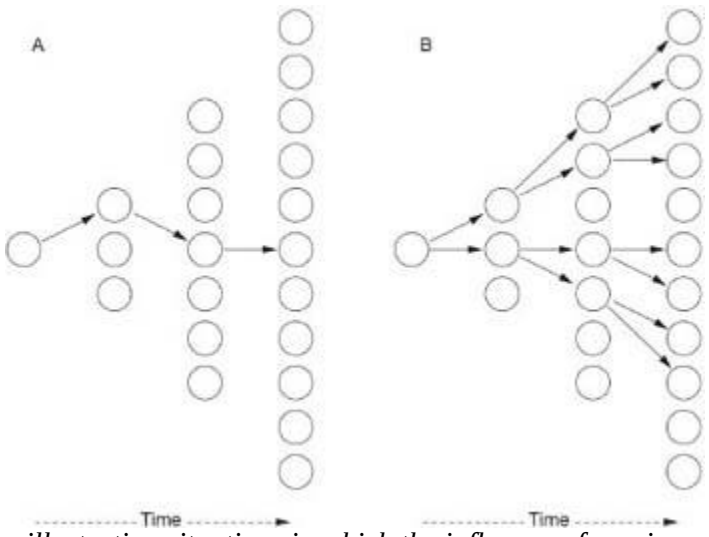

*Figure 16. Diagram illustrating situations in which the influence of previous systems is exhausted by morphic resonance with only one subsequent system (*A*) and two subsequent systems* (B).

For the sake of simplicity, it will be assumed that the morphic influence of systems on subsequent similar systems is *not* exhausted, but this assumption is provisional. The question could eventually be investigated empirically, at least to the extent of distinguishing between a rapid rate of exhaustion of morphic influence on the one hand and a slow or zero rate on the other.

5.6 An experimental test with crystals

According to the conventional theory, the unique forms of chemical and biological systems should be predictable in terms of the principles of quantum mechanics, electromagnetism, energetic causation, etc., before

they come into being for the first time. By contrast, according to the hypothesis of formative causation, unique forms will not be predictable in advance, but only a range of possible forms. Thus, in principle, the failure of the conventional theory to give rise to unique predictions would provide evidence against it and in favor of the hypothesis of formative causation. But in practice this failure could never be conclusive: only approximate calculations are feasible, and therefore defenders of this theory will always be able to argue that unique predictions might be possible if more-refined calculations were carried out in the future.

Fortunately, the hypothesis of formative causation differs from the conventional theory in a second important respect. According to the latter, the laws that give rise to a form on the first occasion, or on the hundredth, or the billionth, should operate in exactly the same way, since they are assumed to be changeless. The same expectation follows from theories that seek to account for empirically observable forms in terms of eternal archetypal forms or transcendent mathematical truths.

But according to the hypothesis of formative causation, the form of a system depends on the cumulative morphic influence of previous similar systems. Thus this influence will be stronger on the billionth occasion than on the thousandth or the tenth. If this cumulative aspect of formative causation could be demonstrated empirically, the hypothesis could be distinguished both from the conventional theory and from theories of the Platonic and Pythagorean types.

In the case of morphic units that have existed for a very long time—billions of years in the case of the hydrogen atom—the morphogenetic field will be so well established as to be effectively changeless. Even the fields of morphic units that originated a few decades ago may be subject to the influence of so many past systems that any increments in this influence will be too small to be detectable. But with brand-new forms, it may well be possible to detect a cumulative morphic influence experimentally.

Consider a newly synthesized organic chemical that has never existed before. According to the hypothesis of formative causation, its crystalline form will not be predictable in advance, and no morphogenetic field for this form will yet exist. But after it has been crystallized for the first time, the form of its crystals will influence subsequent crystallizations by morphic resonance, and the more often it is crystallized, the stronger should this

influence become. Thus on the first occasion, the substance may not crystallize at all readily, but on subsequent occasions crystallization should occur more and more easily as increasing numbers of past crystals contribute to its morphogenetic field by morphic resonance.

In fact, chemists who have synthesized entirely new chemicals often have great difficulty in getting these substances to crystallize. But as time goes on, these substances tend to crystallize with greater and greater ease. Sometimes many years pass before crystals first appear. For example, turanose, a kind of sugar, was considered to be a liquid for decades, but after it first crystallized in the 1920s, it formed crystals all over the world.[7] Even more striking are cases in which one kind of crystal appears and is then replaced by another. For example, xylitol, a sugar alcohol used as a sweetener in chewing gum, was first prepared in 1891 and was considered to be a liquid until 1942, when a form with a melting point of 61°C crystallized out. Several years later another form appeared, with a melting point of 94°C, and thereafter the first form could not be made again.[8]

Crystals of the same compound that exist in different forms are called polymorphs. In many cases they can coexist, like calcite and aragonite, which are both crystalline forms of calcium carbonate, or like graphite and diamond, both crystalline forms of carbon. But sometimes, as in the case of xylitol, the appearance of a new polymorph can displace an old one. This principle is illustrated in the following account, taken from a textbook on crystallography, of the spontaneous and unexpected appearance of a new type of crystal:

> About ten years ago a company was operating a factory that grew large single crystals of ethylene diamine tartrate from solution in water. From this plant it shipped the crystals many miles to another which cut and polished them for industrial use. A year after the factory opened, the crystals in the growing tanks began to grow badly; crystals of something else adhered to them—something which grew even more rapidly. The affliction soon spread to the other factory: the cut and polished crystals acquired the malady on their surfaces . . .

The wanted material was *anhydrous* ethylene diamine tartrate, and the unwanted material turned out to be the *monohydrate* of that substance.

During three years of research and development, and another year of manufacture, no seed of the monohydrate had formed. After that, they seemed to be everywhere.[9]

These authors suggest that on other planets, types of crystal that are common on Earth may not yet have appeared, and add: “Perhaps in our own world many other possible solid species are still unknown, not because their ingredients are lacking, but simply because suitable seeds have not yet put in an appearance.”[10]

The replacement of one polymorph by another is a recurrent problem in the pharmaceutical industry. For example, the antibiotic ampicillin was first crystallized as a monohydrate, with one molecule of water of crystallization per ampicillin molecule. In the 1960s it started to crystallize as a trihydrate, with a different crystal form, and despite persistent efforts, the monohydrate could not be made again.[11]

Ritonavir, an AIDS drug made by Abbott Laboratories, was introduced in 1996. The drug had been on the market for eighteen months when suddenly, during manufacturing, chemical engineers found a previously unknown polymorph. No one knew what had caused the change, and the Abbott team could not find a way to stop the new polymorph forming. Within a few days of its discovery, it was dominating the production lines. Although the two polymorphs had the same chemical formula, their structural dissimilarity made a difference to patients. The second form was only half as soluble as the first, so patients taking the normal prescribed doses would not absorb enough of the drug. Abbott had to pull ritonavir from the market.

The company went on a crash program to try to get its original polymorph back. It eventually succeeded in producing the first form again but could not make the polymorph reliably, and kept getting mixtures of the two forms. The company finally decided to reformulate the drug in the second polymorphic form as a liquid gel capsule containing the drug in a dissolved form. The company spent hundreds of millions of dollars trying to recover the first polymorph, and lost an estimated $250 million in sales the year the drug was withdrawn.[12]

The inability of chemists to control some kinds of crystallization is a serious challenge. As the crystallographer Joel Bernstein remarked, “The loss of control is indeed disturbing, and might even call into question the

criterion of reproducibility as a condition for acceptance of a phenomenon as being worthy of scientific enquiry."[13] Complete reproducibility would be expected on the assumption that all the laws of nature are eternal, the same at all times and in all places. But disappearing polymorphs make it clear that chemistry is not timeless. It is historical and evolutionary, like biology. What happens now depends on what has happened before.

The most obvious explanation for the disappearance of polymorphs is that the new forms were more stable thermodynamically, and hence appeared in preference to the older forms. In competition with each other, the new forms won. Before the new forms existed, there was no competition; after they had come into being, they turned up in laboratories all over the world, and the older forms disappeared. As an American chemist, Charles P. Saylor, commented, it was "as though the seeds of crystallization, as dust, had been carried upon the winds from end to end of the earth."[14]

There is no doubt that small fragments of previous crystals can act as "seeds" or "nuclei" and facilitate the process of crystallization from a supersaturated solution. That is why chemists have always assumed that the spread of new crystallization processes depends on the transfer of seeds from laboratory to laboratory, like a kind of infection. One favorite story in the folklore of chemistry is that these seeds are carried around the world from lab to lab by migrant scientists, especially chemists with beards, which "can harbor nuclei for almost any crystallization process," in the words of a professor of chemical engineering at Cambridge University.[15] Alternatively, the crystal seeds are supposed to have been blown around in the atmosphere as microscopic particles of dust and then settled in crystallizing dishes, where they catalyzed the crystallization of the new substance.

Thus the formation of new kinds of crystals provides one way of testing the hypothesis of morphic resonance. According to the conventional assumption, crystals should not form more readily in a laboratory in Australia after they have been made in a laboratory in Britain if visitors from the British laboratory are rigorously excluded, and if dust particles are filtered out of the atmosphere. If in fact they do form more rapidly, then this result would favor morphic resonance.

The effects of morphic resonance could be investigated by comparing the crystallization of several newly synthesized chemicals, say four. The rate at which the crystals form is determined under standardized conditions. Then one of these four compounds is selected at random, made in large quantities, and crystallized repeatedly. Now, in a different laboratory hundreds of miles away, all four compounds are crystallized again under the same standard conditions as before. The hypothesis of formative causation would predict that the randomly selected compound should now crystallize more rapidly than it did before, but there should be little or no change in the rate of crystallization of the other three.

Further experiments with crystals are discussed in appendix A, and in sections A.2 and A.3. Examples of possible experiments with biological systems are discussed in sections 7.4, 7.6, 7.9, 11.2, and 11.4.

6

FORMATIVE CAUSATION AND MORPHOGENESIS

6.1 Sequential Morphogeneses

After subatomic particles have aggregated into atoms, the atoms may combine together into molecules, and the molecules into crystals. The crystals then retain their form indefinitely as long as the temperature remains below their melting point. By contrast, in living organisms morphogenetic processes continue indefinitely in the endlessly repeated cycles of growth and reproduction.

The simplest living organisms consist of single cells, the growth of which is followed by division, and division by growth. Thus the morphogenetic germs for the chreodes of division must appear within the final forms of the fully grown cells, and the newly divided cells serve as the starting points for the chreodes of cellular growth and development.

In multicellular organisms, these cycles continue in only some of the cells, for example in germ cell lines, stem cells, and meristematic cells. Other cells, and indeed whole tissues and organs, develop into a variety of specialized structures that undergo little further morphogenetic change: they stop growing, although they may retain the ability to regenerate after damage; and sooner or later they die. In fact, they may be mortal precisely because they cease to grow.[1]

The development of multicellular organisms takes place through a series of stages controlled by a succession of morphogenetic fields. At first the embryonic tissues develop under the control of primary embryonic fields. Then, sooner (in “mosaic” development) or later (in “regulative” development), different regions come under the influence of secondary fields, in animals those of limbs, eyes, ears, etc.; in plants of leaves, petals, stamens, etc. Generally speaking, the morphogenesis brought about by the primary fields is not spectacular, but it is of fundamental importance because it establishes the characteristic differences between cells in different regions that enable them to act as the morphogenetic germs of the organ fields. Then in the tissues developing under their influence, germs of subsidiary fields appear, fields that control the morphogenesis of structures within the organ as a whole: in a leaf, the lamina, stipules, petiole, etc.; in an eye, the cornea, iris, lens, etc. And then still lower-level morphogenetic fields come into play: for example, those for vascular differentiation within the lamina of a leaf, and for the differentiation of stomata and hair cells on its surface.

These fields can be, and have been, investigated experimentally by studying the ability of developing organisms to regulate after damage to different regions of the embryonic tissue, and after grafting tissue taken from one region into another. Both in animal embryos and in the meristematic zones of plants, as the differentiation of the tissues proceeds, the different regions behave with increasing autonomy; the system as a whole loses the ability to regulate, but local regulations occur within the developing organs as more numerous secondary fields supplant the primary embryonic fields.[2]

6.2 The polarity of morphogenetic fields

Most biological morphic units are polarized in at least one direction. Their morphogenetic fields, containing polarized virtual forms, will automatically take up appropriate orientations if their morphogenetic germs are also intrinsically polarized; but if they are not, a polarity must first be imposed on them.

For example, the spherical egg cells of the alga *Fucus* have no inherent polarity, and their development can begin only after they have been

polarized by any one of a variety of directional stimuli including light, chemical gradients, and electric currents; in the absence of any such stimuli, a polarity is taken up at random, presumably owing to chance fluctuations.

Nearly all multicellular organisms are polarized in a shoot–root or head–tail direction, many also in a second direction (ventral–dorsal), and some in three (head–tail, ventral–dorsal, and left–right). The latter are consequently asymmetrical and potentially capable of existing in forms that are mirror-images of each other; for example, most people have their heart on the left side, but a few have it on the right. In the condition known as *situs inversus totalis*, the position of all the chest and abdominal organs is reversed.

Structures that are bilaterally symmetrical necessarily occur in both right- and left-handed forms, for example right and left hands. These mirror-image forms have the same morphology, and they presumably develop under the influence of the same morphogenetic field. The field simply takes on the handedness of the morphogenetic germ with which it becomes associated. Thus both right- and left-handed previous systems influence both right- and left-handed subsequent systems by morphic resonance.

This interpretation is supported by some well-known facts of biochemistry. The molecules of amino acids and sugars are asymmetric and are capable of existing in both left- and right-handed forms. Yet in living organisms, all the amino acids in proteins are left-handed, while most of the sugars are right-handed. The perpetuation of these chemical asymmetries is made possible by the asymmetric structures of the enzymes that catalyze the synthesis of the molecules. In nature, most of the amino acids and sugars occur rarely, if at all, outside living organisms. Therefore these particular asymmetric forms should contribute overwhelmingly by morphic resonance to the morphogenetic fields of the molecules. But when they are synthesized artificially, equal proportions of right-and left-handed forms are obtained, indicating that the morphogenetic fields have no intrinsic handedness.

6.3 The size of morphogenetic fields

The dimensions of particular atomic and molecular morphic units are more or less constant; so are those of crystal lattices, although they are repeated indefinitely to give crystals of different sizes. Biological morphic units are

more variable: not only are there differences between cells, organs, and organisms of given types, but also individual morphic units themselves change size as they grow. If morphic resonance is to take place from past systems with similar forms but different sizes, and if a particular morphogenetic field is to remain associated with a growing system, then forms must be capable of being "scaled up" or "scaled down" within the morphogenetic field. Thus their essential features must depend not on the absolute but on the relative positions of their component parts, and on their relative rates of vibration. A simple analogy is provided by the music produced by playing a tape recording at different speeds: it remains recognizable in spite of absolute alterations in all the pitches and rhythms because the relations of the notes and rhythms to each other remain the same.

Although morphogenetic fields may be adjustable in absolute size, the range within which the size of a system can vary is limited by severe physical constraints. In three-dimensional systems, changes in surface area and volume vary respectively as the square and cubic powers of the linear dimensions. This simple fact means that biological systems cannot be magnified or diminished indefinitely without becoming unstable.[3]

6.4 The increasing specificity of morphic resonance during morphogenesis

Energetic resonance is not an "all-or-none" process: a system resonates in response to a range of frequencies that are more or less close to its natural frequency, although the maximum response occurs only when the frequency coincides with its own. Analogously, morphic resonance may be more or less finely "tuned," occurring with greatest specificity when the forms of past and present systems are most closely similar.

When a morphogenetic germ comes into morphic resonance with the forms of countless previous higher-level systems, these forms do not coincide exactly but give rise to a probability structure. As the first stages of morphogenesis take place, structures are actualized at particular places within the regions given by the probability structure. The system now has a more developed and better defined form, and will consequently resemble the forms of some previous similar systems more closely than others; the

morphic resonance from these forms will be more specific and hence more effective. And as development proceeds, the selectivity of morphic resonance will increase still further.

A very general illustration of this principle is given by the development of an organism from a fertilized egg. The early stages of embryology often resemble those of numerous other species, or even families and orders. As development proceeds, the particular features of the order, family, genus, and finally species tend to appear sequentially, and the relatively minor differences that distinguish the individual organism from other individuals of the same species generally appear last.

This increasingly specific morphic resonance will tend to canalize development toward particular variants of the final form that were expressed in previous organisms. The detailed pathway of development will be affected by both genetic and environmental factors: an organism of a particular genetic constitution will tend to develop in such a way that it enters into specific morphic resonance with previous individuals with the same genetic constitution; and environmental effects on development will tend to bring the organism under the specific morphic influence of previous organisms that developed in the same environment.

Previous similar morphic units that were part of the same organism will have an even more specific effect. For example, in the development of leaves on a tree, the forms of previous leaves on the same tree are likely to make a particularly significant contribution to the morphogenetic field, tending to stabilize the leaf form characteristic of this particular tree.

6.5 The maintenance and stability of forms

At the end of a process of morphogenesis, the actual form of a system comes into coincidence with the virtual form given by the morphogenetic field. The continued association of the system with its field is revealed most clearly in the phenomenon of regeneration. The restoration of the form of the system after small deviations from the final form is less obvious, but no less important: its morphogenetic field continuously stabilizes the morphic unit. In biological systems, and to some extent in chemical systems, this maintenance of form enables the morphic units to persist even though their constituent parts change as they are “turned over” and replaced. The

morphogenetic field itself persists, owing to the continuing influence of the forms of similar past systems.

An extraordinarily interesting feature of the morphic resonance acting on a system with a persisting form is that this resonance will include a contribution from the past states of the system itself. Insofar as a system resembles itself in the past more closely than it resembles any other past system, this self-resonance will be highly specific. It may in fact be of the most fundamental importance in maintaining the very identity of the system.

Matter can no longer be thought of as made up of solid particles like tiny billiard balls that endure throughout time. Material systems are dynamic structures that are constantly re-creating themselves. On the present hypothesis, the persistence of material forms depends on a continuously repeated actualization of the system under the influence of its morphogenetic field; at the same time, the morphogenetic field is continuously re-created by morphic resonance from similar past forms. The forms that are most similar and that will consequently have the greatest effect will be those of the system itself in the immediate past. This conclusion would appear to have profound physical implications: the preferential resonance of a system with itself in the immediate past could conceivably help to account for its persistence not only in time, but also at a particular place.[4]

6.6 A note on physical “dualism”

All actual morphic units can be regarded as *forms of energy*. On the one hand, their structures and patterns of activity depend on the morphogenetic fields with which they are associated and under the influence of which they have come into being. On the other hand, their very existence and their ability to interact with other material systems is due to the energy bound within them. But although these aspects of form and energy can be separated conceptually, in reality they are always associated with each other. No morphic unit can have energy without form, and no material form can exist without energy.

This physical “duality” of form and energy that is made explicit by the hypothesis of formative causation has much in common with the so-called

wave-particle duality of quantum theory. According to the hypothesis of formative causation, there is only a difference of degree between the morphogenesis of atoms and that of molecules, crystals, cells, tissues, organs, and organisms. If *dualism* is defined in such a way that the orbitals of electrons in atoms involve a duality of waves and particles, or of form and energy, then so do the more-complex forms of higher-level morphic units; but if the former are not considered to be dualistic, then neither are the latter.[5]

In spite of their similarity, there is of course a difference in kind between the hypothesis of formative causation and the conventional theory. The latter provides no fundamental understanding of the causation of forms, unless equations or "mathematical structures" describing them are assumed to play a causal role; if so, a very mysterious dualism between mathematics and reality is implied. The hypothesis of formative causation overcomes this problem by regarding the forms of previous systems as the causes of subsequent similar forms. From the conventional point of view, this cure may seem worse than the disease insofar as it requires an action across time and space unlike any known type of physical action. However, this is not a metaphysical proposition but a physical one, and is capable of being tested experimentally.

If this hypothesis is supported by experimental evidence, then not only might it allow the various matter fields of quantum field theory to be interpreted in terms of morphogenetic fields, but it could also lead toward a new understanding of other physical fields.

In the morphogenetic field of an atom, a naked atomic nucleus surrounded by virtual orbitals serves as a morphogenetic "attractor" for electrons. Perhaps the so-called electrical attraction between the nucleus and the electrons could be regarded as an aspect of this atomic morphogenetic field. When the final form of the atom has been actualized by the capture of electrons, it no longer acts as a morphogenetic "attractor," and in electrical terminology it is neutral. So it is not inconceivable that electromagnetic fields could be derived from the morphogenetic fields of atoms.

In a comparable manner, it might eventually be possible to interpret the strong and weak nuclear forces in terms of the morphogenetic fields of atomic nuclei and nuclear particles. Morphogenetic fields are part of the

larger category of *morphic fields,* which also includes behavioral and social fields (chapter 9). Much of this summary of the hypothesis of formative causation applies to morphic fields in general, and not just to morphogenetic fields.

6.7 A summary of the hypothesis of formative causation

(1) In addition to the types of energetic causation known to physics, and in addition to the causation due to the structures of known physical fields, a further type of causation is responsible for the forms of all material morphic units (subatomic particles, atoms, molecules, crystals, quasi-crystalline aggregates, organelles, cells, tissues, organs, organisms). Form, in the sense used here, includes not only the shape of the outer surface of the morphic unit but also its internal structure. This causation, called *formative causation*, imposes spatial order on changes brought about by energetic causation. It is not itself energetic, nor is it reducible to the causation brought about by known physical fields (sections 3.3, 3.4, 3.5).

(2) Formative causation depends on *morphic fields,* structures with morphogenetic effects on material systems. Each kind of morphic unit has its own characteristic morphic field. In the morphogenesis of a particular morphic unit, one or more of its characteristic parts—referred to as the *morphogenetic germ*—becomes surrounded by, or embedded within, the morphogenetic field of the entire morphic unit. This field contains the morphic unit's virtual form, which is actualized as appropriate component parts come within its range of influence and fit into their appropriate relative positions. The fitting into position of the parts of a morphic unit is accompanied by a release of energy, usually as heat, and is thermodynamically spontaneous; from an energetic point of view, the structures of morphic units appear as minima or "wells" of potential energy (sections 3.3, 3.4, 4.1, 4.2, 4.4, 4.5).

(3) Most inorganic morphogenesis is rapid, but biological morphogenesis is relatively slow and passes through a succession of intermediate stages. A given type of morphogenesis usually follows a particular developmental pathway; such a canalized pathway of change is called a *chreode*. Nevertheless, morphogenesis may also proceed toward the final form from different morphogenetic germs and by different pathways, as in the

phenomena of regulation and regeneration. In the cycles of cell growth and cell division and in the development of the differentiated structures of multicellular organisms, a succession of morphogenetic processes take place under the influence of a succession of morphogenetic fields (sections 2.4, 4.1, 5.4, 6.1).

(4) The characteristic form of a given morphic unit is determined by the forms of previous similar systems that act upon it across time and space by a process called *morphic resonance*. This influence takes place through the morphic field and depends on the systems' three-dimensional structures and patterns of vibration. Morphic resonance is analogous to energetic resonance in its specificity, but it is not explicable in terms of any known type of resonance, nor does it involve a transmission of energy (sections 5.1, 5.3).

(5) All similar past systems act upon a subsequent similar system by morphic resonance. This action is provisionally assumed not to be attenuated by space or time, and to continue indefinitely; however, the relative effect of a given system declines as the number of similar systems contributing to morphic resonance increases (sections 5.4, 5.5).

(6) The hypothesis of formative causation accounts for the repetition of forms but does not explain how the first example of any given form originally came into being. This unique event can be ascribed to chance, or to creativity inherent in nature, or to a transcendent creative agency. A decision between these alternatives can be made only on metaphysical grounds and lies outside the scope of the hypothesis (section 5.1).

(7) Morphic resonance from the intermediate stages of previous similar processes of morphogenesis tends to canalize subsequent similar morphogenetic processes into the same chreodes (section 5.4).

(8) Morphic resonance from past systems with a characteristic polarity can occur effectively only after the morphogenetic germ of a subsequent system has been suitably polarized. Systems that are asymmetrical in all three dimensions and exist in right- or left-handed forms influence subsequent similar systems by morphic resonance irrespective of handedness (section 6.2).

(9) Morphic fields are adjustable in absolute size and can be "scaled up" or "scaled down" within limits. Thus previous systems influence

subsequent systems of similar form by morphic resonance even though their absolute sizes may differ (section 6.3).

(10) Even after adjustment for size, the many previous systems influencing a subsequent system by morphic resonance are not identical, but only similar in form. Therefore, their forms are not precisely superimposed within the morphic field. The most frequent type of previous form makes the greatest contribution by morphic resonance, the least frequent the least: morphic fields are not precisely defined but are *probability structures* that depend on the statistical distribution of previous similar forms. The probability distributions of electronic orbitals described by solutions of the Schrödinger equation are examples of such probability structures, and are similar in kind to the probability structures of the morphic fields of morphic units at higher levels (sections 4.3, 5.4).

(11) The morphic fields of morphic units influence morphogenesis by acting upon the morphic fields of their constituent parts. Thus the fields of tissues influence those of cells; those of cells, organelles; those of crystals, molecules; those of molecules, atoms; and so on. These actions depend on the influence of higher-level probability structures on lower-level probability structures and are thus inherently probabilistic (sections 4.3, 4.4).

(12) Once the final form of a morphic unit is actualized, the continued action of morphic resonance from similar past forms stabilizes and maintains it. If the form persists, the morphic resonance acting upon it will include a contribution from its own past states. Insofar as the system resembles its own past states more closely than those of other systems, this self-resonance will be highly specific, and may be of considerable importance in maintaining the system’s identity (sections 6.4, 6.5).

(13) The hypothesis of formative causation is capable of being tested experimentally (section 5.6).

7

THE INHERITANCE OF FORM

7.1 Genetics and heredity

Hereditary differences between otherwise similar organisms depend on genetic differences; genetic differences depend on differences in the structure of DNA, or in its arrangement within the chromosomes; and these differences lead to changes in the structure of proteins, or to changes in the control of protein synthesis.

In the twentieth century, these fundamental discoveries, supported by a large body of detailed evidence, provided a satisfyingly straightforward understanding of the inheritance of proteins and of characteristics that depend more or less directly on particular proteins, for example sickle-cell anemia and hereditary defects of metabolism. By contrast, hereditary differences of form generally bear no immediate and obvious relationship to changes in the structure or synthesis of particular proteins. Nevertheless, such changes can affect morphogenesis in various ways through effects on metabolic enzymes, hormone-synthesizing enzymes, structural proteins, proteins in cell membranes, and so on. Many examples of these effects are already known. But granted that various chemical changes lead to alterations or distortions of the normal pattern of morphogenesis, what determines the normal pattern of morphogenesis itself?

According to the mechanistic theory, cells, tissues, organs, and organisms take up their appropriate forms as a result of the synthesis of the right chemicals in the right places at the right times. Morphogenesis is supposed to proceed spontaneously as a result of complex physicochemical

interactions, but in a way that is not yet fully understood because of its complexity. The mechanistic theory leaves open the question of how self-assembly actually works (section 2.2).

The hypothesis of formative causation suggests a new way of answering this question. Insofar as it offers an interpretation of biological morphogenesis that stresses the analogy with physical processes such as crystallization, as well as ascribing an important role to energetically indeterminate fluctuations, it fulfills rather than denies the expectations of the mechanistic theory. But whereas the mechanistic theory attributes practically all the phenomena of heredity to the genetic inheritance embodied in the DNA, the hypothesis of formative causation enables organisms in addition to inherit the morphic fields of past organisms of the same species. This type of inheritance takes place by morphic resonance and not through the genes. So heredity includes *both* the genetic inheritance of proteins *and* morphic resonance from similar past forms.

Consider the following analogy. The music that comes out of the loudspeaker of a radio set depends *both* on the material structures of the set and the energy that powers it *and* on the transmission to which the set is tuned. The music can of course be affected by changes in the wiring, transistors, condensers, etc., and it ceases when the battery is removed. Someone who knew nothing about the transmission of invisible, intangible, and inaudible vibrations through the electromagnetic field might conclude that it could be explained entirely in terms of the components of the radio, the way in which they were arranged, and the energy on which their functioning depended. If he ever considered the possibility that anything entered from outside, he would dismiss it when he discovered that the set weighed the same switched on and switched off. He would therefore have to suppose that the rhythmic and harmonic patterns of the music arose within the set as a result of immensely complicated interactions among its parts. After careful study and analysis of the set, he might even be able to make a replica of it that produced exactly the same sounds as the original, and would probably regard this result as a striking proof of his theory. But in spite of his achievement, he would remain completely unaware that in reality the music originated in a broadcasting studio hundreds of miles away.

In terms of the hypothesis of formative causation, the “transmission” comes from previous similar systems and its “reception” depends on the detailed structure and organization of the receiving system. As in a radio set, two types of change in the organization of the receiver have significant effects.

First, changes in the tuning of the system lead to the reception of quite different morphic transmissions: just as a radio set can be tuned to different radio stations, so a developing system can be tuned to different morphogenetic fields.

Second, changes within a radio set tuned to a particular station can lead to modifications and distortions of the music coming out of the loudspeaker. Likewise, genetic changes within a system developing under the influence of a morphogenetic field can lead to modifications and distortions of the organism’s form.

Thus in developing organisms, both environmental and genetic factors can affect morphogenesis in two different ways: either by changing the tuning of morphogenetic germs or by influencing the habitual pathways of morphogenesis in such a way that variants of the normal forms are produced.

7.2 Altered morphogenetic germs

The morphogenetic germs for the development of organs and tissues consist of cells or groups of cells with characteristic structures and patterns of oscillation, including electrical oscillations in their membranes (sections 4.5, 6.1). Modern scanning techniques like electron spin resonance (ESR) and magnetic resonance imaging (MRI) depend on resonance effects with molecules and with atomic nuclei within the body. In all levels of organization, from atoms, molecules, organelles, membranes, and organs like hearts and brains, there are rhythmic, vibratory patterns of activity, including electromagnetic activity.

If as a result of unusual environmental conditions or genetic alterations the structure and oscillatory pattern of a germ is changed sufficiently, it will no longer become associated with its usual morphogenetic field. Either it will fail to act as a germ at all, in which case an entire structure will fail to appear within the organism; or it will become associated with a different

morphogenetic field, in which case a structure not normally found in this part of the organism will develop instead of the usual one.

Mutations that lead to changes of this kind are called homeotic mutations. Many examples of the loss of an entire structure or of the replacement of one structure by another have been described.[1] Sometimes the same homeotic changes can also be brought about by changes in the environment of the developing organism, as discussed below.

Effects of these types have been studied in great detail in the fruit fly *Drosophila*. Several homeotic mutations lead to transformations of entire regions of the fly; for example, changes to the antennapedia gene change antennae into legs, and mutations within the bithorax gene complex cause the metathoracic segment, which normally bears two halteres, to develop as if it were a mesothoracic segment (see figure 17). The resulting flies bear two pairs of wings on adjacent segments.[2]

Homeotic mutations also occur in plants. In the pea, for example, the leaves normally bear leaflets toward their base and tendrils at their tip. In some leaves, there are tendrils opposite leaflets, indicating that similar primordia are capable of giving rise to both types of structure (figure 18); presumably cells within these primordia are influenced by factors within the embryonic leaf, causing them to take up the structure and oscillatory pattern characteristic of the morphogenetic germ either of a tendril or of a leaflet. In one type of homeotic mutant, the formation of tendrils is suppressed and all the primordia give rise to leaflets; in another mutant (due to a gene on a different chromosome) the formation of leaflets is suppressed and all the primordia give rise to tendrils[3] (figure 18).

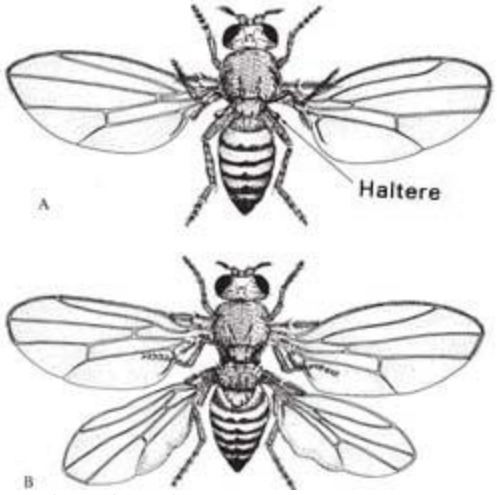

Figure 17. A normal specimen of the fruit fly Drosophila (A) *and a mutant fly* (B) *in which the third thoracic segment has been transformed in such a way that it resembles the second thoracic segment. The fly consequently has two pairs of wings instead of one.*

Homeotic genes code for transcription factors, which are proteins that bind to specific parts of DNA affecting the activity of other genes. The pattern of gene activity they control affects a whole pathway of morphogenesis. In terms of the hypothesis of formative causation, these patterns of gene activity work by influencing morphogenetic germs, affecting their tuning to particular morphogenetic fields. There are many conceivable ways in which they might do this, for example by coding for proteins that modify the properties of cell membranes, affecting the structures or patterns of activity in the cells of the morphogenetic germ in such a way that they no longer resonate with the usual morphogenetic field but tune in to a different one. This is analogous to changing the tuning circuit of a television set: a “mutation” in a transistor or condenser in this circuit could cause the TV set to be tuned to a different channel, or to lose the ability to tune in to any channel at all. Significantly, in evolutionary developmental biology, these homeotic genes are often referred to as “field-specific selector genes.”[4]

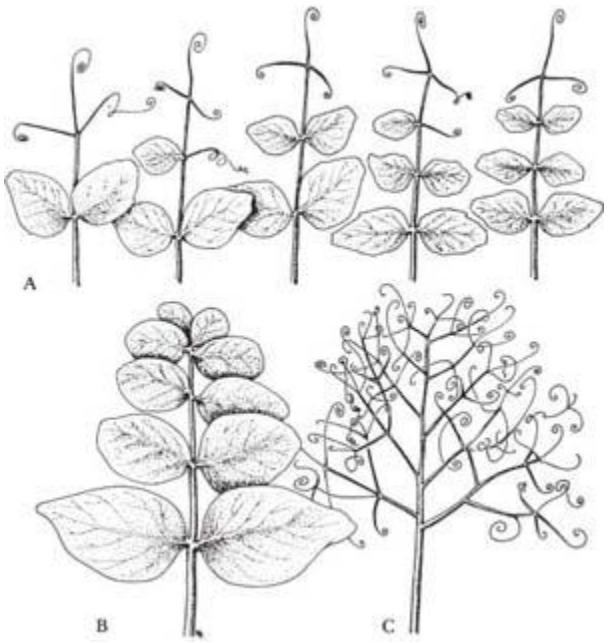

Figure 18. A: *Normal pea leaves, bearing both leaflets and tendrils.* B: *Leaf of a mutant plant in which only leaflets are formed.* C: *Leaf of a mutant plant in which only tendrils are formed.*

7.3 Altered pathways of morphogenesis

Whereas the factors affecting morphogenetic germs have *qualitative* effects on morphogenesis resulting in the absence of a structure or the substitution of one structure for another, many environmental and genetic factors bring about *quantitative* modifications of the final forms of structures through their effects on the processes of morphogenesis. For example, in plants of a given cultivated variety grown under a range of environmental conditions, the overall shape of the shoot and root systems, the morphology of the leaves, and even the anatomy of various organs differ in detail; but nevertheless the characteristic varietal form remains recognizable. Or when different varieties of the same species are grown in the same environment, the plants differ from each other in many details, although they are all recognizably variants of a characteristic specific form.

Genetic and environmental factors influence development through various quantitative effects on structural components, enzymic activity, hormones, etc. (section 7.1). Some of these influences are relatively unspecific and affect several different pathways of morphogenesis. Others may perturb the normal course of development but have little effect on the final form, owing to regulation.

While certain striking genetic effects may be traceable to particular genes, most depend on the combined influence of numerous genes, the individual effects of which are small and difficult to identify and analyze.

According to the hypothesis of formative causation, organisms of the same variety or breed resemble each other not only because they are genetically similar and therefore subject to similar genetic influences during morphogenesis, but also because their characteristic chreodes are reinforced and stabilized by morphic resonance from past organisms of the same kind.

The morphogenetic fields of a species are not fixed, but change as the species evolves. The greatest statistical contribution to the probability structures of the morphogenetic fields is from the most common morphological types, which are also those that develop under the most usual environmental conditions. In the simplest cases, the automatic averaging effect of morphic resonance will stabilize the morphogenetic fields around a single most probable form, or “wild type.” If the species inhabits two or more geographically or ecologically distinct environments in which characteristic growth habits have evolved, the morphogenetic fields of the species will not contain a single most probable form, but a “multi-modal” distribution of forms, depending on the number of morphologically distinct varieties or races and the relative sizes of their past populations.

7.4 Dominance

At first sight, the idea that varietal forms are stabilized by morphic resonance from past organisms of the same variety may appear to add little to the conventional explanation in terms of genetic similarity alone. However, its importance becomes apparent in considering hybrid organisms that are subject to morphic resonance from two distinct parental types.

To return to the radio analogy: Under normal circumstances a set is tuned to only one station at a time, just as an organism is normally “tuned” to similar past organisms of the same variety. But if the radio is tuned in to two different stations simultaneously, the sounds it produces depend on the relative strength of their signals: if one is very strong and the other very weak, the latter has little noticeable effect; but if both are of similar strength, the set produces a mixture of sounds from both sources. Likewise,

in a hybrid produced by crossing two varieties, the presence of genes and gene products characteristic of both will tend to bring the developing organism into morphic resonance with past organisms of both parental types. The overall probability structures within the morphogenetic fields of the hybrid will depend on the relative strength of the morphic resonance from the two parental types. If both parents come from varieties represented by comparable numbers of past individuals, both will tend to influence morphogenesis to similar extents, giving a combination or “resultant” of the two parental forms (figure 19A). But if fewer individuals have represented one variety than the other, their smaller contribution to the overall probability structure will mean that the form of the other parental variety will tend to predominate (figure 19B). And if one of the parents comes from a mutant line of recent origin, morphic resonance from the small number of past individuals of this type will make an insignificant contribution to the probability structure of the hybrids (figure 19C).

These expectations are in harmony with the facts. First, hybrids between well-established varieties or species usually combine features of both, or are of intermediate form. Second, in hybrids between a relatively recent variety and a long-established variety, the characters of the latter are usually more or less dominant. And third, recent mutations affecting morphological characters are nearly always recessive.

Significantly, mechanistic theories of dominance are both vague and speculative, except in the case of characteristics that depend more or less directly on particular proteins. If a mutant gene leads to a loss of function, for example by giving rise to a defective enzyme, it will be recessive, because in hybrids the presence of a normal gene enables the normal enzyme to be produced, and hence the normal biochemical reactions occur. However, in some cases the defective gene product might be positively harmful, for example by interfering with the permeability of membranes, in which case the mutation would tend to be both dominant and lethal.

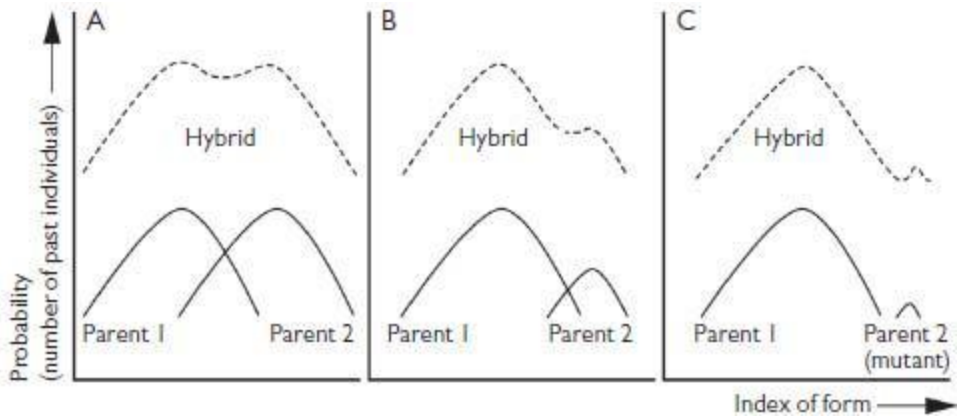

Figure 19. Diagrammatic representation of the probability structures of the morphogenetic fields of parents and hybrids.

These explanations for dominance in terms of molecular biology are satisfactory as far as they go; but in the absence of any mechanistic understanding of morphogenesis, the attempt to account for dominance in the inheritance of form by extrapolation from the molecular level inevitably begs the question.

The conventional genetic theories of dominance are more sophisticated than the purely biochemical theory; they emphasize that dominance is not fixed, but rather evolves.[5] In order to account for the relative uniformity of wild populations, in which most nonlethal mutations are recessive, they assume that the dominance of the “wild type” has been favored by natural selection. One theory postulates the selection of genes that modify the dominance of other genes,[6] and another theory the selection of increasingly effective versions of the genes that control the dominant characters in question.[7] But apart from the fact that there is little evidence in favor of either, and some against both, these theories suffer from the defect that they presuppose rather than explain dominance: they provide only hypothetical mechanisms by which it could be maintained or increased.[8]

According to the hypothesis of formative causation, dominance would evolve for a fundamentally different reason. Types favored by natural selection would be represented by larger numbers of individuals than types of lower survival value; as time went on, the former would become increasingly dominant through the cumulative effect of morphic resonance.

This hypothesis could in principle be distinguished experimentally from all conventional theories of dominance. According to the latter, under a

given set of environmental conditions, dominance depends only on the genetic constitution of a hybrid, whereas according to the former, it depends *both* on the genetic constitution *and* on morphic resonance from the parental types. Therefore, if the relative strength of the resonance from the parental types changed, the dominance of one over the other would change even if the genetic constitution of the hybrid remained the same.

Consider the following experiment. Hybrid seeds are obtained from a cross between plants of a well-defined variety (P1) and a mutant line (P2). Some of these hybrid seeds are placed in cold storage, while others are grown under controlled conditions. The characteristics of the hybrid plants are carefully observed, and the plants themselves are preserved. In these plants the P1 morphology is completely dominant (figure 19C). Then very large numbers of plants of the mutant type (P2) are grown in the field. Subsequently, some of the hybrids are again grown under the same conditions as they were before, from the same batch of seeds. Because P2 now makes a greater contribution by morphic resonance, P1 may be only partially dominant (figure 19B). After growing many more P2 plants, the form of the hybrids may be intermediate between the two parental types (figure 19A). Then still more plants of the P2 type are grown in large numbers; subsequently the hybrids are again grown under the same conditions as the previous hybrids from the same batch of seeds. Now the P2 type will make a greater contribution by morphic resonance than P1, and the P2 morphology will be dominant.

This result would strongly support the morphic resonance hypothesis of dominance, and would be completely incomprehensible from the point of view of orthodox genetic theory. The only problem with this experiment is that it might be difficult to perform in practice, since if P1 is a well-established variety that has existed for a very long time—in the case of a wild variety, perhaps for many thousands or even millions of years—it would not be practicable to grow comparable numbers of the P2 type. The experiment would be feasible only if P1 were a recent variety of which only a relatively small number of individuals had been grown in the past.

7.5 Family resemblances

Within a given variety, organisms differ from each other in all sorts of minor ways. In an interbreeding population, each individual is more or less unique genetically, and thus tends to follow its own characteristic path of development under the various quantitative influences of its genes. Moreover, since morphogenesis depends on the effect of probability structures on probabilistic events, the whole process is somewhat indeterminate. And then local environments vary. As a result of all these factors, each individual has a characteristic form and makes its own unique contribution to subsequent morphogenetic fields.

The most specific morphic resonance acting on a particular organism, apart from self-resonance from its own past, is likely to be that from previous closely related individuals with a similar genetic constitution, accounting for family resemblances. This specific morphic resonance will be superimposed on the less specific resonance from numerous past individuals of the same variety, and this in turn will be superimposed on a general background of morphic resonance from all past members of the species.

In the valley model of a chreode (figure 5), the most specific morphic resonance would determine the detailed course of morphogenesis, corresponding to the bed of a stream, and the less specific morphic resonance from previous individuals of the same variety the bed of a small valley. The variant chreodes of different varieties within the same species would correspond to small divergent or parallel valleys within a larger valley representing the chreode of the species as a whole.

7.6 Environmental influences and morphic resonance

The forms of organisms are influenced to varying degrees by the environmental conditions under which they develop. According to the hypothesis of formative causation, they are also influenced by the environmental conditions under which previous similar organisms developed, because the forms of these organisms contribute to their morphogenetic fields by morphic resonance. In terms of the radio analogy, the music coming out of the loudspeaker is affected not only by changes within the receiver, but also by changes within the broadcasting studio: if an orchestra starts playing a different piece of music, the radio set produces

different sounds even though its tuning and internal structures remain the same.

Consider, for example, a new variety of a cultivated species. If very large numbers of plants of this variety are grown in one environment and very few in others, the former will make a much larger contribution to the probability structures of the varietal morphogenetic fields; their form will be the most probable form of the variety and will therefore tend to influence the morphogenesis of all subsequent plants of the same variety, even when they are grown in different environments.

In order to test this prediction, it would be best to use a new variety of a self-pollinated crop; the plants would be very similar to each other genetically, and there would be no danger of their outcrossing with other varieties. To start with, a few plants would be grown in two very different environments, X and Y, and their morphological characters carefully recorded. Some of the original batch of seeds would be placed in cold storage. Then very large numbers of plants would be grown in environment Y (either in one season or over several generations). Subsequently, using some of the original seeds that had been kept in cold storage, a few plants would once again be grown in environment X. Their morphogenesis should now be influenced by morphic resonance from the large numbers of genetically similar plants grown in environment Y. Consequently, they should show more resemblance to the Y-type morphology than did the original X-type plants. Of course, for a valid comparison of plants grown on different occasions, it would be necessary to ensure that the conditions were practically identical; this would be impossible in the field, but could be achieved relatively easily with an artificially controlled environment in a phytotron.

If this result were actually obtained, it would provide positive evidence for the hypothesis of formative causation, and would be inexplicable in terms of the conventional theories.

7.7 The inheritance of acquired characteristics

The influence of previous organisms on subsequent similar organisms by morphic resonance would give rise to effects that could not conceivably occur if heredity depended on only the transfer of genes and other material

structures from parents to their progeny. This possibility enables the question of the “inheritance of acquired characteristics” to be seen in a new light.

In the fierce controversy at the end of the nineteenth and in the earlier part of the twentieth century, both the Lamarckians on the one hand and the followers of Weismann and of Mendel on the other assumed that heredity depended on only the germ plasm in general or the genes in particular. Therefore, if characteristics acquired by organisms in response to the environment were to be inherited, the germ plasm or the genes would have to undergo specific modifications. The anti-Lamarckians emphasized that such modifications seemed extremely unlikely, if not impossible. Even the Lamarckians themselves were unable to suggest any plausible mechanisms by which these changes could be brought about.

On the other hand, the Lamarckian theory seemed to provide a plausible explanation for hereditary adaptations in animals and plants. For example, camels have callosities on their knees. It is easy to understand how these are acquired in response to abrasion of the skin as the camels kneel down. But baby camels are born with them. Facts of this type would make good sense if acquired characteristics somehow became hereditary.

However, the neo-Darwinians deny this possibility, and offer an alternative interpretation in terms of random mutations: If organisms with the acquired characteristics in question are favored by natural selection, random mutations that happen to produce the same characteristics without the need to acquire them will also be favored by natural selection, and thus the characteristics will become hereditary. This hypothetical simulation of the inheritance of acquired characteristics is sometimes called the Baldwin effect, after one of the evolutionary theorists who first suggested it.[9]

In the early part of the twentieth century, dozens of scientists claimed to have demonstrated an inheritance of acquired characteristics in various species of animals and plants.[10] The anti-Lamarckians replied with counterexamples, citing the well-known experiment of Weismann in which he chopped the tails off mice for twenty-two successive generations and found that their progeny still developed tails. Another argument drew attention to the fact that after many generations of circumcision, Jewish men are still born with foreskins.

After the suicide of one of the leading Lamarckians, Paul Kammerer, in 1926, Mendelism became established in the West as the almost unchallenged orthodoxy.[11] Meanwhile, in the Soviet Union believers in the inheritance of acquired characteristics, led by Trofim Lysenko, gained control of the biological establishment in the 1930s and remained dominant until 1964. During this period, many of their Mendelian opponents were cruelly persecuted.[12] This polarization resulted in bitterness and dogmatism on both sides.

7.8 Epigenetic inheritance

There is now good evidence that acquired characteristics can indeed be inherited. Despite the taboo against Lamarckian inheritance in the West, more and more examples of the inheritance of acquired characteristics continued to accumulate. For example, in the 1960s the British botanist Alan Durrant discovered that when flax plants were grown with different kinds of fertilizer, not only did the plants grow differently, but these differences were inherited by their offspring, even when they were all grown under the same conditions. Some lines were larger than normal, others were smaller, and there were also inherited differences in the hairiness of the seeds. These differences persisted over many generations.[13]

Such cases, although well documented, were generally ignored. However, the taboo began to lift at the turn of the millennium with the widespread recognition of *epigenetics*. In a groundbreaking study with mice, Randy Jirtle and Robert Waterford, at Duke University, found that mice of the agouti strain, which are fat, yellow, and disease-prone, could be transformed by changing the diets of mothers, starting just before conception. After the mothers had been given a food supplement derived from soybeans, many of their offspring were slender, brown, and long-lived. Yet there had been no change in their DNA sequence; instead, the expression of the agouti gene had been modified epigenetically. It is now known that such changes can be passed on to subsequent generations.[14] Epigenetic changes are not always erased when sperm and egg cells are formed, as biologists assumed for decades.

Likewise through epigenetic inheritance, the effects of toxins can echo for generations. In a study at Washington State University, Michael Skinner

and his colleagues found that when pregnant rats were exposed to a commonly used agricultural fungicide, the development of their sons' testes was impaired, and they had a low sperm count later in life. *Their* sons also had lower sperm counts, and this effect was passed on from fathers to sons for four generations.[15]

Epigenetic effects also occur in invertebrates, like *Daphnia*, the water flea. When predators are around, the water fleas develop large defensive spines. When they reproduce, their offspring also have these spines even if they are not exposed to predators.[16]

There are now many examples of epigenetic inheritance in humans. For example, women who were pregnant when a famine hit the Netherlands at the end of the Second World War gave birth to malnourished, underweight babies. When these babies grew up, they too had babies with unusually low birth weights. A study in Sweden of men born between 1890 and 1920 showed that their nutrition in childhood affected the incidence of diabetes and heart disease in their grandchildren. And many common diseases that are inherited within families may also be passed on epigenetically.[17] The Human Epigenome Project was launched in 2003, and is helping to coordinate research in this rapidly growing field of inquiry.[18]

The prefix *epi* means "over and above." Epigenetic inheritance does not involve changes in the genes themselves, but rather changes in gene expression. For example, changes in the configuration of the chromatin—the DNA-protein complex that makes up the structure of chromosomes—can be passed on from cell to daughter cell. When these changes are transmitted through eggs or sperm, they are inherited. In other words, gene activity can be modified heritably without mutation. Another kind of epigenetic change, sometimes called genomic imprinting, involves the methylation of DNA molecules. There is a heritable chemical change in the DNA itself, but the underlying genes remain the same. A third kind of epigenetic inheritance depends on alterations in the cytoplasm, inherited through egg cells and hence only from mothers. In the light of epigenetics, evidence for the inheritance of acquired characteristics that was previously rejected or ignored has been rehabilitated.[19]

The hypothesis of formative causation allows for an inheritance of acquired characteristics through morphic resonance, without the need for

genetic changes or even for epigenetic inheritance. It complements rather than contradicts both these kinds of inheritance, and can be distinguished from them by experiment, as discussed below.

In general, when pathways of morphogenesis are altered by either environmental or genetic factors, similar processes of morphogenesis in subsequent similar organisms will tend to be canalized and stabilized by morphic resonance. The strength of this influence will depend on the specificity of the resonance and on the number of previous similar organisms whose morphogenesis has been altered; this number will tend to be large if the alterations are favored by natural or artificial selection, and small if they are not.

Mutilations of fully formed structures do not alter their pathways of morphogenesis unless they regenerate. Hence mutilations of non-regenerating structures would not be expected to influence the development of subsequent organisms by morphic resonance. This conclusion is in agreement with the findings that the amputation of the tails of mice and the circumcision of Jews have no significant hereditary effects.

7.9 Experiments with phenocopies

C. H. Waddington first introduced the term “epigenetic” into biology in the 1940s. In his laboratory at Edinburgh University in the 1950s he initiated what is still one of the most interesting and important lines of investigation into the inheritance of acquired characteristics, using phenocopies of fruit flies. Phenocopies are organisms whose characteristics resemble those produced as a result of genetic mutations, but which arise in response to a change in the environment instead. For example, the four-winged fruit fly shown in figure 17B has a mutation in the “bithorax” gene complex. Exposing the eggs of normal two-winged fruit flies to fumes of ether three hours after they are laid can produce similar four-winged flies, also known as bithorax flies. This effect occurred not because ether induced specific mutations in the DNA, but because it disturbed the normal pathway of development, much as exposing human embryos to thalidomide resulted in abnormal limbs.

Waddington found that by exposing fruit fly eggs to ether generation after generation, the proportion of bithorax flies increased: the phenocopies

became more frequent. After twenty-nine generations, some of the offspring of these flies showed the bithorax character without any exposure to ether at all. Waddington called this phenomenon “genetic assimilation.” A characteristic that was acquired in response to a changed environment had become hereditary.[20] In Waddington’s words: “All these experiments demonstrate that if selection takes place for the occurrence of a character acquired in a particular abnormal environment, the resulting selected strains are liable to exhibit that character even when transferred back to the normal environment.”[21]

As well as this orthodox-sounding interpretation in terms of natural selection, Waddington considered the possibility that some physical or chemical influence from the altered structures in the abnormal flies could have induced heritable modifications in their genes,[22] but he rejected it because the then prevalent doctrines of molecular biology provided no plausible mechanism by which such modifications could occur.[23] His final interpretation emphasized both the role of selection for the genetic potential to respond to the environmental stress by developing abnormally and the “canalization of development” involved in the modified morphogenesis. “To use somewhat picturesque language, one might say that the selection did not merely lower a threshold, but determined in what direction the developing system would proceed once it got over the threshold.”[24] Waddington himself coined the word *chreode* to express the notion of directed, canalized development (figure 5). He thought of the determination of the direction taken by a chreode in terms of its “tuning.” But he did not explain how this canalization and “tuning” came about, apart from making the vague suggestion that they somehow depended on the selection of genes.[25]

The hypothesis of formative causation complements Waddington’s interpretation: The chreodes and the final forms toward which they are directed depend on morphic resonance from previous similar organisms; the inheritance of acquired characteristics of the kind studied by Waddington depends *both* on genetic selection *and* on a direct influence by morphic resonance from the organisms whose development was modified in response to abnormal environments. Epigenetic inheritance may also play a part.

Mae-wan Ho and her colleagues at the Open University in Britain repeated Waddington's experiments in the 1980s,[26] but unlike Waddington they used an inbred strain of fruit flies with very little genetic variability, so there was very limited scope for genetic selection effects. They did not select bithorax flies as the parents of the next generation either; the great majority of the parents were normal-looking. Nevertheless, in the absence of genetic selection effects, they found that treating eggs with ether generation after generation led to an increase in the proportion of bithorax flies. After ten generations, some of the flies that grew from untreated eggs were bithorax, and so were their descendants, again without ether treatment. The more often that flies developed abnormally, the more probable the bithorax phenocopies became.

Experiments at Stanford University in the 1990s also showed that that the proportion of bithorax phenocopies increased progressively in successive generations treated with ether.[27] The most remarkable finding in Ho's laboratory was that when the experimental flies had been treated with ether for six generations, new batches of flies whose parents had never been exposed to ether reacted more strongly to the same ether treatment: in the first generation, 10 percent of the progeny were bithorax and in the second 20 percent, compared with 2 percent and 6 percent in the first and second generation of the original experimental line. In other words, phenocopies became more probable after similar flies had already developed the bithorax phenocopy, even in flies whose ancestors had never been exposed to ether. This result would be expected on the basis of morphic resonance, but not on any other hypothesis.

In setting up new experiments to test for morphic resonance with fruit flies, two lines could be compared. In one of them, E, the flies would be descended from stressed parents and the eggs would be treated with ether in every generation; in the other, F, the eggs treated with ether would come from unstressed parents, all of whose ancestors were also unstressed (see figure 20). If morphic resonance is at work, the frequency of phenocopies should increase in both lines, but this effect would be stronger in line E because of the combination of morphic resonance with epigenetic inheritance.

In the 1930s, Richard Goldschmidt, one of the most brilliant geneticists of his generation, found that in fruit flies, "it is possible to produce

practically every known type of mutant as phenocopy by the action of different degrees of heat shock during the sensitive periods of the pupa."[28] Waddington followed up Goldschmidt's observations by studying the effects of heat shocks on fruit fly pupae, looking at the development of veins in the wings. He found that exposing twenty-two-hour-old pupae to a temperature of 40°C caused some of them to develop into "crossveinless" phenocopies. In successive generations the proportion increased. After fifteen generations, more than 90 percent of the flies had crossveinless wings. Starting at generation fourteen, some of the untreated pupae developed crossveinless wings too.[29]

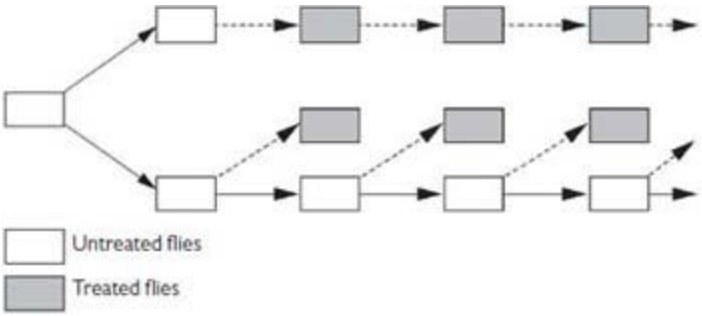

Figure 20. Diagrammatic representation of an experiment with an inbred strain of fruit flies comparing the effect of ether treatment of the eggs (dotted lines) in successive generations of flies descended from ether-treated parents (above) with control lines (below). If the proportion of phenocopies were to increase in successive generations of the control line, this would indicate a morphic resonance effect. An increase in the test line, with flies descended from ether-treated parents, could be due to a combination of morphic resonance and epigenetic inheritance.

Phenocopies occur in many other animals, including butterflies. Wing patterns in butterflies are particularly susceptible to heat or cold shocks. For example, when the pupae of the central European *Vanessa urticae* were exposed to low temperatures, some produced adults with wing patterns indistinguishable from *Vanessa polaris*, found in Lapland.[30] Also, a wide range of plants, including algae and liverworts, form phenocopies in response to physical or chemical changes in their environment.[31]

Phenocopies are of great interest in their own right, and provide many opportunities for research on the role of morphic resonance in morphogenesis.

8

THE EVOLUTION OF BIOLOGICAL FORMS

8.1 The neo-Darwinian theory of evolution

Very little is actually known or ever can be known about the details of evolution in the past. Nor is evolution readily observable in the present. Even on a timescale measured in millions of years, the origin of new species is rare, and of genera, families, and orders rarer still. The evolutionary changes that have actually been observed involve mainly the development of new varieties or races within established species. The best-known examples are of the emergence of dark-colored races of several European moths in areas where industrial pollution led to the blackening of the surfaces on which they settled. Dark mutants were said to be favored by natural selection because they were better camouflaged and hence less subject to predation by birds. But even the textbook example of the peppered moth turns out to be questionable; the often cited evidence includes deceptive photographs and flawed experiments.[1]

With such scanty direct evidence, and with so little possibility of experimental tests, any interpretation of the mechanism of evolution is bound to be speculative: unconstrained by detailed facts, it will largely consist of an elaboration of its initial assumptions about the nature of inheritance and the sources of heritable variation.

The orthodox interpretation is provided by the neo-Darwinian theory, which differs from the original Darwinian theory in two major respects:

first, it denies the inheritance of habits, which Darwin accepted; it asserts that heredity is essentially genetic. Second, it assumes that the ultimate source of heritable variability is random mutations of the genetic material.

Most neo-Darwinian theorists assume that divergent evolution under the influence of natural selection over long periods of time will lead to not only the development of new varieties and subspecies, but also new species, genera, and families.[2] This view has been disputed on the grounds that the differences between these higher taxonomic divisions are too great to have arisen by gradual transformations; apart from anything else, the organisms often differ in the number and structure of their chromosomes. Several authors have suggested that these large-scale evolutionary changes occur suddenly as a result of macro-mutations. Monstrous animals and plants in which structures have been transformed, reduplicated, or suppressed provide contemporary examples of such sudden changes. Occasionally in the course of evolution, “hopeful monsters” could have survived and reproduced.[3] One argument in favor of this view is that whereas gradual changes under selection pressure should result in forms with a definite adaptive value (except perhaps in small populations subject to “genetic drift”), macro-mutations could produce all sorts of apparently gratuitous large-scale variations that would be weeded out by natural selection only if they were positively harmful, thus helping to account for the prodigious diversity of living organisms.[4]

Although these neo-Darwinian theorists emphasize the importance of sudden large changes, they do not disagree with the orthodox assumptions that evolution as a whole depends on only random mutations and genetic inheritance, in combination with natural selection. More-radical critics object to the implicit or explicit mechanistic assumption that evolution as a whole is entirely purposeless.[5]

The metaphysical denial of any creative agency or purpose in the evolutionary process follows from the philosophy of materialism, with which the neo-Darwinian theory is so closely associated.[6] But unless scientific and metaphysical issues are to become hopelessly confounded, within the context of science the neo-Darwinian theory must be treated not as a metaphysical dogma but as a scientific hypothesis. As such it can hardly be regarded as proven: at best it offers a plausible interpretation of

the processes of evolution on the basis of its assumptions about genetic inheritance and the randomness of mutations.

The hypothesis of formative causation enables heredity to be seen in a new light, and therefore leads to a different interpretation of evolution.

8.2 Mutations

Changes are imposed upon organisms both from within, by genetic mutations, and from without, by alterations in the environment.

Mutations are accidental changes in the structure of genes or of chromosomes, individually unpredictable not only in practice but also in principle, because they depend on probabilistic events. Many mutations have effects that are so deleterious as to be lethal. Of those that are less harmful, some exert quantitative influences on pathways of morphogenesis, and give rise to variants of normal forms (section 7.3); others affect morphogenetic germs in such a way that whole pathways of morphogenesis are blocked or replaced by other pathways (section 7.2).

In those rare cases where mutations lead to changes that are favored by natural selection, not only will the proportion of mutant genes in the population tend to increase, in accordance with the neo-Darwinian theory, but also the repetition of the new pathways of morphogenesis in increasing numbers of organisms will reinforce the new chreodes: not only the "gene pools," but also the morphogenetic fields of a species will change and evolve as a result of natural selection.

8.3 The divergence of chreodes

If a mutation or environmental change perturbs a normal pathway of morphogenesis at a relatively early stage, the system may be able to regulate and go on to produce a normal final form in spite of this disturbance. If this process is repeated generation after generation, the chreodic diversion will be stabilized by morphic resonance; consequently a whole race or variety of a species will come to follow an abnormal pathway of morphogenesis while still ending up with the usual adult form.

In fact many such cases have been described; they are called "temporary deviations in development." For example, in the turbellarian worm

Prorhynchnus stagnitilis, the egg cells cleave either in a spiral or in a radial manner, and the developing embryos grow either inside the yolk or on its surface. Owing to these differences in early embryology, some of the organs are formed in different sequences; nevertheless, the adult animals are identical. And in a single species of the annelid worm *Nereis*, two very different kinds of larva are produced, but both develop into the same adult form.[7] In some such cases, the temporary deviations may be adaptive, for example to conditions of larval life, but in most they occur for no apparent reason.

Of much greater evolutionary significance are those divergences of chreodes that are not fully corrected by regulation and that therefore give rise to different final forms. Such changes in the pathway of development could arise as a result of either mutations (section 7.3) or unusual environmental conditions (section 7.6). In the case of mutation in an unchanged environment, if the deviant final form has a selective advantage, the mutant genes will increase in frequency within the population, and also the new chreode will be increasingly reinforced by morphic resonance. In more-complicated cases, where a variant form arises in response to unusual environmental conditions and has a selective advantage, the new chreode will be reinforced as before, and at the same time selection will also operate in favor of those organisms with the genetic capacity to respond in this way (cf. Waddington's experiments on fruit flies, section 7.8). Thus, the acquired characteristics will become hereditary through a combination of morphic resonance and genetic selection.

Under natural conditions, the operation of different selection pressures on geographically or ecologically isolated populations of a species will result in a divergence both of their "gene pools" and of their chreodes. Countless species of animals and plants have in fact diverged into genetically and morphologically distinct races and varieties; familiar examples are provided by domesticated animals and cultivated plants.[8] Think, for instance, of the amazingly diverse breeds of dog, ranging from the Afghan hound to the Pekinese, or the varieties of cabbage, *Brassica oleracea*, like kale, Savoy cabbage, Brussels sprouts, broccoli, and cauliflower.

In some cases, the morphological divergence affects only one particular structure or a small group of structures while others remain relatively unaffected. For example, in the small fish *Belone acus*, the jaws in the early

stages of development resemble those of related species, but subsequently they develop into an enormously elongated snout.[9]

Many structural exaggerations have evolved under the influence of sexual selection, for example the antlers of deer. And flowers provide thousands of examples of the divergent development of different component parts: compare, for instance, the modifications of the petals in different species of orchid.

In other cases, the form of many different structures has changed in a correlated manner. Indeed, if the forms vary in a particularly uniform and harmonious way, they can be compared diagrammatically using the systematic distortion of superimposed coordinates (figure 21), as Sir D'Arcy Thompson showed in the chapter titled "The Theory of Transformations, or the Comparison of Related Forms" from his essay *On Growth and Form.*

These kinds of evolutionary change take place within the context of already existing morphogenetic fields. They produce variations on given themes, but they cannot account for these themes themselves. In Thompson's words:

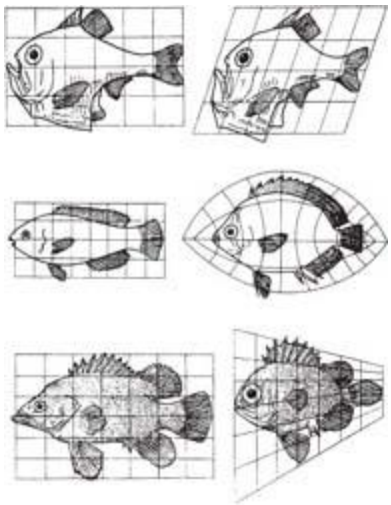

Figure 21. Comparisons of the forms of different species of fish. (From Thompson, 1942. Reproduced by courtesy of Cambridge University Press)

> We *cannot* transform an invertebrate into a vertebrate, nor a coelenterate into a worm, by any simple and legitimate deformation, nor by anything short of reduction to elementary principles . . . Formal

> resemblance, which we depend on as a trusty guide to the affinities of animals within certain bounds or grades of kinship and propinquity, ceases in certain other cases to serve us, because under certain circumstances it ceases to exist. Our geometrical analogies weigh heavily against Darwin's conception of endless small continuous variations; they help to show that discontinuous variations are a natural thing, that . . . sudden changes, greater or less, are bound to have taken place, and new "types" to have arisen, now and then.[10]

8.4 The suppression of chreodes

Whereas the divergence of chreodes within existing morphogenetic fields permits continuous or quantitative variation of form, developmental changes involving the suppression of chreodes or the substitution of one chreode for another result in qualitative discontinuities. According to the hypothesis of formative causation, these effects are caused by homeotic mutations or environmental factors that alter morphogenetic germs (section 7.2). Examples of mutant pea leaves in which leaflets are substituted for tendrils are shown in figure 18, and a bithorax mutant of *Drosophila* in figure 17.

Changes of these types probably occurred frequently in the course of evolution. For example, in certain species of *Acacia*, the leaves have been suppressed and their role taken over by flattened leaf stalks. This process can actually be seen in seedlings, where the first-formed leaves are typically pinnate (see figure 22).

In members of the cactus family, spines have replaced leaves. Among the insects, in almost every order there are species in which the wings have been suppressed either in both sexes, as in certain parasitic flies, or in only one sex, as in the female beetle known as the glowworm. In the case of ants, female larvae develop into either winged queens or wingless workers depending on the chemical constitution of their diet.

In some species, juvenile forms become sexually mature and reproduce without ever producing the characteristic structures of the adult, which are, as it were, short-circuited. The classic example is the axolotl, a tadpole of the tiger salamander, which reaches full size and becomes sexually mature

without losing its larval characteristics. If axolotls are supplied with thyroid hormone, they metamorphose into the air-breathing adult form and move out of the water onto land.

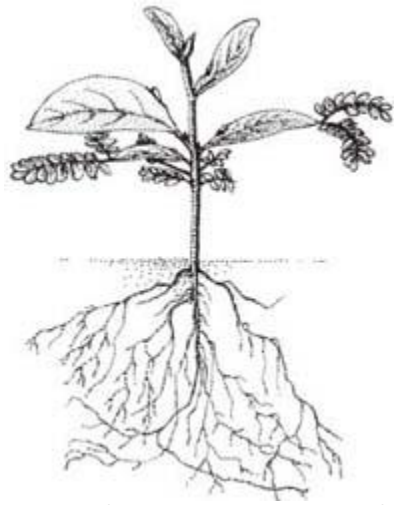

Figure 22. A seedling of an Acacia species. (After Goebel, 1898)

The most extreme examples of the suppression of chreodes are found among parasites, some of which have lost nearly all the structures characteristic of related free-living forms.

8.5 The repetition of chreodes

In all multicellular organisms, some structures are repeated several or many times: the tentacles of *Hydra*, the arms of the starfish, the legs of centipedes, the feathers of birds, the leaves of trees, and so on. Then many organs are made up of repeated structural units: the tubules of kidneys, the segments of fruits, etc. And, of course, tissues contain millions or billions of a few basic types of cell.

If, as a result of mutations or environmental changes, extra morphogenetic germs are formed within developing organisms, then certain structures can be repeated more than usual. A familiar horticultural example is that of “double” flowers, containing additional petals. Human babies are sometimes born with extra fingers or toes. And many instances of abnormally reduplicated structures can be found in the standard texts on teratology, ranging from double-headed calves to monstrous multiple pears (figure 23).

As these additional structures develop, regulation occurs in such a way that they are integrated more or less completely with the rest of the organism: for example, extra petals in double flowers have normal vascular connections, and extra fingers and toes have a proper blood supply and innervation.

Reduplication of structural units must have played an essential role in the evolution of new types of animals and plants, as shown by the structural repetitions within existing organisms. Moreover, many of the structures of animals and plants now different from each other may well have evolved from originally similar units. For example, insects are believed to have evolved from creatures resembling primitive centipedes, with a series of more or less identical segments, each bearing a pair of leglike appendages. The appendages on the segments at the front end may have given rise to the mouthparts and antennae, while the segments fused together to form the head. At the tail end some of the appendages may have been modified to produce structures concerned with mating and manipulation of the eggs. In the abdominal segments the appendages were suppressed, but in the three thoracic segments they were retained, and evolved into the modern insect legs.[11]

Figure 23. A monstrous pear. (After Masters, 1869)

Such a divergence of originally similar chreodes would only have been possible if the segmental morphogenetic germs became differentiated from one another in their structure; otherwise they would all have continued to become associated by morphic resonance with the same morphogenetic fields. Even in modern insects, if this diversification of the segmental primordial fails to occur during the early stages of embryology, then the

normal differences between segments are lost. This is what happens in the fruit fly *Drosophila* as a result of homeotic mutations in the bithorax gene complex: some transform the structures of the third thoracic segment into those of the second, so the fly bears two pairs of wings instead of one (figure 17); some transform abdominal segments into thoracic-type segments, bearing legs; and others have the reverse effect, transforming thoracic segments into segments of the abdominal type.[12]

8.6 The influence of other species

Practical breeders of animals and plants noticed long ago that cultivated varieties from time to time produced offspring resembling the ancestral wild type. Moreover, when two distinct cultivated varieties were crossed, the characters of the offspring sometimes resembled neither of the parental types, but rather those of the wild ancestors. This phenomenon was referred to as “reversion” or “atavism.” Darwin was particularly interested in this phenomenon because it agreed with his ideas about evolution and the inheritance of ancestral habits.[13]

In an evolutionary context, some morphological abnormalities can be thought of as reversions to patterns of development of more or less remote ancestral species. For instance, the formation of two pairs of wings in bithorax mutants of *Drosophila* (figure 17) can be interpreted as a throwback to the four-winged ancestors of flies.[14] Many more examples of putative atavisms can be found in the teratological literature.[15] Of course such interpretations can only be speculative, but they are not necessarily far-fetched. Mutations or abnormal environmental factors could give rise to internal conditions in embryonic tissues that resemble those in ancestral types, with similar morphogenetic consequences.

In most plants and animals, only a small proportion, less than 5 percent, of the chromosomal DNA contains genes coding for the organisms’ proteins. The function of the great majority of the DNA is unknown. Some of this DNA may play a part in the control of protein synthesis; some may have a structural role in the chromosomes; and some may consist of “redundant” ancestral genes that are no longer expressed. It has been suggested that if a mutation—for example due to a rearrangement of chromosome structure—led to the expression of such “latent” genes,

proteins characteristic of remote ancestors might suddenly be produced again, in some cases resulting in the reappearance of long-lost structures.[16]

In terms of the hypothesis of formative causation, if any such changes caused a morphogenetic germ to take up a structure and vibrational pattern similar to that of an ancestral species, it would come under the influence of a morphogenetic field of this species, even though all members of the species have been extinct for millions of years. Moreover, this effect need not be confined to ancestral types. If as a result of mutation (or for any other reason) a germ structure in a developing organism became sufficiently similar to a morphogenetic germ in any other species, contemporary or extinct, it would "tune in" to a chreode characteristic of the other species. And if the cells were capable of synthesizing appropriate proteins, the system would then develop under its influence.

In the course of evolution, closely similar structures sometimes seem to have appeared independently in more or less distantly related lines. For example, among the Mediterranean land snails, species belonging to well-differentiated genera, identifiable by their genitalia, have shells of nearly identical shape and structure; genera of fossil ammonites show the repeated parallel development of keeled and grooved shells; and similar or identical wing patterns occur in quite different families of butterfly.[17]

If a mutation resulted in an organism "tuning in" to another species' chreodes and consequently developing structures characteristic of that other species, it would soon be eliminated by natural selection if these structures reduced its chances of survival. On the other hand, if it were favored by natural selection, the proportion of such organisms in the population would tend to increase. The selection pressures that favored its increase might well resemble those that favored the original evolution of this particular character in the other species. And sometimes the structural resemblance might even be favored for its own sake, precisely because it enabled the organism to mimic members of another species. Thus evolutionary parallelisms may depend on both one species picking up the morphogenetic fields of another and parallel selection pressures.

8.7 The origin of new forms

According to the hypothesis of formative causation, morphic resonance and genetic inheritance together account for the repetition of characteristic patterns of morphogenesis in successive generations of plants and animals. Characteristics acquired in response to the environment can become hereditary through a combination of morphic resonance, epigenetics, and genetic selection.

The morphology of organisms can be changed through the suppression or repetition of chreodes; and some striking instances of parallel evolution can be attributed to the transfer of chreodes from one species to another.

However, neither the repetition, modification, addition, subtraction, nor permutation of existing morphogenetic fields can explain the origin of these fields themselves. During the course of evolution, entirely new morphic units together with their morphogenetic fields must have come into being: those of the basic types of cells, tissues, and organs; of fundamentally different kinds of lower and higher plants and animals, such as mosses, ferns, conifers, spiders, birds, and mammals; and of structures such as feathers and eyes.

As discussed in chapter 12, the origin of new forms can be ascribed to the creative activity of an agency pervading and transcending nature; or to a creative impetus immanent in nature; or to blind and purposeless chance. From the point of view of natural science, the question of evolutionary creativity can only be left open.

9

MOVEMENTS AND BEHAVIORAL FIELDS

9.1 Introduction

The discussion in the preceding chapters concerned the role of formative causation in morphogenesis. The subject of this and the following two chapters is the role of formative causation in the control of movement and behavior.

Some of the movements of plants and animals are spontaneous; that is to say they take place in the absence of any particular stimulus from the environment. Other movements take place in response to environmental stimuli. Of course, organisms respond passively to gross physical forces—a tree may be blown over by the wind, or an animal may be carried away by a strong current of water—but many responses are active, and cannot be explained as gross physical or chemical effects of the stimuli on the organism as a whole: they reveal the organism's *sensitivity* to the environment. This sensitivity generally depends on specialized receptors or sense organs.

The physical and chemical basis of the excitation of these specialized receptors by stimuli from the environment has been worked out in considerable detail; so has the physiology of nerve impulses; and so has the functioning of the muscles and other motor structures. But very little is known about the control and coordination of behavior.

In this chapter I suggest that just as formative causation organizes morphogenesis through the probability structures of fields that impose pattern and order on energetically indeterminate processes, so it organizes movements, and hence behavior. The similarities between morphogenesis and behavior are not immediately obvious, but are easiest to understand in the case of plants and unicellular animals such as *Amoeba*, whose movements are essentially morphogenetic. These are considered first.

9.2 The movements of plants

Plants generally move by growing.[1] This fact becomes easier to appreciate when they are seen on speeded-up films: shoots stretch out and curve toward the light; taproots thrust downward into the soil; and the tips of tendrils and climbing stems sweep out wide spirals in the air until they make contact with a solid support and coil around it.[2]

The growth and development of plants takes place under the control of their morphogenetic fields, which give them their characteristic forms. But the orientation of this growth is determined to a large extent by the directional stimuli of gravity and light. Environmental factors also influence the type of development: for example in dim light, plants become etiolated; their shoots grow relatively rapidly in a spindly manner until they get into brighter light.

Gravity is “sensed” through its effects on starch grains, which roll downward and accumulate in the lowest parts of the cells.[3] The direction from which light is coming is detected by the differential absorption of radiant energy on the illuminated and shaded sides of organs by a yellow carotenoid pigment.[4] The sense of “touch” by which climbing shoots and tendrils locate solid supports may involve the release of a simple chemical, ethylene, from the surface cells when they are mechanically stimulated.[5] The changeover from etiolated to normal growth depends on the absorption of light by a blue protein pigment called phytochrome.[6]

The responses to these stimuli involve complicated physical and chemical changes within the cells and tissues, and in some cases depend on the differential distribution of hormones such as auxin. However, the reactions cannot be explained in terms of these physical and chemical changes alone, but can be understood only within the context of the plants’

overall morphogenetic fields. For example, owing to their inherent polarity, plants produce shoots at one end and roots at the other. The directional stimulus of gravity orients this polarized development so that the shoots grow upward and the roots downward. The action of the gravitational field on starch grains within the cells and consequent changes in hormonal distribution are indeed causes of these oriented growth movements, but cannot in themselves account for the preexisting polarity; nor for the fact that the main shoots and roots respond in exactly opposite senses; nor for the fact that some plants grow into trees, while others are annual or perennial herbs, climbers, or creepers; nor for the particular patterns of branching in the shoot and root systems of different species, such as the patterns in which they spread—the upright form of a Lombardy poplar is very different from the shape of an English oak. All these characteristics depend on the morphogenetic fields.

Although most of the movements of plants occur only in young growing organs, some structures retain the ability to move even when they are mature, for example flowers that open and close again daily, and leaves that fold up at night. These movements are influenced by the intensity of the light and other environmental factors; they are also under the control of a "physiological clock" and continue to take place at approximately daily intervals, even if the plants are placed in an unchanging environment.[7] The leaves or petals open up because specialized cells in the "hinge" region at their bases become turgid; they close when these cells lose water owing to changes in the permeability of their membranes to inorganic ions.[8] The regaining of turgor is an active, energy-requiring process, comparable to growth.

In addition to making "sleep" movements, the leaves of some species move during the course of the day in response to the changing position of the sun. For example, in the pigeon pea, *Cajanus cajan*, the leaflets exposed to the sun are oriented approximately parallel to the sun's rays, exposing the minimum surface area to the intense tropical radiation. But leaves in the shade orient themselves at right angles to the incident radiation, thus intercepting the maximum amount of light. These responses depend on the direction and intensity of light falling on the specialized leaf joints, the pulvini. Throughout the day the leaves and leaflets are continuously adjusting their positions as the sun moves across the sky. At night they take

up their vertical “sleep” positions: the pulvini are sensitive to gravity as well as light.

In the sensitive plant, *Mimosa pudica*, the leaflets close up and the leaves point downward at night, as they do in many other leguminous plants. But these movements also occur rapidly during the daytime in response to mechanical stimulation (figure 24). The stimulus causes a wave of electrical depolarization, similar to a nerve impulse, to pass down the leaf; if the stimulus is strong enough, the impulse spreads to other leaves, which also fold up.[9] Similarly, in the Venus flytrap, *Dionaea muscipula*, mechanical stimulation of the sensitive hairs on the surface of the leaf causes an electrical impulse to travel to the turgid “hinge” cells, which rapidly lose water; the leaf closes like a trap around hapless insects, which are then digested.[10]

Figure 24. Leaves of the sensitive plant, Mimosa pudica. *Left, unstimulated; right, stimulated.*

These movements of leaves and leaflets in response to light, gravity, and mechanical stimulation are made possible by the fact that specialized cells are able to lose water and then “grow” again; they consequently retain a simplified morphogenetic potential, while that of most other tissues is lost when they mature and cease to grow. The reversible movements of these specialized structures are limiting cases of morphogenesis in which the changes of form have become stereotyped and repetitive. But their quasi-mechanistic simplicity is evolutionarily secondary, not primary; it has evolved from a background in which sensitivity to environmental stimuli is associated with the growth and morphogenesis of the plant as a whole.

9.3 Amoeboid movement

Amoebae move by the bulk flow of their cytoplasm into growing projections, the pseudopodia. They normally creep along the surface of solid objects by the continued extension of their front ends. But if the pseudopodia are touched, or if they encounter heat or strong solutions of various chemicals, they stop growing; others develop instead, and so the cells change course. If the new pseudopodia again encounter any potentially harmful stimuli, they too stop, and the amoebae move off in yet another direction. This system of "trial and error" continues until they find a pathway without obstacles or unfavorable stimuli.[11]

In free-floating amoebae not exposed to any particular directional stimulus, there is no consistent orientation of growth; pseudopodia keep developing in various directions until one of them comes into contact with a surface along which it can creep (figure 25).

The extension of pseudopodia presumably occurs under the influence of a specific polarized morphogenetic field. The orientation in which new pseudopodia start to form may depend to a large extent on chance fluctuations within the cell; the virtual pseudopodia projected outward from the cell body are then actualized through the organization of contractile filaments and other structures within the cytoplasm. This process continues until the development of the pseudopodia is inhibited by stimuli from the environment or by competition from pseudopodia growing in other directions.

The fact that amoeboid movements depend on continuous morphogenetic processes is aptly indicated in the specific name *Amoeba proteus* by the allusion to the mythical sea deity who kept changing from one shape to another.

Amoebae feed by engulfing food particles, such as bacteria, by the process of phagocytosis: pseudopodia grow around the particle that is in contact with the surface of the cell; the membranes of the pseudopodia fuse together, and the particle is enclosed within the cell surrounded by a part of the cell membrane. Other vesicles containing digestive enzymes fuse with this phagocytotic vesicle and the food is digested. This type of morphogenesis is distinct from that of cellular locomotion and presumably takes place under the influence of a different morphogenetic field, the orientation of which depends on the contact of the potential food particle with the membrane. This particle in contact with the membrane can be

regarded as the morphogenetic germ; the final form is the particle engulfed within the cell. The chreode of phagocytosis leading to this final form is given by morphic resonance from all similar acts of phagocytosis by similar amoebae in the past.

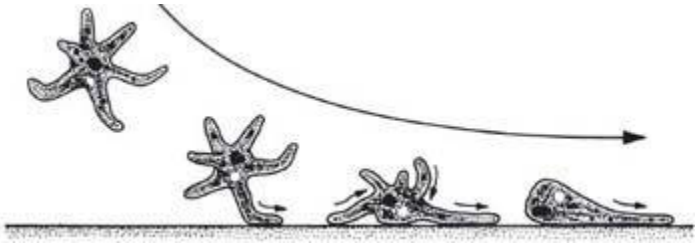

Figure 25. Method by which a floating amoeba passes to a solid surface. After Jennings, 1906)

9.4 The repetitive morphogenesis of specialized structures

The movements of most animals depend on the change of form of certain specialized structures, rather than of the body as a whole.

The beating of whiplike outgrowths, flagella or cilia, propels many unicellular organisms, while the form of the rest of the cell remains more or less fixed (figure 26). These motile organelles contain long tubular elements very similar to cytoplasmic microtubles. Cilia move because the microtubule filaments within them slide relative to one another with the consumption of chemical energy, just as filaments of actin and myosin slide relative to one another in muscle contractions. The change of shape of the microtubule proteins generates a shear force, resulting in the bending of the flagella or cilia.[12]

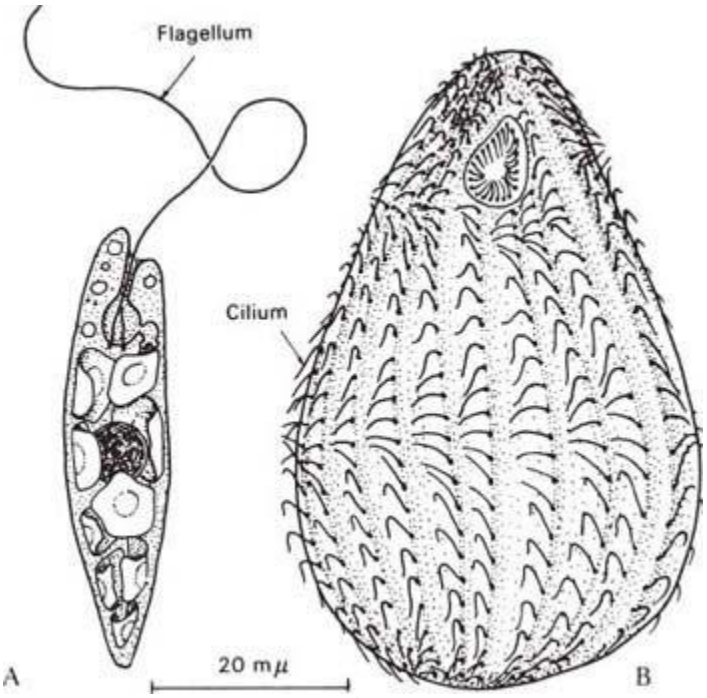

Figure 26. A: A flagellate, Euglena gracilis. (After Raven et al., 1976) B: A ciliate, Tetrahymena pyriformis. (After Mackinnon and Hawes, 1961)

In ciliates, the movements of the many individual cilia are coordinated in such a way that waves of beating pass over the surface of the cell. In some species, this coordination seems to depend on the mechanical influence of the cilia on their neighbors; and in others, on an excitatory system within the cell probably associated with fine fibrils connecting together the bases of the cilia.[13]

If a swimming ciliate, for example *Paramecium*, meets with an unfavorable stimulus, the direction of ciliary beating reverses: the organism backs away and then swims forward again in a new direction.[14] This avoidance reaction is probably triggered by the entry of calcium or other ions into the cell as a result of an alteration in membrane permeability brought about by the stimulus.[15]

The change of form of the beating flagella and cilia, as well as the control of this beating, takes place in such a stereotyped, repetitive way that it seems almost machinelike. This quasi-mechanistic specialization of structure and function is taken still further in the multicellular animals. Entire cells and groups of cells are specialized to undergo a repeated, simplified morphogenesis in their cycles of contraction and relaxation; others have a specialized sensitivity to light, chemicals, pressure, vibration,

or other stimuli; and the nerves, with their enormously elongated axons, are specialized to conduct electrical impulses from place to place, linking the sense organs and the muscles to the nerve net or central nervous system.

9.5 Nervous systems

Just as the beating of the cilia on the surface of a cell is coordinated through fibrils connecting the bases of the cilia, so the contraction of individual muscle cells is coordinated through the nerves. When a single nerve activates several neighboring muscle cells, they can be caused to contract simultaneously. And when the activity of this nerve is part of a higher-level system of control, the contraction of different groups of cells can be coordinated in a rhythmical manner, as it is in a muscle that maintains its tension over a period of time. Then yet higher-level systems control repetitive cycles of contraction in different muscles, for example in the legs of an animal as it runs. Hierarchies of coordination (cf. figure 10) are expressed through the nervous system.

But although nerves transmit definite “all-or-none” impulses from one place to another, formative causation would not be able to control animals’ movements through the nervous system unless the activity of the nerves was at the same time inherently probabilistic. In fact it is.

The firing of nerve impulses depends on changes in the permeability of the membranes of nerve cells to inorganic ions, in particular sodium and potassium. These changes can be brought about either by electrical stimulation or by specific chemical transmitters (e.g., acetylcholine) released from nerve endings at synaptic junctions (figure 27). The excitation of nerves by electrical stimuli around the threshold level has long been known to take place probabilistically.[16] The main reason for this is that the electrical potential across the membrane fluctuates in a random manner.[17] Moreover, the changes in postsynaptic membrane potentials caused by chemical transmitters also show random fluctuations,[18] which seem to be due to the probabilistic opening and closing of ionic “channels” across the membrane.[19]

There is an inherent probabilism not only in the responses of postsynaptic membranes to chemical transmitters but also in the release of the transmitters from the presynaptic nerve endings. Transmitter molecules are

stored in numerous microscopic vesicles (figure 27), and are released into the synaptic cleft when these vesicles fuse with the membrane. This process occurs spontaneously at random intervals, giving rise to discharges of so-called miniature end-plate potentials. The rate of secretion is greatly increased when an impulse arrives at the nerve ending, but here again the fusion of the vesicles with the membrane takes place probabilistically.[20]

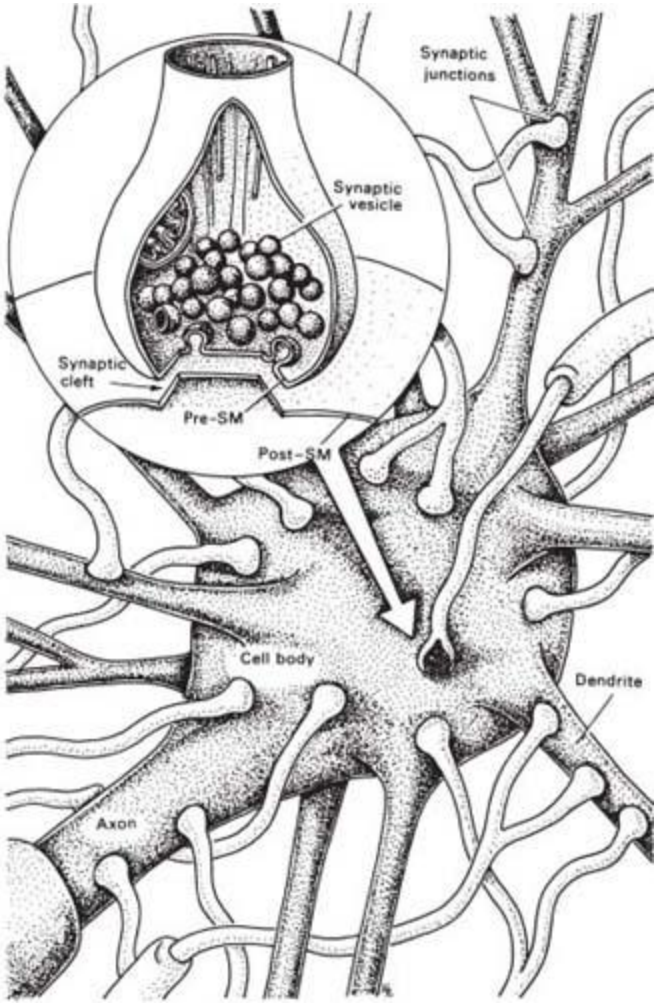

Figure 27. Part of a nerve cell, with numerous synapses on its surface. The inset shows an individual synapse in more detail. Pre SM = presynaptic membrane; Post SM = postsynaptic membrane. (Based on Krstic, 1979)

Within the brain, a typical nerve cell has thousands of fine, threadlike projections that end in synaptic junctions on other nerve cells, and, conversely, projections from hundreds or thousands of other nerve cells end in synapses on its own surface (figure 27). Some of these nerve endings release excitatory transmitters that tend to promote the firing of an impulse;

others are inhibitory and reduce the tendency of the nerve to fire. The triggering of impulses depends on a balance of excitatory and inhibitory influences from hundreds of synapses. At any given time, in many of the nerve cells in the brain this balance is poised so critically that firing either occurs or does not occur as a result of probabilistic fluctuations within the cell membranes or synapses. Thus the deterministic propagation of nerve impulses from place to place within the body is combined with a high degree of indeterminism within the central nervous system, which, on the present hypothesis, is ordered and patterned by formative causation.

9.6 Morphogenetic fields, motor fields, and behavioral fields

Although the fields controlling the changes of form of the specialized motor structures of animals are in fact morphogenetic fields, they bring about movements rather than net changes of form. For this reason it seems preferable to call them *motor fields*. (The word *motor* is used here as the adjective of the noun *motion*.) Motor fields, like morphogenetic fields, depend on morphic resonance from previous similar systems and are concerned with the actualization of virtual forms. Canalized pathways toward a final form or state can be referred to as chreodes in the context of motor fields, just as they can in the context of morphogenetic fields.

Motor fields, like morphogenetic fields, are hierarchically organized, and are in general related to development, survival, or reproduction. Whereas in plants these processes are almost entirely morphogenetic, in animals they also depend on movement. Indeed, in most animals even the maintenance of the normal functions of the body involves continual movement of internal organs such as the gut, the heart, and the breathing system.

Unlike plants, animals need to feed on other organisms in order to develop and maintain their forms. Hence an important high-level motor field in all animals is that of feeding. At this point the motor field becomes a *behavioral field*, responsible for organizing inherited or learned patterns of behavior. Behavioral fields are organized in a nested hierarchy or holarchy (figure 10), with higher-level fields coordinating the activity of lower-level fields, right down to the motor fields that organize the activity of groups of muscle cells.

The behavioral field of feeding organizes subsidiary behavioral fields responsible for finding, securing, and eating the plants or animals that serve as food. Some animals are sedentary and cause food to move toward them in water currents; some simply move around until they find plants they can eat; some stalk and hunt other animals; some make traps to catch their prey; some are parasitic; others are scavengers. All these methods of feeding depend on hierarchies of specific chreodes.

Another fundamental type of motor field is concerned with the avoidance of unfavorable conditions. *Amoeba* and *Paramecium* show the simplest type of reaction: backing or turning away from the unfavorable stimulus and setting off in some other direction. Sedentary animals such as *Stentor* and *Hydra* react to mildly unfavorable stimuli by contracting their bodies, but in response to more-severe stimuli they move away and settle down somewhere else. In addition to general avoidance reactions, more-complex animals also exhibit special types of behavior, shaped by behavioral fields, that help them to escape from predators; for example, they may run away swiftly, or stand their ground and somehow frighten the predator, or "freeze" in such a way that they are less easily seen.

The overall fields of development and survival have as their final form the fully grown animal under optimal conditions. Whenever this state is reached, there is no need for the animal to do anything in particular; but deviations from this state bring the animal under the influence of the various behavioral fields directed toward its restoration. In fact, such deviations are frequent: the animal's continuous metabolism depletes its reserves of food; changes in the environment expose it to unfavorable conditions; and predators approach it unpredictably. These and other changes are detected by the sensory structures and result in characteristic modifications of the nervous system, either directly or through the release of hormones such as adrenaline. These modifications of the activity of the nervous system provide the germ structures for particular behavioral fields, which enter into morphic resonance with previous similar patterns of activity in the same animal or in other, similar animals. Both individual and collective memory depends on morphic resonance.[21]

The attractor for the overall field of reproduction is the establishment of viable progeny. In unicellular organisms, and in simple multicellular animals such as *Hydra*, this is achieved by a morphogenetic process: the

organisms divide into two, or “bud off ” new individuals. Likewise, primitive methods of sexual reproduction are essentially morphogenetic: many lower animals (e.g., sea urchins) as well as lower plants (e.g., the seaweed *Fucus*) simply release millions of ova and sperm cells into the water around them.

In more-complex animals, the sperm are released in the vicinity of the ova as a result of specialized mating behavior. Thus the overall field of reproduction comes to cover the behavioral fields of searching for a mate, of courtship, and of copulation. Organisms may come under the influence of the first motor field in the sequence as a result of internal physiological changes mediated by hormones, as well as olfactory, visual, or other stimuli from potential mates. The end point of the first field constitutes the germ for the second, and so on: searching for a mate is followed by courtship, which, if successful, leads to the copulation chreode. In the simplest cases, the final form of the whole sequence is for the male ejaculation and for the female the laying of eggs. In many aquatic organisms they are simply released into the water, but in land animals the deposition of eggs often involves complex and highly specific patterns of behavior; for example, ichneumon flies inject their eggs into caterpillars of definite species, inside which the larvae develop parasitically, and potter wasps make small “pots” in which they place paralyzed prey before laying their eggs upon the prey and sealing the “pots.”

In some viviparous species the young are simply released and abandoned at birth. But when the young are cared for after they are born or hatched, a new range of behavioral fields comes into play, still under the overall field of reproduction of the parents, but at the same time serving the field of development and survival of the young. Consequently, the behavior of the animals takes on a social dimension. In the simplest cases the societies are temporary and disintegrate when the offspring become independent; in others they persist, with a consequent increase in the complexity of behavior. Special behavioral fields control the various types of communication between individuals and the differentiated tasks that different individuals perform. But the overall field that organizes the society is a field at a higher level: a *social field*.

A social field is the field of a social group. It organizes the form of the society and the interrelations between the individual animals within it. It is

a field in a nested hierarchy of fields (figure 10) at a higher and more inclusive level than the individual animals that make up the society.

In the extraordinarily complex societies of the termites, ants, and social bees and wasps, individuals of similar or identical genetic constitutions perform quite different tasks, and even the same individual may play different roles at different times—for example, a young worker bee may first clean the hive, then after a few days act as a brood nurse, then help build the honeycombs, then receive and store pollen, then guard the hive, and finally go out foraging.[22] Each of these roles must be covered by a social field, which in turn controls the behavioral and motor fields of the individual insects. Within those animals, the overall fields of behavior control the lower-level chreodes involved in the particular specialized tasks. Changes in the insect's nervous system bring it under the influence of one or other of these higher-level fields by causing it to enter into morphic resonance with previous workers that filled that particular role. Such changes depend to some extent on alterations in the physiology of the insect as it grows older, but they are also strongly influenced by the society as a whole: the roles of individuals change in response to disturbances of the hive or society; the whole system regulates.

The behavioral fields of feeding, avoidance, reproduction, etc., generally control a series of lower-level fields that act in sequence, the final form of one providing the germ structure for the next. Motor fields still lower in the hierarchy often act in cycles to give repetitive movements, such as those of the legs in walking, the wings in flying, and the jaws in chewing. At the lowest level are those fields concerned with the detailed control of the contraction of the cells within the muscles.

Behavioral fields embrace the sense organs, the nervous system, and the muscles, but also extend beyond the surface of the animal linking it to objects outside the animal, in the external world. Consider, for example, the field of feeding. The overall process—the capture and ingestion of food—is in fact a special type of aggregative morphogenesis (section 4.1). The hungry animal is the germ structure that enters into morphic resonance with previous fields of feeding. In the case of a predator, these fields are concerned with the capture and ingestion of prey. The field of capture projects into the space around the animal, and includes within it the virtual form of the prey (figure 11). This virtual form is actualized when an entity

corresponding to this virtual form is near the predator: the prey is recognized and the capture chreode initiated.[23] Theoretically, the behavioral field could affect probabilistic events in any or all of the systems it embraces, including the muscles, the perceptual system, and the prey itself. But in most cases its influence is concerned primarily with the modification of probabilistic events in the central nervous system, directing the movements of the animal toward the achievement of the final form, in this case the capture of the prey.

9.7 Behavioral fields and the senses

Through morphic resonance, an animal comes under the influence of specific behavioral fields as a result of its characteristic structure and internal patterns of rhythmic activity. These patterns are modified by changes arising within the body of the animal, and by influences from the environment.

If different stimuli brought about the same changes within the animal, then the same motor and behavioral fields would come into play. This is what seems to happen in unicellular organisms that give the same avoidance reaction to a wide variety of physical and chemical stimuli: probably all of them have similar effects on the physical and chemical state of the cell, for example by modifying the permeability of the cell membrane to calcium or other ions.

In simple multicellular animals with relatively little sensory specialization, the range of reactions to stimuli is not much greater than in unicellular organisms. *Hydra*, for instance, shows the same avoidance reactions to many different physical and chemical stimuli, and it responds to objects such as food particles only as a result of mechanical contact. However, as in certain unicellular organisms, its response to solid objects is modified by chemical stimuli. This can be demonstrated by a simple experiment: If small pieces of filter paper are supplied to the tentacles of a hungry *Hydra*, they evoke no reaction; but if they are first soaked in meat juice, the tentacles carry them toward the mouth, where they are swallowed.[24]

By contrast, animals possessing image-forming eyes can sense objects while they are still some distance away; consequently, the behavioral fields

project far farther outward into the environment; the range and scope of the animals' behavior is greatly increased. The detection of these fields by potential prey animals may underlie the sense of being stared at.[25]

The sense of hearing enables distant objects to be detected and permits an extension of the spatial range of the motor fields even into regions that cannot be seen. In some animals, most notably bats, this sense has replaced sight as the basis of the extended behavioral fields. And in a few aquatic species, such as the Mormyrid and Gymnotid electric fish, specialized receptors detect changes in the electric field set up around themselves by pulses from their electric organs; this sense enables them to locate prey and other objects in the muddy tropical rivers in which they live.

As animals move, the sensory stimuli arising both within their bodies and from the environment change as a consequence of their own movements. This continuous feedback plays an essential part in the coordination of movements by their motor fields.

Behavioral and motor fields, like morphogenetic fields, are probability structures that become associated by morphic resonance with physical systems on the basis of their three-dimensional patterns of oscillation. It is therefore of fundamental significance that all sensory inputs are translated into spatio-temporal patterns of activity within the nervous system. In the sense of touch, the stimuli act on particular parts of the body, which through specific nervous pathways are "mapped" within the brain; in vision, images falling on the retina bring about spatially patterned changes in the optic nerves and visual cortex. Although olfactory, gustatory, and auditory stimuli are not directly spatial, the nerves they excite through the relevant sense organs have specific locations, and the impulses traveling along these nerves into the central nervous system set up characteristic patterns of excitation.[26]

Thus particular stimuli and combinations of stimuli have characteristic spatio-temporal effects that have been revealed in ever-increasing detail by electroencephalographs (EEGs) and brain scans, for example by functional magnetic resonance imaging (fMRI). These dynamic patterns of activity bring the nervous system into morphic resonance with similar past nervous systems in similar states, and consequently under the influence of particular behavioral and motor fields.

9.8 Regulation and regeneration

Behavioral fields, like morphogenetic fields, attract the systems under their influence toward characteristic final forms. They usually do so by initiating a series of movements in a definite sequence. The intermediate stages are stabilized by morphic resonance; in other words, they are chreodes. But chreodes simply represent the most probable pathways toward final forms. If the normal pathway is blocked, or if the system is deflected from it for any reason, the same final form may be reached in a different way: the system regulates (section 4.1). Many, but not all, morphogenetic systems are capable of regulation; and so are social, behavioral, and motor systems.

Regulation occurs under the influence of behavioral and motor fields at all levels in the hierarchy: for example, if a few muscles or nerves in a dog's leg are damaged, the pattern of contraction in the other muscles adjusts so that the limb functions normally. If the leg is amputated, the movements of the remaining legs change in such a way that the dog can still walk, although with a limp. If parts of its cerebral cortex are damaged, after some time it recovers more or less completely. If it is blinded, its ability to move around gradually improves as it comes to rely more on its remaining senses. And if its normal route toward its home, its food, or its puppies is blocked, it changes its habitual sequence of movements until it finds a new way to reach its goal.

The behavioral equivalent of regeneration occurs when the final form of a chreode has been actualized but is then disrupted: think, for instance, of a cat that has caught a mouse, the end point of the capture chreode. If the mouse escapes from its clutches, then the cat's movements are directed toward recapturing it.

Out of all the examples of "behavioral regeneration," the homology with morphogenetic regeneration is shown most clearly in "morphogenetic behavior," concerned with the making of nests and other structures. In some cases animals mend these structures after they have been damaged. For example, potter wasps can fill in holes made by the experimenter in the walls of their pots, sometimes using actions never normally performed when the pots are being constructed.[27] And termites repair damage to their galleries and nests through the cooperative and coordinated activities of many individual insects.[28]

Activities such as these have sometimes been interpreted as evidence of intelligence, on the ground that animals behaving in a rigidly fixed, instinctive manner would not be able to respond so flexibly to unusual situations.[29] But by the same token, regulating sea urchin embryos and regenerating flatworms could also be said to exhibit intelligence. However, this extension of psychological terminology is more confusing than helpful. From the point of view of the hypothesis of formative causation, the similarities can be recognized but interpreted the other way around. Seen against the background of morphogenetic regulation and regeneration, the ability of animals to reach behavioral goals in unusual ways raises no fundamentally new principles. And when, in higher animals, certain types of behavior no longer follow standard chreodes— when behavioral regulation becomes, as it were, the rule rather than the exception—this flexibility can be seen as an extension of the possibilities inherent in morphogenetic and motor fields by their very nature.

9.9 Morphic fields

Morphogenetic fields organize morphogenesis. Motor fields organize movements; behavioral fields organize behavior; and social fields organize societies. These fields are hierarchically ordered in the sense that social fields include and organize the behavioral fields of animals within the society; the animals' behavioral fields organize their motor fields; and the motor fields depend for their activity on the animals' nervous systems and bodies organized by morphogenetic fields.

These are all different kinds of *morphic field.* "Morphic field" is a generic term that includes all kinds of fields that have an inherent memory given by morphic resonance from previous similar systems.[30] Morphogenetic, motor, behavioral, and social fields are all morphic fields, and they are all essentially habitual.

10

INSTINCT AND LEARNING

10.1 The influence of past actions

Behavioral fields, like morphogenetic fields, are given by morphic resonance from previous similar systems. The detailed structure of an animal and the patterns of oscillatory activity within its nervous system will generally resemble *itself* more closely than any other animal. Thus the most specific morphic resonance acting upon it will be that from its own past (section 6.5). The next most specific resonance will come from genetically similar animals that lived in the same environment, and the least specific from animals of other races living in different environments. In the valley model of the chreode (figure 5), the latter will stabilize the general outline of the valley, while the more specific resonance will determine the detailed topology of the valley bottom.

The “contours” of the chreodic valley depend on the degree of similarity between the behavior of similar animals of the same race or species. If their patterns of movement show little variation, morphic resonance will give rise to deep and narrow chreodes, represented by steep-walled valleys (figure 28A). These will have a strongly canalizing effect on the behavior of subsequent individuals, which will therefore tend to behave in very similar ways. Stereotyped patterns of movement brought about by such chreodes at lower levels appear as reflexes, and at higher levels as instincts.

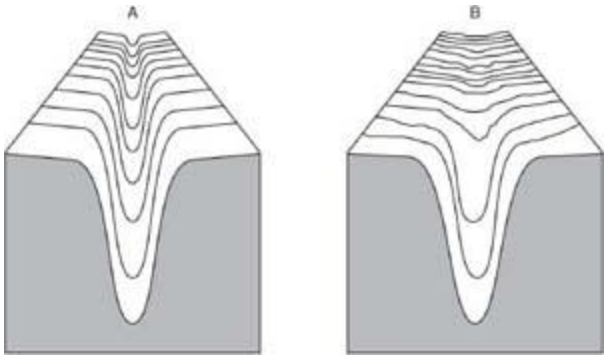

Figure 28. Diagrammatic representation of a deeply canalized chreode (A)*, and a chreode that is weakly canalized in the initial stages* (B).

On the other hand, if similar animals reach the final forms of their behavioral fields by different patterns of movement, the chreodes will not be so well defined (figure 28B); there will therefore be more scope for individual differences in behavior. But once a particular animal has reached the behavioral goal in its own way, its subsequent behavior will tend to be canalized in the same way by morphic resonance from its own past states; and the more often these actions are repeated, the stronger will this canalization become. Such characteristic individual chreodes reveal themselves as habits.

Thus, from the point of view of the hypothesis of formative causation, there is a difference only of degree between instincts and habits: both depend on morphic resonance, the former from countless previous individuals of the same species and the latter mainly from past states of the same individual.

Reflexes and instincts depend on the very specifically patterned morphogenesis of the nervous system in the first place, itself dependent on morphic resonance.

During learning, physical or chemical changes may occur in the nervous system that facilitate the repetition of a pattern of movement. Perhaps in simple nervous systems carrying out stereotyped functions, the potential for such changes may already be “built in” to the “wiring” in such a way that learning occurs quasi-mechanistically. For example, in the snail *Aplysia*, the structure of the nervous system is almost identical in different individuals, down to the fine details of the arrangement of excitatory and inhibitory synapses on particular cells. Very simple types of learning occur in connection with the reflex withdrawal of the gill into the mantle cavity,

namely habituation to harmless stimuli, and sensitization to harmful ones. As the snails learn, the activities of particular excitatory and inhibitory synapses acting on individual nerve cells alter in definite ways.[1] These kinds of changes in nerve cells are called long-term potentiation.[2] Of course, the mere description of these processes does not in itself reveal the reasons for the alterations; these are at present a matter for conjecture. But how is this detailed specialization of structure and function in the nerves and synapses established in the first place? The problem is shifted back to the realm of morphogenesis.

The nervous systems of higher animals are much more variable from individual to individual than in invertebrates such as *Aplysia*, and far more complicated. Very little is known about the way in which learned patterns of behavior are retained,[3] but enough has been found out to make it clear that there can be no simple explanation in terms of specifically localized physical or chemical "traces" within the nervous tissue.

Numerous investigations have shown that in mammals learned habits often persist after extensive damage to the cerebral cortex or to subcortical regions of the brain. Moreover, when loss of memory does occur, it is closely related not to the location of the lesions, but rather to the total amount of tissue destroyed. Karl Lashley summed up the results of hundreds of experiments as follows: "It is not possible to demonstrate the isolated localization of a memory trace anywhere within the nervous system. Limited regions may be essential for learning or retention of a particular activity, but within such regions the parts are functionally equivalent."[4]

A similar phenomenon has been demonstrated in an invertebrate, the octopus: observations on the survival of learned habits after destruction of various parts of the vertical lobe of the brain have led to the seemingly paradoxical conclusion that "memory is both everywhere and nowhere in particular."[5]

These findings are extremely puzzling from a mechanistic point of view. In an attempt to account for them, the neuroscientist Karl Pribram has suggested that memory "traces" are somehow distributed within the brain in a manner analogous to the storage of information in the form of interference patterns in a hologram.[6] But this remains no more than a vague speculation.

The hypothesis of formative causation provides an alternative interpretation, in the light of which the persistence of learned habits in spite of damage to the brain is far less puzzling: the habits depend on behavioral fields that are not stored within the brain at all, but rather are given directly from its past states by morphic resonance.

Some of the implications of the hypothesis of formative causation in relation to instinct and learning are considered in the following sections, and experimental tests are discussed in chapter 11.

10.2 Instinct

In all animals, some patterns of motor activity are inborn rather than learned. The most fundamental are those of the internal organs, such as the heart and gut, but many of the patterns of movement of limbs, wings, and other motor structures are also innate. This is most clearly apparent when animals are able to move around competently almost as soon as they are born or hatched.

It is not always easy to make the distinction between inborn and learned behavior. In general, characteristic behavior that develops in young animals reared in isolation can usually be regarded as innate. However, behavior that appears only when they are in contact with other members of their species may also be innate, but require stimuli from the other animals for its expression.

Studies of the instinctive behavior of a wide range of animals have led to several general conclusions, which constitute the classical concepts of ethology.[7] These can be summarized as follows:

(1) Instincts are organized in a hierarchy of “systems” or “centers” superimposed upon one another. Each level is activated primarily by a system at the level above it. The highest center of each of the major instincts may be influenced by a number of factors including hormones, sensory stimuli from the viscera of the animal, and stimuli from the environment.

(2) The behavior that occurs under the influence of the major instincts often consists of chains of more or less stereotyped patterns of behavior called *fixed action patterns*. When such a fixed action pattern constitutes

the end point of a major or minor chain of instinctive behavior, it is called a *consummatory act*. The behavior in the earlier part of an instinctive chain of behavior, e.g. searching for food, may be more flexible, and is usually called *appetitive behavior*.

(3) Each system requires a specific stimulus in order to be activated or *released*. This stimulus or releaser may come from within the animal's body or from the environment. In the latter case, it is often referred to as a *sign stimulus*. A given releaser or sign stimulus is presumed to act on a specific neurosensory mechanism called the *innate releasing mechanism*, which releases the reaction.

These concepts harmonize remarkably well with the ideas of behavioral and motor fields developed in the previous chapter. The fixed action patterns can be understood in terms of chreodes, and the innate releasing mechanisms as the germ structures of the appropriate motor fields.

10.3 Sign stimuli

The instinctive responses of animals to sign stimuli show that they somehow abstract certain specific and repeatable features from their environments. As the ethologist Niko Tinbergen put it:

> An animal responds "blindly" to only part of the total environmental situation and neglects other parts, although its sense organs are perfectly able to receive them . . . These effective stimuli can easily be found by testing the response to various situations differing in one or other of the possible stimuli. Moreover, even when a sense organ is involved in releasing a reaction, only part of the stimuli that it can receive is actually effective. As a rule, an instinctive reaction responds to only very few stimuli, and the greater part of the environment has little or no influence, even though the animal may have the sensory equipment for receiving numerous details.[8]

The following examples illustrate these principles:[9]

The aggressive reaction of male stickleback fish during the breeding season to other male sticklebacks is released mainly by the sign stimulus of the red

belly: models with very crude shapes but with red bellies are attacked much more than models with the correct shape but no red coloration. Similar results have been obtained in experiments with the red-breasted robin: a territory-holding male threatens very approximate models with red breasts, or even a mere bundle of red feathers, but responds much less to accurate models without red breasts.

Young ducks and geese react instinctively to the approach of birds of prey, in a manner that depends on the shape of the bird in flight. Studies with cardboard models have shown that the most important feature is a short neck—characteristic of hawks and other predatory birds—while the size and shape of the wings and tail are relatively unimportant. In certain moths, the sex odor or pheromone normally produced by females causes males to attempt to copulate with any object bearing it.

In locusts of the species *Ephippiger ephippiger,* males attract females willing to mate by their song. Females are attracted to singing males from a considerable distance, but ignore silent males even when quite near. Males that are silenced by gluing their wings together are incapable of attracting females. Hens come to the rescue of chicks in response to their distress call, but not if they simply see them in distress, for example behind a soundproof glass barrier.

According to the hypothesis of formative causation, recognition of these sign stimuli depends on morphic resonance from previous similar animals exposed to similar stimuli. Owing to the process of automatic averaging, this resonance will emphasize only the common features of the spatio-temporal patterns of activity brought about by these stimuli in the nervous system. The result will be that only certain specific stimuli are abstracted from the environment, whereas others are ignored. Consider, for example, the stimuli acting on hens whose chicks are in distress. Imagine a collection of photographs taken of chicks in distress on many different occasions. Those taken at night will show nothing; those in the daytime will show chicks of different sizes and shapes seen from the front, the rear, the side, or from above; moreover, they may be near to other objects of all shapes and sizes, or even concealed behind them. Now if all these photographs are superimposed to produce a composite image, no features whatever will be reinforced; the result will simply be a blur. By contrast, imagine a series of

tape recordings made at the same time the photographs were taken. All bear the record of distress calls, and if these sounds are superimposed, they reinforce each other to give an automatically averaged distress call. This superimposition of photographs and tape recordings is analogous to the effects of morphic resonance from the nervous systems of previous hens on a subsequent hen exposed to stimuli from a chick in distress: the visual stimuli result in no specific resonance and evoke no instinctive reaction, however pathetic the chick may look to a human observer, whereas the auditory stimuli do.

This example serves to illustrate what seems to be a general principle: *shapes* are very often ineffective as sign stimuli. The probable reason is that they are highly variable because they depend on the angle from which things are seen. By contrast, colors are much less critically dependent on viewpoint, and sounds and odors hardly at all. Significantly, colors, sounds, and odors play important roles as releasers of instinctive reactions; and in those cases where shapes *are* effective, there is a certain constancy of viewpoint. For example, young birds on the ground see predators flying above them in silhouette, and do indeed respond to such shapes. And when shapes or patterns serve as sexual sign stimuli, they do so in courtship displays or “presentations” in which animals take up definite stances or postures in relation to their potential mates. The same is true of displays of submission and aggression.

10.4 Learning

Learning can be said to occur when there is any relatively permanent adaptive change in behavior as a result of past experience. Four general categories can be distinguished:[10]

(1) The most universal type, found even in unicellular organisms,[11] is *habituation*, which can be defined as the waning of a response as a result of repeated stimulation that is not followed by any kind of reinforcement. A common example is the fading of alarm or avoidance responses to new stimuli that turn out to be harmless: animals get used to them.

This phenomenon implies the existence of some sort of memory, which enables the stimuli to be recognized when they recur. On the hypothesis of

formative causation, this recognition is due primarily to the morphic resonance of the organism with its own past states, including those brought about by new sensory stimuli. This resonance serves to maintain, and indeed define, the identity of the organism with itself in the past (section 6.5). Repeated stimuli from the environment to which responses are not reinforced will effectively become part of the organism's own "background." Conversely, any new features of the environment will stand out because they are not so recognized: usually the animal will respond with avoidance or alarm precisely because the stimuli are unfamiliar.

In the case of certain stereotyped responses, such as the withdrawal reflex of the gill in the snail *Aplysia*, habituation may occur in a quasimechanistic manner on the basis of preexisting structural and biochemical specializations in the nervous system (section 10.1). But if so, this specialization is secondary, and seems likely to have evolved from a situation in which habituation depended more directly on morphic resonance.

(2) In all animals, innate patterns of motor activity appear as the individuals grow up. While some are carried out perfectly the first time they are performed, others improve with time. A young bird's first attempts to fly, for example, and a young mammal's first attempts to walk may be only partially successful, but they get better after repeated attempts. Not all such improvement is due to practice: in some cases it is simply a matter of maturation and occurs just as much with the passage of time in animals that have been immobilized.[12] Nevertheless, many types of motor skill do improve in a way that cannot be attributed to maturation.

From the point of view of the hypothesis of formative causation, this type of learning can be interpreted in terms of behavioral regulation. Morphic resonance from countless past members of the species gives an automatically averaged chreode, which governs an animal's first attempts to carry out a particular innate pattern of movement. This standard chreode may give only approximately satisfactory results, for example because of deviations from the norm in the animal's sense organs, nervous system, or motor structures. As the movements are performed, regulation will spontaneously bring about fine adjustments to the overall chreode, and to the lower-level chreodes under its control. These adjusted chreodes will be

stabilized by morphic resonance with the animal's own past states as the pattern of behavior is repeated.

(3) Animals may come to respond to a stimulus with a reaction that is normally evoked by a different stimulus. This type of learning occurs when the new stimulus is applied at the same time as, or immediately before, the original one. The classical examples are the conditioned reflexes established by I. P. Pavlov in dogs. The dogs salivated when they were presented with food. On repeated occasions a bell was rung as the food was presented, and after some time they began to salivate at the sound of the bell even in the absence of food.

An extreme example of this type of learning occurs in the "imprinting" of young birds, especially ducklings and goslings. Soon after hatching, they respond instinctively to any large moving object by following it. Normally this is their mother; but they will also follow foster mothers, human beings, or even inanimate objects dragged in front of them. After a relatively short time, they come to recognize the general features of the moving object, and later the specific features. Then only the particular bird, person, or object with which they have become imprinted elicits their following.

Analogously, animals often learn to recognize the individual features of their mates or their young by sight, sound, smell, or touch. This recognition takes time to develop: for example, a pair of coots with newly hatched chicks will feed and even adopt strange chicks similar in appearance to their own; but when their young are about two weeks old, they recognize them individually, and henceforth tolerate no strangers, however similar.[13]

A comparable process is probably responsible for the recognition of particular places, such as nest sites, by means of landmarks and other features associated with them. Indeed, this type of learning seems likely to play an important part in the development of visual recognition in general. Since the stimuli from an object differ according to the angle from which it is viewed, the animal must learn that they are all connected with the same thing. Likewise, the associations between different kinds of sensory stimulus from the same object—visual, auditory, olfactory, gustatory, and tactile—usually have to be learned.

When the new stimulus and the original stimulus occur simultaneously, it might at first sight seem likely that the different patterns of physical and

chemical change they bring about in the brain gradually become linked with each other as a result of frequent repetition. But two difficulties stand in the way of this apparently simple interpretation. First, the new stimulus might not be simultaneous with the usual one, but precede it. In this case, it seems necessary to suppose that the influence of the stimulus persists for a while, so that it is still present when the usual stimulus occurs. This kind of memory can be thought of by analogy with an echo that gradually dies away. The existence of short-term memory is well established;[14] it could be explicable in terms of reverberating circuits of nervous activity within the brain.[15]

Associative learning seems to involve definite discontinuities: it occurs in steps, or stages. This may be because the linkage between the new and the original stimulus involves the establishment of a new motor field: the field responsible for the original response must somehow be enlarged to incorporate the new stimulus. In effect, a *synthesis* occurs in which a new motor unit comes into being. And a new unit cannot emerge gradually, but only by a sudden jump (or by several successive jumps).

(4) As well as learning to respond to a particular stimulus *after* they have received it, animals may also learn to behave in such a way that they reach a goal as a *result* of their activities. In the language of the Behaviorist school, this is called “operant conditioning.” The response “emitted” by the animal precedes the reinforcing stimulus. Rats in “Skinner boxes” provide the classic examples. These boxes contain a lever that, when pressed, releases a pellet of food. After repeated trials, rats learn to associate the pressing of the lever with the reward. Similarly, they can learn to press a lever in order to avoid the painful stimulus of an electric shock.

The association of a particular pattern of movement with a reward or with the avoidance of punishment usually seems to happen as a result of trial and error. But intelligence of an altogether higher order has been demonstrated in primates, especially chimpanzees. In the early twentieth century, Wolfgang Köhler found that these apes were capable of solving problems in an “insightful” way.[16] For example, chimpanzees were placed in a high chamber with unclimbable walls. From the ceiling hung a bunch of ripe bananas, too high to reach. After a number of attempts to get the fruit by standing on their hind legs and by jumping, they gave up trying

these ways. After a while, one of the apes glanced first at one of a number of wooden boxes that had been placed in the chamber at the beginning of the experiment and then at the bananas. He dragged the box underneath them and stood on it. This did not bring him high enough, so he fetched another box and put it on top of the first, but it was still not high enough; he then added a third, climbed up, and grabbed the fruit.

Many more examples of insightful behavior have been demonstrated by subsequent investigators: in one experiment, for instance, chimpanzees learned to use sticks to rake in food placed outside the cages beyond their reach. They did this sooner if they had been allowed to play with the sticks for several days beforehand; during this period they came to use the sticks as functional extensions of their arms. Thus the use of the sticks to rake in the food represented "the integration of motor components acquired during earlier experience into new and appropriate behavior patterns."[17]

In both trial and error and insight learning, existing chreodes are integrated within new higher-level motor fields. These syntheses can come about only by sudden "jumps." If the new patterns of behavior are successful, they will tend to be repeated. Hence the new motor fields will be stabilized by morphic resonance as the learned behavior becomes habitual.

10.5 Innate tendencies to learn

The originality of learning may be absolute: a new motor field may come into being not only for the first time in the history of an individual, but for the first time ever. On the other hand, an animal may learn something that other members of its species have already learned in the past. In this case, the emergence of the appropriate motor field will be facilitated by morphic resonance from previous similar animals. If a motor field becomes increasingly well established through repetition in many individuals, learning will become progressively easier: there will be a strong innate disposition toward acquiring this particular pattern of behavior.

Thus learned behavior that is repeated very frequently will tend to become semi-instinctive. By a converse process, instinctive behavior may come to be semi-learned. The songs of birds illustrate the intergradations between instinctive and learned behavior particularly clearly.[18] In some species, such as the wood pigeon and the cuckoo, the pattern of the song

varies little from bird to bird and is almost completely innate. But in others, for example the chaffinch, the song has a general structure characteristic of the species, but in its fine detail it differs from individual to individual; these differences can be recognized by other birds and play an important part in the birds' family and social life. Birds raised in isolation produce simplified and rather featureless versions of the chaffinch song, showing that its general structure is innate. However, under normal conditions they develop and improve their singing by imitating other chaffinches. This process is taken much further in mockingbirds, for example, which borrow elements from the songs of other species. And some kinds of birds, notably parrots and mynahs, when kept in captivity often imitate their human foster parents.

In species whose songs are almost entirely innate, the lack of individual variation is both an effect and a cause of the well-defined and highly stabilized motor chreodes (figure 28A): the more the same pattern of movement is repeated, the greater will be its tendency to be repeated in the future. But in species with individual differences in song, morphic resonance will give less well-defined chreodes (figure 28B): the general structure of the chreode will be given by the process of automatic averaging, but the details will depend on the individual and its own experience and habits, remembered through morphic resonance with itself in the past.

11

THE INHERITANCE AND EVOLUTION OF BEHAVIOR

11.1 The inheritance of behavior

On the hypothesis of formative causation, the inheritance of behavior depends on genetic inheritance, *and* on epigenetic inheritance, *and* on the morphogenetic fields that control the development of the nervous system and the animal as a whole, *and* on the behavioral and motor fields given by morphic resonance from previous similar animals. By contrast, according to the conventional theory, innate behavior is supposed to be “programmed” in the DNA.

Relatively few experimental investigations have been carried out on the inheritance of behavior, largely because it is difficult to quantify. Nevertheless, various attempts have been made: for instance, in experiments with rats and mice, behavior has been measured in terms of their running speed on treadmills; the frequency and duration of sexual activity; defecation scores, defined as the number of fecal boluses deposited in a given area in unit time; maze-learning abilities; and susceptibility to audiogenic seizures, caused by very loud noises. A heritable component of these responses has been demonstrated by breeding from animals with high or low scores: the progeny tend to have scores resembling those of their parents.[1]

The trouble with investigations of this type is that they reveal very little about the inheritance of patterns of behavior; moreover, the results are

difficult to interpret because they are open to influence by so many different factors. For example, a lower treadmill speed or a reduced frequency of mating could be due simply to a general reduction in vigor as a consequence of a heritable metabolic deficiency.

In some cases, the reasons for genetic alterations of behavior have been investigated in considerable detail. In the small nematode worm *Caenorhabditis*, certain mutants that wriggle abnormally show structural changes in their nervous systems.[2] In *Drosophila*, various "behavioral mutations" that abolish the normal response to light have been found to affect the photoreceptors or the peripheral visual neurons.[3] In mice, a number of behavioral mutations are known to affect the morphogenesis of the nervous system, leading to defects of whole regions of the brain. In human beings, various congenital abnormalities of the nervous system are associated with abnormal behavior, for example in Down's syndrome. And then behavior can also be affected by hereditary physiological and biochemical defects; for instance, in humans the condition of phenylketonuria, associated with mental retardation, is due to a deficiency of the enzyme phenylalanine hydroxylase.

The fact that innate behavior is affected by genetically determined alterations in the structure and function of the sense organs, nervous system, etc., does not, of course, prove that its inheritance is explicable in terms of genetic factors alone; it shows only that a normal body is necessary for normal behavior. Think again of the radio analogy: Changes within the set affect its performance, but this does not prove that the music that comes out of the loudspeakers originates inside the set itself.

In the realm of behavior, biochemical, physiological, and anatomical changes may prevent the appearance of germ structures, and hence whole motor fields may fail to act; or they may have various quantitative effects on the movements controlled by these fields. And, in fact, investigations on the inheritance of fixed action patterns show that "it is not difficult to find variations that affect the performance in a minor fashion, but the unit still appears in a clearly recognizable form if it appears at all."[4]

The inheritance of behavioral and motor fields is probably dependent on the factors already discussed in connection with the inheritance of morphogenetic fields (chapter 7). Generally speaking, in hybrids between

two races or species, the dominance of the behavioral fields of one over those of the other is likely to depend on the relative strength of the morphic resonance from the parental types (figure 19). If one belongs to a well-established race or species and the other to a relatively new one with a small past population, the behavioral fields of the former would be dominant. But if the parental races of species were equally well established, the hybrids would come under the influence of both to a similar extent.

This is in fact what seems to happen. In some cases, the results are bizarre because the patterns of behavior of the parental types are incompatible with each other. One example is provided by the hybrids produced by crossing two kinds of lovebird. Both parental species make their nests out of strips that they tear from leaves in a similar manner, but whereas one (Fischer's lovebird) then carries these strips to the nest in its bill, the other (the peach-faced lovebird) carries them tucked in among its feathers. Hybrids tear the strips from the leaves normally, but then behave in a most confused manner, sometimes tucking the strips in among their feathers, sometimes carrying them in their bills; but even when they carry them in their bills, they erect the feathers of the lower back and rump and attempt to tuck them in.[5]

11.2 Morphic resonance and behavior: an experimental test

In mechanistic biology, a sharp distinction is drawn between innate and learned behavior: the former is assumed to be "genetically programmed" or "coded" in the DNA, while the latter is supposed to result from physical and chemical changes in the nervous system. There is no conceivable way in which such changes could specifically modify the DNA, as the Lamarckian theory would require; it is therefore considered impossible for learned behavior acquired by an animal to be inherited by its offspring (excluding, of course, "cultural inheritance," whereby the offspring learn patterns of behavior from their parents or other adults).

By contrast, according to the hypothesis of formative causation, there is no difference in kind between innate and learned behavior: both depend on motor fields given by morphic resonance (section 10.1). This hypothesis therefore admits a possible transmission of learned behavior from one animal to another, and leads to testable predictions that differ not only from

those of the orthodox theory of inheritance, but also from those of the Lamarckian theory.

Consider the following experiment. Animals of an inbred strain are placed under conditions in which they learn to respond to a given stimulus in a characteristic way. They are then made to repeat this pattern of behavior many times. *Ex hypothesi,* the new behavioral field will be reinforced by morphic resonance, which will not only cause the behavior of the trained animals to become increasingly habitual, but will also affect, although less specifically, any similar animal exposed to a similar stimulus: the larger the number of animals in the past that have learned the task, the easier it should be for subsequent similar animals to learn it. Therefore, in an experiment of this type it should be possible to observe a progressive increase in the rate of learning not only in animals descended from trained ancestors, but also in genetically similar animals descended from untrained ancestors. This prediction differs from that of the Lamarckian theory, according to which only the descendants of trained animals should learn quicker. And on the conventional theory, there should be no increase in the rate of learning of the descendants of untrained or trained animals.

To summarize: An increased rate of learning in successive generations of both trained and untrained lines would support the hypothesis of formative causation; an increase only in trained lines, the Lamarckian theory; and an increase in neither, the orthodox theory.

Tests of this type have, in fact, already been performed. The results support the hypothesis of formative causation. William McDougall started the original experiment at Harvard in 1920, in the hope of providing a thorough test of the possibility of Lamarckian inheritance. The experimental animals were white rats, of the Wistar strain, that had been carefully inbred under laboratory conditions for many generations. Their task was to learn to escape from a specially constructed tank of water by swimming to one of two gangways that led out of the water. The “wrong” gangway was brightly illuminated, while the “right” gangway was not. If the rat left by the illuminated gangway, it received an electric shock. The two gangways were illuminated alternately, one on one occasion, the other on the next. The number of errors made by a rat before it learned to leave the tank by the non-illuminated gangway gave a measure of its rate of learning.

> Some of the rats required as many as 330 immersions, involving approximately half that number of shocks, before they learnt to avoid the bright gangway. The process of learning was in all cases one that suddenly reached a critical point. For a long time the animal would show clear evidence of aversion for the bright gangway, frequently hesitating before it, turning back from it, or taking it with a desperate rush; but, not having grasped the simple relation of constant correlation between bright light and shock, he would continue to take the bright route as often or nearly as often as the other. Then, at last, would come a point in his training at which he would, if he found himself facing the bright light, definitely and decisively turn about, seek the other passage, and quietly climb out by the dim gangway. After attaining this point, no animal made the error of again taking the bright gangway, or only in very rare instances.[6]

In each generation, the rats from which the next generation were to be bred were selected at random *before* their rate of learning was measured, although mating took place only after they were tested. This procedure was adopted to avoid any possibility of conscious or unconscious selection in favor of quicker-learning rats.

This experiment was continued for thirty-two generations and took fifteen years to complete. In accordance with the Lamarckian theory, there was a marked tendency for rats in successive generations to learn more quickly. This is indicated by the average number of errors made by rats in the first eight generations, which was over fifty-six, compared with forty-one, twenty-nine, and twenty in the second, third, and fourth groups of eight generations, respectively.[7] The difference was apparent not only in the quantitative results, but also in the actual behavior of the rats, which became more cautious and tentative in the later generations.[8]

McDougall anticipated the criticism that in spite of his random selection of parents in each generation, some sort of selection in favor of quicker-learning rats could nevertheless have crept in. In order to test this possibility, he started a new experiment, with a different batch of rats, in which parents were indeed selected on the basis of their learning score. In one series, only quick learners were bred from in each generation, and in

the other series only slow learners. As expected, the progeny of the quick learners tended to learn relatively quickly, while the progeny of the slow learners learned relatively slowly. However, even in the latter series, the performance of the later generations improved very markedly, in spite of repeated selection in favor of slow learning (see figure 29).

These experiments were done carefully, and critics were unable to dismiss the results on the ground of flaws in technique. But they did draw attention to a weakness in the experimental design: McDougall had failed to test systematically the change in the rate of learning of rats whose parents had not been trained.

One of these critics, Francis Crew, of Edinburgh University, repeated McDougall's experiment with rats derived from the same inbred strain, using a tank of similar design. He included a parallel line of "untrained" rats, some of which were tested in each generation for their rate of learning, while others, which were not tested, served as the parents of the next. Over the eighteen generations of this experiment, Crew found no systematic change in the rate of learning either in the trained or in the untrained line.[9] At first, this seemed to cast serious doubt on McDougall's findings. However, Crew's results were not directly comparable in three important respects. First, the rats found it much easier to learn the task in his experiment than in the earlier generations of McDougall's. So pronounced was this effect that a considerable number of rats in both trained and untrained lines "learned" the task immediately without receiving a single shock! The average scores of Crew's rats right from the beginning were similar to those of McDougall's after more than thirty generations of training. Neither Crew nor McDougall was able to provide a satisfactory explanation of this discrepancy. But, as McDougall pointed out, since the purpose of the investigation was to bring to light any effect of training on subsequent generations, an experiment in which some rats received no training at all and many others received very little would not be qualified to demonstrate this effect.[10] Second, Crew's results showed large and apparently random fluctuations from generation to generation, far larger than the fluctuations in McDougall's results, which could well have obscured any tendency to improve in the scores of later generations. Third, Crew adopted a policy of very intensive inbreeding, crossing only brothers with their sisters in each generation. He had not expected this to have

adverse effects, since the rats came from an inbred stock to start with: “Yet the history of my stock reads like an experiment in inbreeding. There is a broad base of family lines and a narrow apex of two remaining lines. The reproductive rate falls and line after line becomes extinct.”[11]

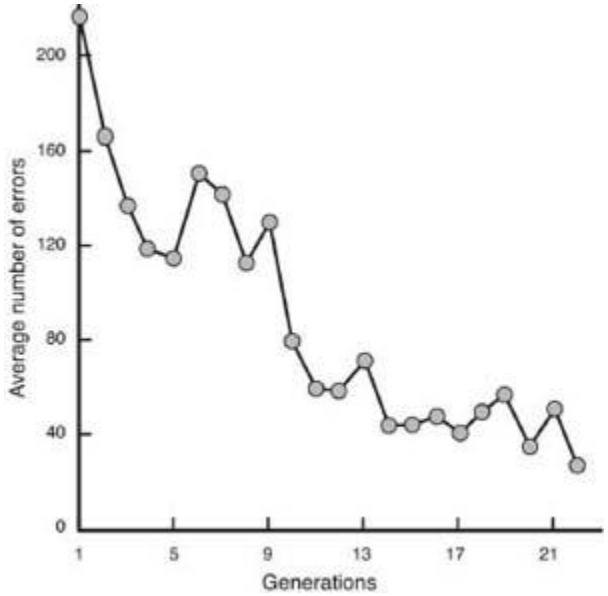

Figure 29. The average number of errors in successive generations of rats selected in each generation for slowness of learning. (Data from McDougall, 1938)

Even in the surviving lines, a considerable number of animals were born with such extreme abnormalities that they had to be discarded. The harmful effects of this severe inbreeding could well have masked any tendency for the rate of learning to improve. Altogether, these defects in Crew’s experiment mean that the results can only be regarded as inconclusive; and in fact he himself was of the opinion that the question remained open.[12]

Fortunately, this was not the end of the story. Wilfred Agar and his colleagues at Melbourne University carried out the experiment again, using methods that did not suffer from the disadvantages of Crew’s. Over a period of twenty years, they measured the rates of learning of trained and untrained lines for fifty successive generations. In agreement with McDougall, they found that there was a marked tendency for rats of the trained line to learn more quickly in subsequent generations. *But exactly the same tendency was also found in the untrained line*.[13]

It might be wondered why McDougall did not also observe a similar effect in his own untrained lines. The answer is that he did. Although he

tested control rats from the original untrained stock only occasionally, he noticed “the disturbing fact that the groups of controls derived from this stock in the years 1926, 1927, 1930 and 1932 show a diminution in the average number of errors from 1927 to 1932.” He thought this result was probably fortuitous, but added: “It is just possible that the falling off in the average number of errors from 1927 to 1932 represents a real change of constitution of the whole stock, an improvement of it (with respect to this particular faculty) whose nature I am unable to suggest.”[14]

With the publication of the final report by Agar’s group in 1954, the prolonged controversy over “McDougall’s Lamarckian Experiment” came to an end. The similar improvement in both trained and untrained lines ruled out a Lamarckian interpretation. McDougall’s *conclusion* was refuted, and that seemed to be the end of the matter. On the other hand, his *results* were confirmed.

These results seemed completely inexplicable; they made no sense in terms of any current ideas, and they were never followed up. But they make good sense in the light of the hypothesis of formative causation. Of course they cannot in themselves prove the hypothesis; it is always possible to suggest other explanations, for example that the successive generations of rats became increasingly intelligent for an unknown reason unconnected with their training.[15]

In future experiments, the most unambiguous way of testing for the effects of morphic resonance would be to cause large numbers of rats (or any other animals) to learn a new task in one location, and then see if there was an increase in the rate at which similar rats learned to carry out the same task at another location hundreds of miles away. The initial rate of learning at both locations should be more or less the same. Then, according to the hypothesis of formative causation, the rate of learning should increase progressively at the location where large numbers are trained; and a similar increase should also be detectable in the rats at the second location, even though very few rats had been trained there. Obviously, precautions would need to be taken to avoid any possible conscious or unconscious bias on the part of the experimenters. One way would be for experimenters at the second location to test the rate of learning of rats in several *different* tasks, at regular intervals, say monthly. Then at the first location, the particular task in which thousands of rats would be trained

would be chosen at random from this set. Moreover, the time at which the training began would also be selected at random; it might, for example, be four months after the regular tests began at the second location. The experimenters at the second location would not be told either which task had been selected or when the training had begun at the first location. If, under these conditions, a marked increase in the rate of learning in the selected task were detected at the second location after the training had begun at the first, then this result would provide strong evidence in favor of the hypothesis of formative causation.

An effect of this type might well have occurred when Crew and Agar's group repeated McDougall's work. In both cases, their rats started off learning the task considerably quicker than McDougall's when he first began his experiment.[16]

If this experiment gave positive results, it would not be fully reproducible by its very nature: in attempts to repeat it, the rats would be influenced by morphic resonance from the rats in the original experiment. To demonstrate the same effect again and again, it would be necessary to change either the task or the species used in each experiment.

11.3 The evolution of behavior

Whereas the fossil record provides direct evidence about the structure of past animals, it reveals practically nothing about their behavior. Consequently, most ideas about the evolution of behavior cannot be based on evidence from the past, but only on comparisons between living species today. Thus, for example, theories can be constructed about the evolution of social behavior in bees by comparing existing social species with solitary and colonial species, which are presumed to be more primitive. But however reasonable such theories may seem, they can never be more than speculative.[17] Moreover, theories of behavioral evolution depend on *assumptions* about the way in which behavior is inherited, since so little is actually known.

The neo-Darwinian theory assumes that innate behavior is "programmed" or "coded" in the DNA, and that new types of behavior are caused by chance mutations. Then natural selection favors favorable mutants; hence instincts evolve. Chance mutations are also assumed to give

animals capacities for particular types of learning. Then animals whose survival and reproduction benefit from these capacities are favored by natural selection. Hence capacities for learning evolve. Even a tendency for learned behavior to become innate can be attributed to chance mutations, by the hypothetical Baldwin effect: animals may respond to new situations by learning to behave in appropriate ways; chance mutations that cause this behavior to appear without the need for learning will be favored by natural selection; hence behavior that was at first learned may become innate, not because of an inheritance of acquired characteristics, but because appropriate mutations happen by chance and are selected for.

There seems to be practically no limit to what can be accounted for by the invocation of favorable chance mutations that change the "genetic programming" of behavior. These neo-Darwinian theories can be developed in a mathematical form by calculations based on the formulae of theoretical population genetics.[18] But insofar as these speculations are untestable, they have no independent value; they merely elaborate the mechanistic assumptions from which they start.

The hypothesis of formative causation leads to very different interpretations of the evolution of behavior. To the extent that genetic changes influence behavior, natural selection would still be expected to lead to alterations in the gene pools of populations. But the specific patterns of behavior themselves depend on the inheritance of behavioral fields by morphic resonance. The more a given pattern of behavior is repeated, the stronger will this resonance become. Thus the repetition of instinctive behavior will tend to fix the instincts more and more. On the other hand, if patterns of behavior vary from individual to individual, morphic resonance will not produce well-defined chreodes; hence the behavior will be less stereotyped. The greater the variety of behavior, the greater will be the scope for variation in future generations. This kind of evolution permitting the emergence of intelligence has taken place to some extent among the birds, more so in the mammals, and most of all in humans.

In some cases, behavior that is semi-learned may have evolved from a background in which it was fully instinctive. One way in which this could have happened is through the hybridization of races with different chreodes, giving rise to composite motor fields with more scope for individual variation.

Conversely, semi-instinctive behavior could have evolved from behavior that was originally learned, as a result of frequent repetition. Consider, for example, the behavior of different breeds of dog. Sheepdogs have been trained and selected over many generations for the ability to round up sheep, retrievers to retrieve, pointers to point, fox hounds to chase foxes, and so on. Dogs often show an innate tendency toward the behavior characteristic of their breed even before they are trained.[19] Perhaps these tendencies are not quite strong enough to be called instincts, but they are strong enough to show that there is only a difference of degree between instinct and a hereditary predisposition to learn particular types of behavior. Of course, breeds of dogs have evolved under conditions of artificial rather than natural selection, but the same principles apply in both cases.

While it is relatively easy to imagine how some types of instinctive behavior could have developed by the repetition of learned behavior generation after generation, this cannot feasibly account for the evolution of all types of instinct, especially in animals with a very limited capacity for learning. Possibly some new instincts emerged from new permutations and combinations of preexisting instincts; one way in which this could occur would be through hybridization between races or species with different patterns of behavior. Another way in which new combinations might come about is through the incorporation of "displacement activities," the seemingly irrelevant actions performed by animals "torn" between conflicting instincts. Certain elements of courtship rituals may well have originated in this way.[20] It is also conceivable that mutations or exposure to unusual environments could enable an animal to tune in to other species' motor chreodes (section 8.6).

But in addition to the recombination of existing chreodes, there must be some way in which entirely new motor fields come into being in animals whose behavior is almost entirely instinctive. New patterns of behavior could emerge only if the usual repetition of ancestral behavior was blocked, either by a change in the environment or by a mutation that altered the normal physiology or morphogenesis of the animal. In most such cases the animal might act in an uncoordinated and ineffective manner, but occasionally a new motor field might come into being. And whenever a new field appears for the first time, there must be a jump that cannot be

fully accounted for in terms of preceding energetic or formative causes (sections 5.1, 8.7).

If the pattern of behavior due to a new behavioral field impairs the ability of animals to survive and reproduce, it will not be repeated very often; animals that persist in this behavior will be eliminated by natural selection. But if the new pattern of behavior helps animals to survive and reproduce, it will tend to be repeated and reinforced by morphic resonance.

11.4 Human behavior

Higher animals often behave more flexibly than do lower animals. However, this flexibility is confined to the early stages of a behavioral sequence, and especially to the initial appetitive phase; the later stages, and in particular the final stage, the consummatory act, are performed in a stereotyped manner as fixed action patterns (section 10.1).

In terms of the landscape model, a major motor field can be represented by a broad valley, which then narrows down and becomes increasingly steep-walled, finally ending up in a deep canyon (figure 28B). The broad valley corresponds to the appetitive phase, in which many alternative pathways can be followed; these pathways then converge as they are funneled toward the final highly canalized chreode of the consummatory act.

In human behavior the ranges of ways in which behavioral goals are reached are far wider than in any other species, but the same principles seem to apply: under the influence of the higher-level behavioral fields, patterns of action are funneled toward stereotyped consummatory acts that are generally innate. For example, people obtain their food by all sorts of different methods, either directly by hunting, gathering, fishing, herding, or farming, or earn it indirectly by performing various tasks or jobs. Then the food is prepared and cooked in many different ways, and placed in the mouth by a variety of means, for instance by hand, or with chopsticks, or on a spoon. But there is little difference in the way the food is chewed, and the consummatory act of the whole motor field of feeding, swallowing, is similar in all people. Likewise, in the behavior governed by the motor field of reproduction, methods of courtship and systems of marriage differ widely, but the consummatory act of copulation toward which they lead is

more or less stereotyped. In the male, the final fixed action pattern, that of ejaculation, proceeds automatically, and is in fact innate.

Thus the very varied patterns of human behavior are usually directed toward a limited number of goals given by the motor fields inherited from past members of the species by morphic resonance; in general, these goals are related to the development, maintenance, or reproduction of the individual or social group. Even play and exploratory activity not immediately directed toward such goals often help achieve them later on, as they do in other species, for neither play nor "generalized exploratory appetitive behavior" in the absence of immediate reward is confined to humans: rats, for example, explore their environment and investigate objects even when they are satiated.[21]

However, not all human activity is subordinated to the motor fields that canalize it toward biological or social goals; some is explicitly directed toward transcendent ends. This kind of behavior is shown in its purest form in the lives of saints.

Although the range of variation in human behavior is very wide when the species as a whole is considered, in any given society the activities of individuals tend to fall into a limited number of standard patterns. People usually repeat characteristically structured activities that have already been performed over and over again by many generations of their predecessors. These include the speaking of a particular language; the skills associated with hunting, farming, weaving, tool-making, cooking, and so on; songs and dances; and the types of behavior specific to particular social roles. All of these can be thought of as morphic fields.

Richard Dawkins coined the word *meme* to refer to "a unit of cultural transmission, or a unit of *imitation*."[22] He deliberately chose a word that sounded rather like gene to stress the analogy between genes and memes as replicators. But one of the problems with this term is that it is atomistic: it implies that memes are independent units, at the same level as each other. By contrast, thinking of cultural inheritance in terms of morphic fields has no such implication: morphic fields are organized in nested hierarchies (figure 10).[23]

All the patterns of activity characteristic of a given culture can be regarded as morphic fields. The more often they are repeated, the more

strongly stabilized they will be. But because of the bewildering variety of culture-specific morphic fields, each of which could potentially canalize the movements of any human being, morphic resonance cannot by itself lead an individual into one set of chreodes rather than another. So none of these patterns of behavior expresses itself spontaneously: all have to be learned. An individual is initiated into particular patterns of behavior by other members of the society. Then as the process of learning begins, usually by imitation, the performance of a characteristic pattern of behavior brings the individual into morphic resonance with all those who have carried out this pattern in the past. Consequently, learning is facilitated as the individual "tunes in" to specific morphic fields.[24]

Processes of initiation are indeed traditionally understood in terms rather similar to these. Individuals are thought to enter into states or modes of existence that precede them and have a transpersonal reality. The facilitation of learning by morphic resonance would be difficult to demonstrate empirically in the case of long-established patterns of behavior, but a change in the rate of learning should be more readily detectable with motor patterns of recent origin. Thus, for example, it should have become progressively easier to learn to ride a bicycle, drive a car, ski, or play a video game, owing to the cumulative morphic resonance from the large number of people who have already acquired these skills. However, even if reliable quantitative data showed that the rates of learning had in fact increased, the interpretation would be complicated by the probable influence of other factors like improved machine design, better teaching methods, and a higher motivation to learn. But with specially designed experiments in which precautions are taken to hold these other factors constant, it might well be possible to obtain persuasive evidence for the predicted effects. Recent experimental research on morphic resonance in human learning is summarized in appendix A.

The hypothesis of formative causation applies to all aspects of human behavior in which particular patterns of movement are repeated. But it cannot account for the origin of these patterns in the first place. Here, as elsewhere, the problem of creativity lies outside the scope of natural science, and an answer can be given only on metaphysical grounds (sections 5.1, 8.7, and 11.3).

12

FOUR POSSIBLE CONCLUSIONS

12.1 The hypothesis of formative causation

The hypothesis of formative causation is a testable hypothesis about objectively observable regularities of nature. It cannot explain the origination of new forms and new patterns of behavior, nor can it explain subjective experience. Such explanations can be given only by theories of reality more far-reaching than those of natural science, in other words by metaphysical theories.

At present, scientific and metaphysical questions are frequently confused with each other because of the close connection between the mechanistic theory of life and the metaphysical theory of materialism. The latter would still be defensible if the mechanistic theory were to be superseded within biology by the hypothesis of formative causation, or indeed by any other hypothesis. But it would lose its privileged position; it would have to enter into free competition with other metaphysical theories.

In order to illustrate the important distinction between the realms of science and of metaphysics, in the following sections four different metaphysical theories are briefly outlined. All four are compatible with the hypothesis of formative causation. From the point of view of natural science, the choice among them is open.

12.2 Modified materialism

Materialism starts from the assumption that only matter is real; hence everything that exists is either matter or entirely dependent upon matter for its existence. However, the concept of matter has no fixed meaning: in the light of modern physics, it has already been extended to include physical fields, and material particles have come to be regarded as forms of energy. The philosophy of materialism has been modified accordingly, and is sometimes called physicalism to reflect this change.

Morphic fields are associated with material systems; they too can be regarded as aspects of matter (section 3.5). Thus materialism or physicalism could be further modified to incorporate the idea of formative causation.[1] In the following discussion, this new form of the materialist philosophy will be referred to as "modified materialism."

Materialism denies a priori the existence of any nonmaterial causal agency; the physical world is causally closed. Hence there can be no such thing as a nonmaterial self that acts upon the body, as there seems to be from a subjective point of view. Rather, conscious experience is either in some sense the same thing as material states of the brain or it simply runs parallel to these states without affecting them.[2] But whereas in conventional materialism brain states are determined by a combination of energetic causation and chance events, in modified materialism they are, in addition, determined by formative causation. Indeed, conscious experience would probably best be thought of as an aspect or epiphenomenon of the morphic fields acting on the brain.

The subjective experience of free will cannot, *ex hypothesi*, correspond to the causal influence of a nonmaterial self upon the body. However, it is conceivable that some of the random events within the brain might be subjectively experienced as free choices; but this apparent freedom is nothing but an aspect or epiphenomenon of the chance activation of one morphic field rather than another.

If all conscious experience is simply an accompaniment of, or runs parallel to, the morphic fields acting upon the brain, then conscious memory, like the memory of habits (section 10.1), must depend on morphic resonance from past states of the brain. Neither conscious nor unconscious memories would be stored within the brain.

In the context of conventional materialism, the evidence for parapsychological phenomena can only be denied, ignored, or explained away. But modified materialism permits a more positive attitude. In particular, it is possible to formulate an explanation of telepathy in terms of morphic fields,[3] and of psychokinesis in terms of the modification of probabilistic events within objects under the influence of behavioral fields.[4]

The origin of new forms, new patterns of behavior, and new ideas cannot be explained in terms of preexisting energetic and formative causes (sections 5.1, 8.7, 11.3, and 11.4). Moreover, materialism denies the existence of any nonmaterial creative agency that could have given rise to them. Hence they have no cause. Their origin must therefore be attributed to chance, and evolution can be seen only in terms of the interplay of chance and physical necessity.

In summary, according to modified materialism, conscious experience is either an aspect of or runs parallel to the morphic fields acting on the brain. All human creativity, like evolutionary creativity, must ultimately be ascribed to chance. Human beings adopt their beliefs (including the belief in materialism) and carry out their actions as a result of chance events and physical necessities within their brains. Human life has no purpose beyond the satisfaction of biological and social needs; nor has the evolution of life, nor the universe as a whole, any purpose or direction.

12.3 The conscious self

Contrary to the philosophy of materialism, the conscious self can be admitted to have a reality that is not merely derivative from matter. One can accept, rather than deny, that one's own conscious self has the capacity to make free choices. Then, by analogy, other people can also be assumed to be conscious beings with a similar capacity.

This "common sense" view leads to the conclusion that the conscious self and the body *interact*. But then how does this interaction take place?

In the context of the mechanistic theory of life, the conscious self can only be a kind of "ghost in the machine."[5] To materialists this notion seems inherently absurd. And even the defenders of the interactionist position have been unable to specify how the interaction takes place, beyond the

suggestion that it might somehow depend on a modification of quantum events within the brain.[6]

The hypothesis of formative causation enables this long-standing problem to be seen in a new light. The conscious self can be thought of as interacting not with a machine, but with morphic fields. These morphic fields are associated with the body and depend on its physical and chemical states. But the self is neither the same as the morphic fields nor does its experience simply parallel the changes brought about within the brain by energetic and formative causation. It “enters into” the morphic fields, but it remains over and above them.

Through these fields, the conscious self is closely connected with the external environment and with the states of the body in perception and in consciously controlled activity. Subjective experience that is not directly concerned with the present environment or with immediate action—for example, in dreams, reveries, and discursive thinking—need not necessarily bear any particularly close relationship to the energetic and formative causes acting on the brain.

At first sight, this conclusion might appear to contradict the evidence showing that states of consciousness are often associated with characteristic physiological activities. Dreams, for instance, tend to be accompanied by rapid eye movements and by electrical rhythms of particular frequencies within the brain.[7] But such evidence does not prove that the specific details of the dreams run parallel to these physiological changes: the latter could simply be a nonspecific consequence of the entry of consciousness into the dream state.

This point is easier to grasp with the help of an analogy. Consider the interaction between a car and its driver. Under certain conditions, when the car is actually being driven, its movements are closely connected with the actions of the driver, and depend on his perceptions of the road ahead, road signs, dials indicating the internal state of the car, and so on. But under other conditions, this connection is less close: for example, when the car is stationary with its engine turning over, the driver might be looking at a map. Although there would be a general relationship between the state of the car and what he was doing—he could not read when driving—there would be no specific connections between the vibrations of the engine and the features of the map he was studying. Likewise the rhythmical electrical

activity in the brain need bear no specific relationship to the images experienced in dreams.

Granted that the self has properties of its own, how does it act upon the body and the external world through morphic fields? There are two ways in which it could do so: first, by selecting between different possible morphic fields, causing one course of action to be adopted rather than another; and second, by serving as a creative agency through which new morphic fields come into being, for example in "insight" learning (section 10.4). In both cases it would act like a formative cause, but one that is, within limits, free and undetermined from the point of view of physical causation. It could indeed be thought of as a formative cause of formative causes.

On this interpretation, consciously controlled actions depend on three kinds of causation: conscious causation, formative causation, and energetic causation. By contrast, traditional interactionist theories, of the "ghost in the machine" type, admit only two, conscious and energetic causation, with no formative causation in between. Modified materialism admits a different two, formative and energetic, and denies the existence of conscious causation. And conventional materialism admits only one: energetic causation.[8]

In the lower animals, the strong canalization of instinctive patterns of behavior probably leaves little or no room for conscious causation; but among the higher animals, the relatively weak innate canalization of appetitive behavior may well provide a limited scope. In humans, the enormous range of possible actions gives rise to many ambiguous situations in which conscious choices can be made, both at lower levels, between possible methods of reaching goals already given by higher-level morphic fields, and at higher levels, between competing morphic fields.

On this view, consciousness is directed primarily toward the choice between possible actions, and its evolution has been intimately connected with the increasing scope of conscious causation.

At an early stage in human evolution, this scope must have increased enormously with the development of language, both directly through the capacity to produce an indefinite number of patterns of sounds in the speaking of phrases and sentences and indirectly through all those actions made possible by this detailed and flexible means of communication. In the

associated development of conceptual thought, the conscious self must at some stage, in a qualitative leap, have become aware of itself as the agent of conscious causation.

Although conscious creativity reaches its highest development in the human species, it probably also plays an important part in the development of new patterns of behavior in the higher animals, and may even be of some significance in the lower animals. But conscious causation takes place only within already established frameworks of formative causation given by morphic resonance from past animals; it cannot account for the behavioral fields in the context of which it is expressed, nor can it be regarded as a cause of the characteristic form of the species. Still less can it help to explain the origin of new forms in the plant kingdom. So the problem of evolutionary creativity remains unsolved.

The reality of the conscious self as a source of creativity can be admitted, but the existence of any creative agency transcending individual organisms can be denied. All other forms of creativity can be ascribed to chance. To go further involves admitting sources of creativity that transcend individual organisms, as discussed below.

12.4 The creative universe

Although a creative agency capable of giving rise to new forms and new patterns of behavior in the course of evolution would necessarily transcend individual organisms, it need not transcend all nature. It could, for instance, be immanent within life as a whole; in this case it would correspond to what Henri Bergson called the *élan vital*,[9] or vital impetus. Or it could be immanent within the planet as a whole, or the solar system, or the entire universe. There could indeed be a hierarchy of immanent creativities at all these levels.

Such creative agencies could give rise to new morphic fields by a kind of causation very similar to the conscious causation considered above. In fact, if such creative agencies are admitted at all, then it is difficult to avoid the conclusion that they must in some sense be conscious selves.

If such a hierarchy of conscious selves exists, then those at higher levels might well express their creativity through those at lower levels. And if such a higher-level creative agency acted through human consciousness, the

thoughts and actions to which it gave rise might actually be experienced as coming from an external source. This experience of *inspiration* is in fact well known.

Moreover, if such “higher selves” are immanent within nature, then it is conceivable that under certain conditions human beings might become directly aware that they were embraced or included within them. And in fact the experience of an inner unity with life, or the Earth, or the universe, has often been described, to the extent that it is expressible.

But although an immanent hierarchy of conscious selves might account for evolutionary creativity within the universe, it could not possibly have given rise to the universe in the first place. Nor could this immanent creativity have any goal if there were nothing beyond the universe toward which it could move. So the whole of nature would be evolving continuously, but blindly and without direction.

This metaphysical position admits the causal efficacy of the conscious self, *and* the existence of creative agencies transcending individual organisms, but immanent within nature. However, it denies the existence of any ultimate creative agency transcending the universe as a whole.

12.5 Transcendent reality

The universe as a whole could have a cause and a purpose only if it were itself created by a conscious agent that transcended it.

If this transcendent conscious being were the source of the universe and of everything within it, all created things would in some sense participate in its nature. The more or less limited “wholeness” of organisms at all levels of complexity could then be seen as a reflection of the transcendent unity on which they depended, and from which they were ultimately derived.

Thus this fourth metaphysical position affirms the causal efficacy of the conscious self, *and* the existence of a hierarchy of creative agencies immanent within nature, *and* the reality of a transcendent source of the universe.

APPENDIX A

NEW TESTS FOR MORPHIC RESONANCE

There are two approaches to testing the hypothesis of formative causation: first, through morphic fields, which connect together parts of a morphic unit in *space;* second, through morphic resonance and its cumulative influence in *time*.

Research on the spatial aspect of morphic fields has been concerned mainly with social and perceptual fields. I have summarized the findings in my books *Seven Experiments That Could Change the World*, *Dogs That Know When Their Owners Are Coming Home*, and *The Sense of Being Stared At*.[1] The full texts of my scientific papers on these subjects, published in peer-reviewed journals, are available on my website, www.sheldrake.org

In this appendix, I suggest a range of new tests for morphic resonance itself. When new morphic fields first come into being, they are weak. They are not stabilized by morphic resonance from similar past systems. The more frequently a morphic process occurs, the greater the morphic resonance, the stronger the morphic field, and the more compelling the force of habit. As morphic resonance increases, morphic processes become faster, and morphic fields more stable. These predictions of the hypothesis of formative causation are testable in a wide range of systems, ranging from low-temperature physics to human learning.

A.1 Bose-Einstein condensates

When morphic units have occurred for billions of years and been repeated innumerable times, no changes in the rate of their formation will be

detectable. Nor will their stability change. Their habits are fixed. For example, the formation of hydrogen atoms, methane molecules, and sodium chloride crystals will not show any measurable changes. In order to detect morphic resonance, it is necessary to study *new* self-organizing systems.

In the realm of physics, what processes observable here on Earth are unlikely ever to have occurred anywhere else in the universe? Phenomena at very low temperatures.

The background temperature of the universe, as revealed by the cosmic background microwave radiation, is 2.7°K, or in other words 2.7 degrees C above absolute zero. But in laboratories, it is now possible to cool systems to less than 1°C above absolute zero, far colder than the rest of the universe, as far as we know. At these ultra-low temperatures, physical systems behave very strangely.

The best-known low temperature phenomenon is the formation of Bose-Einstein condensates, a new state of matter, over and above the familiar solid, liquid, gas, and plasma states. Satyendranath Bose and Albert Einstein first predicted the existence of these condensates in 1927. The first to be investigated was helium-4 in 1938. When cooled to 2.17°K, it became a superfluid, flowing without friction. The first “pure” Bose-Einstein condensate was made in 1995, with rubidium-87. Such condensates have many strange properties and are effectively superatoms, groups of atoms that behave as one.

Presumably Bose-Einstein condensates made in modern physics laboratories are entirely new to nature, and have never occurred before in the history of the universe (unless they have been made in physics laboratories by aliens on other planets). Since they behave as unified wholes, they may be a point at which quantum fields and morphic fields converge.

If Bose-Einstein condensates are indeed organized by morphic fields, then the more often a given kind of condensate is made in a laboratory, the easier it should be to make it under similar conditions all over the world, and the more stable it should be.

To test for morphic resonance, a new kind of condensate is prepared, and then made again repeatedly under standard conditions. The rate at which it forms is monitored. If morphic resonance is at work, the condensate will

form more readily the more often this process is repeated, and the stability of the condensate will increase.

A.2 Melting points

As discussed in chapter 5, morphic resonance should lead to an increased rate of crystallization the more often a compound is crystallized. Through resonance from previous similar crystals, the field of any particular type of crystal should be strengthened.

An increase in morphic field strength should also cause crystals to be more stable; it should be harder to destroy them. Crystals break up when they are heated to their melting point. Morphic resonance should cause the melting points of new kinds of crystals to increase.

This is a shocking prediction. Melting points are called “physical constants” because they are supposed not to change. Although they are affected by a number of variables, such as atmospheric pressure and the presence of impurities, it is generally taken for granted that pure samples of a given substance at standard atmospheric pressure have the same melting point at all times and in all places. Everyone knows that the melting point of ice is, always has been, and always will be 0°C. Weighty handbooks of physical constants list the melting points of many thousands of substances. Few aspects of science seem more certain. Having studied chemistry, I too used to take the constancy of melting points for granted.

After the first edition of this book was published, I gave a seminar on morphic resonance in the Chemistry Department of the University of Vermont in which I discussed the increasing rates of crystallization of new compounds. A chemist pointed out to me that if morphic fields of crystals grew stronger by morphic resonance, then melting points should also rise. He was right. I began to investigate whether this really happened.

I started by asking several synthetic chemists if they had ever noticed a tendency for the melting points of new substances to increase. Yes, they had; this seemed to be a common observation. But they had a ready explanation: as time goes on, chemists’ skills improve. Impurities reduce melting points, and therefore melting points rise as chemists make purer samples. I asked, “How we can be sure that the later samples were in fact

purer?" The usual answer was: "They must be purer because they have higher melting points." The argument was reasonable, but circular.

I then looked up the melting points of a wide range of organic chemicals in early-, mid-and late-twentieth-century handbooks and chemical journals. My aim was to compare the melting points of compounds that have crystallized in nature for millions of years with those of compounds that first crystallized in laboratories. If there is a general tendency for chemists' samples to be purer, then both kinds of crystals should show similar increases in their melting points. But if melting points are influenced by morphic resonance, only the melting points of recently crystallized substances would be expected to rise. Compounds that crystallize under natural conditions should not show this tendency, for two reasons.

First, there are likely to be limits beyond which melting points can increase no more. Other factors become limiting. This is true of all processes. For example, after Roger Bannister first ran a four-minute mile in 1954, speeds have continued to increase; the current record is 3 minutes 43 seconds. But it is very unlikely that records would keep being broken until the mile is run in 3 minutes, or 1 minute, or 1 second. Other factors become limiting—the muscular system, the ability of the heart to pump enough blood, and even friction—a point would come at which the athletes' jockstraps burst into flames. In general, morphic resonance would be expected to lead to changes that reach limits. And this would be as true of melting points as of everything else.

Second, there will be so much morphic resonance from past crystals that no further change will be observable. Against a background of resonance from quadrillions of past crystals, the resonance from a few thousand more makes no detectable difference.

Obviously the melting points in handbooks are based on reports in the chemical literature that predate the handbooks themselves, and melting points in one edition of a handbook are often copied into the next edition, or copied from other handbooks. Hence the dating of changes in melting points is not precise, and the value quoted in a given handbook could refer to a determination carried out years or even decades earlier.[2] Nevertheless, the handbooks are updated from time to time, and new melting points substituted for old ones.

The most up-to-date melting points are to be found in chemical catalogs. I concentrated on the Aldrich Chemical Company's *Catalogue Handbook of Fine Chemicals*. In many cases, the Aldrich melting points were higher than in the standard reference books in libraries. But how reliable were the Aldrich values? In 1991, I purchased samples of forty different chemicals from Aldrich and arranged for their melting points to be measured in the Materials Department of Imperial College of Science and Technology, University of London.[3] The values were in close agreement with the company's claims, usually with differences of less than one or two degrees C. Thus the melting points in the Aldrich catalog seemed to be a reliable guide to contemporary values.

Many increases in melting points over the course of the twentieth century were greater than five degrees. For example, saccharin, the oldest artificial sweetener, was first synthesized in 1878. In 1902, its melting point was 220°C. By 1996 it was 229°C—a nine-degree increase. Phenolphthalein, used in chemistry laboratories as an indicator of acidity, was first made in 1880. In 1907 its melting point was 252°C; in 1989 it was 262°C—a ten-degree increase. The crown ethers are a family of crown-shaped molecules used as chelating agents, first synthesized in 1976. The most widely used member of the family, 18-crown-6, started with a melting point of 39°C. By 1989, it was 45°C—a six-degree increase.

Other compounds with rising melting points were chemicals naturally occurring in living organisms, but too dilute to crystallize in nature. Although the chemicals themselves have existed for many millions, even billions, of years, they probably crystallized for the first time when they were isolated and concentrated in laboratories from the nineteenth century onward. For example adrenaline, first isolated in 1895, had a melting point of 201°C in 1901. By 1989 it was 215°C—a fourteen-degree increase. Cortisone, isolated in the 1930s from the adrenal cortex, had a melting point of 205°C in 1936; in 1989 it was 225°C—a twenty-degree rise.

In the course of this research I found one glaring anomaly: vitamin B_2, also known as riboflavin, crystallizes in nature, for example, in the eyes of lemurs, and also in some fungal cells.[4] Riboflavin crystals should therefore show little or no change in melting point. Yet there was an increase from 275°C in 1940 to 290°C in contemporary samples. However, the 1940 figure, taken from the fifth edition of the *Merck Index*, was only one of a

range of melting points, from 271°C to 293°C, reported between the 1930s and 1950s.[5] This confusing variability may have a simple explanation: riboflavin is now known to have three different crystal forms with different melting points.[6]

In my survey of a wide range of chemicals, I found some with constant melting points and others whose melting points increased. Very few went down. In the early 1990s, I corresponded with the editor of one of the leading handbooks to ask whether he had ever studied the pattern of change from edition to edition. He had not. He was surprised by the widespread tendency for melting points to go up, having assumed that any changes would be a result of random errors, equally likely to go up or down. But this was not the case.

Figure A.1 compares the historical melting points of compounds that have been crystallizing in nature for millions of years with chemical derivatives of these compounds that did not exist until the nineteenth or twentieth century. Salicin is found in the bark of willows, poplars, and in other plants and has been used medicinally since the time of the ancient Greeks. It was first isolated in 1827. Its chemical derivative acetylsalicylic acid, also known as aspirin, was first synthesized in 1853. Aspirin was introduced into medical practice in 1899 and subsequently became one of the world's most popular drugs, with an annual consumption of around 50,000 tons.[7] The melting point of salicin was constant throughout the twentieth century, while the melting point of aspirin increased by eight degrees C.

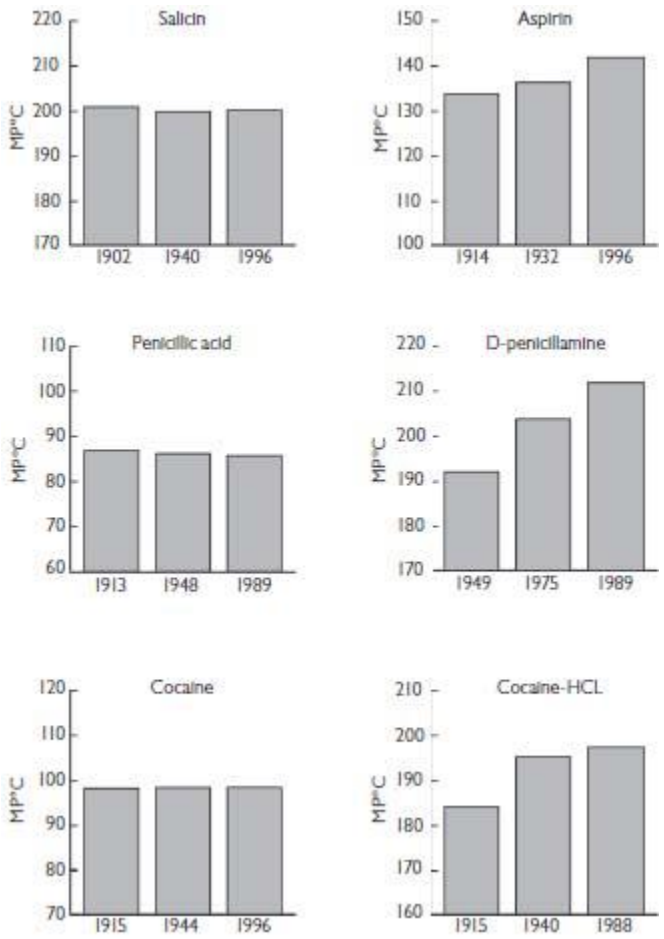

Figure A.1. Changes in melting points over time in natural compounds (left) and related synthetic compounds (right).

Penicillic acid is excreted naturally by several species of fungi in the genus *Penicillium*, and was first isolated and identified in 1913, years before the discovery of the antibiotic properties of penicillin in 1929. A range of related compounds were isolated and synthesized in the 1940s.[8] One of them was D-penicillamine, a breakdown product of penicillin antibiotics, which is used therapeutically as an antirheumatic drug. The melting point of penicillic acid has remained more or less constant, whereas that of D-penicillamine has increased by twenty degrees C.

Cocaine occurs in the leaves of coca plants at a concentration of up to 1 percent.[9] Presumably, over millions of years it has often crystallized as leaves dried up. By contrast, cocaine hydrochloride, the cocaine of commerce, is new; it is produced by treating coca-leaf extracts with hydrochloric acid. The melting point of cocaine has remained constant, whereas that of cocaine hydrochloride has increased by thirteen degrees C.

In 1997, a Dutch Skeptical organization, Stichting Skepsis, wrote to me challenging my observations about changes in melting points. I sent them my data. They checked the literature and came up with very similar values. They conceded that some melting points had indeed increased, but then fell back on the argument that these increases must have been due to improvements in purity rather than to morphic resonance. They had no evidence to support their assumption. In an article they wrote in the *Skeptical Inquirer*, they simply asserted, "There is no other explanation."[10]

Much more research could be done on the history of melting points. I have surveyed only a small part of the huge chemical literature. But unfortunately these records do not usually include any information on purity, and therefore this historical evidence can never be conclusive. The only way forward is to do special tests.

Here is an example. Take six new chemicals recently made in a university or chemical company. Crystallize all six and measure their melting points. Store the samples in a refrigerator. Now, in another laboratory, make one of these chemicals, selected at random, in large quantities, and crystallize it repeatedly. This should lead to an increase in the melting point of this particular compound, but not of the other five. In the first laboratory, now measure the melting points of all six samples again. Does the melting point of the test sample increase? Do the melting points of the other five samples stay the same?

A.3 Crystal transformations

Many chemical substances take more than one crystalline form. The best-known examples are the alternative forms of chemical elements, called allotropes. Graphite and diamond are both crystalline forms of carbon, with the atoms bonded together as a hexagonal lattice in graphite and as a tetrahedral lattice in diamond. Graphite can be transformed into diamond at high temperature and pressure, which is how artificial diamonds are made. Tin has a gray allotrope with a cubic crystal structure and no metallic properties. When heated above 13.2°C, it changes into white tin, which is metallic and has a tetragonal lattice structure. Other elements with allotropic crystal forms are sulfur, phosphorus, and plutonium.

The crystals of many salts and molecules also exist in alternative forms, which are called polymorphs rather than allotropes. For example, calcium carbonate occurs in rocks as calcite or aragonite. Aragonite is more soluble, and occurs as small crystals within basalts, and also in the shells of mollusks. Calcite is found in sedimentary rocks, such as limestone, in Iceland spar crystals, and in the shells of bivalves such as oysters. Aragonite changes to calcite when heated to 470°C.

Potassium nitrate also exists in two alternative forms similar to calcite and aragonite. The aragonite type changes to the calcite type at 127.5°C. The transition has been studied in detail in single crystals slowly heated up and then cooled down while being observed continuously by means of light reflected by the crystals: the polymorphs have different reflection patterns. The aragonite-type crystals took several minutes just above the transition temperature to transform into calcite. When the calcite crystals were cooled down again, the original aragonite structure was restored within a few minutes in surprising detail, with the atoms lined up in the same way they had been in the original crystal, leading the investigators to conclude there was a "memory effect."[11]

Transformations between polymorphs also occur in many crystals of organic chemicals. For example, a sulfur-containing compound called N-methyl-1-thia-5-azoniacyclooctane-1-oxide perchlorate (NMTAOP) has two polymorphs, alpha and beta, with a transition temperature of 17°C. In studies with single crystals, when the alpha form was warmed to a few degrees above 17°C, it changed to the beta form in a few minutes, as measured by the optical properties of the crystals. The reverse transformation occurred when the beta form was cooled to 14°C, but took several days to go to completion. This transformation cycle could be repeated over and over again.[12]

Just as the crystallization of a compound from solution should occur more readily the more often this process is repeated (as discussed in section 5.6), so should the transformation of one polymorph to another occur more readily the more often this polymorph has formed. Hence, crystal transformations could provide a way of testing for morphic resonance.

The transformations need to be monitored continuously, either through optical properties, as in the examples of potassium nitrate and NMTAOP, or by other means: some crystals change color as they transform, while in

other cases their electrical or magnetic properties change.[13] The transformations can be brought about by heating or cooling, or by applying high pressure, or by both combined. Do the transformations occur more rapidly under standard conditions the more often a new polymorph is made?

As in the case of melting points, it is important to choose synthetic compounds for this study. Changes would not be expected in the rate of transformation of naturally occurring polymorphs like calcite and aragonite, because they have existed naturally for millions of years; transformations under high pressure and temperature have often occurred within the Earth's crust through geological processes. Fortunately, there are plenty of synthetic organic compounds that have never existed in nature, as far as we know, and whose polymorphs are of recent origin.

A.4 Adaptations in cell cultures

Plant and animal cells can be grown outside the organisms they come from, and some can be propagated in cell cultures within laboratory glassware for years. Through morphic resonance, if some cells from the culture adapt to a new challenge, similar cells should be able to adapt to the same challenge more rapidly even when they are separated.

There is already evidence that such an effect takes place. Miroslav Hill, a cell biologist, made a very surprising discovery in the 1980s when he was director of research at the Centre National de la Recherche Scientifique in Villejuif, France. Cells seemed to be influencing other similar cells at a distance.

Hill and his colleagues were working with cell cultures derived from hamsters. They were trying to find mutant cells resistant to thioguanine, a toxin. The standard procedure was to expose cells to the poison and see if any survived as a result of rare random mutations enabling them to resist it. None did.

At this stage the normal procedure would have been to expose the cells to mutation-causing chemicals in order to increase the number of random mutations and then try again. The conventional assumption is that mutations take place at random; they have nothing to do with adaptation to the environment. Instead, Hill's group decided to follow a trick of the trade of laboratory technicians, not mentioned in official laboratory manuals.

Instead of testing large numbers of cells at a single time to find rare mutants resistant to attack, the technicians tested successive generations of cells. At regular intervals they routinely subcultured the cells, taking rapidly growing cells and putting some of them in a fresh culture medium. This process is called a passage. At the time of each passage, they also put some of the cells on top of dying cells in flasks containing the toxin. Sooner or later, resistant cells began to appear.

Hill and his colleagues decided to look for thioguanine resistance using a "serial assay" method, which differed from the technicians' procedure in that fresh flasks of toxic medium were used at each passage. The hamster cells were grown in a normal culture medium, and while still growing were divided into two samples. One was put into a fresh culture medium so that it could go on growing; the other was put into a new flask of the toxic medium. Thus at each passage, some cells were assayed for thioguanine resistance, while the others went on growing normally (figure A.2).

To start with, all the cells they placed in the thioguanine assay medium were killed. But after several passages, some cells were able to survive in the toxic medium. They had mutated. At the next passage, even more of the cells survived the toxin. The mutation rate was increasing. The descendants of these cells were also able to grow in the toxic assay medium; they inherited this resistance.[14]

Hill and his colleagues did another experiment to see if the same process could be repeated with a different poison, ethionine, not previously used in toxicity studies with hamster cells. For the first thirty passages, over a fifteen-week period, all the cells exposed to ethionine died.

> The subsequent passages were characterized by a sudden appearance of mutants. These were more frequent from passage to passage . . . Thus ethionine-resistant mutants occurred in cultures growing without selection, and arose, in those growing cultures, in response to an ethionine attack on cells in parallel, physically separated cultures.[15]

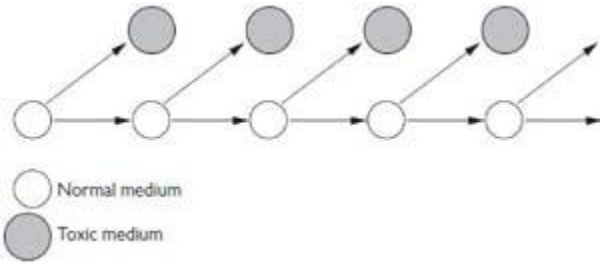

Figure A.2. Miroslav Hill's serial assay technique. At each passage, some cells were transferred to the toxic medium and some to the normal medium.

The ethionine-resistant cells gave rise to descendants that inherited their resistance.

Hill's team then investigated whether the same techniques would enable hamster cells to adapt to high temperatures. The cells were grown, as usual, at 37°C, and at each passage, a sample was withdrawn and assayed for growth at 40.6°C.

> Cells in the first sample died within three days, in the second they survived a profound crisis and gave rise to eleven colonies, and in the third they became established after a barely noticeable crisis. These cells then grew continuously as a cell line at 40.6°C.

In a second phase of the experiment, this cell line was kept growing at 40.6°C and samples were withdrawn at each passage and assayed at 41.3°C. No cells survived at this elevated temperature for thirty-one passages. Then tolerant cells began to occur in small numbers, then more frequently, and finally in large numbers. This new strain could thereafter be grown indefinitely at 41.3°C. In further experiments, Hill's team succeeded in establishing a strain that could grow at an even higher temperature, 42.0°C, but were not able to go higher.

Hill's conclusion was that "cells are more likely to survive an attack if their close relatives have already experienced such an attack." He argued that this showed that "there is an additional flow of information, not mediated by DNA, which may be referred to as adaptive information."

How was this adaptive information transmitted to close relatives? Hill suggested it happened because some of the cells under attack and some of the cells in the normal culture were sisters, separated at the most recent

passage. Because they were descendants of the same mother cell, they were “entangled” in the sense of quantum physics.

According to quantum theory, systems that were part of the same system in the past remain linked, even when miles apart, such that a change in one is immediately accompanied by a change in the other, a phenomenon that Albert Einstein described as “spooky action at a distance.” There is good experimental evidence that entanglement (also known as quantum non-locality, or quantum non-separability) really happens. Hill suggested that sister cells are not just analogous to but actually are entangled quantum systems.

Hill proposed that some of the cells struggling for survival adapted in such a way that they could resist the toxin, and their entangled sister cells underwent a similar adaptation even though they were not exposed to the toxin. Some of the descendants of these unexposed sister cells were carried over at the next passage to the assay conditions, and when they came under attack they were already resistant. Thus, passage by passage, the proportion of resistant cells increased in cells growing under normal conditions (figure A.3a).

The hypothesis of morphic resonance provides an alternative interpretation. Some cells under attack may undergo adaptive changes, as Hill suggests. Then cells currently under attack tune in to the adaptation via morphic resonance from past cells under attack. Hill’s proposal involves a transmission of adaptive information across space, from sister cells under attack to sister cells in the normal culture. Morphic resonance involves a transmission of adaptive information across time, from past cells under attack to present cells under attack (figure A.3b).

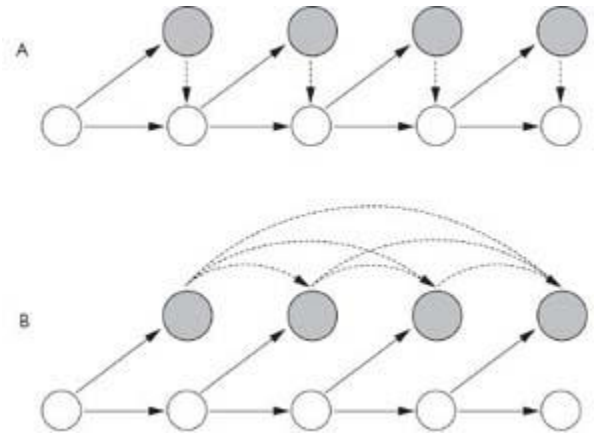

Figure A.3. Above: The "entanglement" interpretation of the Hill effect. Adapted cells in the toxic medium affect their sisters in the normal medium through entanglement (dotted lines). Below: The morphic resonance interpretation of the Hill effect. Adapted cells in the toxic medium influence subsequent cells in the toxic medium by morphic resonance (curved dotted lines).

These interpretations make different predictions that can be tested by experiment. Mouse cells could be used instead of hamster cells to avoid any morphic resonance from Hill's previous experiments.

Two cell lines, A and B, are derived from a common ancestral culture. Line A is simply transferred to a new normal medium in passage after passage, with no samples subjected to attack. B is subcultured following the Hill serial assay procedure, with some of the cells put under attack at each passage (figure A.4). Say that resistant cells in line B arise at passage five. The entanglement hypothesis predicts that adaptation should increase in the normal cells in line B but not in line A. Starting at passage five, line A is now subcultured at each passage following the Hill procedure, and subcultures are subject to the same attack as those in line B (figure A.4). The entanglement hypothesis suggests that there will be about five passages before the cells under attack begin to develop resistance, as before. But the morphic resonance hypothesis suggests that resistance should begin to appear within one or two passages, because of morphic resonance from cells in line B.

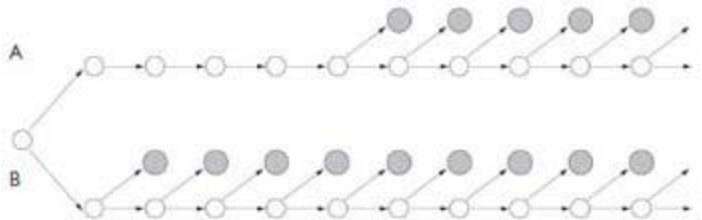

Figure A.4. An experiment to distinguish between entanglement and morphic resonance effects in the adaptation of cells to a toxic medium. Below: At successive passages, cells in line B are placed in a toxic medium. After, say, five passages, adapted cells begin to appear and the proportion of adapted cells increases in subsequent passages. Above: The serial assay procedure begins after five passages in line A. If only entanglement was at work, adapted cells would not appear in the toxic medium for about five passages; if morphic resonance was at work, they would appear almost immediately.

A.5 Heat tolerance in plants

Animals and plants often adapt to changes in their environment. For example, humans who move to high altitudes acclimatize through a variety of physiological responses, including making more red blood cells. Sheep moved to cold, damp climates grow thicker wool. Plants moved to new climates adjust their physiology and growth habits.

Gardeners are familiar with these changes, and know that plants grown in greenhouses may need “hardening off ” if they are to survive outdoors. The plants are moved to a cold frame and gradually exposed to outdoor conditions during the daytime, then at night, before they are planted out in the open air. Hardening off may take two or three weeks. A range of biochemical changes occurs within the plants and they often grow thicker coatings of wax on their leaves. Under natural conditions, plants undergo cold hardening at the beginning of winter as temperatures drop, helping them to resist damage by frost that kills unhardened plants.

When plants are introduced into new environments by gardeners or farmers, the plants may continue to adapt over several generations. Charles Darwin was convinced that the new habits that plants acquired as they acclimatized were inherited, for example when spring-sown varieties of cereals were planted in the autumn and changed into winter varieties.

> In the reciprocal conversion of summer and winter wheat, barley, and vetches into each other, habit produces a marked effect in the course of a very few generations. The same thing apparently occurs with the varieties of maize, which, when carried from the Southern States of America into Germany, soon become accustomed to their new homes.[16]

Trofim Lysenko and his colleagues in the Soviet Union continued to study the interconversion of winter and spring wheat varieties, and they applied these principles to Soviet agriculture on a large scale, with some success. But the subject became intensely politicized, and neo-Darwinians in the West denounced the findings of the Soviet researchers as bogus.[17] The inheritance of adaptive habits is prohibited by neo-Darwinism; only genes can be inherited.

Darwin was not a neo-Darwinian. In his book *The Variation of Animals and Plants Under Domestication,* he brought together impressive evidence

for the inheritance of acquired characteristics. He thought heritable habits played an important part in evolution, along with spontaneous variation and natural selection: “We need not . . . doubt that under nature new races and new species would become adapted to widely different climates, by variation, aided by habit, and regulated by natural selection.”[18]

Morphic resonance provides a means whereby habits can be inherited and is in accord with Darwin’s own ideas. But however much it agrees with Darwin, it is still only a hypothesis. Does it really play a part in the adaptation of plants to new conditions?

I propose a simple test in which plants of an inbred strain, say a standard variety of pea, are grown from seed in a controlled environment under near lethal high temperatures. The proportion that survives is recorded. The same procedure is repeated again and again. An increasing proportion of plants should survive because of morphic resonance from those that adapted successfully in previous trials.

This experiment could be done with two parallel lines. In C, plants are grown from the original stock of seeds, so there is no possibility that any adaptive changes are passed on through the genes (figure A.5 above). Any increase in adaptation over time would be a result of morphic resonance.

In line D, seeds are taken from plants that have survived the high temperature and are used for growing the next generation (figure A.5 below). In this line, any increase in adaptation from generation to generation would be due to a combination of morphic resonance and epigenetic inheritance.

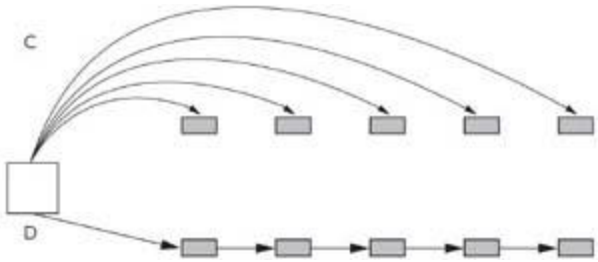

Figure A.5. An experiment on heat tolerance in plants. Above: Seeds of an inbred variety are grown under high temperature conditions in successive trials. If morphic resonance is at work, they should show greater adaptation in successive trials. Below: Seeds taken from heat-adapted plants are used in the next trial. Increased heat adaptation in successive generations could be due to a combination of epigenetic inheritance and morphic resonance.

The recognition of epigenetic inheritance took place only after the turn of the millennium, and it provides a legitimately mechanistic basis for the inheritance of acquired characteristics. Now that a mechanistic explanation is available, the taboo against the inheritance of acquired characteristics has been lifted (section 7.7). Evidence for the inheritance of acquired characteristics that was previously anomalous and rejected or ignored has been rehabilitated.[19]

If heat adaptation has heritable epigenetic effects, the progeny of adapted plants will tolerate high temperatures better than plants grown from the original batch of seeds (figure A.5). Epigenetic inheritance will not only pass on patterns of gene activation and inactivation but also, at the same time, make the progeny of adapted plants more similar to previous adapted plants, and hence more strongly affected by morphic resonance from them. Any improvements in the adaptation of plants in line D would be a result of both direct epigenetic inheritance and increased morphic resonance.

The conventional expectation would be that line C would show no change. The hypothesis of formative causation predicts that both lines C and D will show a progressive heat tolerance in successive trials, but line D will show this effect more strongly.

A.6 The transmission of aversion

Conditioned aversion is a rapid and long-lasting form of learning. Animals avoid eating something that has made them ill. If you eat a new kind of food and are sick soon afterward, you will probably avoid that food thereafter. Conditioned aversion occurs in invertebrates, too. Its evolutionary advantages are obvious—it helps animals avoid harmful foods, and hence survive better.

Conditioned aversion is associated with the brain stem, the part of the brain that helps control the gut, the secretion of gastric juices, and vomiting. Learning at this level operates unconsciously. If a cancer patient receives chemotherapy that makes her feel sick, and eats something just before the sickness starts, she will probably find its smell nauseating for the rest of her life, even though she knows that the cancer treatment and not the food was the cause of her illness. Conditioned aversion overrides conscious understanding.

Could conditioned aversion be transmitted by morphic resonance? If animals of a particular species have become averse to eating a harmful kind of food, will animals of the same species tend to avoid that food as a result of morphic resonance from similar animals that have already become averse to it? Some preliminary experiments suggest this might happen.

In 1988, I wrote an article about morphic resonance in the *Guardian*, a British newspaper. Soon afterward the same newspaper published a response by Steven Rose, a neuroscientist, who challenged me to test "this seemingly absurd hypothesis" in his laboratory at the Open University. Rose was well known in Britain for his strong political views—he was a Marxist—and his robust polemical style.

I accepted his challenge, and raised the funding for the tests to be carried out by a student, Amanda Harrison, in Rose's laboratory. She worked under Rose's supervision and was not informed of the hypothesis being tested.

At that time, Rose was studying changes in the brains of day-old chicks as a result of conditioned aversion. Chicks instinctively peck at small bright objects in their environment, and Rose's standard procedure was to expose chicks to a test stimulus, for example a small yellow light–emitting diode (LED). Soon after the chicks pecked it, they were made mildly sick with an injection of lithium chloride. As a result, they developed an aversion to pecking the same kind of bead again. Control chicks were exposed to a control stimulus, say a chrome bead. After pecking at the chrome bead, the control chicks were injected with a harmless saline solution, and developed no aversion to it. This form of learning was different from conditioned taste aversion in that it involved a visual stimulus, but like taste aversion it provided a rapid form of learning that needed only one trial.

Rose and I designed an experiment with a new stimulus, a yellow LED not previously used in experiments of this kind, to avoid any carryover of morphic resonance from previous aversion experiments with green LEDs. Indeed, we found that the chicks pecked a yellow LED much more readily than a green LED: there was an average delay of 4.1 seconds before they pecked the yellow LED and 19.0 seconds with green.[20] For the control stimulus, we used a chrome bead.

Every day for thirty-seven days the same tests were performed with fresh batches of day-old chicks. Half the batch of chicks, selected at random, was

tested with the yellow LED, the other half with the chrome bead. Then the chicks exposed to the yellow LED were made mildly sick. Three hours later they were tested again, and exposed to both the yellow LED and the chrome bead. Most avoided pecking the yellow LED, but had no aversion to the chrome bead. The control chicks that had pecked at the chrome bead were injected with saline solution, and they too were tested three hours later with both the chrome bead and the yellow LED.

I predicted that if morphic resonance was taking place, successive batches of day-old chicks should show an increasing aversion to the yellow LED when first exposed to it. No such aversion to the chrome bead would be expected with the control chicks. Rose predicted that there would be no increase in aversion with the control *or* the test chicks.

What did the data show? First of all, there was an effect that neither of us had predicted, though in retrospect we should have done so. The student carrying out the tests had never worked with chicks before, and took about a week before she learned how to handle the chicks and carry out the tests properly. The data from the first few days showed a big learning effect—not by the chicks but by the student. From then on, after she had learned the techniques, there was a consistent pattern. Relative to the controls, the test chicks exposed for the first time to the yellow LED became progressively averse to pecking it (figure A.6). This effect was statistically significant.

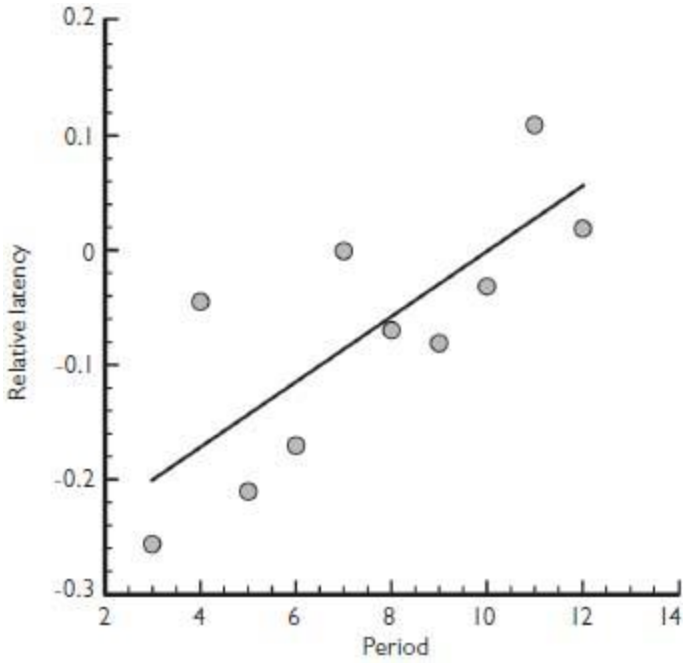

Figure A.6. An experiment on conditioned aversion with day-old chicks. "Test" chicks were exposed to a yellow light–emitting diode (LED) and control chicks to a chrome bead. There was an increased delay in pecking at the LED relative to the control stimulus in successive three-day periods. The measure of delay, or " latency," was the proportion of chicks that did not peck at the stimulus within ten seconds. (Data from Sheldrake, 1992a)

In my view, the data were consistent with the operation of morphic resonance. In Rose's view, they were not.[21]

Perhaps the best opportunity for further research on the transmission of aversion is with rats. Conditioned aversion is an important practical problem for the rat-control industry. If rats are fed bait laced with a quick-acting toxin, the poison kills a few rats to start with, but the other rats soon avoid it. They rapidly become "bait shy." For this reason, the most effective rat poisons are slow-acting, like warfarin, which does not cause illness soon after being eaten. Warfarin, first licensed for use as a rodenticide in 1952, is an anticoagulant and works slowly because it kills rats through internal bleeding. Some bleed to death after being bitten by other rats.

After warfarin had been in use for about ten years, resistant strains of rats began to appear in Britain, then in other parts of Europe, the United States, and Asia. In the 1970s, poison manufacturers rose to the challenge by producing a second generation of rodenticides, "superwarfarins" such as brodifacoum. Resistance to these new toxic agents is now increasing all over the world.[22]

When anticoagulant poisons fail to eradicate all the rats in an infestation, pest-control operatives usually revert to using a fast-acting, old-style rat poison like zinc phosphide. Because rats so rapidly become bait shy, a technique called prebaiting is used. The rats are fed on an attractive food that does not contain poison and when they are used to it, zinc phosphide is added. The rats are no longer cautious and eat enough to kill them.

Without prebaiting, individual rats may eat only a small amount of poisoned bait. They fall ill but then recover and are bait shy because of conditioned taste aversion. Rats are social animals, and bait-shy individuals communicate their aversion to other members of the group by "social learning." One component of social learning is imitation, especially the imitation of parents by their young. Another component is the smelling of

the breath of other members of the colony, enabling other rats to know what they have been eating. Morphic resonance may also play an important role in social learning. But as bait shyness spread through a colony, it would be impossible to tease apart the relative contributions of morphic resonance and other kinds of information transmission. To test for morphic resonance, it would be necessary to compare the behavior of separate colonies miles apart.

Here is a simple experimental design. Two new kinds of food, designated G and H, are given unusual flavors that rats are unlikely to have encountered before. Ten colonies are selected for this experiment, located miles away from each other. Five of these colonies are selected at random and both G and H are made available to them. The rates at which the rats eat them are recorded. Now one of the foods is selected at random—say G—and poisoned with low doses of zinc phosphide. The rats become bait shy and avoid G.

Now rats in the other five colonies are given unpoisoned G and H to eat. If morphic resonance is at work, the rats should show a tendency to avoid G but not H.

Similar experiments could be done under more-controlled conditions with captive colonies of rats or mice, but to minimize unnecessary suffering, it would be better to do these experiments in situations where the animals are going to be poisoned anyway.

A.7 The evolution of animal behavior

In *The Presence of the Past,* I described the spread of a new pattern of behavior suggestive of morphic resonance: the stealing of cream by blue tits. In Britain, fresh supplies of milk were (and still are) delivered to the doorsteps of houses every morning except Sunday. In the 1920s, blue tits and several related species of birds began to steal cream by removing the caps and drinking from the tops of the bottles.

The first record of this habit was in 1921 from Southampton, and its spread throughout Britain was monitored by amateur bird-watchers between 1930 and 1947. The main cream-stealing species were blue tits, great tits, and coal tits. Once cream-stealing had been discovered in a particular place, it spread locally by imitation.

Tits do not usually move more than a few kilometers from their nesting place, and appearances of this habit over gaps of more than twenty-five kilometers probably represented new discoveries by individual birds. A detailed analysis of the records by scientists at Cambridge University showed that cream-stealing was probably discovered independently at least eighty-nine times in the British Isles. The spread of the habit accelerated as time went on.[23]

This habit also appeared in continental Europe, particularly Sweden, Denmark, and Holland. The Dutch records are particularly interesting. Milk deliveries stopped during the Second World War and began again in 1947. Tits live only a few years, and probably none that had learned this habit before the war would have survived until this date. Nevertheless, attacks on milk bottles began again rapidly. “It seemed certain that the habit was started in many different places by many individuals.”[24]

Incidentally, cream-stealing now seems to be dying out. In the late 1980s, tits regularly attacked milk bottles delivered to our family house in London. In the early 1990s, we switched from full-cream to semi-skimmed milk, like many other people, and the attacks soon stopped. I have not seen a milk bottle attacked by tits for more than ten years, although there are still plenty of tits in the neighborhood. The birds seem to have given up now that there is so little cream to steal.

Several other examples of the rapid evolution of new patterns of behavior suggest morphic resonance could have played a part.

According to an eminent Texas naturalist, Roy Bedichek, when barbed wire was first introduced, in the late nineteenth century, skeptics predicted that it would never be suitable for horse pastures. Horses dashed right into it and “cut their own throats, tore great slugs of flesh from their breasts, while wounds not fatal or mere scratches became infested with screw worms.” In 1947 he wrote, “I can remember the time when there was hardly a horse to be found in Texas farming or ranching sections that was not scarred from encounters with barbed wire.”[25] Yet by the middle of the twentieth century, this was now no longer a serious problem: “In half a century the horse has learned to avoid barbed wire. Colts rarely dash into it. The whole species has been taught a new fear.”

Bedichek also commented on the changed reactions of horses to cars.

> When automobiles first appeared, horse-drawn traffic was disorganized. The more considerate autoist would drive out of the road and cut off the motor immediately a team of horses hove in sight. Not only that, the motorist would get out of his car and help the driver lead the rearing, snorting horses by it. Many the vehicle wrecked and many the neck broken in making the introduction of horse to automobile and establishing his tolerance for it. Loud were the demands for laws to keep automobiles in their place . . . We no longer have breakneck runaways every time a team of horses meets an automobile.[26]

Another example of behavioral evolution in farm animals concerns cattle grids (known as cattle guards in the United States), which are pits with a series of parallel steel tubes or rails over the top. They make it physically impossible for cattle to walk across them, and serve as both a gate and a fence; they keep livestock from passing, but allow vehicles and people to cross freely. Cattle guards were invented in the United States in the nineteenth century to stop animals wandering onto railway lines. They began to be used on American roads around 1905,[27] and are now widely used in many other countries.

When cattle grids were first introduced, animals may have had to learn the hard way that they could not pass. But this is no longer the case. Farm animals seem to avoid these grids instinctively and do not even try to cross them.

Several decades ago, ranchers throughout the American West found that they could save money on cattle grids by using fake grids instead, consisting of stripes painted across the road. The painted grids worked because the animals did not even try to cross them.

In response to my inquiries, several ranchers in the western United States told me that there is no need for herds to be exposed to real cattle grids first. Animals that have never seen a real cattle grid avoid the fake ones. When young cattle approach a painted grid, they “put on brakes with all four feet,” as one rancher expressed it. I corresponded with researchers in the Departments of Animal Science at Colorado State University and at Texas Agricultural and Mechanical University (A&M) who confirmed this observation.

Professor Ted Friend, of Texas A&M, systematically tested the responses of several hundred head of cattle to painted grids, and found that naive animals avoided them just as much as those previously exposed to real grids. Sheep and horses also showed an innate aversion to crossing painted grids. Nevertheless, the spell of a fake grid could be broken. When cows were driven toward one under pressure, or when food was placed on the other side, sometimes one of them examined the stripes closely and then walked across. When one member of a herd did this, the others soon followed. Thereafter, the phony grid ceased to act as a barrier.[28]

Perhaps painted cattle grids work simply because they create the illusion of a drop. In this case, they should have worked all along, and ranchers need not have used real grids in the first place. It would be interesting to find out if *wild* species never before exposed to cattle grids show a comparable aversion to crossing them. It would also be good to find out whether cattle respond equally well to a variety of striped patterns, or just stripes that look like cattle grids.

Interestingly, a new response to cattle grids is currently evolving. In 1985, sheep near Blaenau Ffestiniog, in Wales, started escaping from their pastures by rolling over grids. So did sheep in Sweden, around Malmohus. An editorial in the *Guardian* in 1985 commented:

> To the best of our knowledge the sheep in the Yorkshire Dales, which are mostly Swaledales or Dalesbred, have yet to master the technique of crossing cattle grids by curling up and rolling over them. Yet the sheep of Blaenau Ffestiniog, which are a different breed, have learned how to do it (to the annoyance of the town, which may have to put up a fence) and so have the lowland sheep of southern Sweden. Among the questions that immediately arise are how long will it take the Swaledales to learn and whether, when they do, they will be demonstrating the theory of formative causation.[29]

Twelve years later, sheep started crossing cattle grids in Hampshire. To start with, they used a “commando” technique, with one of them lying on the cattle grid while others scrambled across her. But then they started crossing by rolling across the bars of the grid, like the Welsh sheep.[30] Similar behavior was observed in the Valais region of Switzerland.[31]

In 2004, nineteen years after the editors of the *Guardian* had anticipated the possibility, sheep on the Yorkshire moors began escaping from the moors by rolling over cattle grids and grazing on the nearby gardens of villagers.[32]

Animals, both wild and domesticated, continue to evolve in response to man-made changes in their environment, and the emergence of new patterns of behavior provides opportunities for documenting how these patterns spread. The monitoring of cream-stealing in Britain by amateur bird-watchers in the 1930s and 1940s provides a good precedent for research with widespread participation. Such studies will never be able to provide such clear-cut data as laboratory experiments, but they could shed light on the possible role of morphic resonance in evolution, with very different implications from the neo-Darwinian theory.

A.8 Collective human memory

According to the hypothesis of morphic resonance, that which has been learned by many people in the past should be easier for people to learn today. Everyone draws upon and in turn contributes to a collective human memory.

In 1982, the British magazine *New Scientist* held a competition for ideas for tests of morphic resonance. The winning entry was by a psychologist, Richard Gentle, for an experiment involving Turkish nursery rhymes. He suggested that English-speaking people be asked to memorize two short rhymes in Turkish, a traditional nursery rhyme known to millions of Turks over the years and the other a new rhyme made by rearranging the words in the genuine nursery rhyme. The participants would not be told which was which. After equal periods spent memorizing each of the rhymes, they would be tested to find out which they remembered better. If morphic resonance were at work, the traditional rhyme would be easier to memorize than the new one.

This is an example of an “old field” test, whereby learning of something with a long-established morphic field is compared with learning something new. Many old-field tests of morphic resonance have been conducted. Most have given positive results.

I took up Gentle's suggestion but used Japanese rather than Turkish nursery rhymes. A leading Japanese poet, Shuntaro Tanikawa, kindly supplied me with a genuine nursery rhyme known to generations of Japanese children and two others specially composed to resemble it in its structure, one meaningful and the other meaningless. In tests conducted in Britain and America, people did indeed remember the genuine rhyme significantly better than the others.[33] But this experiment raised a difficulty that applies to all old-field experiments. How can one be sure that the new rhymes, with which the old one was compared, were of similar intrinsic structure? Perhaps real nursery rhymes became popular precisely because they have features that made them easier to memorize in the first place. Although a poet is more likely to be able to produce comparable new rhymes than an amateur, it is hard to know whether the new rhymes would be intrinsically comparable to the old ones in the absence of any morphic resonance effects.

Most old-field tests have involved foreign scripts. Gary Schwartz, a professor of psychology at Yale University, carried out one of the first. His idea was that ordinary words should be associated with morphic fields that facilitate their recognition. For example, the English word *cat* is recognized as a whole—as a Gestalt—and involves a morphic field sustained by resonance from millions of readers in the past. By contrast, a meaningless anagram of the same letters, like "tca," has no such resonance. Schwartz reasoned that people who are unfamiliar with a foreign script might find it easier to recognize real words in this language than false words.

Schwartz selected forty-eight three-letter words from the Hebrew Old Testament, twenty-four common and twenty-four rare, and then produced a meaningless anagram of each word, giving ninety-six words in all. Over ninety participants who were ignorant of Hebrew were shown these words one by one, projected on a screen in a random order. They were asked to guess the meaning of each word by writing down the first English word that came to mind. Then they estimated on a 0–4 scale the confidence they felt in their guess. They were not told the purpose of the experiment, nor that some of the words were scrambled. This test depended entirely on the visual pattern of the written words; it did not involve hearing the words or attempting to pronounce them.

A few participants did in fact guess the meanings of some of the Hebrew words correctly, but Schwartz excluded them from his analysis on the grounds that they might have had some knowledge of Hebrew. He then examined the replies of the participants who always guessed the wrong meanings. Remarkably, on average, they were more confident about their guesses when viewing real words than scrambled words, even though they did not know that some of the words were real and others false. The effect was roughly twice as strong with the common words as with the rare words. The results were very significant statistically.[34]

Only after Schwartz had tested his participants did he inform them that half the words were real and the other half were scrambled. He then showed them the words again, one by one, asking them to guess which was which. The results were no better than chance. The participants were unable to do consciously what they had already done unconsciously. Schwartz interpreted the greater confidence participants felt about their guesses of the meanings of the real words in terms of an "unconscious pattern recognition effect."

Alan Pickering, a psychologist at Hatfield Polytechnic in England, used Persian words rather than Hebrew words, written in Persian script. His test, like Schwartz's, involved real and scrambled words. Participants were shown a word and asked to look at it for ten seconds. They were then asked to draw it. Independent judges evaluated the reproductions of real and false words. Neither the experimenter nor the judges knew which words were real and which were scrambled. The real words were reproduced significantly more accurately than the false words.

Subsequent experiments carried out as student projects by Nigel Davidson with Persian words and by Geraldine Chapman with Arabic words gave similar positive results.

Arden Mahlberg, an American psychologist, performed an analogous test with Morse code. He constructed a new version by assigning dots and dashes to different letters of the alphabet. The participants did not know Morse code. He compared their ability to learn the new code and genuine Morse code, presenting the material in a written form. (The letters *S* and *O* were excluded because many people who do not know Morse code are nevertheless familiar with the code for S.O.S.) On average, participants learned real Morse code significantly more accurately than the new code.[35]

Suitbert Ertel, a professor of psychology at Göttingen University, Germany, investigated the possible effects of morphic resonance on the recognition of Japanese hiragana script, a phonetic component of the Japanese writing system. Participants were shown nine different hiragana characters in a random order, projected on a screen for eight seconds. They then turned to an answer sheet with twenty hiragana characters, among which the nine characters they had just seen were randomly mixed. They were asked to mark the characters they thought they had just seen. The same test was repeated with the characters in different random orders. Each participant did six trials, and the recognition of the hiragana characters generally improved trial by trial.[36]

Ertel predicted that if morphic resonance were playing a part, hiragana characters should be recognized more readily when they were the right way up than when they were upside down, because millions of Japanese were used to recognizing these characters in their normal position. Sure enough, this is what he found.

In a further experiment, he used artificial hiragana characters invented by a graphic designer. Before running the learning tests, he and his students showed participants genuine and artificial hiragana characters and asked them to pick the genuine ones. They could not tell the difference. The Göttingen team then carried out its standard memory tests, and found that the real characters were remembered better than the false ones, in accordance with the predictions of the hypothesis of morphic resonance.

Ertel and his team then carried out a further test, which they regarded as crucial. They compared the effect of putting the real characters upside down with that of putting fake characters upside down. Ertel argued that with fake Hiragana characters, rotation should have no effect because morphic resonance plays no part in the recognition of these characters either way up.

The results were confusing and Ertel’s interpretation was hard to follow. In the first two trials, there was indeed almost no difference in the recognition of the upside-down and right-way-up fake hiragana characters (figure A.7). But in the subsequent trials, the false hiraganas were remembered better the right way up. Ertel argued that the faster rate of learning in the later trials with the fake hiraganas the right way up was because of “intrinsic factors” that had nothing to do with morphic

resonance. Surprisingly, he provided no statistical analysis to show whether this effect was significant.

However, Ertel's fake hiragana characters were *designed* to look like real hiragana characters when they were the right way up. Insofar as they resembled real hiragana characters, it may be that they did so precisely because they had a "right-way-up" feel to them, because of a generic resemblance to real characters. This "intrinsic factor" may not be an alternative to morphic resonance, but rather may depend on their generic resemblance to right-way-up hiragana characters, which was built in from the start.

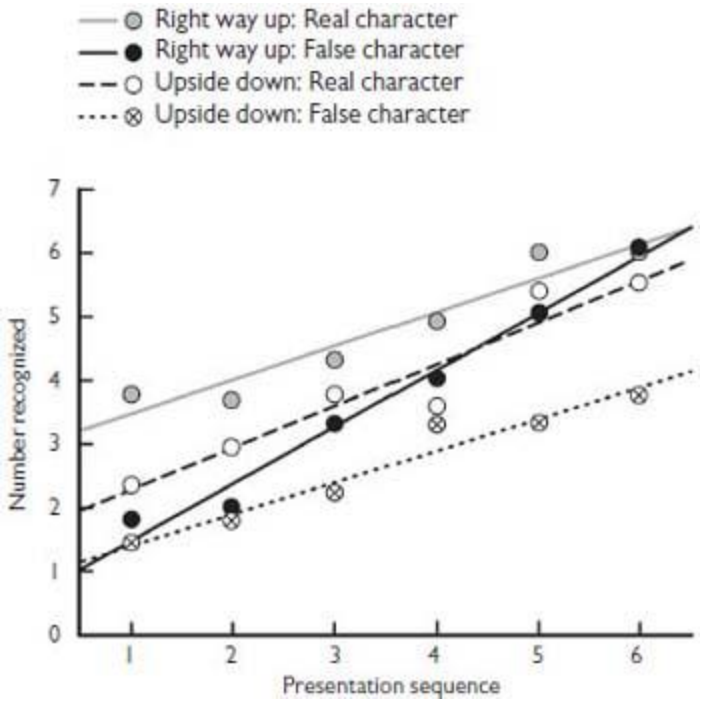

Figure A.7. The results of Suitbert Ertel's experiment on the recognition of hiragana characters. The vertical axis shows the number of words recognized in six successive trials. The four sets of data points refer to real and false characters, right way up and upside down. (Reproduced by courtesy of Suitbert Ertel)

In retrospect, Ertel thought that he and his students had made a mistake in their initial tests when they were trying to find out whether the fake hiragana characters were indeed similar to real ones: "It gradually dawned on us that we had not instructed the participants of the preliminary tests optimally. We should have asked them to look at the 40 symbols on the piece of paper and mark those that seemed *simpler, more pleasant, and less strange* to them. These were the intrinsic features that another test had

already revealed to be relevant. Instead we had informed the participants that there were 20 genuine and 20 artificial Japanese symbols and asked them to mark the 20 genuine." The complexity of Ertel's interpretations illustrates how difficult it is to obtain clear-cut results in old-field experiments.

Robert Schorn, Gottfried Tappeiner, and Janette Walde recently carried out an old-field test at the University of Innsbruck. They used stimuli consisting of political, religious, and economic symbols such as flags, emblems, and trademarks that were once well known but have now fallen into oblivion, or ones that are familiar to many people in foreign countries, such as the Chinese Coca-Cola symbol, Indian trademarks, and Far Eastern religious symbols. For each of the symbols, a designer created a corresponding control symbol with a similar general pattern and similar complexity.

In order to find out whether the new symbols were indeed comparable, the experimenters conducted seven pretests with more than two hundred participants, who were asked to indicate which of the symbols in each pair they found less credible or real. In their main experiment, they employed false symbols that were as credible as the originals, if not more credible. Participants were shown pairs of symbols, one real and one false, in a randomly determined order, and they were asked to judge which of each pair had more "spirit." They selected the real symbols significantly more often than the fake ones.[37]

In a second test, the Innsbruck team compared real Russian words written in Cyrillic script with meaningless anagrams of these words. Again the real and false stimuli were presented in pairs, and the participants were asked to judge which had more "spirit." The real words were selected significantly more than the anagrams.

Some of these tests took place through the Internet, illustrating the potential for widespread public participation in automated morphic resonance tests. Kimberly Robbins and Chris Roe, at the University of Northampton, England, carried out the most recent old-field experiment using genuine and false Chinese characters. The experimental design was similar to Ertel's. Participants were first shown a PowerPoint presentation consisting of five real and five false Chinese characters in a random sequence, seeing each character for three seconds. They were not told that

some characters were real and others false. They were then given a sheet with twenty characters on it and asked to circle the ten they had just seen. The other ten characters were “decoys,” and again five were real and five false. The participants recognized the real characters significantly better than the false ones. With the decoys, participants had significantly more false memories of real than false characters, consistent with a morphic resonance effect.[38]

Nevertheless, all old-field tests face the difficulty of controlling for “intrinsic factors” that might make old symbols, words, or rhymes more memorable or more attractive than newly invented ones. But are intrinsic factors and morphic resonance genuine alternatives? Intrinsic factors may themselves depend on morphic resonance.

A.9 Improving human performance

The simplest new-field tests start with two new patterns. The first step is to find out how easily they can be learned or recognized. The second step is to build up morphic resonance from one and not the other. If morphic resonance is playing a part, the one that has been “boosted” should subsequently be easier to learn or recognize; there should be no such change with the control.

The first new-field test was carried out with hidden images, following a suggestion by Nicholas Humphrey. Such pictures seem to make no sense at first, or contain only vague hints of patterns (see figure A.8a). Seeing the underlying image (figure A.8b) involves a sudden Gestalt shift; the picture takes on a definite meaning. After this has happened, it is difficult not to recognize the hidden image and hard to believe that others cannot see it. If morphic resonance is at work, a hidden image should become easier to recognize if many people have already seen it.

In the summer of 1983, a British television company, Thames Television, made it possible for me to conduct an experiment of this kind. The two puzzle pictures were specially produced by an artist and designed to be difficult, so that only a small minority of people could spot the hidden images. Before the television broadcast in Britain, I sent both these pictures to collaborators in Europe, Africa, and the Americas. Each experimenter showed both pictures for one minute each to a group of participants before

the transmission, and afterward to another group of comparable participants. The number of people who recognized the hidden image was recorded.

The experimenters did not know which of the pictures was going to be shown on television, and nor did I. On the TV show itself, one was picked at random and shown to about two million viewers. After several seconds the answer was revealed, and this then "melted" back into the puzzle picture so that the previously hidden image was now readily apparent. The same picture was shown once more at the end of the program.

The percentage of participants recognizing the control picture before and after the TV broadcast did not change, while the percentage recognizing the image shown on TV in Britain increased. This effect was statistically significant, with a probability of less than one in a hundred that the result arose by chance.[39]

The experiment was repeated, using different images, on BBC television in November 1984 on a popular science program called *Tomorrow's World*. Again there were two puzzle pictures with hidden images. Experimenters all over the world tested groups of participants to find out what proportion could recognize the hidden images within thirty seconds. Such tests were carried out in a five-day period before the TV transmission in Britain, and with comparable participants in a five-day period afterward. On the TV show, one of the two images was selected at random and shown to eight million viewers, to whom the answer was revealed.

This picture did in fact become significantly easier to recognize elsewhere while there was no change with the control. But this positive effect was confined to participants in continental Europe; there was no effect in North America. The disparity was surprising. Morphic resonance should not be distance-dependent. One possible explanation was that in Europe, where the time difference from Britain is only one hour, people were more "in phase" with the British TV audience than people in America, with a five- to eight-hour time difference.

Figure A.8a. A hidden image, as used in a television test for morphic resonance. The image is revealed in figure A.8b.

A new hidden-image experiment was carried out in February 1985, with a TV transmission in Germany by Norddeutscher Rundfunk. Again there were two pictures, of which only one was shown on television. This experiment was coordinated by Susan Fassberg, in Freiburg im Breisgau. She arranged for thousands of participants to be tested in various parts of the world, predominantly in Britain. As in the previous experiments, there was no significant change in the proportion of people recognizing the control picture, but the proportion recognizing the test picture *declined* in Britain and elsewhere after being seen by about half a million people in North Germany! The decline was significant, at the 2 percent level of probability. From the point of view of morphic resonance, there should have been an increase. From a Skeptical point of view, there should have been no change. Nobody predicted a decrease.

Figure A.8b. The image hidden in figure A.8a.

This result showed that other factors were coming into play, but what were they? No one knew. This puzzling finding discouraged anyone from doing more tests on television, which were complicated to arrange.

In 1987, the Institute of Noetic Sciences (IONS), near San Francisco, announced an award for the best student research on morphic resonance. An independent panel of judges assessed the entries, and the results were announced in 1991.[40]

The winner of the undergraduate award was Monica England, a psychology student at the University of Nottingham, England. Her test was stimulated by anecdotal evidence that some people find it easier to do newspaper crosswords the day after they have been published than when they first appear, an effect that could be due to morphic resonance from thousands of people who have already done the puzzle.

The experiment involved two puzzles from a London newspaper, the *Evening Standard*, which was not distributed in Nottingham. The newspaper kindly cooperated by supplying two unpublished puzzles a week before they appeared: the "easy crossword" and the "quick crossword." The easy crossword had simple cryptic clues and the quick crossword had single-word clues that required synonyms as answers.

Monica England tested about fifty students the day before the crossword puzzles were published in London, and a further fifty the day after. Both groups of participants were also given two control puzzles, which had been published in the *Evening Standard* two weeks earlier. The participants were given ten minutes with each crossword to solve as many clues as possible.

On average, participants solved significantly more clues with the easy puzzle after it had been published than before. There was no change with the control crossword. By contrast, with the quick crossword there was no significant difference in the test crossword relative to the control.

I repeated this experiment in 1990, again using easy and quick crosswords from the *Evening Standard*, and testing people with the help of experimenters who lived far from London, where the participants would not have seen this London newspaper. Again, the scores with both crosswords were compared with controls. There was a slight improvement in scores with the easy crossword after it was published, but this change was not statistically significant. By contrast, there was a statistically significant increase with the quick crossword. Thus the results were inconsistent, giving a positive effect with one crossword but not the other, as in Monica

England’s experiment. In her test the easy crossword showed a positive effect, and in mine the quick crossword.

While reflecting on these results, I realized that I taken it for granted that all the crossword puzzles were new, and I had assumed that they would be unaffected by morphic resonance from crosswords in the past. I then inquired how the crosswords were put together, and found that the compilers frequently recycled clues from previous crosswords. Hence these simple crosswords did not provide a good test for morphic resonance, since many of the clues were in fact not new.

Zoltan Dienes, then in the psychology department at the University of Oxford, won the IONS award for graduate students. His participants were required to decide quickly whether a string of letters they saw on a computer screen was a meaningful English word or a nonword. This experiment involved a phenomenon known to psychologists as “repetition priming,” which occurs when a word (or other stimulus) is recognized more quickly after repeated exposure to it. Dienes reasoned that later participants might find it easier to recognize stimuli if others had done so earlier.

The participants saw strings of letters flashed on a computer screen and had to indicate whether a string was a real word or a nonword by pressing computer keys as fast as possible. Dienes used two sets of words and non-words. One “shared” word set was presented to all ninety participants, while the second “unique” set was shown only to every tenth participant. The experiment thus involved eighty “boosters” who viewed only the shared stimuli and ten “resonators” who saw the shared stimuli together with the unique ones. If morphic resonance were at work, the speed at which the shared stimuli were correctly judged should increase relative to the speed at which unique stimuli were correctly judged. In order to maximize the resonance between participants, all experimental trials were conducted in a controlled environment with distinctive visual, olfactory, and auditory cues.

The outcome was positive and statistically significant. The more often a nonword had been seen before, the faster subsequent resonators responded to it. However, when Dienes tried to repeat this experiment at the University of Sussex, there was no significant effect.[41]

Professor Suitbert Ertel carried out two new-field experiments in addition to the old-field tests discussed above. The first took place through a magazine called *Übermorgen*. The experiment was based on anagrams, such as "*Seterleirei*" for "*Reiseleiter,*" and the task for the magazine's readers was to find the normal words. Readers were asked to repeat each anagram and its corresponding word as often as possible. When they had memorized them, they sent a postcard to the experimenters on which they gave their telephone numbers. Their names were entered for a raffle, and thirty of them received free copies of one of my books. They had to face a possible checkup by telephone to see whether they knew the words, and a random sample of fifty people was actually called, with satisfying results. About a thousand readers participated.

These readers did not know the experiment also included sixty students at Dresden University, where the magazine was not distributed. These students were tested with the same ten anagrams and with ten additional anagrams that had not been boosted by readers of *Übermorgen*. Could the students solve the boosted anagrams better than the controls? On average they could, but the effect was not statistically significant.[42]

Ertel's second experiment took place through another magazine, *PM*. This was designed to be fun for readers, and used artificial German words in standard phrases or proverbs. The meaning of the artificial words had to be guessed from their context, like "*Die blampe Leier*," "*Das ist doch ein blamper Hut*," in which "*blampe*" was invented to replace "*alt*." Ten new words had to be learned in this way, in a total of one hundred phrases. The count of new meanings (e.g., the meaning *alt = blampe* occurred eight times) resulted in a ten-digit telephone number that the readers called. If the number was correct, they received a confirmation from an answering machine. Sending a postcard with the correct number enabled the participants to take part in a raffle for fifty copies of my book. Altogether 1,017 readers of *PM* magazine participated.

Again, the influence of this boosting was tested in Dresden, where participants had to push a button saying "artificial" or "real" as quickly as possible after a word had appeared on a computer screen. The boosted artificial words were mixed with twenty other artificial words that had not been boosted. The students in Dresden were tested before and after the *PM*

boosting. There was no difference in their success with the boosted and the control words.

One problem with this test was that the conditions in which the participants saw the words were very different from the context in which the *PM* readers learned them. One group of people were doing puzzles in a magazine at home or in other informal settings. The others were being tested for their reaction speed on computers in a laboratory. These dissimilarities could have weakened any resonance effect.

In summary, small-scale new-field tests have not given consistent, repeatable results. But perhaps they are not sensitive enough; the resonance may be too weak to be detectable with only a few hundred or a few thousand boosters.

Morphic resonance can be investigated on a much larger scale by studying changes in human performance over time. Does the performance of new skills show a tendency to improve as time goes on? Do video games get easier to play? Do new sports such as skateboarding and windsurfing become easier to learn? Anecdotal evidence suggests that they do, but such changes are not documented quantitatively, and the situation is complicated by other factors, like improvements in equipment, fashion, better teaching methods, and so on.

One of the few areas in which detailed data are available over many years is for the scores of IQ (Intelligence Quotient) tests. Around 1980, I realized that if morphic resonance occurs, average performance in IQ tests should be rising, not because people are becoming more intelligent, but because IQ tests should be getting easier to do as a result of morphic resonance from the millions who have done them before. I searched for data that would enable this prediction to be tested, but could not find any published figures. I was therefore intrigued in 1982 by the finding that average IQ test scores in Japan had been increasing by 3 percent a decade since the Second World War.[43] Soon afterward, it turned out (to the relief of many Americans) that IQs had been rising at a similar rate in the United States.

The psychologist James Flynn first detected this effect in America in his study of intelligence tests by U.S. military authorities. He found that recruits who were merely average when compared with their contemporaries were above average when compared with recruits in a

previous generation who had taken exactly the same test (figure A.9). No one had noticed this trend because testers routinely compared an individual's score with others of the same age, tested at the same time; at any given time, the average IQ score is set to one hundred by definition.[44]

Comparable increases are now known to have occurred in twenty other countries, including Australia, Britain, France, Germany, and Holland.[45] Many attempts have been made to explain this "Flynn effect," but none has succeeded.[46] For example, very little of this effect can be ascribed to practice at taking such tests. If anything, such tests have become less common in recent years. Improvements in education cannot explain it either. Nor, as some have suggested, can increasing exposure to television. IQ scores began rising decades before the advent of television in the 1950s, and as Flynn has commented wryly, television was usually considered "a dumbing down influence until this effect came along."[47] The more research there has been, the more mysterious the Flynn effect has become. Flynn himself describes it as "baffling."[48] But morphic resonance could provide a natural explanation.

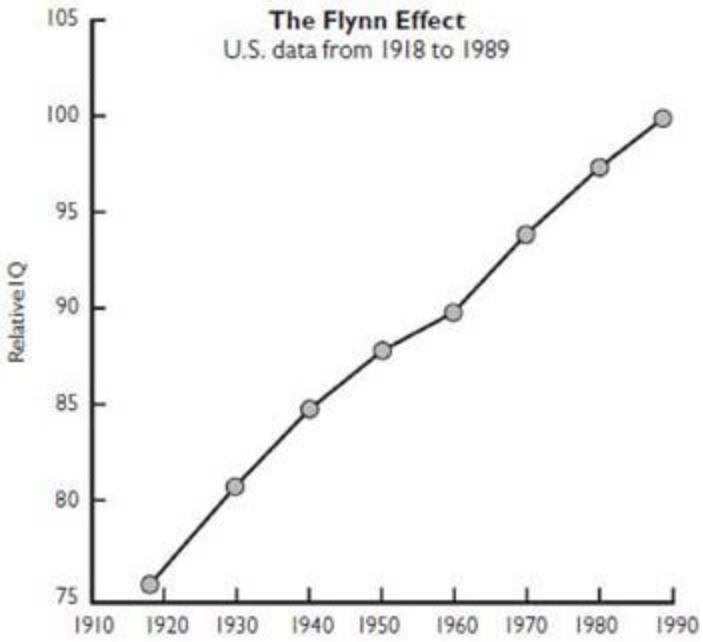

Figure A.9. Changes in average IQ test scores in the United States from 1918 to 1989, relative to 1989 values. (Data from Horgan, 1995)

If the Flynn effect is indeed explicable in terms of morphic resonance, it shows that such resonance effects are relatively small. If millions of people

taking IQ tests lead to increased scores of only a few percentage points, then in experiments involving a few hundred people, or at most a few thousand, the morphic resonance effects may be too small to detect against the “random noise” due to wide variations in performance from participant to participant.

Morphic resonance might also have a bearing on “grade inflation,” the phenomenon of increasing academic grades over time. An evaluation of the grading practices in American colleges and universities shows that since the 1960s, grades in the United States have risen at a rate of 0.15 per decade in a 4.0 scale. In Britain, the proportion of students achieving A grades in school examinations and first-class university degrees has also been increasing steadily. This phenomenon has caused an intense debate: some people lament that standards are becoming more lax, while others assert that students are producing better work. But morphic resonance would again provide a simple explanation. Standard examinations are becoming easier to do because so many people have already done them.

When my elder son, Merlin Sheldrake, was about to take the British GCSE (General Certificate of Secondary Education) examinations at age sixteen, he and a group of his school friends came up with an ingenious plan for increasing their scores with no extra effort. In each exam, they would do the last questions first, and then return to the beginning and follow the normal sequence. Hence they would be about ten minutes behind everyone else in Britain sitting the same exam at the same time, and should therefore receive a boost by morphic resonance. They actually put this idea into practice, reasoning that if morphic resonance existed, they might score better, and if it did not, they had nothing to lose.

This idea raises the possibility of an experiment within the framework of large-scale examinations. The order of questions carried out by a random sample of students could be changed. Are scores significantly higher on questions that other students have answered earlier?

New-field tests for morphic resonance could also be done on a large scale using newly released puzzles, such as Sudokus and computer games. Such tests would require the cooperation of the game or puzzle companies. As in the hidden-image and crossword experiments, there would need to be a control puzzle or game that was not released during the period of the test. Groups of participants would need to be tested in places where they do not

have access to the newly released puzzles or games, and such tests would be done before and after the puzzles or games were released elsewhere.

A.10 Resonant computers

The hypothesis of formative causation applies to self-organizing systems such as crystals, cells, and animal societies. Morphic fields work by imposing patterns on otherwise indeterminate events. Hence morphic resonance does not apply to machines. Machines are not self-organizing systems, but are made of components put together in factories according to human designs. Their functioning is strictly determinate—they are designed to be predictable, and to do the same things over and over again. Even when computers incorporate "randomness" in their programming, the random numbers are generally provided by pseudorandom algorithms, rather than by sources of genuine random "noise."

Francisco Varela, a neuroscientist, tried to test for morphic resonance in a computer in the 1980s. He programmed it to carry out the same sequence of operations with 100 million repetitions, and measured how long these repetitions took.

There was no speeding up. Varela published this result in the *Skeptical Inquirer,* claiming that it falsified the hypothesis of formative causation.[49] He argued that the changes imposed on silicon chips by the workings of the computer were equivalent to repeated crystallizations, and hence they should happen faster if the hypothesis of morphic resonance was correct.

I replied that there was a difference in kind between the spontaneous formation of a crystal and the changes imposed on a silicon chip in a computer. But most important, the experiment was technically misconceived. Computers work by a rapid series of operations pulsed by the computer's internal clock. In Varela's computer, the clock paced the instructions in the program with a unit time of one microsecond. Even if the silicon chips had responded faster to the pulsed instructions as a result of morphic resonance, the sequence of operations was fixed by the clock and could not have speeded up.[50] Many readers of the *Skeptical Inquirer* independently pointed out this fatal flaw in the test.[51]

One morning in the spring of 1990, I was suddenly deluged with telephone calls from science journalists and computing science departments

at universities. The cause of the excitement was an article in a British magazine, *Computer Shopper,* describing some remarkable results about which I knew nothing. The report stated that an Italian computer scientist, Dr. Lora Pfilo, had recently been carrying out an experiment with genetic algorithms, trying to find the best solution to an engineering problem by letting prospective solutions play against each other over successive "generations." The simulation was running on the Bologna University connection machine, a massive parallel computer with 256,000 processors. The article stated that Dr. Pfilo noticed that the first time the program ran it took forty minutes to complete, yet the second time she ran it, it took twenty-three.

She found the sudden decrease in processing time a little worrying—perhaps it had not run correctly owing to a power surge. So she ran the program again, and although the results matched up with results from the second run, the processor time had decreased to thirteen minutes. She ran the program repeatedly, and eventually the time decreased to one minute and twelve seconds. The result amazed her. What was this extra causal factor that led to the decrease in programming time? In January she contacted a colleague visiting Milan University—Professor Kvitlen Duren, a professor of mathematics from the Institut Svit Chotiri in Kiev.

The article continued with an interview with Professor Duren, who was in London to address the Royal Society. He too claimed to have noticed a decrease in processing time in one of his computers, but not in another that was running the same genetic algorithm program. He found that the computer that ran more quickly had some additional circuitry, including a hardware random number device based on a reversed Zener diode that generated quantum randomness. The other computer worked on a standard pseudorandom number algorithm. Professor Duren was reported as saying:

> We had great difficulty accepting this at first but what must have been happening was that, in some sense, information from previous runs was being "stored" out there. At this point, my good friend Lora Pfilo contacted me and in the course of a general conversation we discovered we had both observed the same effect. What I had been calling causal acceleration, she called morphic resonance . . . What happens on the connection machine is that there is a quantum

> indeterminacy in the scheduling of the multiple processes. The indeterminacy is sufficient to produce the effects that Lora saw.

I rang up *Computer Shopper* magazine and asked to be put in touch with the author of the article. Soon afterward, he rang me back and said, “Before we go any further, please look at the date on this issue of *Computer Shopper*.” I did. It was April 1.

The author, Adrian Owen, and his colleague John Kozak invited me to meet them in a local pub soon afterward. Professor Duren, pictured in the article, was none other than John Kozak with a false beard. Lora Pfilo was an anagram of April fool. They told me that they were both intrigued by morphic resonance and had been thinking about ways in which it might apply to computers. They had also tried to think of an April fool article that would be sufficiently plausible to stimulate widespread interest, without being recognized immediately as a spoof. They succeeded beyond their wildest expectations.

In 1993, Steven Rooke, of Tucson, Arizona, an experienced computer programmer, explored the possibility of carrying out in reality what the report in *Computer Shopper* had described. He used a computer graphics system, a reversed Zener diode as a source of quantum noise, and a genetic algorithm program that converged on a target image. The question was whether, in a randomized series of runs, the rate of convergence on this target would increase. Rooke had to overcome a variety of technical problems, and the results of the morphic resonance tests were inconclusive. But his programs generated extraordinarily beautiful graphic images, which he then produced commercially.

Looking back on his experience, in October 2007, he doubted whether the quantum event generator and the computer programs were tightly coupled enough to constitute a morphic field.

> Even if a genetic program convergence process can resonate with processes occurring fleetingly in time previously, it seems likely that there will need to be a much tighter coupling between the thing generating the source of randomness (the quantum event generator) and the novel thing being produced. Designing such experiments is fraught with difficulties, including keeping track of previous solutions,

> so as to know whether a new solution is really new; all preparatory work should be done solely with pseudo-random numbers.[52]

In morphic fields, all the different parts of the system are linked together and the fields work by affecting random processes. The problem Rooke highlighted is that the random numbers were fed into the computer, but the random number generator was not linked to the system in any other way. To make it more closely coupled, the random number generator would have to be affected by the processes it was itself affecting. One way to make a more closely coupled system, suggested by the mathematician Ralph Abraham, would be to use optical feedback—the simplest model being to point a video camera at a screen that displays the output from the camera at low definition, leaving scope for random noise.

But there may be a surprising new possibility. We are used to the idea that all computers are digital; but in the early days of computing, in the 1950s, analog computers were serious contenders for the path of the future. They enabled complex, self-organizing patterns of activity to develop through sometimes chaotic, oscillating circuits in electronic devices. William Ross Ashby, a British pioneer of cybernetics, published in 1952 an influential book called *Design for a Brain*, which showed how analog cybernetic circuits could model brain activity, including leaps from one state or level to another. Then digital computers took over, and analog systems were forgotten.

In a recent revival, the analog approach has led to astonishing results in the creation of “living machines” in the form of insectlike analog robots. These machines achieve feats of self-organization, and even of learning and memory, whose complexity belies the fact that these machines contain less than ten transistors and have no computers within them at all. Mark Tilden, the inventor of these machines, built electronic systems that rely on inputs from sensors as the robots move. The activity of the wavelike, rhythmic circuits is partly chaotic and unpredictable, and is influenced by what has gone before. As Tilden put it, “When conditions are repeated exactly the same way twice, a digital computer will respond in exactly the same way. These analog devices may or may not do the same thing twice! You can influence them, but you don’t actually have any power over them.”[53] Tilden’s work has inspired a new kind of “reaction-based” machine

building, called BEAM robotics (Biology Electronics Aesthetics Mechanics, or Biotechnology Ethology Analogy Morphology).

Can morphic fields be established in electronic machines? No one knows. But for research on this question, a good starting point might be self-organizing, wave-based analog robots that include truly random elements.

If morphic fields were to come into being within such probabilistic analog systems, they would automatically have an inherent memory, without the need for special memory-storage devices like hard drives and memory chips. They would also enter into morphic resonance with similar computers around the world, without the need for communication through wires, cables, or radio signals. They would share a collective memory. An entirely new technology would be born.

APPENDIX B

■

MORPHIC FIELDS AND THE IMPLICATE ORDER

A Dialogue with David Bohm

David Bohm was an eminent quantum physicist. As a young man, he worked closely with Albert Einstein at Princeton University. With Yakir Aharonov he discovered the Aharonov-Bohm effect. He was later professor of theoretical physics at Birkbeck College, University of London, and was the author of several books, including Causality and Chance in Modern Physics[1] *and* Wholeness and the Implicate Order.[2] *He died in 1992. This dialogue was first published in* ReVision Journal, *and the editorial notes are by Renée Weber, the journal's editor.*[3]

Bohm: Suppose we look at the development of the embryo, at those problems where you feel the present mechanistic approach doesn't work. What would the theory of morphogenetic fields do that others don't?

Sheldrake: The developing organism would be within the morphogenetic field, and the field would guide and control the *form* of the organism's development. The field has properties not just in space but in time. Waddington demonstrated this with his concept of the chreode [see figure 5], represented by models of valleys with balls rolling down them toward an end point. This model looks mechanistic when you first see it.

But when you think about it for just a minute you see that this end point, which the ball is rolling down the valley toward, is in the *future,* and it is, as

it were, attracting the ball to it. Part of the strength of this model depends on the fact that if you displace the marble up the sides of the valley, it will roll down again and reach the same end point; this represents the ability of living organisms to reach the same goal, even if you disrupt them—cut off a bit of embryo and it can grow back again; you'll still reach the same end point.

Bohm: In physics the Lagrangian law is rather similar; the Lagrangian falls into a certain minimum level, as in the case of the chreode. It's not an exact analogy, but you could say that in some sense the classical atomic orbit arises by following some sort of chreode. That's one way classical physics could be looked at. And you could perhaps even introduce some notion of physical stability on the basis of a chreode. But from the point of view of the implicate order, I think you would have to say that this formative field is a whole set of potentialities, and that in each moment there's a selection of which potential is going to be realized, depending to some extent on the past history, and to some extent on creativity.

Sheldrake: But this set of potentialities is a limited set, because things do tend toward a particular end point. I mean cat embryos grow into cats, not dogs. So there may be variation about the exact course they can follow, but there is an overall goal or end point.

Bohm: But there would be all sorts of contingencies that determine the actual cat.

Sheldrake: Exactly. Contingencies of all kinds, environmental influences, possibly genuinely chance fluctuations. But nevertheless the end point of the chreode would define the general area in which it's going to end up.

Anyway, the point about Waddington's concept of the chreode, which is taken quite seriously by lots of biologists, is that it already contains this idea of end point, in the future, in time; and the structure, the very walls of the chreode, are not in any normal sense of the word material, physical things. Unfortunately Waddington didn't define what they were. In my opinion, they represent this process of formative causation through the morphogenetic field. Waddington in fact uses the term "morphogenetic field." Now the problem with Waddington's concept is that, when he was

attacked by mechanists, who maintained that this was a mystical or ill-defined idea, he backed down and said, well, this is just a way of talking about normal chemical and physical interactions. René Thom, who took up the concepts of chreodes and morphogenetic fields and developed them in topological models (where he called the end points "morphogenetic attractors"), tried to push Waddington into saying more exactly what the chreode was. Waddington, whenever pushed by anyone, even René Thom, backed down. So he left it in a very ambiguous state.

Now Brian Goodwin and people like him see chreodes and morphogenetic fields as aspects of eternal Platonic forms; he has a rather Platonic metaphysics. He sees these formative fields as eternally given archetypes, which are changeless and in some sense necessary. It is almost neo-Pythagorean; harmony, balance, form, and order can be generated from some fundamental mathematical principle, in some sort of necessary way, that acts as a causal factor in nature in an unexplained but changeless manner.

The difference between that and what I'm saying is that I think these morphogenetic fields are built up causally from what's happened before. So you have this introjection, as it were, of explicit forms, to use your language, and then projection again.

Bohm: Yes. What you are talking about—the relation of past forms to present ones—is really related to the whole question of time—"How is time to be understood?" Now, in terms of the totality beyond time, the totality in which all is implicate, what unfolds or comes into being in any present moment is simply a projection of the whole. That is, some aspect of the whole is unfolded into that moment and that moment is just that aspect. Likewise, the next moment is simply another aspect of the whole. And the interesting point is that each moment resembles its predecessors but also differs from them. I explain this using the technical terms "injection" and "projection." Each moment is a projection of the whole, as we said. But that moment is then injected or introjected back into the whole. The next moment would then involve, in part, a re-projection of that injection, and so on indefinitely. [*Editor's note: As a simplistic analogy, take the ocean and its waves: each wave arises or is "projected" from the whole of the ocean; that wave then dips back into the ocean, or is "injected" back into the*

whole, and then the next wave arises. Each wave is affected by past waves simply because they all rise and fall, or are projected and injected, by the whole ocean. So there is a type of "causality" involved, but it is not that wave A linearly causes *wave B, but that wave A* influences *wave B by virtue of being absorbed back into the totality of the ocean, which then gives rise to wave B. In Bohm's terms, wave B is in part a "re-projection" of the " injection" of wave A, and so on. Each wave would therefore be similar to previous waves, but also different in certain aspects—exact size, shape, etc. Bohm is suggesting that there is a type of "causality," but one that is mediated via the totality of the implicate ocean, and not merely via the separated, isolated, explicate waves. This means, finally, that such "causation" would be non-local, because what happens at any part of the ocean would affect all other parts.*] Each moment will therefore contain a projection of the re-injection of the previous moments, which is a kind of memory; so that would result in a general replication of past forms, which seems similar to what you're talking about. [*Editor's note: This is according to Bohm's reformulations of present-day quantum mechanics. In the following discussion, Bohm will point out that present-day quantum mechanics, as it is usually interpreted, completely fails to account for the replication of past forms, or the notion of temporal process, a failure that in part led Bohm to propose "injection" and "projection" via the implicate order.*]

Sheldrake: So this re-injection into the whole from the past would mean there is a causal relationship between what happens in one moment and what subsequently happens?

Bohm: Yes, that is the causal relation. When abstracted from the implicate order, there seems to be at least a tendency, not necessarily an exact causal relationship, for a certain content in the past to be followed by a related content in the future.

Sheldrake: Yes. So if something happens in one place at one time, what happens there is then re-injected into the whole.

Bohm: But it has been somewhat changed; it is not re-injected exactly, because it was previously projected.

Sheldrake: Yes, it is somewhat changed, but it is fed back into the whole. That can have an influence that, since it is mediated by the whole, can be felt somewhere else. It doesn't have to be local.

Bohm: Right, it could be anywhere.

Sheldrake: Well, that does sound very similar to the concept of morphic resonance, where things that happen in the past, even if they're separated from each other in space and time, can influence similar things in the present, over, through, or across—however one cares to put it—space and time. There's this non-local connection. This seems to me to be very important because it would mean that these fields have causal (but non-local) connections with things that have happened before. They wouldn't be somehow inexplicable manifestations of an eternal, timeless set of archetypes. Morphogenetic fields, which give repetitions of habitual forms and patterns, would be derived from previous fields (what you call "cosmic memory"). The more often a particular form or field happened, the more likely it would be to happen again, which is what I am trying to express with this idea of morphic resonance and automatic averaging of previous forms. It's this aspect of the theory that makes it empirically testable, because this aspect leads to predictions, such as: if rats learn something in one place, say a new trick, then rats everywhere else should be able to learn the same trick faster. That makes it different from Goodwin's theory of eternal archetypes, which wouldn't lead to that prediction, because they would always be the same. And this is where what I'm saying grows out of the tradition of thought that has been around in biology for sixty years, the idea of morphogenetic fields. These fields have always been very ill-defined, and have been interpreted either as Waddington did, to be just a way of speaking about conventional mechanistic forces, or by a Goodwin-type metaphysical approach.

Bohm: Yes. Now if we were to use the analogy of the radio wave receiver which you discussed in your book: if you take a receiver, it has the ability to amplify very small radio wave signals. As you say, we can regard the radio wave as a morphogenetic field. And the energy in the receiver (which comes from the wall socket) is being given shape or form by the

information in the radio wave itself, so you get a musical sound coming out of the speaker. Now in that case you could say the radio wave possesses a very tiny energy compared to the energy in the radio coming from the wall socket. Thus, roughly speaking, there are two levels of energy: one is a kind of energy that is unformed but which is subject to being formed by very tiny impulses. The other is a field that is very much more subtle and which has very little energy in the usual sense of the word, but has a quality of form that can be taken up by the energy of the radio receiver. The point is that one might look at the implicate order that way; the subtler levels of the implicate order are affecting the energy in the less subtle levels. The implicate energies are very fine; they would not ordinarily even be counted as energies, and these implicate energies are giving rise to the production of electrons and protons and the various particles of physics. And these particles have been replicating so long that they are pretty well determined, or fixed in "cosmic memory."

Sheldrake: Yes, I think one could look at it that way. But whether these morphogenetic fields have a subtle energy or not, I don't really know what to think about that. When I wrote my book, I tried to draw a very sharp distinction between formative causation and the ordinary kind of causation (energetic causation), the kind that people are familiar with (e.g., pushing things, electricity), for two reasons: first, I wanted to make it clear that this formative causation is a different kind of thing from what we usually think of as causation. (It may not be so different when one takes into account causation through fields, as in physics.) But the second reason was that it is an important part of my theory that these morphic fields can propagate across space and time, that past events could influence other events everywhere else. Now if these fields are conceived of as energetic, in any normal sense of the word, most people assume that they could only propagate locally according to some sort of inverse square law, because most known energies—light, gravity, magnetism, etc.—fade out over distance.

Bohm: But that doesn't necessarily follow, you see. One of the early interpretations of the quantum theory I developed was in terms of a particle moving in a field.

Sheldrake: The quantum potential.

Bohm: Yes. Now the quantum potential had many of the properties you ascribe to morphogenetic fields and chreodes; that is, it guided the particle in some way, and there are often deep valleys and plateaus, and particles may start to accumulate in plateaus and produce interference fringes. Now the interesting thing is that the quantum potential energy had the same effect regardless of its intensity, so that even far away it may produce a tremendous effect; this effect does not follow an inverse square law. Only the form of the potential has an effect, and not its amplitude or its magnitude. So we compared this to a ship being guided by radar; the radar is carrying form or information from all around. It doesn't, within its limits, depend on how strong the radio wave is. So we could say that in that sense the quantum potential is acting as a formative field on the movement of the electrons. The formative field could not be put in three-dimensional [*or local*] space, it would have to be in a three-n dimensional space, so that there would be non-local connections, or subtle connections of distant particles (which we see in the Einstein-Podolsky-Rosen experiment). So there would be a wholeness about the system such that the formative field could not be attributed to that particle alone; it can be attributed only to the whole, and something happening to faraway particles can affect the formative field of other particles. There could thus be a [*non-local*] transformation of the formative field of a certain group to another group. So I think that if you attempt to understand what quantum mechanics means by such a model, you get quite a strong analogy to a formative field.

Sheldrake: Yes, it may even be a homology; it may be a different way of talking about the same thing.

Bohm: The major difference is that quantum mechanics doesn't treat time, and therefore it hasn't any way to account for the cumulative effect of past forms. To do so would require an extension of the way physics treats time, you see.

Sheldrake: But don't you get time in physics when you have a collapse of the wave function?

Bohm: Yes, but that's outside the framework of quantum physics today. That collapse is not treated by any law at all, which means that the past is, as it were, wiped out altogether. [*Editor's note: This is the point where, as earlier mentioned, Bohm discusses some of the inadequacies of present-day quantum mechanics—in particular, its incapacity to explain process, or the influence of the past on the present. He then suggests his re-formulations—injection, projection, the implicate order, etc.—that might remedy these inadequacies. And these re-formulations, apparently, are rather similar to Sheldrake's theories.*] You see, the present quantum mechanics does not have any concept of movement or process or continuity in time; it really deals with one moment only, one observation, and the probability that one observation will be followed by another one. But there is obviously process in the physical world. Now I want to say that that process can be understood from the implicate order as this activity of re-projection and re-injection. So, the theory of the implicate order, carried this far, goes quite beyond present quantum mechanics. It actually deals with process, which quantum mechanics does not, except by reference to an observing apparatus that in turn has to be referred to something else.

Sheldrake: Would you say that process at that level is a re-projection?

Bohm: Yes.

Sheldrake: And a re-injection at the same time?

Bohm: Re-injection is exactly what the Schrödinger equation is describing. And re-projection is the next step, which quantum mechanics doesn't handle (except by the arbitrary assumption that the wave function "collapses" in a way that has no place in the physical laws, such as Schrödinger's equation).

Now, there's one other thing that modern quantum mechanics doesn't handle. Oddly enough, physics at present has no contact with the notion of actuality. You see, classical physics has at least some notion of actuality in saying that actuality consists of a whole collection of particles that are moving and interacting in a certain way. Now, in quantum physics, there is no concept of actuality whatsoever, because quantum physics maintains that its equations don't describe anything actual, they merely describe the

probability of what an observer could see if he had an instrument of a certain kind, and this instrument is therefore supposed to be necessary for the actuality of the phenomenon. But the instrument, in turn, is supposed to be made of similar particles, obeying the same laws, which would, in turn, require another instrument to give them actuality.

That would go on an infinite regress. Wigner has proposed to end the regress by saying it is the consciousness of the actual observer that gives actuality to everything.

Sheldrake: But that doesn't seem very satisfactory to me.

Bohm: Nor to me, but apparently Wigner feels happy with this, as do some others. The point is, unless you extend quantum mechanics, there is no room in it for actuality, no room for any of the things you are talking about. So quantum mechanics as it stands now, I want to say, is a very truncated, limited, abstracted set of formulae that gives certain limited results having to do with only one moment of an experiment. But out of this truncated view, physicists are trying to explain everything, you see; the whole thing simply has no meaning at all. Think about it: modern physics can't even talk about the actual world!

Sheldrake: But how do you think we can get to a concept of actuality?

Bohm: Well, I think through the implicate order. We have a projection of the whole to constitute a moment; a moment is a movement. And we can say that that projection is the actualization. In other words, the thing that physics doesn't discuss is how various successive moments are related, and that's what I say the implicate order is attempting to do. If we extended quantum mechanics through the implicate order, we would bring in just that question of how past moments have an effect on the present (i.e., via injection and re-projection). At present, physics says the next moment is entirely independent, but with some probability of being such and such. There's no room in it for the sort of thing you're talking about, of having a certain accumulated effect of the past; but the implicate order extension of quantum mechanics would have that possibility. And further, suppose somehow I were to combine the implicate order extension of quantum mechanics [*which would account for the accumulated effects of the past*]

with this quantum potential [*which would account for these effects being non-local in nature*], then I think I would get things very like what you are talking about.

Sheldrake: Yes, that would be very exciting! Of all the ways I've come across, I think that's the most promising way of being able to mesh together these sort of ideas. I haven't come across any other way that seems to show such possible connections.

Bohm: If we can bring in time, and say that each moment has a certain field of potentials (represented by the Schrödinger equation) and also an actuality, which is more restricted (represented by the particle itself); and then say that the next moment has its potential and its actuality, and we must have some connection between the actuality of the previous moments and the *potentials* of the next—that would be introjection, not of the wave function of the past, but of the actuality of the past into that field from which the present is going to be projected. That would do exactly the sort of thing you're talking about. Because then you could build up a series of actualities introjected that would narrow down the field potential more and more, and these would form the basis of subsequent projections. That would account for the influence of the past on the present.

Sheldrake: Yes, yes. Now how do you think this ties in with the alleged matter waves in de Broglie's equation?

Bohm: That's exactly where we started. These matter waves are the formative cause, and that was what de Broglie originally suggested. However, he wanted to regard the matter wave as just simply a real three-dimensional wave in time, and that doesn't work well. The formative field is a far better interpretation. The quantum potential is the formative field that we derive from the generalized de Broglie waves. And we say that the particle is the actuality, affected by the formative field. The set of particles, the whole structure of all the particles forming a system, is the actuality of that formative field.

But that model by itself still ignores time, so the next step is to bring in time, to say that there's a succession of moments of time in which there is a recurrent actuality. And we would say that what recurs is affected by the

formative field. But then that formative field is affected by what has previously happened, actually. Now, that would help to remove most of the problems in physics, if we can manage it. And it would tie up closely with the sort of thing that you're talking about.

See, at present we say that the wave function as potential spreads out very fast and then it suddenly collapses into some definite actual state for reasons totally outside the theory. So we say it requires a piece of measuring apparatus to do so. Then another collapse, and the only continuity of this system would be achieved by an infinite set of measuring apparatuses that would keep it in observation all the time, and these observation apparatuses in turn would have to be observed to allow them to exist actually, and so on. And the whole thing vanishes in a fog of confusion. Because people take the present mathematics as sacred, they say these equations in their general form are never to be altered, and then they say here we are with all these strange problems. But you see almost no one wants to introduce anything fundamentally different into this general framework.

Sheldrake: So the de Broglie interpretation is the way you're thinking of developing. You'd have this recurrent actualization of something that is continually associated with the formative field.

Bohm: And the present formative field is affected by past actualizations. In the present quantum mechanics there is no way to have the formative field affected by anything at all, including the past, because there's only one moment that you can talk about. You can't find anything that would affect the formative field, and that's the problem.

Sheldrake: Yes, I see. Now this is a closely related topic: what I'm talking about with morphogenetic fields has to do with physical forms and habitual patterns of behavior. The connection of these ideas to the thought process itself is not obvious, although they're certainly related. If you start framing the whole topic in physical terms, as I do with morphogenetic fields, then you have to speak in terms of morphic resonance, the influence of past forms on present ones through the morphogenetic field by a kind of resonance. If, however, you start using psychological language, and you start talking in terms of thought, then you've got a handier way of thinking

of the influence of the past, because with mental fields you have memory. And one can extend this memory if one thinks of the whole universe as essentially thoughtlike, as many philosophical systems have done. You could say that if the whole universe is thought-like, then you automatically have a sort of cosmic memory developing. There are systems of thought that take exactly this view. One of them is a Mahayana Buddhist system—the idea of the Alayavijnana, store consciousness, is rather similar to the idea of cosmic memory. And the Theosophists I think took over some of that in the idea of the Akashic record. The entire universe is, in one school of Hindu thought, Vishnu's dream. Vishnu dreams the universe into being—it has the same kind of reality as a dream, and because Vishnu is a long-lasting god, who goes on dreaming for a long time, it retains a certain consistency. There's memory within that dream; what he dreamed about in the past tended to repeat itself, having its own laws and dynamics. All of those systems of thought have memory built into them. So you could phrase the whole thing in psychological language. But that doesn't really help to make much contact with modern physics and our modern scientific way of looking at the world. So, in a sense, notions like the implicate order seem to be a better way of approaching the problem, because implicate order is neutral in connotation. It is something that can underlie both physical reality and thought. So it transcends the usual materialist-idealist dichotomy, which says either all of reality is thoughtlike or all of reality is matterlike. The implicate order idea has the big advantage of transcending that distinction.

Bohm: In fact its very essence is that transcendence.

Sheldrake: If we take a broader view of creativity, we have the idea of the overall evolutionary process; now that's clearly a creative process. How do you think that kind of evolutionary creativity is related to this model?

Bohm: You could speculate that a great deal of life is the constant replication of forms that are given with small variations, and that's similar to our experience of thought: a constant replication of pattern within variation. But then we wonder, "How does it ever come about that we get variations—that we get beyond that pattern?"

Sheldrake: Yes, creative “jumps.”

Bohm: “Jumps”—yes; you see we call it “jumps” when it’s projected into the fixed categories of thought. If you were to say that there’s a proto-intelligence or implicit intelligence in matter as it evolves, that it’s actually not moving causally in a sequence but is constantly created and replicated, then there is room for such a creative act to occur, and to project and introject a creative content.

Sheldrake: The thing that’s involved in this creativity seems to be something that links things together, a wholeness that embraces parts and sets up relationships between them. They’re linked together within a new whole that didn’t exist before. In this creative realization, two previously separate things have been linked together within a whole.

Bohm: Yes. They’re now seen as mere aspects of the whole rather than independent existences. You have realized a new whole, and from that realization you may create an external reality as well.

Sheldrake: So the creative process, which gives rise to new thought, through which new wholes are realized, is similar in that sense to the creative reality that gives rise to new wholes in the evolutionary process. The creative process could be seen as a successive development of more-complex and higher-level wholes, through previously separate things being connected together.

Bohm: And being realized now as not only independent parts but also aspects of a greater whole that has new qualities.

Sheldrake: Right, and that realization of a greater whole is what actually creates the greater whole—

Bohm: Yes, and it could even propose it, as in imagination, or a flash of insight, you realize the whole in the mind and you further realize it outside by work. So you might suppose, say, that somehow nature realizes that it’s being presented with various things that now have to be brought together. Nature realizes this greater whole at a deeper level, which is analogous to

imagination, and then it unfolds it into the external environment. In a way, a flash of creative insight occurs in the biological system.

Sheldrake: Exactly. Now do you think that these relations between things that make them part of the greater whole could, way back in time, have given rise to the fundamental forces of physics? For example, could the gravitational forces that link together all matter have arisen through an original creative insight that all matter was one?

Bohm: One could say that in bringing together various things that previously had been disparate, suddenly there was a realization of their oneness and this created a new whole that is the universe, as we know it, anyway. We can say that nature has an intent, you see, that is much deeper than what appears on the surface.

Sheldrake: Now, as to whether natural laws are eternally given or whether they are gradually built up—how do you see that?

Bohm: I think, in view of the implicate order, that the notion of formative fields gradually becoming necessary is what is called for. Even modern physics is pointing to that idea by saying there was a time (i.e., prior to the big bang) before any of these units (molecules, quarks, atoms), on which we are basing the necessity, even existed. So, if you said there were certain fixed and everlasting laws of the molecules and atoms, then what would you say if you traced it back to the time before the atoms and molecules existed? Physics can say nothing about that, right? It can say only that there was a formation of these particles at a certain stage. So there would have to be an actual development in which the necessity in a certain field grew more and more fixed. You can even see that happening as you cool down a substance that liquefies; at first you get little clumps of liquid that are transient, and then they get bigger and more determinate. Now physicists explain all this by saying that the laws of the molecules are eternal; molecules are merely consequences of those laws, or derived from those laws. But if you follow that back and ask, “Where were molecules?”: well, they were originally protons and electrons, which were originally quarks, which were originally sub-quarks. And it goes right back to a stage where none of the units we know even existed, so the whole scheme sort of fades

out. It's then open to you to say that, in general, fields of necessity are not eternal; they are constantly forming and developing.

Sheldrake: I think that the current conventional and scientific picture hasn't really faced up to this at all. You see, science started with a sort of neo-Platonic, neo-Pythagorean notion—the idea of timeless laws— which has been taken for granted in science for a very long time. I think that when the evolutionary theory in biology came in, it triggered the beginning of change. We then had an evolutionary view of reality regarding animals and plants, but it was still considered that there was a timeless background of the physical world, the molecular and atomic world. Now we've gone to the cosmology of the big bang, which is widely accepted. So now we've got the idea of the entire universe as being a radically evolutionary universe. And this, I think, provokes a crisis, and should provoke a crisis. The idea of timeless laws that have always been there, somehow pervading space and time, ceases to have much meaning when you have an actual historical big bang, because you then have this problem: where were the laws before the big bang?

Bohm: There is also the belief, commonly accepted, that at the core of black holes the laws as we know them would also vanish. As you say, scientists haven't faced up to it because they are still thinking in the old way, in terms of timeless laws. But some physicists realize that. One cosmologist was giving a talk and he said, "Well, you know, I used to think everything was a law of nature, and it's all fixed, but as far as a black hole is concerned, anything can happen. You see, if it suddenly flashed a Coca-Cola sign, this would still be a possibility." [*Laughter*.] So, the notion of timeless laws doesn't seem to hold, because time itself is part of the necessity that developed. The black hole doesn't involve time and space as we know it; they all vanish. It's not just matter that vanishes, but any regular order that we know of vanishes, and therefore you could say anything goes, or nothing goes.

Sheldrake: The interesting thing about the big bang theory is that the minute you have to address the question of the origins of the laws of nature, you're forced to recognize the philosophical assumptions underlying any sort of science. People who think of themselves as hard-nosed mechanists

or pragmatists regard metaphysics as a waste of time, a useless speculative activity, whereas supposedly they are practical scientists getting on with the job. But you can force them to realize that their view of the laws of nature as being timeless, which is implicit in everything they say or think or do, is in fact a metaphysical view. And it's one possible metaphysical view, it's not the only possible one. I talk with biological friends, and they say, "Oh, what you're doing is metaphysics." So I say, "Wait a minute, let's look at what you're doing." And then you confront them with the question of where were the laws of nature before the big bang. And most of them say, "Well, they must have always been there." And you say, "Where? There's no matter in any sense that we know of before the big bang. Where were these laws of nature, sort of free-floating?" And they say, "Well, they must have been there somehow." And then you say, "Don't you think this is a rather metaphysical concept, in any literal sense of metaphysics, because it's quite beyond existing physics?" They have to admit it sooner or later. As soon as you get into that sort of area, the certainty that so many scientists think their view of the world is founded on simply disappears. It becomes clear that current science presupposes uncritically one possible kind of metaphysics. When one faces this, one can at least begin to think about it rather than accepting one way of thinking about it as self-evident, taken for granted. And if one begins to think about it, one might be able to deepen one's understanding of it.

ENDNOTES

Preface to the 2009 Edition

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Introduction

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Chapter 1. The Unsolved Problems of Biology

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7 G. Wolff, *Mechanismus and Vitalismus* (Leipzig, Germany: 1902).

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12 M. Ricard, *The Mystery of Animal Migration* (London: Constable, 1969).

13 C. Darwin, *The Variation of Animals and Plants Under Domestication*, vol. 2 (London, UK: John Murray, 1905).

14 B. Rensch, *Evolution above the Species Level* (London: Methuen, 1959); E. Mayr, *Animal Species and Evolution* (Cambridge, Mass.: Harvard University Press, 1963); G. L. Stebbins, *Flowering Plants: Evolution above the Species Level* (Cambridge, Mass.: Harvard University Press, 1974).

15 R. Goldschmidt, *The Material Basis of Evolution* (New Haven, Conn.: Yale University Press, 1940); J. C. Willis, *The Course of Evolution* (Cambridge: Cambridge University Press, 1940); S. M. Stanley, *Macroevolution: Pattern and Process* (San Francisco: W. H. Freeman, 1981).

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18 M. Eigen and P. Schuster, *The Hypercycle* (Heidelberg, Germany, and New York: Springer-Verlag, 1979).

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22 E. Wigner, "Remarks on the Mind-Body Question," in *The Scientist Speculates*, I. J. Good, ed. (London: Heinermann, 1961); E. Wigner, "Epistemology in Quantum Mechanics," in *Contemporary Physics. Trieste Symposium 1968*, vol. II (Vienna, Austria: International Atomic Energy Authority, 1969), 431–38; R. Penrose, *The Emperor's New Mind:*

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23 P. Churchland, *A Neurocomputational Perspective: The Nature of Mind and the Structure of Science* (Cambridge, Mass.: MIT Press, 1992).

24 M. Pauen, A. Staudacher, and S. Walter, "Epiphenomenalism: Dead End or Way Out?" *Journal of Consciousness Studies* 13 (2006): 7–19.

25 A. Hyslop, "Methodological Epiphenomenalism," *Australasian Journal of Philosophy* 76 (1998): 61–70.

26 E.g., J. B. Watson, *Behaviorism* (Chicago: Chicago University Press, 1924); B. F. Skinner, *The Behaviour of Organisms* (New York: Appleton Century, 1938); D. E. Broadbent, *Behaviour* (London: Eyre and Spottiswoode, 1961).

27 For critical discussions, see J. Beloff, *The Existence of Mind* (London: MacGibbon and Kee, 1962); A. Koestler, *The Ghost in the Machine* (London: Hutchinson, 1967); and K. R. Popper and J. C. Eccles, *The Self and its Brain* (Berlin: Springer International, 1977).

28 E.g., A. Damasio, *Descartes' Error: Emotion, Reason and the Human Brain* (New York: Putnam, 1994).

29 F. Varela, E. Thompson, and E. Risch, *The Embodied Mind: Cognitive Science and Human Experience* (Cambridge, Mass.: MIT Press, 1991).

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31 C. G. Jung, *The Archetypes and the Collective Unconscious* (London: Routledge and Kegan Paul, 1959), 43.

32 Ibid., 75.

33 R. Sheldrake, *The Sense of Being Stared At, And Other Aspects of the Extended Mind* (New York: Crown, 2003).

34 C. Carter, *Parapsychology and the Skeptics* (Pittsburgh, Pa.: Sterling House, 2007).

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and R. Radin, *The Conscious Universe: The Scientific Truth of Psychic Phenomena* (San Francisco: Harper, 1997).

36 R. H. Thouless, *From Anecdote to Experiment in Psychical Research* (London: Routledge and Kegan Paul, 1972); D. Radin, *Entangled Minds: Extrasensory Experiences in a Quantum Reality* (New York: Pocket Books, 2006); R. Radin, *The Conscious Universe: The Scientific Truth of Psychic Phenomena* (San Francisco: Harper, 1997); R. Sheldrake, *The Sense of Being Stared At, And Other Aspects of the Extended Mind* (New York: Crown, 2003).

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Chapter 2. Three Theories of Morphogenesis

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3 L. Wolpert, "Pattern Formation in Biological Development," *Scientific American* 239, no. 4 (1978): 154–64.

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5 M. V. Olsen and A. Varki, “The Chimpanzee Genome—A Bittersweet Celebration,” *Science* 305 (2004): 191–92.

6 Ibid.

7 H. K. MacWilliams and J. T. Bonner, “The Prestalk–Prespore Pattern in Cellular Slime Moulds,” *Differentiation* 14 (1979): 1–22.

8 R. Sheldrake, “The Production of Hormones in Higher Plants,” *Biological Reviews* 48 (1973): 509–59.

9 For a theoretical discussion of this problem, see H. Meinhardt, “Space-dependent Cell Determination under the Control of a Morphogen Gradient,” *Journal of Theoretical Biology* 74 (1978): 307–21.

10 K. Roberts and J. S. Hyams, eds. *Microtubules* (London: Academic Press, 1979).

11 G. Nicolis and I. Prigogine, *Self-Organization in Nonequilibrium Systems* (New York: Wiley-Interscience, 1977).

12 S. Kauffman, *Origins of Order: Self-Organization and Selection in Evolution* (Oxford, U.K.: Oxford University Press, 1994).

13 T. H. Huxley, *Hardwicke’s Science Gossip* 3 (1867): 74.

14 In Driesch, *History and Theory of Vitalism* (London: Macmillan, 1914), 119.

15 H. Driesch *Science and Philosophy of the Organism* (London: A. & C. Black, 1908, 2nd ed., 1929), 290.

16 Ibid., vol. 1, 203.

17 Ibid., 152–24, 293.

18 Ibid., 135, 291.

19 Ibid., 246.

20 Ibid., 103.

21 Ibid., 246.

22 Ibid., 266.

23 Ibid., 262.

24 A. Eddington, *The Nature of the Physical World* (London: Dent, 1935), 302.

25 J. C. Eccles, *The Neurophysiological Basis of Mind* (Oxford: Oxford University Press, 1953).

26 E.g., E. H. Walker, "Foundations of Paraphysical and Parapsychological Phenomena," in *Quantum Physics and Parapsychology*, ed. L. Otera (New York: Parapsychology Foundation, 1975); J. H. M. Whiteman, "Parapsychology and Physics, in *Handbook of Parapsychology*, ed. B. B. Wolman (New York: Van Nostrand Reinhold, 1977); J. B. Hasted, "Speculations about the Relation between Psychic Phenomena and Physics," *Psychoenergetic Systems* 3 (1978): 243–57; D. F. Lawden, "Possible Psychokinetic Interactions in Quantum Theory," *Journal of the Society for Psychical Research* 50 (1980): 399–407.

27 For a discussion of these influences, and an account of the subsequent development of organismic ideas, see Haraway (1976). The best early summary of the organismic approach to morphogenesis is by von Bertalanffy (1933).

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29 For a systematic statement of Weiss's ideas, see his *Principles of Development.*

30 C. H. Waddington, *The Strategy of the Genes* (London: Allen & Unwin, 1957), chap. 2.

31 R. Thom, *Structural Stability and Morphogenesis* (Reading, Mass.: Benjamin, 1975a).

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33 R. Abraham and C. D. Shaw, *Dynamics: The Geometry of Behavior* (Santa Cruz, Calif.: Aerial Press, 1984).

34 R. Thom, *Mathematical Models of Morphogenesis* (New York: Wiley, 1983).

35 Waddington did not even make explicit the organismic background of his concepts, for the reason explained in the following passage, which was written toward the end of his career: "Since I am an unaggressive

character, and was living in an aggressively anti-metaphysical period, I chose not to expound publicly these philosophical views. An essay I wrote around 1928 on “The Vitalist-Mechanist Controversy and the Process of Abstraction” was never published. Instead I tried to put the Whiteheadian outlook to use in particular experimental situations. So biologists uninterested in metaphysics do not notice what lies behind—though they usually react as though they feel obscurely uneasy.” *Toward a Theoretical Biology. 2: Sketches*, ed. C. H. Waddington (Edinburgh: Edinburgh University Press, 1969), 72–81.

36 In C. H. Waddington, ed., *Toward a Theoretical Biology. 2: Sketches* (Edinburgh, UK: Edinburgh University Press, 1969), 238, 242.

37 W. M. Elsasser, *Atom and Organism* (Princeton, N.J.: Princeton University Press, 1966); W. M Elsasser, *The Chief Abstractions of Biology* (Amsterdam: North-Holland, 1975); L. von Bertalanffy, *General Systems Theory* (London: Allen Lane, 1971). For a discussion of this mechanistic organicism, see Sheldrake, “Three approaches to biology. III. Organicism.”

38 B. C. Goodwin, “On Morphogenetic Fields,” *Theoria to Theory* 113 (1979): 112–13. See also B. C. Goodwin, *How the Leopard Changed Its Spots: The Evolution of Complexity* (London: Weidenfeld and Nicholson, 1994).

39 S. B. Carroll, J. K. Grenier, and S. D. Weatherbee, *From DNA to Diversity: Molecular Genetics and the Evolution of Animal Design* (Oxford: Blackwell, 2001), 47.

40 J. A. Bolker, “Modularity in Development and Why It Matters to Evo-devo,” *American Zoologist* 40 (2000): 770–76.

41 S. F. Gilbert, J. M. Opik, and R. A. Raff, “Resynthesizing Evolutionary and Developmental Biology,” *Developmental Biology* 173 (1996): 357–72.

Chapter 3. The Causes of Form

1 An excellent introduction to the problem of organic form is provided by E. W. Sinnott, *The Problem of Organic Form* (New Haven, Conn.: Yale University Press, 1963).

2 For a discussion of this problem, see R. Thom, *Structural Stability and Morphogenesis* (Reading, Mass.: Benjamin, 1975b).

3 Ibid., 320.

4 R. Thom, “D’un modele de la science a une science des modeles,” *Synthese* 31 (1975): 359–74.

5 For a discussion of the limited relevance of information theory to biology, see C. H. Waddington, *The Evolution of an Evolutionist* (Edinburgh: Edinburgh University Press, 1975), 209–30.

6 Some mathematicians make it explicit, as in R. Penrose, *The Emperor’s New Mind: Concerning Computers, Minds and the Laws of Physics* (Oxford, U.K.: Oxford University Press, 1989).

7 Numerous examples of the combination of aspects of the organismic philosophy with explicitly neo-Platonic speculation are provided in *La Gnose de Princeton* (Paris: Fayard, 1974), in Ruyer’s account of a small neo-gnostic group in the United States whose members include a number of prominent scientists.

8 See Emmet, D., *Whitehead’s Philosophy of Organism* (London: Macmillan, 1966).

9 L. Pauling, *The Nature of the Chemical Bond*, 3rd ed. (Ithaca, N.Y.: Cornell University Press, 1960), 220.

10 T. Helgaker, et al., “A Priori Calculation of Molecular Properties to Chemical Accuracy, *Journal of Physical Organic Chemistry* 17 (2004): 913–33.

11 L. Pauling, *The Nature of the Chemical Bond*, 3rd ed. (Ithaca, N.Y.: Cornell University Press, 1960), 543.

12 J. Maddox, “Crystals from First Principles,” *Nature* 335 (1988): 201.

13 J. D. Dunitz and H. A. Scheraga, “Exercises in Prognostication: Crystal Structures and Protein Folding, *Proceedings of the National Academy of Sciences USA* 101 (2004): 14, 309–11.

14 Ibid.

15 K. Sanderson, “Model Predicts Structure of Crystals,” *Nature* 450 (2007): 771.

16 C. B. Anfinsen and H. A. Scheraga, “Experimental and Theoretical Aspects of Protein Folding, *Advances in Protein Chemistry* 29 (1975): 205–300.

17 For a review, see Nemethy and Scheraga, “Protein Folding.”

18 6th Community Wide Experiment on the Critical Assessment of Techniques for Protein Structure Prediction, Categories of Prediction (CASP1–6), see www.predictioncenter.org/casp6/doc/categories.html.

19 J. D. Dunitz and H. A. Scheraga, “Exercises in Prognostication: Crystal Structures and Protein Folding, *Proceedings of the National Academy of Sciences USA* 101 (2004): 14, 309–11.

20 C. B. Anfinsen and H. A. Scheraga, “Experimental and Theoretical Aspects of Protein Folding, *Advances in Protein Chemistry* 29 (1975): 205–300.

21 Cf. the “principle of finite classes” in Elsasser, *The Chief Abstractions of Biology.*

22 This distinction between formative causation and energetic causation resembles Aristotle’s distinction between “formal causes” and “efficient causes.” However, the hypothesis of formative causation developed in the following chapters differs radically from Aristotle’s theory, which presupposed eternally given forms.

23 From a theoretical point of view, the causal role of morphogenetic fields can be analyzed in terms of “counterfactual conditionals.” For a discussion of the latter, see, for example, J. L. Mackie, *The Cement of the Universe* (Oxford, U.K.: Oxford University Press, 1974).

24 Arthur Koestler has suggested the use of the term *holon* for such “self-regulating open systems which display both the autonomous properties of wholes and the dependent properties of parts.” *Beyond Reductionism*, eds. A. Koestler and J. R. Smythies (London: Hutchinson, 1969), 210–11. This term is wider in its application than the term *morphic unit*—it included, for example, linguistic and social structures—but it represents a very similar concept.

Chapter 4. Morphogenetic Fields

1 The identification of morphogenetic fields with electromagnetic fields is responsible for much of the confusion inherent in H. S. Burr's theory of electrodynamic "life fields." In *Blueprint for Immortality* (London: Neville Spearman, , Burr cites indisputable evidence that living organisms are associated with electromagnetic fields, which change as the organisms change, but then goes on to argue that these fields control morphogenesis by acting as "blueprints" for development, which is a very different matter.

2 For a review of the literature on conformational changes in proteins in solution, see R. J. P. Williams, "The Conformational Properties of Proteins in Solution," *Biological Reviews* 54 (1979): 389–437.

3 C. B. Anfinsen, "Principles That Govern the Folding of Protein Chains," *Science* 181 (1973): 228.

4 For a general discussion of probabilistic causality, see P. Suppes, *A Probabilistic Theory of Causality* (Amsterdam: North-Holland, 1970).

5 Cf. Karl Popper's concept of probability or propensity fields. K. R. Popper, "Quantum Mechanics without 'the Observer,'" in *Quantum Theory and Reality*, ed. M. Bunge (Berlin: Springer-Verlag, 1967); K. R. Popper and J. C. Eccles, *The Self and Its Brain* (Berlin: Springer International, 1977)

6 This suggestion might fit in with the approach to quantum physics advocated by D. Bohm in "Some Remarks on the Notion of Order," *Towards a Theoretical Biology 2: Sketches*, Waddington, ed. (Edinburgh: Edinburgh University Press, 1969), and *Wholeness and the Implicate Order* (London: Routledge and Kegan Paul, 1980), and by B. J. Hiley in "Toward an Algebraic Description of Reality," *Annales de la Foundation Louise de Broglie* 5 (1980): 75–103.

7 This and other instances of what Thom (1975a) calls "generalized catastrophes" are discussed in chapter 6 of his *Structural Stability and Morphogenesis.*

8 W. A. Bentley and W. J. Humphreys, *Snow Crystals* (New York: Dover, 1962).

9 See G. Nicolis and I. Prigogine, *Self-Organization in Nonequilibrium Systems* (New York: Wiley-Interscience, 1977). A different but related

approach to these problems is outlined by H. Haken in *Synergetics* (Berlin: Springer-Verlag, 1977).

10 C. F. Stevens, “Study of Membrane Permeability Changes by Fluctuation Analysis,” *Nature* 270 (1977): 391–96.

11 Chaos theory provides models for some kinds of formative processes in terms of “strange attractors,” according to J. Gleik, *Chaos: Making a New Science* (London: Heinemann, 1988).

12 For a discussion of Thompson’s theories, see Medawar, *The Art of the Soluble.*

13 For recent accounts of the properties and functions of microtubules, see P. Dustin, *Microtubules* (Berlin: Springer-Verlag, 1978), and K. Roberts and J. S. Hyams, eds., *Microtubules* (London: Academic Press, 1979).

14 One suggestion is that the smooth endoplasmic reticulum plays a role in the transport of microtubule subunits to the regions in which they aggregate, as in J. Burgess and D. H. Northcote, “The Relationship between the Endoplasmic Reticulum and Microtubular Aggregation and Disaggregation,” *Planta* 80 (1968): 1–14. The existence of “nucleating elements” that may or may not be bound together in “microtubule organizing centers” has also been suggested; see U. B. Tucker in *Microtubules,* K. Roberts and J. S. Hyams, eds. (London: Academic Press, 1979).

15 H. E. Street and G. G. Henshaw, “Introduction and Methods Employed in Plant Tissue Culture,” in *Cells and Tissues in Culture*, vol. 3, ed. E. N. Willmer (London: Academic Press, 1965), 459–532.

16 For examples, see E. N. Willmer, *Cytology and Evolution,* 2nd ed. (London: Academic Press, 1970).

17 In some instances, the nuclei are destroyed in the final stages of differentiation (e.g., xylem vessels in plants, red blood cells in mammals). In these cases, the nuclei could act as morphogenetic germs for the differentiation process up to the point at which they were still intact; then the final stages of differentiation could proceed purely mechanistically by straightforward chemical processes not subject to morphogenetic ordering, through the release of hydrolytic enzymes.

18 In some algae (e.g., *Oedogonium*), the nuclear membrane remains intact during mitosis; this is probably an evolutionarily primitive feature, as noted in J. D. Pickett-Heaps, *Green Algae* (Sunderland, Mass.: Sinauer Associates, 1975).

19 J. D. Pickett-Heaps, *Green Algae* (Sunderland, Mass.: Sinauer Associates, 1969).

20 F. A. L. Clowes, *Apical Meristems* (Oxford, U.K.: Blackwell, 1961).

21 L. Wolpert, "Pattern Formation in Biological Development," *Scientific American* 239 (4) (1978): 154–64.

Chapter 5. The Influence of Past Forms

1 J. L. Mackie, *The Cement of the Universe* (Oxford, U.K.: Oxford University Press, 1974), 19.

2 M. B. Hesse, *Forces and Fields* (London: Nelson, 1961), 285.

3 Many examples of oscillations within biological systems have been described. See, for example, the review of oscillations at the cellular level in P. E. Rapp, "An Atlas of Cellular Oscillations," *Journal of Experimental Biology* 81 (1979): 281–306.

4 The vibration of a system brought about by a "one-dimensional" energetic stimulus can in fact give rise to definite forms and patterns: simple examples are the Chladni figures produced by sand or other small particles on a vibrating diaphragm. Illustrations of numerous two-and three-dimensional patterns on vibrating surfaces can be found in H. Jenny, *Cymatics* (Basel, Switzerland: Basileus Press, 1967), and A. Lauterwasser, *Water Sound Images* (Newmarket, N.H.: Macromedia Publishing, 2006). However, this effect is not comparable to the type of morphogenesis brought about through morphic resonance.

5 For discussions of the possibility of causal influences from future events, see M. B. Hesse, *Forces and Fields* (London: Nelson, 1961), and J. L. Mackie, *The Cement of the Universe* (Oxford, U.K.: Oxford University Press, 1974).

6 Evidence for precognition would be relevant to this argument only if mental states were assumed, on metaphysical grounds, to be an aspect of physical states of the body, to run parallel to them, or to be

epiphenomena of them. However, from the point of view of interactionism, an influence from future *mental* states would not necessarily require a *physical* influence to pass "backward" in time. These metaphysical alternatives are discussed further in chapter 12.

7 G. D. Woodard and W. C. McCrone, "Unusual Crystallization Behavior," *Journal of Applied Crystallography* 8 (1975): 342.

8 Ibid.

9 A. Holden and P. Singer, *Crystals and Crystal Growing* (London: Heinemann, 1961), 80–81.

10 Ibid., 81.

11 G. D. Woodard and W. C. McCrone, "Unusual Crystallization Behavior," *Journal of Applied Crystallography* 8 (1975): 342.

12 A. Goho, "The Crystal Form of a Drug Can Be the Secret of Its Success, *Science News* 166 (2004): 122–24.

13 J. Bernstein, *Polymorphism in Molecular Crystals* (Oxford: Clarendon Press, 2002), 90.

14 Quoted in G. D. Woodard and W. C. McCrone, "Unusual Crystallization Behavior," *Journal of Applied Crystallography* 8 (1975): 342.

15 P. V. Danckwerts, letter, *New Scientist*, November 11, 1982, 380–81.

Chapter 6. Formative Causation and Morphogenesis

1 It seems probable that an important cause of aging, at least at the cellular level, is the buildup of harmful waste products that cells are unable to eliminate. If cells grow fast enough, they can keep "one step ahead" of this buildup simply because these substances are diluted by growth. Furthermore, in asymmetric cell divisions, which are common in higher animals and plants, these substances may be passed on unequally to the daughter cells: one may be rejuvenated at the expense of the increased mortality of the other. Thus, rejuvenation depends on growth and cell division: morphogenetic end points—the differentiated cells, tissues, and organs of multicellular organisms—are necessarily mortal, as noted in Sheldrake, "The Ageing, Growth and Death of Cells."

2 For animal examples, see P. Weiss, *Principles of Development* (New York: Holt, 1939); for plants, see C. W. Wardlaw, *Organization and Evolution in Plants* (London: Longmans, 1965).

3 The classic discussion of this elementary but important point is in the chapter titled “On Magnitude” in D’Arcy W. Thompson, *On Growth and Form* (Cambridge, U.K.: Cambridge University Press, 1942).

4 If the system “identifies” itself with a particular location and if its persistence at that location depends on morphic resonance with itself in the immediate past, its resistance to being moved from that location—its *inertial mass*—should be related to the frequency with which this self-resonance occurs, for resonance depends on characteristic cycles of vibration; it cannot occur in an instant because a cycle of vibration takes time. The higher the frequency of vibration, the more recent will be the past states with which self-resonance occurs; thus, the greater will be the tendency of the system to be “tied” to its position in the immediate past. Conversely, the lower the frequency of vibration, the less strong will be the tendency of a system to “identify” itself with itself at a particular location: it will be able to move farther relative to other objects before it “notices” that it has done so.

5 Karl Popper, among others, has argued that talking of a dualism of particle and wave has led to much confusion, and has suggested that the term *dualism* should be abandoned: “I propose that we speak instead (as did Einstein) of the particle and its *associated* propensity fields (the plural indicates that the fields depend not only on the particle but also on other conditions), thus avoiding the suggestion of a symmetrical relation. Without establishing some such terminology as this (‘association’ in place of ‘dualism’), the term *dualism* is bound to survive, with all the misconceptions connected with it; for it does point to something important: the association that exists between particles and fields of propensities.” K. R. Popper, “Quantum Mechanics without ‘the Observer,’” in *Quantum Theory and Reality*, ed. M. Bunge (Berlin: Springer-Verlag, 1967), 41. This proposal would appear to harmonize well with the hypothesis of formative causation if propensity fields are taken to include morphogenetic fields.

1 The classic work on this subject is W. Bateson, *Materials for the Study of Variation: Treated with Especial Regard to Discontinuity in the Origin of Species* (London: Macmillan, 1894).

2 G. Morata and P. A. Lawrence, "Homoeotic Genes, Compartments and Cell Determination in *Drosophila*," *Nature* 265 (1977): 211–16.

3 B. Snoad, "A Preliminary Assessment of 'Leafless Peas,' *Euphytica* 23 (1974): 257–65.

4 S. B. Carroll, J. K. Grenier, and S. D. Weatherbee, *From DNA to Diversity: Molecular Genetics and the Evolution of Animal Design* (Oxford, U.K.: Blackwell, 2001).

5 D. Bourguet, "The Evolution of Dominance," *Heredity* 83 (1999): 1–4.

6 R. A. Fisher, *Genetical Theory of Natural Selection* (London: Clarendon Press, 1930).

7 J. B. S. Haldane, "The Theory of the Evolution of Dominance," *Journal of Genetics* 37 (1939): 365–74.

8 J. A. Serra, *Modern Genetics*, vol. II (London: Academic Press, 1966).

9 J. M. Baldwin, *Development and Evolution* (New York: Macmillan, 1902).

10 Much of this evidence is summarized in R. Semon, *Das Problem der Vererbung Erworbener Eigenschaften* (Leipzig: Engelmann, 1912), and P. Kammerer, *The Inheritance of Acquired Characteristics* (New York: Boni and Liveright, 1924).

11 A. Koestler, *The Case of the Midwife Toad* (London: Hutchinson, 1971).

12 Z. A. Medvedev, *The Rise and Fall of T. D. Lysenko* (New York: Columbia University Press, 1969).

13 A. Durrant, "The Association of Induced Changes in Flax," *Heredity* 32 (1974): 133–43.

14 H. D. Morgan, H. G. E. Sutherland, D. I. K. Martin, and E. Whitelaw, "Epigenetic Inheritance at the Agouti Locus in the Mouse," *Nature Genetics* 23 (1999): 314–18.

15 M. D. Anway, A. S. Cupp, M. Uzumcu, and M. K. Skinner, "Epigenetic Transgenerational Actions of Endocrine Disruptors and Male Fertility,"

Science 308 (2005): 1466–69; and R. H. Ashby, *The Guidebook for the Study of Psychical Research* (London: Rider, 1972).

16 E. Young, “Rewriting Darwin: The New Non-genetic Inheritance,” *New Scientist* (9 July 2008).

17 C. Dennis, “Altered States,” *Nature* 421 (2003): 686–88.

18 J. Qiu, “Unfinished Symphony,” *Nature* 441 (2006): 143–45.

19 G. Vines, “Hidden Inheritance,” *New Scientist,* November 28, 1998, 27–30.

20 C. H. Waddington, “Genetic Assimilation of the Bithorax Phenotype,” *Evolution* 10 (1956): 1–13.

21 Ibid., 65.

22 C. H. Waddington, *The Strategy of the Genes* (London: Allen & Unwin, 1957).

23 See the discussion between C. H. Waddington and A. Koestler in Koestler and Smythies, *Beyond Reductionism,* 382–91.

24 C. H. Waddington, *The Evolution of an Evolutionist* (Edinburgh: Edinburgh University Press, 1975), 87.

25 Ibid., 87–88.

26 M. W. Ho, et al., “Effects of Successive Generations of Ether Treatment on Penetrance and Expression of the Bithorax Phenocopy in *Drosophila melanogaster*,” *Journal of Experimental Zoology* 225 (1983): 357–68.

27 See G. C. Gibson and D. S. Hogness, “Effect of Polymorphism in the *Drosophila* Regulatory Gene Ultrabithorax on Homeotic Stability,” *Science* 271 (1996): 200–203.

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3 R. Goldschmidt, *The Material Basis of Evolution* (New Haven, Conn.: Yale University Press, 1940), 267; S. J. Gould, "Return of the Hopeful Monster," in *The Panda's Thumb* (New York: Norton, 1980).

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6 See, for example, J. Monod, *Chance and Necessity* (London: Collins, 1972).

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Chapter 10. Instinct and Learning

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17 C. Loizos, "Play Behaviour in Higher Primates: A Review," in *Primate Ethology,* D. Morris, ed. (London: Weidenfeld and Nicolson, 1967), 203.

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13 W. E. Agar, et al., "Fourth (Final) Report on a Test of McDougall's Lamarckian Experiment on the Training of Rats," *Journal of Experimental Biology* 31 (1954): 307–21.

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15 A number of possible explanations were suggested at the time these experiments were being carried out; they are discussed in McDougall’s papers, to which the interested reader should refer. None of these explanations turned out to be plausible on closer examination. Agar and colleagues noticed that fluctuations in the rates of learning were associated with changes, extending over several generations, in the health and vigor of the rats; see W. E. Agar, et al., “Fourth (final) Report on a Test of McDougall’s Lamarckian Experiment on the Training of Rats,” *Journal of Experimental Biology* 31 (1954): 307–21. McDougall had already noted a similar effect. A statistical analysis showed that there was indeed a low but significant (at the 1 percent level of probability) correlation between vigor (measured in terms of fertility) and learning rates in the “trained” line, but not in the “untrained” line. However, if only the first forty generations were considered, the coefficients of correlation were somewhat higher: 0.40 in the trained line and 0.42 in the untrained. While this correlation may help to account for the fluctuations in the results, it cannot plausibly explain the overall trend. According to standard statistical theory, the proportion of the variation that is “explained” by a correlated variable is given by the square of the correlation coefficient, in this case $(0.4)^2 = 0.16$. In other words, variations in vigor account for only 16 percent of the changes in the rate of learning.

16 McDougall estimated that the average number of errors in his first generation was more than 165. In Crew’s experiment, this figure was 24, and in Agar’s, 72; see the discussions in F. A. E. Crew, “A Repetition of McDougall’s Lamarckian Experiment,” *Journal of Genetics* 33 (1936): 61–101; and W. E. Agar, et al., “Fourth (final) Report on a Test of McDougall’s Lamarckian Experiment on the Training of Rats,” *Journal of Experimental Biology* 31 (1954): 307–21. If Agar’s group had used rats of identical parentage and followed the same procedures as Crew, their initial score might have been expected to be even lower than his. However, owing to the different parentage of their rats and to differences in their testing procedure, the results are not fully comparable.

Nevertheless, the greater facility of learning in these later experiments is suggestive.

17 J. L. Brown, *The Evolution of Behavior* (New York: Norton, 1975).

18 Numerous examples of this type of speculation can be found in Wilson, *Sociobiology: The New Synthesis*, and Dawkins, *The Selfish Gene*.

19 E.g., R. Clarke, "Two Men and Their Dogs," *New Scientist* 87 (1980): 303–4.

20 N. Tinbergen, *The Study of Instinct* (Oxford, U.K.: Oxford University Press, 1951), 201.

21 W. H. Thorpe, *Learning and Instinct in Animals*, 2nd ed. (London: Methuen, 1963); M. Bekoff and J. A. Byers, eds., *Animal Play: Evolutionary, Comparative and Ecological Perspectives* (Cambridge: Cambridge University Press, 1998).

22 R. Dawkins, *The Selfish Gene* (Oxford: Oxford University Press, 1976), 206.

23 Language in particular provides an excellent example of the hierarchical organization of motor fields, and a beginning has already been made by René Thom in developing a theory of language in terms of chreodes; see R. Thom, *Structural Stability and Morphogenesis* (Reading, Mass.: Benjamin, 1975), chap. 6.

24 For a fuller discussion of the role of morphic resonance in cultural inheritance, see my book *The Presence of the Past*, chapters 14 and 15.

Chapter 12. Four Possible Conclusions

1 Some versions of the philosophy of dialectical materialism would probably provide a good starting point for the development of a modified materialism in this sense. They already include many aspects of the organismic approach and are based on the idea that reality is inherently evolutionary; see L. A. Graham, *Science and Philosophy in the Soviet Union* (New York, Knopf, 1972), 332.

2 For a historical account and critical discussion of the various materialist theories, see K. R. Popper, in *The Self and Its Brain*, K. R. Popper and J. C. Eccles, eds. **(**Berlin: Springer International, 1977).

3 The hypothesis that both telepathy and memory might be explicable in terms of a new type of trans-temporal and trans-spatial resonance between similar complex systems has in fact already been put forward by Marshall in “ESP and Memory: A Physical Theory”; indeed, his suggestion anticipates in several important respects the idea of morphic resonance. For a discussion of the role of morphic fields in animal and human telepathy, see my books *Dogs That Know When Their Owners Are Coming Home* and *The Sense of Being Stared At*.

4 Although telepathy and psychokinesis might conceivably be explicable in terms of formative causation, it is difficult to see how this hypothesis could help to account for certain other phenomena, such as clairvoyance, that seem to pose insurmountable problems for any physical theory. For a review of various theories, physical and nonphysical, that have been proposed to account for the phenomena of parapsychology, see K. R. Rao, “On the Nature of Psi,” *Journal of Parapsychology* 41 (1977): 294–351.

5 G. Ryle, *The Concept of Mind* (London: Hutchinson, 1949).

6 E.g., A. Eddington, *The Nature of the Physical World* (London: Dent, 1935); J. C. Eccles, *The Neurophysiological Basis of Mind* (Oxford: Oxford University Press, 1953); E. H. Walker, “Foundations of Paraphysical and Parapsychological Phenomena,” in *Quantum Physics and Parapsychology*, ed. L. Otera (New York: Parapsychology Foundation, 1975). For a review of quantum theories of consciousness, see H. Stapp, “Quantum Mechanical Theories of Consciousness,” in *The Blackwell Companion to Consciousness*, M. Velmans and S. Schneider, eds. (Oxford, U.K.: Blackwell, 2007).

7 M. Jouvet, “The States of Sleep,” *Scientific American* 216 (2) (1967): 62–72.

8 Two different types of dualistic or vitalist theory can be recognized in the light of this classification. The first, exemplified in the writings of Driesch, postulated the existence of a new type of causation responsible for repetitive and regular biological processes, corresponding to formative causation in the present sense; see H. Driesch, *Science and Philosophy of the Organism*, 2nd ed., first published 1908 (London: A. & C. Black, 1929), and H. Driesch, *Mind and Body* (London: Methuen,

1927). The second, developed most brilliantly by Bergson, emphasized conscious causation on the one hand, in H. Bergson, *Matter and Memory* (London: Allen and Unwin, 1911), and evolutionary creativity on the other, in H. Bergson, *Creative Evolution* (London: Macmillan, 1911), neither of which could be explained in terms of physical causes.

9 H. Bergson, *Creative Evolution* (London: Macmillan, 1911).

Appendix A. New Tests for Morphic Resonance

1 R. Sheldrake, *Seven Experiments That Could Change the World: A Do-It-Yourself Guide to Revolutionary Science* (London: Fourth Estate, 1994); R. Sheldrake, *Dogs That Know When Their Owners Are Coming Home, and Other Unexplained Powers of Animals* (London: Hutchinson, 1999); R. Sheldrake, *The Sense of Being Stared At, and Other Aspects of the Extended Mind* (New York: Crown, 2003a).

2 The main sources for melting points at different dates were various editions of the following handbooks: *Beilsteins Handbuch der Organischen Chemie, British Pharmacopoeia, British Pharmaceutical Codex, CRC Handbook of Chemistry and Physics,* and the *Merck Index.* In addition to these handbooks, I referred to many original papers in a range of chemical journals.

3 These melting point determinations were carried out using a heated-stage microscope melting-point apparatus by A. Datta, working "blind." I supplied replicates of coded samples and also an indication of a ten-degree-Celsius range within which the melting point of each sample was expected to lie. This work was carried out under the supervision of Gwyn Hocking.

4 For example, *Eremothecium ashbyii;* see W. H. Sebrell and R. S. Harris, *The Vitamins,* vol. 5, 2nd ed. (New York: Academic Press, 1972).

5 W. H. Sebrell and R. S. Harris, *The Vitamins*, vol. 5, 2nd ed. (New York: Academic Press, 1972).

6 *Merck Index* (Rahway, N.J.: Merck Publishing Group, 1996).

7 R. Aldstädter, *100 Years of Acetyl-Salicylic Acid* (Leverkusen, Germany: Bayer AG, 1997).

8 H. T. Clarke, J. R. Johnson, and R. Robinson, eds., *The Chemistry of Penicillin* (Princeton, N.J.: Princeton University Press, 1949).

9 J. W. Purseglove, *Tropical Crops: Dicotyledons* (London: Longmans, 1968).

10 M. van Genderen, B. Koene, and J. W. Nienhuys, "Sheldrake's Crystals," *Skeptical Inquirer,* October/November 2002, 35–40; R. Sheldrake, "Are Melting Points Constant? *Skeptical Inquirer,* September/October 2002, 40–41; M. van Genderen, B. Koene, and J. W. Nienhuys, "A Last Reply to Sheldrake," *Skeptical Inquirer,* October/November 2002, 41.

11 B. L. Davis and E. M. Oshier, "Memory Effect in Single-crystal Transformations of Aragonite-type to Calcite-type Potassium Nitrate," *The American Mineralogist* 32 (1967): 957–73.

12 J. D. Dunitz, "Phase Transitions in Molecular Crystals from a Chemical Viewpoint, *Pure and Applied Chemistry* 63 (1991): 177–85.

13 J. Bernstein, *Polymorphism in Molecular Crystals* (Oxford: Clarendon Press, 2002).

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15 Ibid., 214–15.

16 C. Darwin, *The Variation of Animals and Plants Under Domestication,* popular ed., vol. 2 (London: John Murray, 1905), 377.

17 N. Roll-Hansen, *The Lysenko Effect: The Politics of Science* (Amherst, N.Y.: Humanity Books, 2005).

18 C. Darwin, *The Variation of Animals and Plants under Domestication,* vol. 2 (popular ed.) (London: John Murray, 1905), 379.

19 G. Vines, "Hidden Inheritance," *New Scientist,* November 28, 1998, 27–30.

20 R. Sheldrake, "Rose Refuted," *Biology Forum* 85 (1992): 455–60.

21 R. Sheldrake, "An Experimental Test of the Hypothesis of Formative Causation," *Biology Forum* 85 (1992): 431–43; R. Rose, "So-Called 'formative causation': A Hypothesis Disconfirmed," *Biology Forum* 85 (1992): 444–53.

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24 R. A. Hinde and J. Fisher, “Further Observations on the Opening of Milk Bottles by Birds,” *British Birds* 445 (1951): 393–96.

25 R. Bedichek, *Adventures with a Texas Naturalist*, new ed. (Austin: University of Texas Press, 1961), 157.

26Ibid., 157–58.

27 J. F. Hoy, *The Cattle Guard: Its History and Lore* (Lawrence: University Press of Kansas, 1982).

28 R. Sheldrake, “Cattle Fooled by Phony Grids,” *New Scientist* (1988b): 65.

29 *The Guardian*, February 28, 1985.

30 *Daily Telegraph*, March 3, 1997.

31 *Daily Telegraph*, March 23, 1997.

32 *Huddersfield Daily Examiner*, July 27, 2004.

33 R. Sheldrake, *The Presence of the Past* (New York: Times Books, 1988a), chap. 10.

34 G. Schwartz, “Morphische Resonanz und systemetisches Gedächtnis,” in *Rupert Sheldrake in der Diskussion*, H.-P. Dürr and F.-T. Gottwald, eds. (Bern: Scherz Verlag, 1997).

35 A. Mahlberg, “Evidence of Collective Memory: A Test of Sheldrake’s Theory,” *Journal of Analytical Psychology* 32 (1987): 23–34.

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38 K. Robbins and C. A. Roe, “An Empirical Test of the Theory of Morphic Resonance Using Recognition for Chinese Symbols,” *Proceedings of Presented Papers: The Parapsychological Association 51st Annual Convention and the Incorporated Society for Psychical Research 32nd Annual Convention*, 2008, 176–86.

39 R. Sheldrake, “Formative Causation: The Hypothesis Supported,” *New Scientist* 27 (1983).

40 *Institute of Noetic Sciences Bulletin*, Autumn 1991.

41 Z. Dienes, “A Test of Sheldrake’s Claim of Morphic Resonance,” *Journal of Scientific Exploration* 8 (1994), 578.

42 S. Ertel, “Morphische Resonanz auf dem Prüfstand des Experimentes,” in *Rupert Sheldrake in der Diskussion*, eds. H.-P. Dürr and F.-T. (Bern: Gottwald Scherz Verlag, 1997).

43 A. M. Anderson, “The Great Japanese IQ Increase,” *Nature* 297 (1982), 180–81.

44 J. R. Flynn, “Now the Great Augmentaton of the American IQ,” *Nature* 301 (1983): 655; J. R. Flynn, “The Mean IQ of Americans: Massive Gains 1932 to 1978,” *Psychological Bulletin* 95 (1984): 29–51.

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48 Ibid.

49 F. C. Varela and J. Letelier, “Morphic Resonance in Silicon Chips,” *Skeptical Inquirer*, Spring 1988, 298–300.

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51 A. Fedanzo and I. S. Wingfield, “Morphic Resonance Test,” *Skeptical Inquirer,* Fall 1988, 100–101.

52 S. Rooke, personal communication with author, October 2007.

53 P. Trachtman, “Redefining Robots, *Smithsonian Magazine*, February 2000, 97–112.

Appendix B. Morphic Fields and the Implicate Order

1 D. Bohm, *Causality and Chance in Modern Physics* (New York: Harper, 1957).

2 D. Bohm, *Wholeness and the Implicate Order* (London: Routledge and Kegan Paul, 1980).

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