1 The Transformations of Darwinism

No sphere of knowledge is free of controversy, and science is no exception. If anyone imagines that scientists are dispassionate and impartial people, discussing theories and ideas unemotionally in the cool clear light of reason, they have been seriously misled. Passion and fervor accompany all worthwhile scientific discussions. This is particularly evident when the discussion is about something like the theory of evolution, which bears directly on human history and our relationships with each other and the world around us. Because such discussions are tied up with ideas about "human nature," and impinge on moral judgments and ethical issues, they can be very emotional, as well as intellectually exciting.

We are not referring here to the arguments between people who accept evolutionary ideas and those who prefer to believe that the world was created by God in six real or metaphorical days. Such arguments have considerable sociological and political interest, but they are not really part of science, so we need say no more about them. What we are referring to are the heated discussions that have gone on and still go on among the evolutionary biologists themselves.

When you read popular accounts of new discoveries in biology, you often come across phrases such as "according to Darwin's theory of evolution . . . ," or "evolutionary biologists explain this as . . . ," or "the evolutionary explanation is. . . ." You get the impression that there is a tidy, well-established theory of evolution—Darwin's theory of natural selection—which all biologists accept and use in the same way. The reality is very different, of course. Ever since Darwin's book *On the Origin of Species* appeared in 1859, scientists have been arguing about whether and how his theory of evolution works. Can competition between individuals with heritable differences in their ability to survive and reproduce lead to new features? Is natural selection the explanation of all evolutionary change? Where does all the hereditary variation on which Darwin's theory depends

come from? Can new species really be produced by natural selection? Darwin's book was crammed with observations that supported his theory, but there were some glaring gaps in his evidence. The biggest was that he could say little about the nature and causes of hereditary variation. Right from the outset, even those who accepted Darwin's evolutionary theory questioned its completeness and sufficiency, and struggled to try to find answers to the questions it raised about heredity and variation. In subsequent decades, as new discoveries were made and new theoretical approaches were developed, the debates continued. Existing ideas were constantly being challenged and revised, with the result that profound changes have occurred in the ways the concepts of evolution and heredity have been understood.

Today, most biologists see heredity in terms of genes and DNA sequences, and see evolution largely in terms of changes in the frequencies of alternative genes. We doubt that this will be the situation in twenty years' time. More and more biologists are insisting that the concept of heredity that is currently being used in evolutionary thinking is far too narrow, and must be broadened to incorporate the results and ideas that are coming from molecular biology and the behavioral sciences. We share this view, and in later chapters will explain why. But before doing so, we want to outline some of the history of evolutionary thinking over the last 150 years to see how the present gene-centered version of Darwinian theory came into being, and what it means for today's evolutionary biologists. Since we cannot even attempt to look at all of the many twists and turns in the pathway of ideas that led to the present position, we will focus on some of the major turning points and the arguments that influenced the direction taken.

Darwin's Darwinism

Darwin summarized his view of evolution in the last paragraph of *The Origin*. In what was for him an unusually poetic paragraph, he wrote:

> It is interesting to contemplate an entangled bank, clothed with many plants of many kinds, with birds singing on the bushes, with various insects flitting about, and with worms crawling through the damp earth, and to reflect that these elaborately constructed forms, so different from each other, and dependent on each other in so complex a manner, have all been produced by laws acting around us. These laws, taken in the largest sense, being *Growth with Reproduction*; *Inheritance* which is almost implied by reproduction; *Variability* from the indirect and direct action of the external conditions of life, and from use and disuse; a Ratio of Increase so high

as to lead to a *Struggle for Life*, and as a consequence to Natural Selection, entailing Divergence of Character and the Extinction of less-improved forms. Thus, from the war of nature, from famine and death, the most exalted object which we are capable of conceiving, namely, the production of the higher animals, directly follows. There is grandeur in this view of life, with its several powers, having been originally breathed into a few forms or into one; and that, whilst this planet has gone cycling on according to the fixed law of gravity, from so simple a beginning endless forms most beautiful and most wonderful have been, and are being, evolved. (Darwin, 1859, pp. 489–490)

The italics in the rather less poetical sentence in the middle are ours, not Darwin's. They are there to stress the "laws" to which Darwin pointed: the laws of reproduction, inheritance, variability between individuals, and a struggle for existence. By using these laws, it is possible to formulate Darwin's theory in a very general and abstract way, without referring to our own world or to the types of reproduction, inheritance, variation, and competition with which we are familiar. For example, in British evolutionary biologist John Maynard Smith's generalization, the properties that any group of entities and their world must have in order for evolution by natural selection to occur are the following:

- *Multiplication*—an entity can reproduce to give two or more others.
- *Variation*—not all entities are identical.
- *Heredity*—like begets like. If there are different types of entities in the world, the result of the multiplication of entity of type A will be more entities of type A, while the result of the multiplication of entity B will be more of type B.
- *Competition*—some of the heritable variation affects the success of entities in surviving and multiplying.

If all these conditions are met, evolution by natural selection is inevitable: the type of entity that has the greatest ability to survive and multiply will increase in frequency (figure 1.1). Eventually, evolution in this world will stop, because all the entities will be of the same type. However, if heredity is not always exact, so that from time to time new variants arise, then variations in a certain direction may accumulate and produce a complex functional system. Historically, the eye is the classic example of cumulative evolution in the living world, and the modern PC is a good example from the world of technology.

When formulated in Maynard Smith's way, Darwin's theory of evolution by natural selection is an extremely general theory. It says nothing about the processes of heredity and multiplication, nothing about the origin of

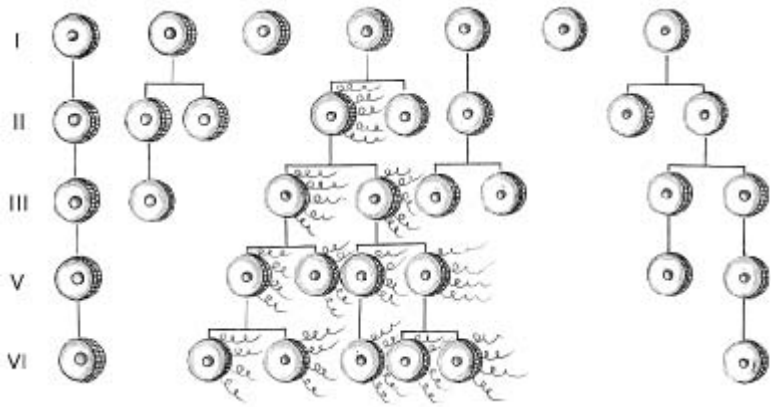

Figure 1.1
Universal Darwinism: the frequency of the hairy entity, which first appears in generation II, increases in subsequent generations because it survives better and multiplies more than its competitors.

the heritable variation, and nothing about the nature of the entity that is evolving through natural selection. Appreciating this is going to be crucial for the arguments that we develop in later chapters. Although we are not advocating it, we want it to be clear that it is possible to be a perfectly good Darwinian without believing in Mendel's laws, mutating genes, DNA codes, or any of the other accoutrements of modern evolutionary biology. That is why Darwin's theory can be and is so widely applied—to aspects of cosmology, economics, culture, and so on, as well as to biological evolution.

Darwin himself knew nothing about genes, Mendelian laws, and DNA, of course. These did not become part of evolutionary theorizing until the twentieth century. In fact, in Darwin's day, there was no good theory of heredity at all, and this was a problem. At that time, most people assumed that the characteristics of two parents blended in their young, so if you started out with a population with two types in it (say black and white), you would end up with a population in which everything was the same (gray). There would be no variability left. Yet Darwin's theory depends on the presence of heritable differences between individuals. Even without blending, if you continually selected one type (say black), the proportion of that type would increase until eventually all in the group would again be identical (this time black). So where does new variation come from? For the theory of natural selection to be believable, Darwin and his followers had to explain the origin and maintenance of variation.

As the quotation from the last paragraph of *The Origin* indicates, Darwin thought that heritable variation stems from the effects the conditions of life have on the organism, and from "use and disuse." Discovering that this is what Darwin thought surprises some people, because they associate the idea of evolutionary change through use and disuse with the name of Lamarck. Lamarck, they have been told, put forward a theory of evolution fifty years before Darwin did, but got the mechanism all wrong. Foolishly (somehow, Lamarck is always made to seem foolish), Lamarck believed that giraffes have long necks because their ancestors were constantly striving to reach the leaves on tall trees, stretching their necks as they did so. They passed on these stretched necks to their young, so that over many generations necks became longer and longer. Lamarck, the story goes, saw evolution as the result of the inherited effects of use (or disuse). His big mistake was to assume that "acquired characters"—changes in structures or functions that occur during an animal's life—could be inherited. Fortunately, the story continues, Darwin showed that natural selection, not use and disuse, is the cause of evolutionary change, so the idea that acquired characters can be inherited was abandoned.

This often repeated version of the history of evolutionary ideas is wrong in many respects: it is wrong in making Lamarck's ideas seem so simplistic, wrong in implying that Lamarck invented the idea that acquired characters are inherited, wrong in not recognizing that use and disuse had a place in Darwin's thinking too, and wrong to suggest that the theory of natural selection displaced the inheritance of acquired characters from the mainstream of evolutionary thought. The truth is that Lamarck's theory of evolution was quite sophisticated, encompassing much more than the inheritance of acquired characters. Moreover, Lamarck did not invent the idea that acquired characters can be inherited—almost all biologists believed this at the beginning of the nineteenth century, and many still believed it at its end. It was certainly part of Darwin's thinking, and his theory of natural selection certainly did not lead to the idea being abandoned. On the contrary, it led to endless acrimonious arguments (and even a few experiments) about whether or not acquired characters are inherited. For as long as there was no satisfactory and agreed theory of heredity, and no explanation of the origin of variation, the inheritance of acquired characters retained a place in evolutionary thinking.

The lack of a good theory of heredity and an explanation of variation were a constant frustration for Darwin and his followers, and Darwin tried to do something about it. From the 1840s onward, he collected together everything that was known about inheritance, and used it to develop his

own heredity theory. He called it the "provisional hypothesis of pangenesis," and eventually described it in his massive book *The Variation of Animals and Plants under Domestication*. It wasn't very original, and was never very popular, but despite the criticism, Darwin never deserted it. It is worth describing Darwin's pangenesis theory, because most other heredity theories in the second half of the nineteenth century were quite similar. All were totally different from the theory of inheritance that we accept today.

What Darwin suggested was that every part of the body, at each developmental stage, sheds tiny particles, which he called "gemmules." These circulate in the body, sometimes multiplying as they do so. Some gemmules are used for regenerating damaged or missing parts, but most eventually aggregate in the reproductive organs. In asexual organisms, the gemmules in the egg, seed, spore, or whatever piece of the parent produces the next generation organize themselves and eventually each develops into the same type of part as that from which it originally came. In sexually reproducing organisms, the gemmules stored in the egg and sperm join together before development starts (figure 1.2). The offspring therefore become a blend of the parental characters, although sometimes, according to Darwin, gemmules are not used immediately, but remain dormant and reappear either later in life or in future generations.

Initially the gemmules present in a fertilized egg are not ordered in any particular way, but during development, as they grow and multiply, they are incorporated into the appropriate place at the appropriate time because they have certain special affinities for each other. Gemmules are therefore units of both heredity and development. According to this notion of heredity, what is inherited is the actual character itself. It is transmitted from one generation to the next in the form of its miniature representatives, the gemmules. In Darwin's words, "inheritance must be looked at as merely a form of growth" (Darwin 1883, vol. 2, p. 398).

Pangenesis could account for most of the things Darwin had found out about heredity, regeneration, hybridization, developmental abnormalities, and much else. But what about variation? Pangenesis should lead to blending and uniformity, so how did Darwin explain variation? First, he suggested, a change of nutrition or climate could affect growth and alter the proportions of the different gemmules in the reproductive organs; it could also reawaken dormant gemmules. Second, changed conditions or new experiences could at any stage lead to changes in the gemmules themselves. If parts of a parent were modified, for example through use or disuse, correspondingly modified gemmules would be produced. The new,

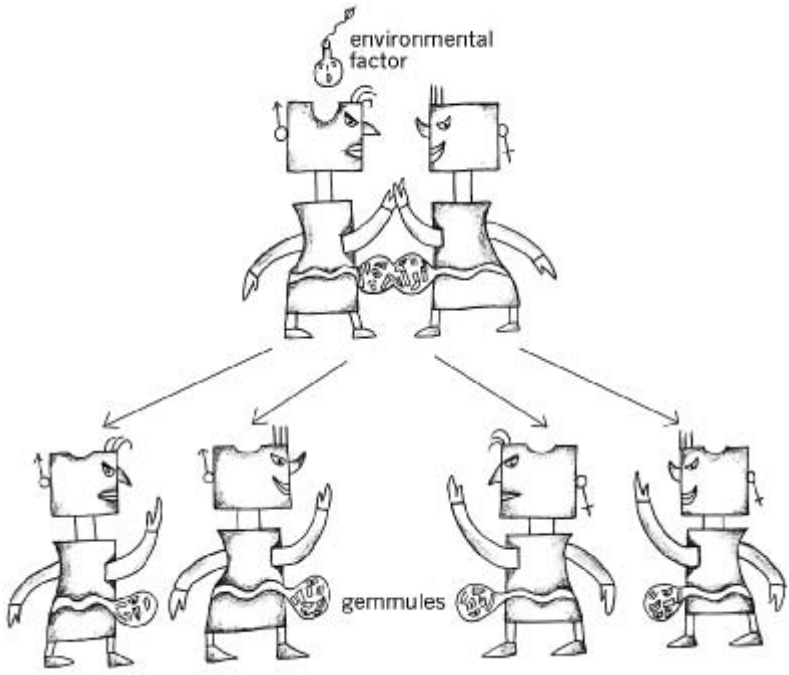

Figure 1.2
Sexual pangenesis. Representative particles (gemmules) from the male (left) and female (right) parents accumulate in their reproductive organs. Following insemination, these gemmules mix and together produce the next generation. An environmental effect (the bomb) induced a change in the male parent. This change is inherited, because the gemmules from the modified body parts are also modified, but the effect is diluted by gemmules from the unaffected female parent.

acquired character would be inherited, although it might not be expressed very strongly, because the modified gemmules would be mixed with those already present in the reproductive organs and with those contributed by the mate.

Obviously, accepting that the environment has a role in inducing variation in no way weakens Darwin's theory of evolution by natural selection. On the contrary, if new variation can arise in response to the conditions of life, it increases the amount of variation and the scope for natural selection. Darwin would no doubt have been amazed to hear that many biologists today think that Lamarckian views about the inheritance of acquired characters contradict the fundamental assumptions of his theory of natural selection. They do not. Darwin's pangenesis

hypothesis shows that the theory of natural selection is really not very fussy. Gemmules turned out to be no more than fascinating figments of Darwin's imaginative mind, but as a cause of the heredity and variation needed for animals and plants to evolve through natural selection, they did very nicely. Darwin's theory of natural selection is a very general theory; it is not tied to any particular mechanism of heredity or cause of variation.

Weismann's Neo-Darwinian Theory: Acquired Characters Discarded

We tend to assume that the great increase in the rate of scientific progress began in the twentieth century, but imagine what it must have been like to be a biologist in the late 1850s. First, Rudolph Virchow propounds the theory that cells come only from other cells; they cannot arise from non-cellular matter. Soon after, Darwin tells the world that species arise only from other species; they are not produced by special creation, but by natural selection. Then Louis Pasteur reports his experiments showing that living things are not generated spontaneously; organisms come only from other organisms. Trying to keep up with all that was going on must have been as big a nightmare for scientists in the mid-nineteenth century as it is now. So it is not surprising that when Darwin was dealing with the finer points of his pangenesis theory, he left the question of the formation of cells rather vague, "as I have not especially attended to histology." Given how much else he was attending to, no one can really blame him for deciding that he didn't know enough to evaluate the various ideas about the origin of cells. It was left to others to try to relate the new cell biology to heredity and evolution. Among those who tried to do so was the German biologist August Weismann, one of the most profound and influential evolutionary thinkers of the nineteenth century.

Weismann's ideas about heredity and development changed over time, but the essentials were in place by the mid-1880s. By then it was generally recognized that organisms are made of cells, and that cells have a nucleus containing threadlike *chromosomes* (the word itself was not invented until 1888). It was known that ordinary body cells divide by *mitosis*, a process in which each chromosome doubles and then splits longitudinally, with one half going to each of the daughter cells. Once this rather precise method of allocating the nuclear material had been recognized, it became clear to Weismann and several other people that the chromosomes probably contain the hereditary substance that determines the characteristics of the cell and its descendants.

Weismann realized, however, that if the chromosomes of the nucleus contain the hereditary materials, then there is a problem when it comes to inheritance between generations. The link between generations of sexually reproducing organisms is through the eggs and sperm (or as we would now say, the gametes). Yet, if both egg and sperm have the same chromosomal content as other cells, the fertilized egg and the new organism it produces will have twice as much chromosomal material per cell as either parent had. Obviously, this cannot be what is happening. Weismann therefore concluded that during sperm and egg production, the cells in the reproductive system must undergo a different kind of division from that of other cells. It has to be a "*reduction* division," he said, in which each daughter cell receives only half of the parent cell's chromosomal material. Then, when the nuclei of sperm and egg are united during fertilization, the two halves become a new whole with the same amount of nuclear material as other cells. When Weismann first suggested this, there was no real evidence for a reduction division, although it was known that odd things happen during the cell divisions that produce eggs. It took some years for people to unravel the nature of the process that was eventually called *meiosis*, and recognize its significance in inheritance. As Weismann guessed, the amount of chromosomal material is indeed halved, but there is a lot more to meiosis than that.

How did Weismann's deductions about cell division relate to his ideas about heredity and evolution? The first thing to be said is that Weismann emphatically rejected any possibility that acquired characters are inherited. The big muscles that the blacksmith develops through his hard work cannot be transmitted to his sons and daughters. If his sons want to be blacksmiths, they will have to go through the muscle-building process themselves, because they do not inherit their father's big muscles. There is no way, according to Weismann, in which properties that reside in the cells and tissues of the arms can be transmitted to the father's sperm cells. The same is true for circumcision. Although for three thousand years Jews have been circumcising their newborn boys, this has not resulted in their male babies being born without a foreskin. Eight-day-old baby boys still have to undergo the painful ritual operation. There is no route through which information about a cut-off foreskin can be passed to the sperm. Not only are there no empirical data of any kind to support the conjecture that acquired characters are inherited, claimed Weismann, there is no way in which it could happen.

Weismann's insistence that it is impossible to inherit acquired characters was tied up with the way in which he saw heredity and embryonic

development. He devised a scheme that was founded on what he called "the continuity of the germ plasm," which we have shown in figure 1.3. It involved a division of labor between the elements that maintain individual life and the elements that are devoted to producing future generations—a division between the soma (the body) and the germ line. He argued that right from the beginning of development, a part of the chromosomal material, which he called the "germ plasm," is set aside for the production of the eggs, sperm, spores, or whatever else gives rise to the next generation. In many animals it is separated off into special gamete-producing cells—germ cells—very early in development. Sometimes the germ cells are the very first cells to form, but even if they form later, they still have germ plasm that is identical to that in the fertilized egg. According to Weismann, the other cells of the body, the somatic cells, do not.

Weismann's scheme for development was quite complicated and, as it turned out, quite wrong. It involved a whole hierarchy of units, each present in the chromosomes in multiple copies. In essence, what Weismann thought was that when embryonic cells divide, each daughter cell can receive different parts of the nuclear material—a different set of "determinants." That is why daughter cells develop into different cell types. Determinants move out of the nucleus to impose their characteristics on the cells, so the nuclear material gets simpler and simpler as cells continue to divide and produce the different tissues. Development therefore depends on gradual, regulated, qualitative changes in the nuclear substance. Only the germ plasm in the germ line retains the full hereditary potential—a full set of determinants. It is this unaltered and untainted

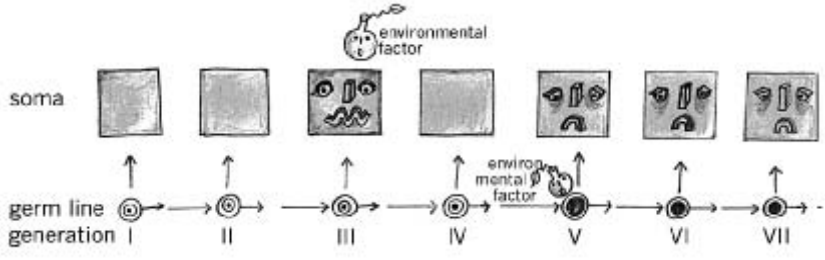

Figure 1.3
Weismann's doctrine: hereditary continuity is through the germ line. An environmentally induced change in the soma (bomb in generation III) does not affect the offspring, whereas a change in the germ line (bomb in generation V) affects all subsequent generations.

germ plasm that is used for the sperm and eggs that will produce the next generation.

If, as Weismann maintained, acquired characters cannot be inherited because bodily events do not affect the protected germ line, where did he think all the variation that Darwin's theory demanded came from? Here he had an important insight: it comes from sexual reproduction, he said. He reasoned that since the father's germ plasm in the sperm mixes with the mother's germ plasm in the egg, there are two mingled germ plasms in their offspring. In the next generation, the two mingled germ plasms in these and similar offspring's eggs will mix with two mingled germ plasms from their sperm to give offspring with four mingled germ plasms; and in the next generation four mingled germ plasms in the egg will mix with four more from the sperm to give eight. And so it will go on. Every individual is thus the product of a mixture of minute quantities of vast numbers of ancestors' germ plasms. Now, since the amount of nuclear material is kept constant by the reduction division that halves the amount of germ plasm during sperm and egg formation, what Weismann cleverly suggested was that the half of the germ plasm that is eliminated is not the same for every egg or sperm. In each a different group of ancestral germ plasms is retained. It is like a card game in which a deck of ancestral germ plasms is shuffled before a gamete is formed, and the gamete is then dealt half of the deck. Since there is an enormous number of possible combinations of ancestral germ plasms, no two gametes will be the same. Thus there is always a lot of variability in sperm and eggs, and even more in the offspring they produce when they fuse. There have been some wonderful words written about sex, but what Weismann rather prosaically said was "The object of this process [sex] is to create those individual differences which form the material out of which natural selection produces new species" (Weismann, 1891, p. 279).

Sex could provide endless variability by recombining the hereditary material from different ancestors, but Weismann still had to explain how ancestral germ plasms came to be different in the first place. The ultimate origin of variation, he said, was in changes in the quantity and qualities of the many growing and multiplying determinants for each character that are present in the germ line. From time to time, small random accidents would alter determinants. Some would survive and multiply better than others, so through natural selection among the determinants, the germ plasm would gradually change. Weismann called this process "germinal selection." Exactly which determinants were selected would depend on factors such as nutrition and temperature, said Weismann.

It is worth noting two things about Weismann's germinal selection. The first is that although he was adamant that environmental effects on the body cannot be inherited, he did accept that the conditions of life had heritable effects. They did so because they affected the determinants in the germ plasm *directly*. The second is that the germinal selection idea shows that Weismann appreciated how very general Darwin's theory is: he recognized that natural selection can occur between units other than individual organisms. As well as believing that it occurs between determinants in the germ plasm, he accepted that selection must also occur between the cells within a tissue. Like Darwin, he also recognized that natural selection must occur between groups of organisms, because this was the only satisfactory way of explaining the evolution of sterile worker ants and bees. We will come to the evolutionary problems with social insects later, but here we just want to point out that in applying Darwinian theory to other levels of biological organization, Weismann and some of his contemporaries were really way ahead of their time. It took another three-quarters of a century for the idea of multilevel selection to be incorporated into mainstream evolutionary biology.

In summary we can say that the key differences between Darwin's original theory and Weismann's version of it are as follows:

- Weismann gave natural selection an exclusive role. He completely excluded change through use and disuse, and every other form of the inheritance of acquired characters.
- Weismann's heredity theory was totally different from Darwin's. His heredity determinants were transmitted from generation to generation only through the germ line. In contrast to Darwin's gemmules, determinants were not derived anew in each generation, but were stable, replicating entities. Not only were they not derived from the parent's body structures, those retained in the germ line were totally immune from anything occurring in the body.
- For Weismann, the only source of new heritable variation was accidental or environmentally induced changes that directly affected the quantity or quality of determinants in the germ line itself.
- Weismann recognized that it was the sexual process, which brought together different combinations of the parent's determinants, that produced the heritable differences between individuals that were needed for evolution through natural selection.

Historically, one of the most interesting things about Weismann's theories is that although many of his contemporaries hated them, they were

still extremely influential. His theory of heredity and development was far too speculative and complicated to gain much acceptance, yet elements of it were incorporated into the new science of genetics in the early twentieth century. Similarly, Weismann's version of Darwinism was seen as far too restricted, yet it had long-lasting effects on the direction ideas about evolution took in later years.

Doubts about Darwinism

By the 1880s, although most biologists accepted the idea of evolution, Darwin's theory of natural selection was thought to be on its deathbed. It didn't recover until well into the twentieth century. One reason for its decline was probably Weismann's dogmatic insistence that natural selection is the *only* mechanism of evolution. This hardened the attitudes of those who preferred Darwin's more pluralistic views, which included the inheritance of acquired characters. Some people rejected Darwin's natural selection almost completely, assigning to it the minor role of merely weeding out the oddities and mistakes. In place of natural selection, various "neo-Lamarckian" mechanisms were proposed.

The term *neo-Lamarckism* was invented in 1885, but was never well defined and meant different things to different people. A dominant element in neo-Lamarckism was the idea that adaptation could occur through the inherited effects of use and disuse. In addition, however, many neo-Lamarckians believed that there were internal forces that made evolution progressive and goal-directed, just like embryonic development. Ideas like these seemed to provide a better basis for adaptation and what was known of evolutionary history. They also fitted better with many peoples' deep-seated religious or moral beliefs. To some the idea of human beings improving as a result of experience was much more attractive than change through ruthless Darwinian competition.

People from both within and outside the scientific community attacked Weismann's ideas from all sides, not always in moderate language. Prominent figures such as Herbert Spencer, Samuel Butler, and later even George Bernard Shaw, ensured that the Lamarckian aspects of evolution were given the widest publicity. Herbert Spencer, one of the leading thinkers of the second half of the nineteenth century, was a believer in biological evolution even before *The Origin*. In fact he was the person who brought the term *evolution* into general currency, using it for all sorts of developmental processes that lead from the simple to the more complex. It was an explanatory concept that united events in the solar system, in society, in

the development of mind and body during an individual's lifetime, and in structures and functions in lineages over generations. For Spencer, evolution extended beyond biology, and he assumed that all evolutionary change was fueled by similar mechanisms. He was convinced that the inheritance of acquired characters played a major role in both biological and social evolution, and battled publicly with Weismann about it in the pages of the widely read *Contemporary Review*.

Since Lamarckians rejected Weismann's ideas about inheritance, they needed a heredity theory that would allow the effects of use and disuse to be transmitted. Darwin's pangenesis hypothesis might have done, because it was compatible with the inheritance of acquired characters, but it never found favor, partly because of some work done by Darwin's cousin, Francis Galton. Galton tested pangenesis experimentally by making massive blood transfusions between rabbits with different-colored fur. If Darwin was right, he reasoned, then when blood from white rabbits is transfused into gray rabbits, white-fur gemmules should be transferred too, and some should reach the gray rabbits' reproductive organs. The offspring of these gray rabbits should therefore have some white fur. Unfortunately for Darwin, Galton found that they did not. Although Darwin tried to wriggle out of this embarrassment for his theory by pointing out that he had never said that gemmules circulate *in the blood*, Galton and many others saw it as evidence against pangenesis. However, the main reason why pangenesis-type theories fell from favor was probably not so much that there was no experimental evidence for them, but that they didn't fit with cell biology. As the cell theory became better established, it was impossible to reconcile gemmules or similar hereditary particles coming from all parts of the body with the idea that all cells, including the sperm and egg, come only from other cells. Increasingly, heredity theories had to be seen to be consistent with the growing knowledge of the behavior of cells.

Lamarckians suggested various ways in which what happened in the body could influence the hereditary material in the germ cells, but their theories were extremely speculative. They and their opponents also made many attempts to show experimentally that the inheritance of acquired characters did or did not occur, and such attempts continued until well into the twentieth century. It is not worth going into the details of these experiments and the arguments about them here, however, because in the long run they had little influence on the debate about Lamarckism. As Peter Bowler, one of the leading historians of biology of this period, has stressed, it was not the lack of experimental evidence that eventually led to the

demise of Lamarckism, but the lack of a good theoretical model of inheritance.

Neo-Lamarckians were not the only people who were attacking Darwinism in the latter part of the nineteenth century. The idea of *gradual* evolution through the selection of *small* variations was also under attack. People began to argue that evolutionary change was saltatory—it occurred by big jumps, not through the selection of many little differences. Once again, Darwin's cousin Francis Galton was in the forefront of those who caused problems for Darwin's ideas. In an effort to understand human heredity better, Galton applied statistical reasoning to characters that show continuous variation. Continuous characters—characters such as height, for which there is a whole range of possible values—were those which Darwin believed were really important in evolution. According to Darwin, it was selection of small differences over many generations that led to gradual change. Galton, however, decided that this type of selection simply would not work. He did some calculations that suggested (incorrectly) that because you inherit not only from your parents but also from your grandparents and more distant ancestors, the average value of a character could never be permanently changed by selection. He concluded that for permanent change you needed a "sport"—a large, qualitative change in the hereditary material.

Galton's conclusions were fiercely contested by other biometricians, who said he had made a logical mistake in his mathematics. They claimed that selection *could* shift a population average, in exactly the way Darwin had suggested. However, support for the idea that evolution occurred through big jumps also came from a totally different direction. Hugo de Vries in Holland and William Bateson in England had both studied variation in nature, and recognized that a lot of it is discontinuous. Often there are just a few distinct, alternative types, with no intermediates. The same is true if you compare species—there are distinct differences between them; they do not grade into each other. Bateson and de Vries therefore agreed with Galton that discontinuous variation is of greatest importance in evolution, and that evolution occurred through sudden big jumps, not slow crawling. According to de Vries, the driving force in evolution was *mutation*, a process that suddenly and without cause irreversibly changed the germ plasm. Mutation produced a new type of organism in a single step.

De Vries and Bateson were to be significant figures in the development of Mendelism in the first decade of the twentieth century, and it is worth remembering that almost all of the pioneers of the new science of genetics were, like them, "mutationists." Although the term *mutation* didn't

mean exactly the same then as it does now, it did relate to a quantum change in the hereditary material. Among most of the founders of modern genetics, both Lamarckism and Darwinism were deemed irrelevant to evolution—mutations were believed to be the important factor.

The Modern Synthesis: Development Vanishes

Debates about the relative importance of selection, mutation, and the inheritance of acquired characters continued until well into the 1930s, but during that decade a far more specific version of Darwin's theory began to be established. Biologists from several disciplines started to shape what became known as the "Modern Synthesis" of evolutionary biology. Weismann's ultra-Darwinism was combined with Mendelian genetics, which had adopted the concept of the gene as the hereditary unit of biological information. Using this framework, many aspects of comparative anatomy, systematics, population biology, and paleontology were explained in terms of natural selection. We are not going into all the details of this, but want to look quite closely at the theory of heredity that was incorporated into the Modern Synthesis, because it was this that began to bias many biologists' approach to evolution.

Mendel gave the world the laws that now bear his name in 1865, when he told the Brno Scientific Society about the hybrids he had made between varieties of the garden pea. His paper was published in the society's journal in the following year, but its significance was not appreciated until decades later. It was not until 1900 that three botanists—Hugo de Vries (the mutationist), the German Carl Correns, and the Austrian Erich von Tschermak—published results from their own breeding experiments which confirmed the validity of the laws that Mendel had established more than thirty years earlier. The year 1900 is now regarded as the birthdate of the discipline for which William Bateson a few years later coined the term *genetics*.

According to the formulation of Mendel's theory that was produced in the early years of the twentieth century, individuals contain hereditary units that determine the development of their characteristics. The crucial thing about these heredity units, which were called *genes*, is something that Weismann (and initially de Vries) had failed to recognize—they exist in pairs. One member of each pair is inherited from the male parent, the other from the female parent. The members of a pair can be identical or somewhat different, but both can affect the development of a particular trait, such as the color of pea seeds or the shape of human ear lobes. The

different versions of a gene are known as *alleles*. When sperm or pollen and eggs are formed, they contain only one allele from each pair because, just as Weismann had said, the formation of gametes involves a reduction division, which halves the hereditary material. During fertilization, when the sperm and egg, or pollen and egg, unite, the full hereditary complement is reestablished, and there are once again two alleles for each character.

Mendel's "laws" describe the regularity of the distribution of alleles in the gametes and at fertilization. The "first law" asserts that during the formation of gametes, the two alleles of each pair separate. They have not been changed by being with their partner or by being in that particular body. They leave it in exactly the same condition as they entered it. The "second law" asserts that alleles that belong to different pairs segregate independently of each other. This means that if you are thinking about a lot of characters and a lot of pairs of alleles, there is a vast amount of hereditary variation in the gametes. The argument is basically the same as that which Weismann used—any two eggs or two sperm are very unlikely to get exactly the same combination of alleles. Mendel's laws also assume that which particular sperm and egg unite is not influenced by the alleles they carry, so even more variation is present in the fertilized eggs.

A crucial part of Mendel's findings was that, with the strains he chose to use (and he made his choice very carefully), hybrid offspring did not show intermediate characteristics; they resembled one or other of the parents. For example, in crosses between a pure breeding strain with yellow seeds and one with green seeds, all the offspring were yellow, not yellowish-green. In Mendelian jargon, yellow is dominant, green is recessive. The explanation is simple: if the allele that determines yellowness is given the symbol *Y*, and that determining greenness is given the symbol *y*, seeds have to have two copies of *y* to be green, but a single *Y* allele is enough for yellowness. So with parents that are pure yellow (*YY*) and pure green (*yy*), the offspring inherit a *Y* allele from the yellow parent and a *y* allele from the green parent, so they are *Yy*. The single *Y* allele is enough to make them yellow. When these are self-fertilized, you get the famous Mendelian ratio of three yellow to one green. The reason why can be seen in figure 1.4, which shows the behavior of characters in a typical Mendelian cross and its genetic interpretation.

Within a few years of the rediscovery of Mendel's laws, hundreds of crosses confirming them had been made using a variety of animals and plants. It was quickly realized that the behavior of the hypothetical hereditary units, the genes, which was deduced from breeding experiments, was

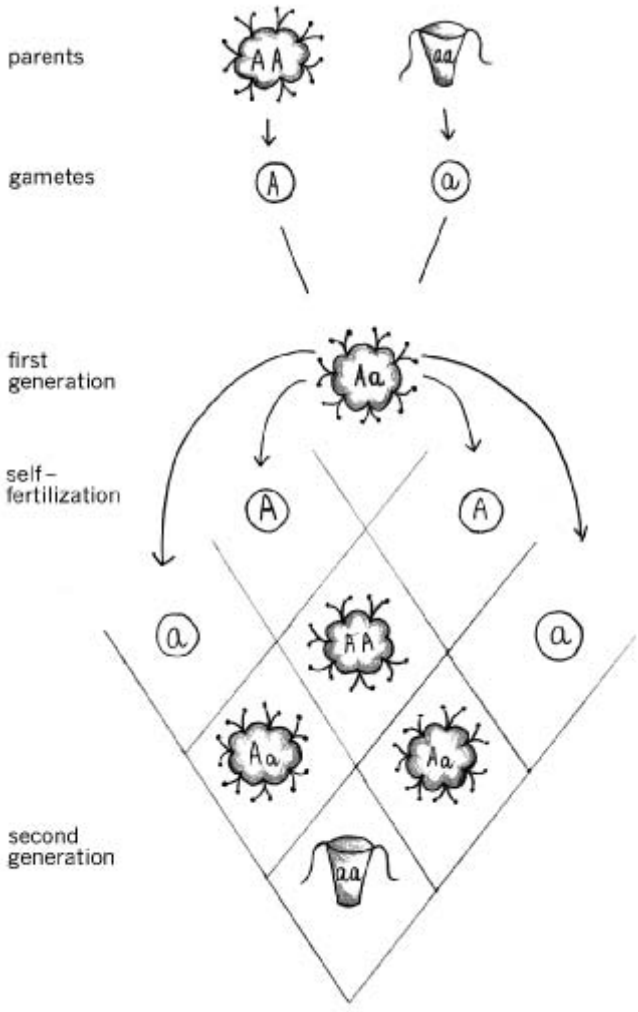

Figure 1.4
A Mendelian cross between two strains with different alleles for a structural feature. Crossing *AA* and *aa* produces *Aa*, which resembles the *AA* parent. *A* is therefore dominant, *a* recessive. When individuals of type *Aa* are self-fertilized, three-quarters of their offspring resemble the dominant parental type, and one-quarter resemble the recessive type.

paralleled by the behavior of chromosomes during gamete formation and fertilization. Alleles come in pairs, and so do the chromosomes in body cells; in the gametes there is only a single allele of each gene, and there is only a single copy of each chromosome. From this starting point it did not take long to show that genes are linearly arranged on the chromosomes. This has some consequences when you are looking at more than one trait, but we do not have to worry about this at the moment. We just need to appreciate that genes were soon being regarded as discrete particles, organized rather like beads on a string.

Before moving on, we need to stress something that at first glance may seem rather trivial. It is that Mendelian genetics is based on the analysis of differences. When differences in alleles lead to differences in appearance, we can deduce something about the genetic constitution of parents and offspring. From the ratios of the different types of offspring, we can say which alleles the parents probably have. Conversely, if we know the parents' genetic constitution, we can predict the expected proportions of each type of offspring. But if there are no visible differences, we can say nothing about the genetic constitution, and we know nothing about inheritance.

At first, the distinct character differences that genetics dealt with so well—yellow or green, tall or short, long wings or vestigial wings—reinforced the mutationists' view that evolution depends on discrete qualitative jumps. Mendelism lent no support to Darwinism. Later, however, it was realized that genes can also explain the inheritance of characters such as height or weight, which show continuous variation. All that is necessary is to assume that the character is controlled by many genes, each having a small effect. When there are many genes involved, genetic differences between individuals can supply all the variation needed for adaptive evolution through Darwinian selection.

How genes brought about their effects was at first totally unknown, and for Mendelian analysis and evolutionary theorizing it seemed unimportant. Many of the pioneer geneticists made a conscious decision to ignore development. The newly formed departments of genetics concentrated on counting the different types of progeny obtained in crosses between plants or animals with visible differences, and from their numbers deducing the relationship of the underlying genes to each other and to the chromosomes. Thomas Hunt Morgan and his students at Columbia University launched the small, rapidly breeding, fruit fly *Drosophila* on its career as the geneticists' favorite experimental animal, and used it to produce a wealth of information about the transmission of genes and the

chromosomes that carried them. It was the Mendelist-Morganist view of heredity that was later adopted by the architects of the Modern Synthesis of evolution. It was a view that was based on genes located firmly and exclusively in the nucleus, and ignored the surrounding cytoplasm.

The conceptual basis of the Morgan school's view of heredity was provided in the very early days of genetics by Wilhelm Johannsen, a Danish botanist. It was Johannsen who coined the term *gene* as part of his attempt to formulate a biological concept of heredity. Johannsen worked with pure lines of plants—strains that are initiated from a single individual, and maintained by repeated self-fertilization. They can differ from each other, but within any particular line there is very little variation among individuals, and any differences that there are, are not inherited. Johannsen found that if he selectively bred from the extremes—say the tallest and the shortest—it had absolutely no effect: the selected lines still had the same average height as those from which they came. This work led Johannsen to define two key concepts—genotype and phenotype. The genotype is an organism's inherited potential—the potential to have green seeds, green eyes, or to be tall. Whether or not this potential is realized depends on the conditions in which the organism is raised. For example, the height of a plant will depend on the quality of the soil, the temperature, how much water it gets, and so on. So even if a plant has the genotype to be tall, it will not manifest this potential tallness unless the conditions are right. How tall the plant actually is—its phenotype—depends on both its genotype and environmental conditions. Johannsen's interpretation of his pure-line work was simple: all individuals in a pure line have the same genotype. Because they all have the same genes, any differences in their phenotypes cannot be passed on. Differences in phenotype can be inherited and selected only if they are the result of differences in genotype.

The distinction between genotype and phenotype is fundamental to classical genetics. According to Johannsen, heredity does not involve the transmission of characters, but of the potential for characters. As early as 1911, he said quite clearly, "Heredity may then be defined as *the presence of identical genes in ancestors and descendants . . .* " (Johannsen, 1911, p. 159; the italics here are Johannsen's, not ours). His unit of heredity, the gene, was neither a part of the phenotype nor a representation of it. It was a unit of information about the potential phenotype. Genes are not affected by the way that the information is used. They are extremely stable, although occasionally an accident happens and a gene mutates to a new allele, which is then inherited.

The architects of the Modern Synthesis adopted these chromosomal genes as the foundation of the revised neo-Darwinian theory. They rejected both de Vries's type of mutationism and all forms of Lamarckism. By the late 1930s, the mathematical geneticists had shown theoretically how the frequencies of different alleles in a population would alter in response to changes in the mutation rate, the intensity of selection, or when migrants entered the population or its size was restricted. Laboratory experiments and natural populations were soon showing how, give or take a bit, when there are two genetically controlled alternative characters, they behaved as the mathematical geneticists' equations predicted. So, according to the Modern Synthesis:

- Heredity is through the transmission of germ-line genes, which are discrete units located on chromosomes in the nucleus. Genes carry information about characters.
- Variation is the consequence of the many random combinations of alleles that are generated by the sexual processes, with each allele usually having a small phenotypic effect. New variations in genes—mutations—are the result of accidental changes; genes are not affected by the developmental history of the individual.
- Selection occurs among individuals. Gradually, through the selection of individuals with phenotypes that make them more adapted to their environment than others, some alleles become more numerous in the population.

One of the major figures of the Modern Synthesis, the Russian-American geneticist Theodosius Dobzhansky, in 1937 described evolution as "a change in the genetic composition of populations" (Dobzhansky, 1937, p. 11). The genes he was thinking about were, at that time, entirely hypothetical units whose existence had been deduced from numerical data obtained in breeding experiments. What a gene was chemically, and what went on between the genotype and the phenotype, were entirely unknown.

The view of heredity that was taken into the Modern Synthesis did not go unchallenged. Many embryologists maintained that heredity involves more than the transmission of nuclear genes from generation to generation. They argued that the egg cytoplasm is crucial for the inheritance and the development of species characteristics. Moreover, some European biologists, particularly those making crosses between plant varieties, insisted that their results showed that the cytoplasm influences heredity and must carry hereditary factors of some kind. They rejected what was called the

"nuclear monopoly" of the Morgan school. But in the English-speaking world their protests went largely unheeded. The influence of the Mendelist-Morganists spread as genetics was taken up by plant and animal breeders, and by the eugenicists, who wanted to "improve" human populations.

Molecular Neo-Darwinism: The Supremacy of DNA

Even though rumblings of dissent about the exclusively nuclear location of the hereditary material continued, the influence of the American and British schools of genetics grew. During the 1940s and 1950s, biochemistry developed rapidly, and many of the chemical processes that go on in cells and tissues were worked out. Geneticists began to recognize the value of microorganisms for their work, and adopted various bacteria and fungi to help them discover what genes are and what they do. Fungi have a few genetic quirks, many of which turned out to be useful, but their genetics can be studied by the classical methods of Mendelian analysis. Bacteria, on the other hand, have no proper nucleus and no pairs of chromosomes, so Mendel's rules do not apply to them. However, they do have a type of sexual process, so genetic analysis is possible. It showed that for the bacteria being studied, genes were linearly arranged on a single circular chromosome.

Through a combination of biochemical and genetic analyses using a variety of organisms, it became clear that genes are involved in the production of proteins. By the early 1950s, it was accepted that the hereditary substance was not the many chromosomal proteins, but a rather simple molecule, deoxyribonucleic acid (DNA). In 1953 Watson and Crick deciphered its structure, the famous double helix, and pointed to how it might do the job required of the genetic material. At amazing speed, molecular biology raced forward. The way DNA replicates was characterized, and the relationship between the DNA of genes and the production of proteins began to be worked out. We shall have to go into this in more detail later, but in essence what was discovered was that a DNA molecule consists of two strings of four different units, called nucleotides. Proteins are made up of one or more polypeptide chains, which are strings of another kind of unit, amino acids, of which there are twenty types. The sequences of nucleotides in DNA encode the sequences of amino acids in the polypeptide chains of protein molecules. However, the translation from DNA into proteins is not direct; the DNA sequence is first copied into mRNA (messenger ribonucleic acid, another linear sequence of nucleotides), and only then is it translated into proteins.

As the code and the way it is translated were worked out, it became clear that a change in the sequence of nucleotides in DNA often brings about a corresponding change in the sequence of amino acids in the protein it encodes. However, the way the process works seemed to offer no way in which a change in a protein could alter the corresponding nucleotides in DNA. "Reverse translation" was deemed impossible. In 1958 Francis Crick proclaimed this unidirectional flow of information from DNA to protein as the "central dogma" of molecular biology. As figure 1.5 shows, the central dogma is conceptually very similar to Weismann's doctrine, which says that somatic events cannot influence the germ line.

Up to this point, the discoveries of molecular biology had little effect on the Modern Synthesis of evolutionary biology that had been developed in the 1930s and 1940s. The gene was interpreted as a DNA sequence, which produced its phenotypic effects by coding for the proteins involved in cell structure and function. Mutations were random changes in the nucleotide sequences of DNA in the nucleus. And just as evolutionary biologists had believed for a long time, because of the central dogma there was no way in which induced phenotypic changes could have any effects on the genetic material. However, soon things began to change, and the Modern Synthesis version of neo-Darwinian evolution had to be updated.

The undercurrent of dissent about the hegemony of the nuclear gene that had been rumbling since the early days of genetics intensified.

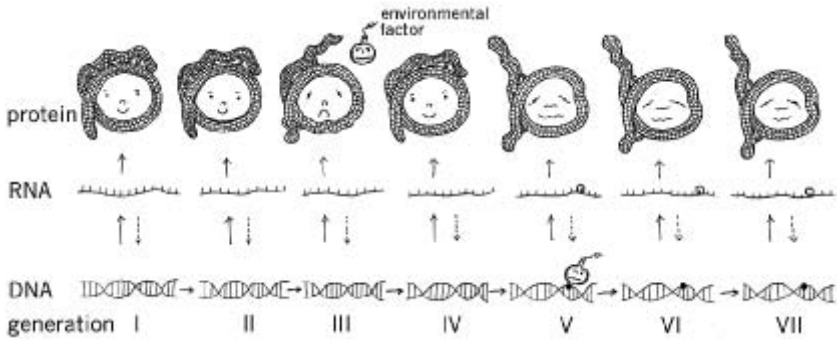

Figure 1.5
The central dogma. Induced changes in the protein product of DNA (bomb in generation III) do not affect the protein in the offspring, whereas changes in the DNA (bomb in generation V) affect the protein in all subsequent generations. Information flows from DNA to RNA to proteins (solid arrows), and possibly from RNA to DNA (dashed arrows), but never from protein to RNA or DNA.

Eventually, studies made in the 1960s confirmed what a few people had been saying for years—there are perfectly good hereditary units outside the nucleus. Genes, made of DNA, were identified in the cytoplasmic organelles known as mitochondria and chloroplasts. This meant that nuclear chromosomes could no longer be regarded as the sole repository of hereditary information.

Molecular studies also showed that there was much more variation in populations than had previously been thought. In fact, there was an embarrassingly large amount of it. It had generally been assumed that any new variant allele that cropped up in a population would either have beneficial effects, in which case it would spread through natural selection and eventually replace the original allele, or, more commonly, it would have detrimental effects and be selectively eliminated. It was recognized that occasionally two or more alleles might persist in a population, and theories about why and when this might happen had been worked out. But in the mid-1960s it was found that for many proteins there were often several allelic variants in a single population. As a result, a new spate of arguments erupted in the evolutionary community. Do all small differences in the amino acid sequence of a protein matter, as the selectionists claimed, or are most of them selectively irrelevant, and kept in the population by chance, as the neutralists said? It was not the first time that chance effects had been given a place in evolutionary theory: ever since the 1930s, Sewall Wright had maintained, somewhat controversially, that differences between small populations would arise by chance, not just by selection. His reasoning was mathematical, but now there were real biochemical data to argue about.

Eventually, after several years of heated debate, it was more or less agreed that many differences in proteins and alleles are, on average, selectively equivalent. In other words, if you think about a genetically diverse population over many generations, it will experience a lot of slightly different conditions, and a small difference in a protein will sometimes improve and sometimes reduce the survival chances or fertility of the organisms carrying it, but on average it will have no effect. As often happens, both sides in the controversy could claim to have been right.

Another cause of controversy was the result of the realization that most DNA in higher organisms does not code for proteins at all. What does this noncoding DNA do? Is it just "junk," or does it have a regulatory function? There has been a lot of argument about both the term and the idea that DNA can be "junk," and the matter is still being discussed. Some noncoding sequences are undoubtedly control sequences, which help regulate

when and where the information in DNA is processed to form proteins, but it is also true that a vast amount of DNA has no obvious function. Part of it consists of sequences that are present in many copies, either clustered together or dispersed all over the genome. Some have been found to be similar in organization to the genomes of viruses, and can change their location, moving around the genome. We will say more about these "mobile elements" and "jumping genes" in later chapters, but here we just want to note that their discovery complicated the Modern Synthesis view of the causes of changes in genes and gene frequencies.

As it was recognized that a lot of DNA is concerned with regulating gene activities rather than coding for proteins, the way people thought about hereditary information changed. They began thinking in terms of a genetic program—a set of instructions, written in the genes, which guides the development of traits. The relationship between genotype and phenotype was transformed into the relationship between a plan and a product. John Maynard Smith, an aeronautical engineer by training, likened the genotype to a plan for building an airplane, and the phenotype to the actual plane. Another British biologist, Richard Dawkins, likened the genotype to a recipe for a cake, and the phenotype to the actual cake that is baked. Changes in the recipe or in the plan lead to changes in the product, but changes in the product do not affect the recipe or plan. If a cake is accidentally burnt during its baking, it does not change the recipe; modifications made while building an airplane do not change the written plan. Only changes in plans or recipes—the programs—are inherited, not changes in products.

The discoveries in molecular biology inevitably led to a partial revision of the Modern Synthesis version of Darwinian evolution:

- The gene, the unit of heredity in the Modern Synthesis, became a DNA sequence, which codes for a protein product or an RNA molecule.
- Inheritance became associated with DNA replication, a complex but precise copying process that duplicates chromosomal DNA.
- It was recognized that in higher organisms DNA-containing chromosomes are present in the cytoplasmic organelles as well as in the nucleus.
- Mutations were equated with changes in DNA sequence, which arise through rare mistakes during DNA replication, through chemical and physical insults to the DNA and imprecise repair of the damage, and through the movement of mobile elements from one DNA site to another. Some physical and chemical agents (mutagens) increase the rate of mutation, but since they do not increase specifically those variations that are adaptive,

these induced variations, like all others, were still considered to be random, or blind.

Selfish Genes and Selfish Replicators

While the molecular biologists were busy working out what genes are and what they do, some evolutionary biologists became preoccupied with another problem—the problem of the level at which selection acts. As we mentioned earlier, in the nineteenth century Weismann and others had recognized that natural selection can occur among units other than individuals, but interest in the subject had waned. It revived in the early 1960s, when people began looking more seriously at who benefits from certain types of behavior found in group-living animals. For years most biologists had been happy to accept that some behaviors were "for the good of the species," or "for the good of the group," because they were certainly (or so it seemed) of no benefit to the individual. The most famous and extreme examples are worker ants and bees, where females work for the good of other members of their colony, but do not themselves have young. There are other less extreme examples, such as the alarm notes of birds. The bird that calls out, thereby warning others when it sees potential danger, often is not doing itself any good; on the contrary, it may make it more likely that it will be spotted and killed. It was therefore argued that this type of "altruistic" action must have been selected because it benefits the group, rather than the individual.

Not everyone agreed. A few evolutionary biologists had been pointing out for some time that the for-the-good-of-the-group argument is beset with problems. The most obvious one is that if genes crop up that make individuals selfish—that turn a bird into a noncaller, for example—then those genes will spread in the population and replace the genes for altruistic behavior. Compared with altruistic callers, who keep drawing attention to themselves, noncallers are less likely to be caught, so they will, on average, produce more offspring. Noncaller genes will increase in frequency, and eventually the population will end up as all noncallers. The only way for the altruistic calling behavior to survive in spite of this is if groups of individuals with calling behavior do very much better than groups without it. The question that had to be asked, therefore, was could a behavior (or any other characteristic) be maintained because selection between groups overrides the effects of selection between individuals within the group?

At first the mathematical evolutionists said no. So compelling were their arguments that group selectionists tended to be derided and accused of mathematical illiteracy. Later, however, different equations with different assumptions showed that evolution through group selection was possible after all. Others took different approaches to the problem of why altruism and the genes underlying it do not disappear. Bill Hamilton, one of the most original evolutionary biologists of the second half of the twentieth century, provided an answer that was seen initially as a viable alternative to the idea of group selection. He realized that the beneficiaries of most altruistic behavior tend to be the altruist's own kin. The significance of this is that an animal and its kin are likely to have inherited copies of the same genes. How many genes family members have in common depends on the closeness of their genetic relationship: it is 50 percent among parents and children, 50 percent among brothers and sisters, 25 percent among grandparents and grandchildren, and the same among half brothers and sisters; cousins share only 12½ percent of their genes. The genes that relatives have in common include, of course, any gene or genes that underlie altruistic behavior. So, if altruistic behavior leads to a large increase in the number of offspring reared by members of the altruist's family, the genes underlying the behavior may increase in frequency, even if the altruist has fewer offspring than it would have had had it not helped its kin.

Whether or not altruism genes increase in frequency depends on first, how close the relationship is (and therefore the chances that relatives carry the genes for altruism); second, by how much the altruistic behavior decreases the number of the altruist's own offspring; and third, by how much it increases the number of offspring reared by the beneficiaries of its altruistic actions. It may sound complicated, but the basic idea is very simple. From the point of view of a gene for altruism, it can increase its representation in the next generation if it makes the animals carrying it help their kin to survive and reproduce, because kin are likely to carry copies of it.

Richard Dawkins took up Hamilton's approach, extended it, and popularized it. He suggested that taking a gene's-eye view can help us to understand the evolution of *all* adaptive traits, not just the paradoxical ones like altruism. He coined the term *the selfish gene*, which recognizes that the "interests" of a gene may not coincide with the interests of the individual carrying it. Metaphorically speaking, the gene is "selfish" because the effects it has on the well-being or the reproductive success of the individual carrying it do not matter so long as they enhance the chances that it,

the gene, will have more representatives in the next generations. Adaptations are always "for the good of" the gene. They are all outcomes of competition between selfish genes.

According to Dawkins, thinking about evolution in terms of competition between rival genes, rather than between individuals or other units such as genomes, groups, or species, unifies many aspects of evolution. The gene is not just the unit that is inherited, it is also the unit that is ultimately selected. Genes have the stability and permanence that is required for units of selection, whereas most other potential units do not. If you think about individual bodies, then a child is really a rather poor copy of its parent: it does not inherit most of the features that the parent has acquired during its lifetime, and parental characters get separated and mixed up during sexual reproduction. So individual bodies are not faithfully inherited, whereas genes usually are. The living and breathing body is just a carrier—a vehicle—for selfish genes.

On the basis of his image of the selfish gene, Dawkins has constructed a unifying scheme in which he has generalized the molecular neo-Darwinian approach. He argues that genes belong to a category of entities (not necessarily made of DNA) that he calls "replicators." He defines the replicator as "anything in the universe of which copies are made" (Dawkins, 1982, p. 83). At first sight this definition seems very general, and capable of including many types of entities and processes, because "copying" is a conveniently vague word. But Dawkins immediately restricted what he meant by "copying." Bodies are not replicators, because an acquired feature, such as a scar, is not copied to the next generation. But a stretch of DNA or a sheet of paper that is photocopied is a replicator, because any change in DNA or the scribbles on a sheet of paper will be copied. "Copying" is thus restricted so that the term *replicator* cannot be applied to entities that are changed by their own development or product. To make this point Dawkins defined another entity, the "vehicle":

> A vehicle is any unit, discrete enough to seem worth naming, which houses a collection of replicators and which works as a unit for the preservation and propagation of those replicators. (Dawkins, 1982, p. 114)

Individual bodies are therefore vehicles, not replicators.

The replicator concept fits the gene so well because it is a generalization of the properties of the classical gene. The distinction between gene and body, and more generally between replicator and vehicle, is derived from Johannsen's distinction between genotype and phenotype, which was built on Weismann's view that the inheritance of acquired characters is impos-

sible. The gene-replicator in the germ line has a special status: it is the unit of heredity, of variation, of selection, and of evolution. It causes the vehicle-body to behave in a way that will increase its frequency, even at the price of sacrificing the body. The move is unidirectional: variations in genes affect corresponding variations in the body, while variation in the body, resulting from the history of the body and from the environment, do not cause corresponding variations in the gene. Development is a process that vehicles (bodies) undergo, and it is controlled by genes that replicate to ensure their own further propagation.

Notice that there is a claim here about the nature of the relationship between genes and development. According to Dawkins, heredity and variation cannot be influenced by adaptive processes that go on in individuals. There is therefore a big difference between this neo-Darwinian generalization and the version of Darwinism with which we started, which was not committed to any type of replicator-vehicle distinction or to assumptions about the origin of heritable variation. In addition to the gene, Dawkins discusses another type of replicator, the *meme*, which is a cultural unit of information that is passed among individuals and generations through cultural replication processes. We shall have more to say about this replicator in chapter 6.

Needless to say, Dawkins's selfish-gene view of evolution has not gone unchallenged. In fact it has been aggressively attacked (and defended) ever since *The Selfish Gene* was published in 1976. But as Hamilton, Dawkins, and others soon realized, a lot of the initial disagreements between those who went along with the selfish-gene view and those who insisted that individuals and groups are the focus of natural selection was the result of scientists talking past each other. The two ways of viewing evolution are not incompatible. Dawkins centers his evolution on the gene-replicator, a permanent unit whose frequency changes during evolutionary time. Other biologists center their evolutionary ideas on the targets of selection, the vehicles—the organism or groups of organisms that survive and multiply. But whatever the targets of selection—whether individuals, interacting groups of kin, or larger groups—biologists still assume that the underlying hereditary units that affect the properties of these targets are genes. Today's models of group selection are as gene-centered as any other models of natural selection, including Hamilton's explanation of the evolution of altruistic traits. Many biologists are now quite comfortable with the idea that kin selection is a form of group selection, in which the interacting kin group is the target of selection, and the unit whose frequency changes during selection is the gene.

One of Dawkins's most bitter critics was the American paleontologist Stephen Jay Gould, who insisted that focusing evolutionary ideas on genes is misleading. According to Gould, tracing the fate of genes through generations is no more than bookkeeping, because it can tell us little about evolution. It is individuals, groups, or species that survive or fail to survive, that reproduce or fail to reproduce, not genes. Moreover, said Gould, we cannot explain the varieties of animals and their adaptations solely in terms of natural selection, whether of genes, individuals, or anything else. We have to take into account historical events such as catastrophic climate changes; we have to think about accidents that affect the amount of genetic variation in populations and lineages; we have to appreciate the way evolutionary change is constrained by development, and remember the side effects that are an inevitable consequence of selection. Natural selection is just one of the many factors that have brought about the wonderful adaptations and patterns of evolution that we see in the living world. For Gould, the central focus of evolutionary studies had to be organisms, groups, and species, which are the targets of natural selection and the entities that develop. For Dawkins, it has to be the gene, the unit of heredity.

The controversy between Gould and Dawkins continued until Gould's death in 2002. Like many of the controversies that punctuated the earlier history of evolutionary thinking, it was bitter, venomous, and often unfair. Arguments were pushed *ad absurdum*, and the ambiguities of language were used and misused to erect and demolish straw men. We cannot and need not go into the details here, because for us what is important is not the disagreements, but what Gould's and Dawkins's ideas have in common. What is interesting for us is that although their different perspectives put them at opposite ends of the spectrum of views held by orthodox evolutionary biologists, they were in agreement when it came to the nature of hereditary variation. Gould and Dawkins were united in assuming that genes are the only units of heredity relevant to the evolution of organisms other than humans, and that acquired characters are not inherited.

The Transformations of Darwinism

Our account of the history of Darwinism has been sketchy, but we hope that we have said enough to show that Darwin's theory is not something set in stone. Ever since the publication of *The Origin*, the theory of natural selection has been the subject of intense debate, and its fortunes have

Table 1.1

Type of theory	Hereditary transmission	Unit of variation	Origin of variation	Target of selection	Unit of evolution
Darwin's Darwinism	Gemmules transferred from the soma to sex cells	Gemmule	Random + induced in the soma	Individual (sometimes also the group)	The population of individuals
Weismann's neo-Darwinism	Transfer of determinants through the germ line	Determinant	Random + induced in the germ line	Individual (mainly) + determinants, cells, organs	The population of individuals, cells, or determinants
Modern Synthesis neo-Darwinism	Transfer of genes in the germ line	Genes in the germ line	Random mutation	Individual	The population of individuals
Molecular neo-Darwinism	DNA replication	DNA sequence	Random DNA changes; rarely also directed changes (see chapter 3)	Mainly the individual (also the gene, the group, lineage, and species)	Mainly the population of individuals
Selfish gene neo-Darwinism	DNA replication	DNA sequence	Random DNA changes	The gene, the individual, the group	The population of alleles of the gene

waxed and waned. Sometimes the predominant view has been that it has played only a minor role in evolution; at other times, it has been seen as the most important part of the evolutionary process.

Not only has opinion about the theory of natural selection as a whole changed over the years, there have also and inevitably been changes in the details. We have summarized the various historical transformations of Darwin's theory that we have described in table 1.1. It shows how ideas about the nature of the hereditary process, the unit of heritable variation, the origin of variation, the target of selection, and the units of evolution have changed. New facts and new scientific fashions, often promoted by powerful and persuasive voices, have molded Darwin's theory of evolution into its present form.

Today, the gene-centered view of evolution predominates. It certainly provides a tidy framework for evolutionary thinking, and biologists are generally comfortable with it. That does not mean, of course, that it is the final, correct, and complete interpretation of Darwin's theory. In fact, there is a growing feeling that Darwinism is due for another transformation. We shall be putting the case for this in subsequent chapters.

Dialogue

I.M.: I am not entirely comfortable with the implications of the characterization of evolution by natural selection that you borrowed from Maynard Smith. If I am not mistaken, both Maynard Smith and Dawkins see natural selection not only as the mechanism underlying adaptive evolution but also as a kind of litmus paper for life. The conditions for natural selection—multiplication, heritable variation, and competition—are the conditions for life itself. According to this view, if we ever make robots that are able to produce robots like themselves, you will have to define them as evolving and hence alive. This contradicts our intuitions. What is your position?

M.E.: The "definition of life" issue is a really messy subject. First of all, self-production is not sufficient for there to be evolution by natural selection. You also need a mechanism through which variation that is generated during the production of robots is transmitted. Only then can you have evolution by natural selection. You have to have heritable variation. And the variation has to affect the chances of self-production.

I.M.: Let's say my robots can produce themselves and also transmit some variants that occur during the production process. But let us also assume that the number of variations is very limited—let's say that four possible

robot variants can arise, and each variant affects self-production in a different manner, which depends on the environment. Nothing very exciting can happen—you can have one of four possibilities reoccurring and changing in frequency as the environment cycles. But that's all. Would you call these robots "living"?

M.E.: John Maynard Smith and Eörs Szathmáry call these cases in which you have only a very few variants "limited heredity" systems. With them you can certainly have evolution by natural selection, but very restricted and boring evolution. Functional complexity and the evolution of functional complexity are the hallmarks of living organisms. Maybe we should be talking about different manifestations of life, rather than about whether there is a clear distinction between life and nonlife. Maybe there is no simple line of demarcation.

I.M.: Since you obviously accept the principle of natural selection, and seem to be prepared to generalize it even to self-producing and varying robots, why do you imply that Dawkins's generalization is insufficient and that Darwinism is due for another transformation? As you showed, Dawkins has suggested a unifying scheme, which allows us to understand the evolution of many different traits, both the straightforward ones and the seemingly paradoxical ones like altruism. It seems very logical to me. What is your problem with it?

M.E.: Our problem is with Dawkins's replicator/vehicle concepts. There are several difficulties. First, he assumes that a replicator has to have a high level of permanence to be a unit of evolutionary change. It has to be copied with very high fidelity. He rightly pointed out that a particular individual—Charles Darwin, for example—is unique and is never replicated, whereas his genes are. It is his faithfully replicated genes that are passed on and effect evolutionary changes. That is why, according to Dawkins, genes, not individuals, are the units of evolution. However, like many other people, we think this argument is misleading, because no one ever thought that individuals are units of heredity and selection in the sense implied by Dawkins. When looking at levels of organization above the gene, evolutionary biologists have focused on traits—for example, on Darwin's square jaw or the shape of his nose, or an aspect of his intelligence—not on whole individuals. So the alternative units should be genes or traits, not genes or individuals. Alternative traits can be traced from one generation to the next and their frequency may change. They have sufficient permanence through time to be units of evolution, even though many genes concurrently affect them and these genes are reshuffled in every generation through sex.

Our second difficulty is with Dawkins's assumption that the relation between replicator and vehicle is unidirectional—variations in the replicator (gene) affect the vehicle (body), but not vice versa. He assumes that development does not impinge on heredity, and we take issue with this assumption. Our third problem is that Dawkins assumes that the gene is the only biological (noncultural) hereditary unit. This simply is not true. There are additional biological inheritance systems, which he does not consider, and these have properties different from those we see in the genetic system. In these systems his distinction between replicator and vehicle is not valid. We will come to them in later chapters.

I.M.: So I shall wait for you to develop these arguments. Meanwhile, I want to ask you about your historical reconstruction. I realize that it is very sketchy, but you pictured the historical trend as one in which Darwinian thinking has become more and more specific about the nature of heredity and the origins of variation. Now that biology has gone so molecular, ideas about heredity and evolution are presented in ever more molecular terms. I see this as progress, and surely so do you. Yet there is a note of discontent in your story.

M.E.: Of course we welcome the molecular level of description. In fact some of the new ideas and the challenges to orthodoxy that we are going to describe in the next chapters are consequences of the new findings in molecular biology. But the molecular-genetic description does not come instead of other levels of description. We shall be making the case that some variations at the physiological and behavioral levels are heritable, and can lead to interesting processes of heredity and evolution even when there is no variation at the genetic level. At this point in time, as at most previous stages of the history of evolutionary ideas, certain findings in biology are being ignored or underplayed. That is why we decided to present today's standard view of Darwinian evolution and how it was reached historically.

I.M.: I have a question about this claim of yours that findings were underplayed or ignored at certain times in the history of evolutionary theory. It is not difficult to be wise in retrospect, and see imperfections and dogmatism, but what does it mean? It seems to me that the most important turning point in the history of twentieth-century evolutionary thinking was the formulation of the Modern Synthesis, so I'll focus on that. You mentioned the rumblings of disagreement about the importance of nuclear genes that came from certain Europeans, but there was nothing in your depiction of the Modern Synthesis to suggest that it did not accurately reflect the biology of the time. The biologists involved in the Synthesis had

a certain concept of heredity and evolution, which was derived, I assume, from what they found. It was not as if it was an ideological decision, like it was with the Lysenko doctrine in the USSR, where there was only one politically correct genetic theory. Surely the Synthesis had a wide empirical basis? What was wrong or misleading in the Modern Synthesis? Are you claiming that the view of evolution that emerged was the consequence of scientific ideology?

M.E.: It depends on how you think about ideology. At a very basic level, there is no scientific activity that is totally free from ideology. You can't build a theory without assumptions, and some of them stem from a sociopolitical general worldview, and feed into that worldview. This doesn't mean that it is a cynical and conscious type of process—that scientists are just puppets in the hands of politicians, or that power-hungry and amoral scientists are recruited for the service of an explicit ideology. Of course this can happen, as the sad story of Russian genetics during the Stalinist era testifies. German eugenics also showed it in a dreadful manner. But in many and perhaps most cases, everything is rather more subtle. Even in nontotalitarian regimes, ideological considerations appear in various guises, and they are important in determining the route of science. This occurred in the United States. There is a fascinating book written in 1966 by Carl Lindegren, an American microbial geneticist. The book is called *The Cold War in Biology*. It describes the political attitudes that surrounded the study of genetics in the West, and the discussions about the nature of the gene and the gene-environment relationship that took place during the Cold War. Self-evidently there were also scientific-ideological presuppositions about the genetic research in which some of the architects of the Synthesis were engaged. They decided what were the important things, and what belonged to the unimportant fringe.

I.M.: And what, for example, did they decide?

M.E.: The Synthesis was based on genetic research that focused on traits that could be studied using the methods of Mendelian analysis. Mendelian analysis depended on discrete qualitative traits that showed fairly regular segregation. Traits that did not behave like that were pushed aside. It was easy to believe that they were the consequences of experimental mistakes, or the overcomplexity of the system. If there are a lot of genes and they interact, it was said that the trait is obviously too difficult to analyze. Extra genes, called "modifiers," which interact with the main gene, were readily evoked whenever there were problems of interpretation. As early as 1949, Lindegren was pointing out that in the bread mould *Neurospora*, two-thirds of the mutations he found did not show Mendelian segregation. But most

scientists ignored these cases, even though they were in fact the majority. They were considered to be part of the "noise" in the system. When these deviant traits were acknowledged at all, they were excused, not studied. And even when there was agreement that there are indeed some strange phenomena—jumping genes in maize, for example, or strange inheritance of cortical structures in unicellular organisms—they were brushed under the carpet. At best they were considered to be eccentric cases that did not alter the general picture, and at worst they were simply ignored.

Animal geneticists worked mainly on the mouse or the fruit fly, and organisms that reproduced asexually were of little interest to them. They worked largely with traits that, in the jargon, show "strong developmental canalization." In other words, the organisms develop the same phenotype whatever the environmental conditions. Moreover, much of the genetics of the Synthesis was based on organisms in which the germ cells are separated off from the rest of the body early in development. In plants the germ line and soma do not separate early—you can often take a piece of stem or a leaf from a mature plant and grow another plant from it, and this plant can then produce pollen and eggs. There is no real segregation of germ line and soma in plants, and of course they are much less canalized. On the whole, the botanists were always much less dogmatic about heredity than the zoologists, but their influence on the Synthesis was not great.

I.M.: And do you think that the choices geneticists made were ideological? They seem to me to be good practical decisions.

M.E.: Of course they were not *just* ideological, and usually it was not a conscious and simple process. There was certainly an element of historical continuity. A lot of the early work was done with fruit flies, for example, and this no doubt led to a tendency to generalize from them and see all genetic phenomena in the light of this research. Again, it depends on what you mean by ideology and choice. There were conservatives, liberals, and communists participating in the Modern Synthesis. But there was also a commitment to the Mendelian view and the conception of heredity promoted by Johannsen, and a rejection of the possibility of the inheritance of acquired characters. And these views hardened as a result of the Cold War and the discovery of the charlatanism of Lysenko in the USSR, where the inheritance of acquired characters was fundamental and Mendelism was seen as a bourgeois perversion.

I.M.: What is wrong with generalizing from the genetics of the fruit fly to other species? I thought Mendel's laws were general.

M.E.: They are, but the fruit fly is really peculiar in many ways. Some of these peculiarities were a great help to genetic research, but some were a

handicap to evolutionary theorizing. For example, in the fruit fly there is very early segregation between somatic cells and germ cells; the cells of the adult fly do not divide and, in general, development is very stable. So it is difficult to see the effects of the environment on the phenotype, especially any long-term, transgenerational effects. These are much clearer in plants, for example. But there was another more human element too—there was a struggle over the way heredity should be studied. People argued over what kind of research really yields the most significant results, about the status of nuclear genes relative to cytoplasmic factors, about the place of developmental research in the study of heredity. The Mendelist-Morganists, who focused on nuclear genes and on the transmission rather than the expression of characters, won this battle. There were others who took a different approach, especially in prewar Germany, but they lost the battle for various reasons, both scientific and extrascientific.

I.M.: Today biologists are excited about what is happening in genetic engineering and molecular biology, and I know that battles are going on about what work should and should not be done, because some of it has social implications. But whatever the ideologies and whatever the decisions made, isn't it inevitable that this emphasis on molecular biology will lead to a hardening of the gene-centered approach to evolution?

M.E.: We think not. There is a lot more to molecular biology than genes, and the current selfish-gene view does not fit easily with some of the things that molecular studies are turning up. In the next chapters we will look at what molecular biology is telling us about genes and development, and you will see that what has been found is not compatible with an exclusively gene-centered view of heredity and evolution. In addition, although molecular biology is hogging the limelight and the money at present, new facts and ideas are still coming from other areas of biology, and these too are having repercussions on evolutionary thinking.