10.1 Natural selection is the only known explanation for adaptation

Before Darwin, adaptation was explained theologically, . . .

The fact that living things are adapted for life on Earth is sufficiently obvious that philosophers did not have to wait for Darwin to point it out. In Section 1.2 (p. 6) we looked at a classic example of adaptation, the woodpecker's beak. In later chapters we have met many more examples, such as camouflage in moths, mimicry in butterflies, and drug resistance in HIV. Living creatres are, in many ways, well adjusted for living in their natural environments. Adaptation was a crucial concept in *natural theology*—a school of thought that was highly influential from the eighteenth century until Darwin's time. Natural theologians explained the properties of nature, including adaptation, theologically (that is, by the direct action of God). John Ray and William Paley were two important thinkers of this type. In our time, the ideas of natural theology are still used by certain kinds of modern creationist.

Darwin himself was much influenced by the examples of adaptation, such as the vertebrate eye, discussed by Paley. Paley explained adaptation in nature by the creative action of God: when God miraculously created the world and its living creatures, he or she miraculously created their adaptations too. Natural theology was influential as a way of understanding adaptations in nature, but its main influence — beyond biology — was as an argument to prove that God exists, called the "argument from design." This is one of several classic philosophical arguments for the existence of God. Part of the reason why Darwin's theory was so controversial was that it wrecked one of the most popular (at that time) arguments for the existence of God. The key difference between natural theology and Darwinism is that the former explains adaptation by supernatural action, and the latter by natural selection.

. . . or by directed variation

Natural theology and natural selection are not the only explanations that have been put forward for adaptation. The inheritance of acquired characters ("Lamarckism") suggests that the hereditary process produces adaptations automatically. Other theories suggest that the hereditary mechanism itself produces designed, or directed, mutations and adaptation results as the consequence. These theories differ from Darwinism. In Darwinism, variation is not directed toward improved adaptation. Instead, mutation is undirected and selection provides the adaptive direction in evolution (Section 4.8, p. 88).

These alternatives can be ruled out philosophically, . . .

It is one of the most fundamental claims in the Darwinian theory of evolution that natural selection is the only explanation for adaptation. The Darwinian, therefore, has to show that the alternatives to natural selection either do not work or are scientifically unacceptable. Let us consider the natural theologians' supernatural explanation first. We can accept that an omnipotent, supernatural agent could create well adapted living things: in that sense the explanation works. However, it has two defects. One is that supernatural explanations for natural phenomena are not used in science (Section 3.13, p. 67). The second is that the supernatural Creator is not explanatory. The problem is to explain the existence of adaptation in the world, but the supernatural Creator already possesses this property. Omnipotent beings are themselves well designed, adaptively complex, entities. The thing we want to explain has been built into the explanation. Positing a God begs the question of how such a highly adaptive and well

designed thing could in its turn have come into existence. Natural theology is therefore arguably non-explanatory, and its use of supernatural causes is unscientific.

. . . factually, . . .

The "Lamarckian" theory — the inheritance of acquired characters — is not unscientific.[1] It posits a hereditary mechanism that can be tested for, and that could give rise to adaptations. Biologists generally reject Lamarckism for two reasons. One is factual. Since Weismann, in the late nineteenth century, it has generally been accepted that acquired characters are, as a matter of fact, not inherited. More than a century of genetics since Weismann has supported this view. (A few minor exceptions are known, but they do not challenge the general principle.)

. . . or theoretically

The second objection is theoretical. Lamarckism by itself arguably cannot account for the evolution of adaptation. Consider the adaptations of zebras to escape from lions. Ancestral zebras would have run as fast as possible to escape from lions. In doing so, they would have exercised and strengthened the muscles used in running. Stronger legs are adaptive as well as being an individually acquired character: if the acquired character was inherited, the adaptation would be perpetuated. Superficially, this looks like an explanation, whose only defect is that acquired characters happen not to be inherited.

Directed variation assumes, rather than explains, adaptation

Now let us imagine (for the sake of argument) that acquired characters are inherited, and look more closely at the explanation. The adaptation arises because zebras, within their lifetimes, become stronger runners. However, muscles do not by some automatic physical process become stronger when they are exercised. The muscles might just as well become weaker, because they are used up. Muscle strengthening in an individual zebra requires explanation and cannot be taken for granted. Muscles, when exercised, grow stronger because of a pre-existing mechanism which is adaptive for the organism. But where did that adaptive mechanism come from? The theoretical defect in Lamarckism is that it has no good answer to this question. To provide a complete explanation for adaptation, it would have to fall back on another theory, such as God or natural selection. In the former case it would run into the difficulties we discussed above. In the latter case it is natural selection, not Lamarckism, that is providing the fundamental explanation of adaptation. Lamarckism could work only as a subsidiary mechanism; it could only bring adaptations into existence in so far as natural selection had already programed the organism with a set of adaptive responses. Pure Lamarckism does not by itself explain adaptation.

All theories of directed or designed mutation have the same problem. A theory of directed mutation, if it is to be a true alternative to natural selection, must offer a mechanism for adaptive change that does not fundamentally rely on natural selection to provide the adaptive information. Most alternatives to natural selection do not explain adaptation at all. For example, in the early twentieth century, some paleontologists, such as Osborn, were impressed by long-term evolutionary trends in the fossil record. The titanotheres are a classic example. Titanotheres are an extinct group of Eocene and Oligocene perissodactyls (the mammalian order that includes horses). In a number of

[1] I put "Lamarckian" in quotes because, as we saw in Chapter 1, the inheritance of acquired characters was not especially important in Lamarck's own theory; nor did he invent the idea. However, the inheritance of acquired characters has generally come to be called Lamarckism and we can conveniently follow normal usage, outside purely historic discussion.

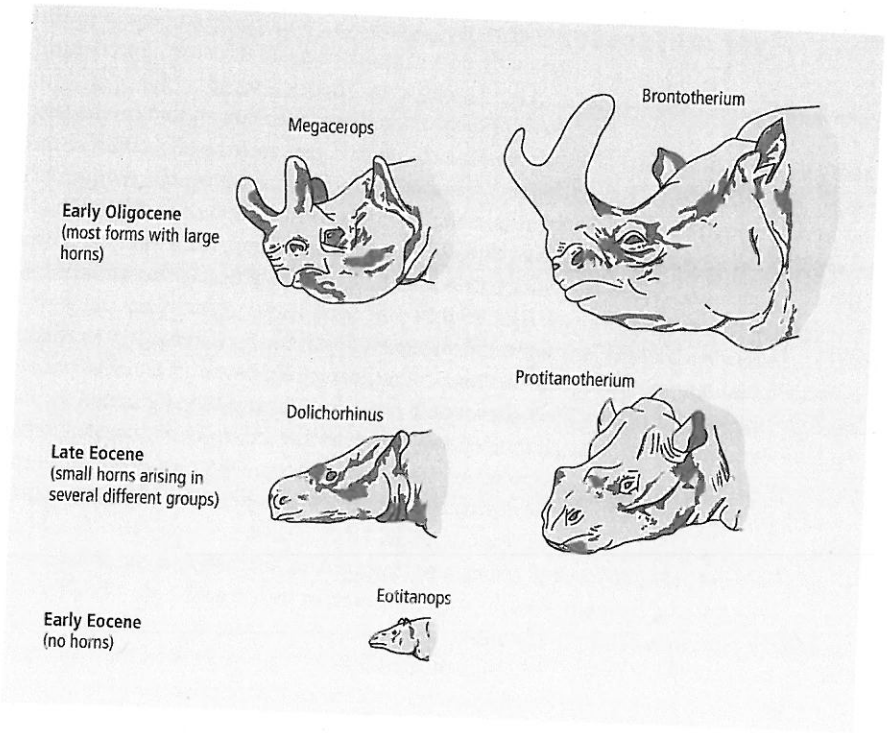

Figure 10.1
Two lineages of titanotheres showing parallel body size increase and the evolution of horns. Only two of many lineages are illustrated. Reprinted, by permission of the publisher, from Simpson (1949).

The fossil record shows some apparently directed trends . . .

lineages, the earlier forms lacked horns whereas later ones had evolved them (Figure 10.1). Osborn, and others, believed that the trend was *orthogenetic*: that it arose not because of natural selection among random mutations but because titanotheres were mutating in the direction of the trend.

Directed mutation could explain a simple, adaptively indifferent trend. If a titanothere was equally well adapted no matter what size its horns were, then a trend toward larger horns might be generated by directed mutation. In fact, the horns are thought to be adaptive, and that makes directed mutation an implausible explanation. Mutation is random with respect to adaptation (Section 4.8, p. 88). If mutation is directed, it is in a non-adaptive way. Thus, if someone explains a trend by orthogenesis (or directed mutation) we can ask how the "orthogenetic" mutations could keep on occurring in the direction of adaptive improvement. If the reply is that variation just happens to be that way, then adaptation is being explained by chance — and chance alone cannot explain adaptation, almost by definition.

. . . but they are unlikely to have been driven by directed variation

This objection is not all that strong for titanothere horns, because their adaptive function is little understood. The trends might have been possible by simple increases in size. However, for other known trends in the fossil record, such as the evolution of mammals from mammal-like reptiles (Section 18.6.2, p. 542), the objection is much more powerful. Mammals evolved over about 100 million years, during which time changes occurred in the teeth, jaws, locomotion, and physiology. Almost every feature of the animals was altered in an integrated way. Directed mutation alone would be highly unlikely to drive a complex, multicharacter, adaptive trend of this kind. A

random process alone will not explain adaptation. For this reason, directed mutation on its own, like Lamarckism, is ruled out as an explanation for adaptation.

In conclusion a strong argument can be made that natural selection is the only currently available theory of adaptation. The alternatives variously rely on chance, on unscientific causes, on processes that do not operate in fact, or are non-explanatory.

Not all evolution is adaptive

10.2 Pluralism is appropriate in the study of evolution, not of adaptation

So natural selection is our only explanation for adaptation. This statement, however, applies only to adaptation and not to evolution as a whole. Biologists, such as Gould & Lewontin (1979), have pointed out that Darwin did not himself rely exclusively on natural selection, but admitted other processes too; and they urge that we should accept a "pluralism" of evolutionary processes, rather than relying exclusively on natural selection. For evolution as a whole, this is a sensible idea. In Chapter 7, for instance, we saw that many evolutionary changes in molecules may take place by random drift. The molecular sequences among which drift takes place are not different adaptations. They are different variants of one adaptation, and natural selection does not explain why one organism has one sequence variant, and another organism has another. We need drift as well as selection in a full theory of evolution.

The fact that processes beside natural selection can cause evolutionary change does not alter the argument of Section 10.1. It just goes to show that not all evolution need be adaptive. This being so, we should be pluralists about evolution; but when we are studying adaptation, it is sensible to concentrate on natural selection.

10.3 Natural selection can in principle explain all known adaptations

The argument so far has been negative: we have ruled out the alternatives to natural selection, but we have not made the positive case for natural selection itself. We have seen before (Chapter 4) that natural selection can explain adaptation, but we can also ask a stronger question: can it explain *all* known adaptations?

The question is important historically, and it still often rises in popular discussions of evolution. The case against selection would run something like this. There is no doubt that natural selection explains some adaptations, such as camouflage. However, the adaptation in this case, as well as in other famous examples of natural selection, are all simple. In the peppered moth it is just a matter of adjusting external color to the background. The problem arises in complex characters that are adapted to the environment in many interdependent respects. Darwin's explanation for complex adaptations is that they evolved in many small steps, each analogous to the simple evolution in the peppered moth; that is what Darwin meant when he called evolution gradual. Evolution has to be gradual because it would take a miracle for a complex organ,

requiring mutations in many parts, to evolve in one sudden step. If each mutation arose separately, in different organisms at different times, the whole process becomes more probable (we look at this further in Section 10.5).

In Darwin's theory, complex adaptations evolve in many small steps

Darwin's "gradualist" requirement is a fundamental property of evolutionary theory. The Darwinian should be able to show for any organ that it could, at least in principle, have evolved in many small steps, with each step being advantageous. If there are exceptions, the theory is in trouble. In Darwin's (1859) words, "if it could be demonstrated that any complex organ existed which could not possibly have been formed by numerous successive slight modifications, my theory would absolutely break down."

Darwin argued that all known organs could have evolved in small steps. He took examples of complex adaptations and showed for them how they could have evolved through intermediate stages. In some cases, such as the eye (Figure 10.2), these intermediates can be illustrated by analogies with living species, in other cases they can only be imagined. Darwin only had to show that the intermediates could possibly have existed. His critics had the more difficult task of showing that the intermediates could not have existed. It is very difficult to prove negative statements. Nevertheless, many critics suggested, for various adaptations, that natural selection cannot account for them. These types of adaptation can be considered under two headings.

Coadaptations

Critics suggest that complex organs cannot evolve by natural selection

Coadaptation here refers to complex adaptations, the evolution of which would have required mutually adjusted changes in more than one of their parts. (Coadaptation is a popular word: it has already been used in a different sense in Chapter 8, and will be used in a third sense in Chapter 20!) In a historic dispute in the 1890s, Herbert Spencer and August Weismann discussed the giraffe's neck as an example. Spencer supposed that the nerves, veins, bones, and muscles in the neck were each under separate genetic control. Any change in neck length would then require independent, simultaneous changes of the correct magnitude in all the parts. A change in the length of the neck-bones would malfunction without an equal change in vein length, and evolution by natural selection on one part at a time would be impossible. The example is unconvincing now because of the obvious retort that the lengths of all the parts could be under common genetic control.

The other standard example of a complex coadaptation is the eye. When one eye part, such as the distance from the retina to the cornea, changes during evolution, changes in other parts, such as lens shape, would (it is said) be needed at the same time. Because of the improbability of simultaneous correct mutations in both parts at the same time, a complex, finely adjusted engineering device like the eye could not therefore have evolved by natural selection. The Darwinian reply (illustrated in Figure 10.2) is that the different parts could evolve independently in small steps: it is not necessary for all the parts of an eye to change at the same time in evolution.

A computer model study by Nilsson & Pelger (1994) illustrates the power of Darwin's argument. Although the eye of a vertebrate or an octopus looks so complex that it can be difficult to believe it could have evolved by natural selection, in fact light-sensitive organs (not all of them complex) have evolved 40–60 times in various invertebrate groups — which suggests either that the Darwinian explanation faces a 40- to

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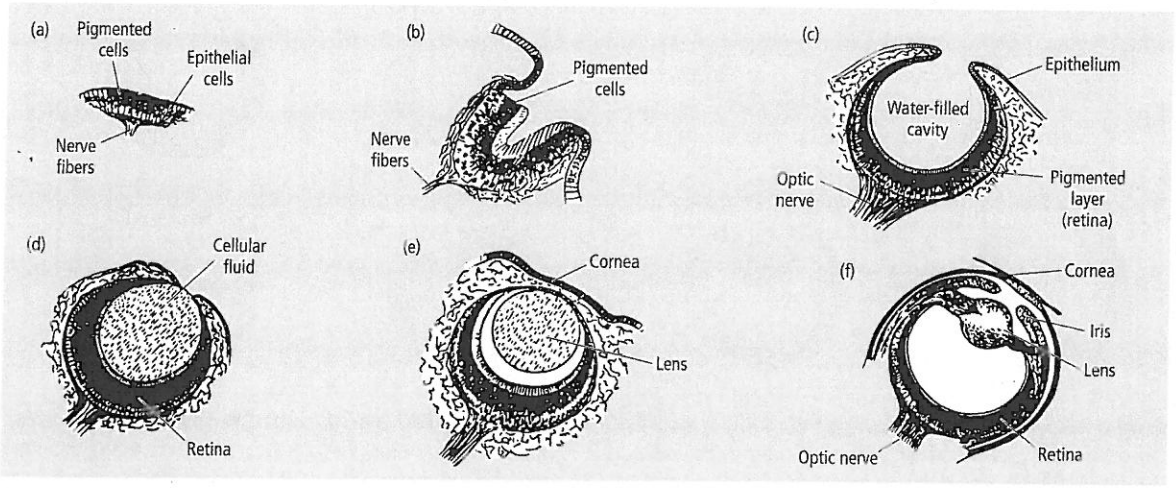

Figure 10.2
Stages in the evolution of the eye, illustrated by species of mollusks. (a) A simple spot of pigmented cells. (b) A folded region of pigmented cells, which increases the number of sensitive cells per unit area. (c) A pin-hole camera eye, as is found in *Nautilus*. (d) An eye cavity filled with cellular fluid rather than water. (e) An eye is protected by adding a transparent cover of skin and part of the cellular fluid has differentiated into a lens. (f) A full, complex eye, as found in the octopus and squid. Redrawn, by permission of the publisher, from Strickberger (1990).

A simulation suggests that the eye could easily evolve in gradual advantageous stages . . .

60-fold more difficult problem than the vertebrate eye alone presents, or that it may not be so difficult for the things to evolve after all.

Nilsson and Pelger simulated a model of the eye to find out how difficult its evolution really is. Their simulation began with a crude light-sensitive organ consisting of a layer of light-sensitive cells sandwiched between a darkened layer of cells below and a transparent protective layer above (Figure 10.3). The simulation, therefore, does not cover the complete evolution of an eye. To begin with it takes light-sensitive cells as given (which is an important but not absurd assumption, because many pigments are influenced by light) and at the other end it ignores the evolution of advanced perceptual skills (which are more a problem in the evolution of the brain than the eye). It concentrates on the evolution of eye shape and the lens; this is the problem that Darwin's critics have often pointed to, because they think it requires the simultaneous adjustment of many intricately related parts.

From the initial simple stage, Nilsson and Pelger allowed the shape of the model eye to change at random, in steps of no more than 1% change at a time. One percent is a small change, and fits in with the idea that adaptive evolution proceeds in small, gradual stages. The model eye then evolved in the computer, with each new generation formed from the optically superior eyes in the previous generation; changes that made the optics worse were rejected, just as selection would reject them in nature.

The particular optical criterion used was visual acuity or the ability to resolve objects in space. The visual acuity of each eye in the simulation was calculated by methods of optical physics. The eye is particularly well suited to this kind of study because optical

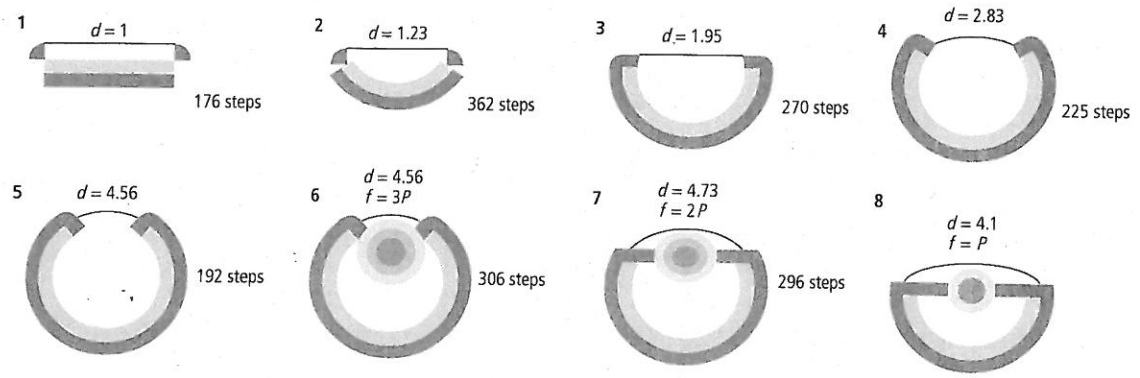

Figure 10.3
Eight stages in the evolution of the eye in a computer model. The initial stage has a transparent cell layer, a light-sensitive cell layer, and a dark pigmented bottom cell layer. It first improves its optical properies by buckling in (up to stages 4–5); by stage 5 it approximately corresponds to the pin-hole camera eye (see Figure 10.2c). It then improves by the evolution of a lens (stage 6). The lens shape then changes, and the iris flattens, to improve the focusing properties. *f* is the focal length of the lens; it has the best optical properties when *f* equals the distance from the lens to the retina (*P*): this feature gradually improves in the final three phases (stages 6–8). *d* indicates the change in shape and is the normalized diameter of the eye. Redrawn, by permission of the publisher, from Nilsson & Pelger (1994).

qualities can readily be quantified: it is possible to show objectively that one model eye would have better acuity than another. (It is not so easy to imagine how to measure the quality of some other organs, such as a liver or a backbone.) The simulated eye duly improved over time, and Figure 10.3 shows some of the phases along the way. After 1,000 or so steps the eye had evolved to be rather like a pin-hole camera eye (Figure 10.2c shows a real example). Then, the lens started to evolve by a local increase in the refractive index of the layer that had started out simply as transparent protection. The lens to begin with had poor optical qualities but its focal length improved until it equaled the diameter of the eye, at which point it could form a sharply focused image.

. . . in less than half a million generations

How long did it take? The complete evolution of an eye like that of a vertebrate or octopus took about 2,000 steps. What had looked like an impossibility actually turns out to be possible in a short interval of time. Nilsson and Pelger (1994) used estimates of heritability and strength of selection (Section 9.7, p. 236) to calculate how long the change might take; their answer was about 400,000 generations. With a generation time of 1 year, the evolution of an eye from a rudimentary beginning would take less than 0.5 million years. Far from being difficult to evolve, the model shows that it is rather easy.

The work also illustrates the value of building models to test our intuitions. Darwin himself referred to the evolution of complex organs by natural selection as presenting a problem for the imagination, not the reason. Nilsson and Pelger's computer study supports his remark.

Functionless, or disadvantageous, rudimentary stages

An organ has to be advantageous to its bearer at all stages in its evolution if it is to be produced by natural selection. Some adaptations, it is said, although undoubtedly

Some critics suggest that the initial stages of a character would be disadvantageous

advantageous in their final form, could not have been when in a rudimentary form: "What is the use of half a wing?" is a familiar example. The anatomist St George Jackson Mivart particularly stressed this argument in his *The Genesis of Species* (1871). The Darwinian reply has been to suggest ways in which the character could have been advantageous in its rudimentary form. In the case of the wing, partial wings might have broken the force of a fall from a tree, or protowinged birds might have glided from cliff tops or between trees — as many animals, such as flying foxes, do now. These early stages would not have required all the muscular back up of a full, final wing. The concept of preadaptation (see below) provides another solution to the problem.

Evolutionary biologists are sometimes challenged with arguments about functionless rudimentary stages or the impossibility of complex adaptive evolution. It is impossible to imagine, someone will insist, how such-and-such a character could have evolved in small, advantageous steps. In reply, the evolutionary biologist may offer a possible series of stages by which the character might have evolved. We need to keep in mind the status of the evolutionary biologist's argument here. The series of stages may in some cases not be particularly plausible, or well supported by evidence, but the argument is put forward solely to refute the suggestion that we cannot imagine how the character could have evolved.

A second order of critics may latch on to the argument at this point and accuse evolutionists of making speculative, even fanciful, suggestions about the stages through which individual adaptations could have evolved. But the critics overlook the original point of the discussion. The speculations are not the prize specimens of evolutionary analysis. It is not being claimed that the series is particularly profound or realistic, or even very probable. The long evolutionary history that precedes any complex modern adaptation will appear, with hindsight, to be an improbable series of accidents: the same point is as true for human history as evolution. Given the state of our knowledge at any one time, for some characters we can reconstruct their evolutionary stages with some rigor (Chapter 15), but for others we cannot — and for these it is only possible to make guesses to illustrate possibilities, not conduct a careful scientific investigation.

It is fair to conclude that there are no known adaptations that definitely could not have evolved by natural selection. Or (if the double negative is confusing!), we can conclude that all known adaptations are in principle explicable by natural selection.

10.4 New adaptations evolve in continuous stages from pre-existing adaptations, but the continuity takes various forms

10.4.1 *In Darwin's theory, no special process produces evolutionary novelties*

We saw in the previous section that Darwin's theory of adaptation is "gradualist." New adaptations evolve in small stages from pre-existing organs, behavior patterns, cells, or molecules. Another way of saying the same thing is to say that there is continuity between all the forms of adaptation that we see in the world today. This view of continuity contrasts with, for example, a creationist view of life in which the

adaptations of different species originate separately and there is no continuity between them.

Gradual change produces novelties

The continuity of adaptive evolution can challenge our understanding of novelty. During evolution, organs do arise that can be described as evolutionary novelties. The vertebrate eye, for example, exists in vertebrates including ourselves, but is not found in all life. It was in some sense newly evolved during vertebrate ancestry. It is eventually recognizable as a new structure that did not exist before. However, as we saw in the previous section, the eye evolved in continuous small stages ultimately from ancestral photoreceptive cells on the body surface. There is no distinct stage at which the "eye" sudenly and distinctly came into existence. The vertebrate eye evolutionarily blurs out through multiple ancestral stages. Thus something that we recognize as a novelty can arise even though it evolved through the modification of previously existing structures.

In Darwin's theory, no special evolutionary process operates to create new structures. The same evolutionary process of adaptation to the local environment is at work throughout. The cumulative effect of many small modifications can be such that something "new" has arisen. (This view of evolutionary novelty is not universally agreed by biologists. Some biologists do argue that evolutionary novelty is a special process: however, they would probably agree that theirs is a minority view.)

10.4.2 *The function of an adaptation may change with little change in its form*

During the evolution of the eye, the function of the organ was relatively constant throughout. From simple photoreceptive cells to full eyes, the organ was a sense organ — sensitive to light. Probably, many organs evolve in this way, by gradual transformation of a structure that has a constant function. In other cases, organs can change their function with relatively little change in structure. Feathers are an example, suggested by dramatic, recently excavated, evidence from fossils in China. Feathers are found in modern birds and mainly function in flight. Birds likely evolved from a group of dinosaurs, and dinosaur fossils typically lack feathers. We might therefore infer that feathers evolved along with flight during the origin of birds.

Feathers preceded flight in the evolution of birds

However, in the past 5 years or so a series of fossils have been described from China (Prum & Brush 2002). The fossils are described as non-avian dinosaurs, but they have feathers or rudimentary feathers. Feathers probably originally evolved for some function other than flight — thermoregulation, perhaps, or display. Later on, flight evolved and feathers turned out to be useful aerodynamically. Feathers then took on their modern function. (Feathers are still used in display and thermoregulation so it might be more accurate to say a function was added, rather than changed. Alternatively, we could say that a function of flight plus display is a change from a function of display alone.)

The classic Darwinian term for a case such as the feathers in non-avian dinosaurs is *preadaptation*. A preadaptation is a structure that happens to be able to evolve some new function with little change in structure. A second example is the tetrapod leg. Fish lack legs, which evolved during the evolution of amphibians and are now used for walking on land. Fossil evidence, such as from *Acanthostega*, suggests that legs originally evolved for underwater swimming. The bone structure of swimming paddles in one

Box 10.1
Molecular Cooption

The term *cooption* is often used to describe the evolutionary process in which a molecule takes on a new function, but with little change in structure. Cooption is conceptually much the same as preadaptation. One term (cooption) happens to be used more about molecules, and the other (preadaptation) about morphology.

The crystallins are a remarkable example. These are the molecules that make up the lens in the eye. Many unrelated molecules appear to be able to function as lens proteins, and the exact molecule that is found in the lens can change during evolution. The lens of human eyes, like those of many vertebrates, contains α-crystallin, which is very similar to a heat-shock protein and probably evolved by gene duplication from a gene coding for a heat-shock protein. The eye lenses of some other vertebrate taxa contain other crystallins that are unrelated to the heat-shock protein. A few birds, and crocodiles, use ε-crystallin, which has much the same sequence as (and indeed is) lactate dehydrogenase. The usual crystallin of birds and reptiles is δ-crystallin, which is arginosuccinate lyase. Other odd crystallins are found in individual taxa such as elephant shrews. Apparently all that is needed for a molecule to serve as a crystallin is that it forms a certain globular shape. Many enzymes meet this requirement, and during evolution the molecules that have been used as lens proteins have chopped and changed while the lens itself has remained much the same. (The crystallins make an interesting case study in homology. The lens of human eyes is homologous with the lens of a crocodile eye. The molecules that make up the lenses are not. On homology, see Section 15.3, p. 427.)

The emerging subject of "evo-devo" (Chapter 20) is documenting many examples of molecular cooption. In embryonic development, certain regulatory genes code for subroutines that can be useful in many circumstances. A gene that regulates how far a limb grows before the feet start to develop may also prove useful in regulating the size of an "eyespot" pattern on a butterfly wing. In both cases, some embryonic process has to operate for a certain time, or across a certain space, and then come to a stop. Much the same genetic instructions may be able to control the development of both limbs and eyespots.

Further reading: Raff (1996), Carroll *et al.* (2001), Gould (2002b).

group of creatures turned out to be appropriate for a leg that could walk on land. (Section 18.6.1, p. 540, describes the fish–amphibian transition.)

Many further examples of preadaptation are being discovered in molecular evolution and Box 10.1 gives an example.

10.4.3 *A new adaptation may evolve by combining unrelated parts*

One enzyme used in milk synthesis evolved from two unrelated enzymes

So far we have seen how new adaptations may evolve by changes in structures that have a constant function, or by changes in the function of a structure. A third possibility is that a novelty may result when two pre-existing parts are combined. For example, the use of milk to feed the young is a unique feature in mammals. Mammals evolved from reptiles, who did not produce milk. The full story of the evolution of lactation has many components. One of them is the evolution of the enzymatic machinery to synthesize milk. Milk contains large amounts of a sugar, lactose, and mammals have evolved a new enzyme — lactose synthetase — to manufacture it. Lactose synthetase catalyzes the conversion of glucose into lactose and is made up of modified versions of two pre-existing enzymes, galactosyl transferase and α-lactalbumin. Galactosyl transferase functions in the golgi apparatus of all eukaryotic cells and α-lactalbumin is related to the enzyme lysozyme that all vertebrates use in their antibacterial defenses. In this example, an

evolutionary novelty resulted from the combination of two pre-existing parts with unrelated functions. A lactose-manufacturing enzyme evolved by combining a golgi enzyme and an antibacterial enzyme.

Evolution can proceed by symbiosis

The evolution of milk digestion is a molecular example in which a new enzyme evolved by combining two pre-existing enzymes. A related process operates at a higher level when two whole species merge by symbiosis and evolve into a new species with a new combined physiology. For example, the mitochondria and chloroplasts in eukaryotic cells each originated when one bacterial cell engulfed another bacterial cell. In the case of mitochondria, the combined cell was capable (or soon evolved to be capable) of burning carbohydrates in oxygen — a process that releases more energy than anerobic respiration. The new cell had a more complex metabolism than either ancestral cell by itself.

Evolution by symbiosis, or combining several genes into new composite genes, can violate the letter, but not the spirit, of Darwinian gradualism. According to the gradualist requirement, new adaptations evolved in many small, continuous stages. When two cells merge, there may be a relatively sudden transition to a new adaptation in one big step. However, no deep principle in Darwinism has been violated because the adaptive information within each ancestral cell was built up in gradual stages.

10.5 Genetics of adaptation

10.5.1 *Fisher proposed a model, and microscope analogy, to explain why the genetic changes in adaptive evolution will be small*

Adaptations have been suggested to evolve in few, large genetic steps, or many, small ones

Evolutionary biologists distinguish between a "Fisherian" and a "Goldschmidtian" view of the genetic steps by which adaptations evolve. Goldschmidt (1940) argued that new adaptations, and new species, evolve by macromutations (or "hopeful masters"). A macromutation is a mutation with a large phenotypic effect, such that the individual carrying the mutation is outside the normal range of variation for its population (Figure 1.7, p. 14). Fisher doubted whether macromutations contribute much to evolution, and argued that adaptive evolution mainly proceeds in many small steps. The mutations that contribute to adaptive evolution have small phenotypic effects.

Fisher's argument begins by noting that living things are fairly well adapted to their environments. They must be at least reasonably well adjusted, or they would be dead. Next, Fisher assumes that most characters are in an optimally adapted state. If the character is larger or small than the optimum, the organism's fitness declines (Figure 10.4a). Because living organisms are at least fairly well adapted, they are somewhere near the peak in Figure 10.4a. We now assume that the direction of mutations is random with a mutation having a 50% chance of increasing the character state, and a 50% chance of decreasing it. A small mutation therefore has a 50% chance of improving the adaptation. But a large mutation would make things worse either way. It either is directed away from the optimum, or shoots past the optimum down the slope on the other side (Figure 10.4a). Fisher calculated, on the assumption that the organism is near the adaptive peak, that an indefinitely small mutation has a half chance of improving the adaptation, and the

10.5.4 *Conclusion: the genetics of adaptation*

We have met four theories about the genetic changes that occur during adaptive evolution. The "Goldschmidt" theory, that adaptations evolve by macromutations, has been rejected because of its theoretical implausibility. Macromutations will almost always reduce the quality of adaptation. Wright's theory, that adaptations evolve by the shifting balance process, has not been the topic of this section; but should be included for completeness. We saw in Section 8.13 (p. 216) that the shifting balance theory continues to inspire research, but no one has yet shown it to be important in evolution. Fisher's original theory suggested that adaptive evolution proceeds only by many mutational steps each of small effect. This theory has never been ruled out (or ruled in), and has been highly influential. However, modern research is looking at an expanded theory, that builds additional factors onto Fisher's basic model. Experimental work may be able to test what mix of large or small mutations contribute to adaptive evolution, depending on the ecological conditions.

10.6 Three main methods are used to study adaptation

"Whether" and "how" a character is adapted are different questions

We should distinguish two questions about any character of an organism. One is *whether* it is adaptive. The other is (if the character is an adaptation) *how* it is an adaptation. The first question is complicated, because the answer will depend on what definition of adaptation is used. Several definitions exist, and the methods of recognizing adaptations vary from definition to definition. We shall return to the question in Section 10.8 below. Here we can look at the methods used to study adaptations, to work out how the attribute in question is adaptive.

The study of adaptation proceeds in three conceptual stages. The first is to identify, or postulate, what kinds of of genetic variant the character could have. Sometimes, as in peppered moths (Section 5.7, p. 108) for instance, this is done empirically. Other characters do not vary genetically and for them it is necessary to postulate appropriate theoretical mutant forms. For example, when we come in Chapter 12 to look at why sex exists, we shall postulate mutant forms that reproduce clonally, or asexually.

Adaptation can be studied . . .

The second stage is to develop a hypothesis, or a model, of the organ or character's function. The original hypothesis for peppered moths was that coloration functioned as camouflage. Hypotheses are of varying quality, but they can be improved on as work proceeds. As we saw in Section 5.7 (p. 108), melanic coloration in peppered moths seems to have some other advantage in addition to camouflage in polluted areas. Another example comes from beak shape in birds. In this book, we shall often consider beak shape as an adaptation to the food supply. Larger beaks are better at eating larger and tougher food items, as we saw from the Grant's research on Darwin's finches (Section 9.1, p. 223). However, beaks have other functions too, including lice preening, and beak shape matters for those other functions (Clayton & Walther 2001).

. . . by engineering models, . . .

A good hypothesis is one that predicts the features of an organ exactly, and makes testable predictions. In morphology, these predictions are often derived from an engineering model. For example, hydrodynamics is used to understand fish shape, while

Table 10.1
The wing stripe of some butterflies was painted over, and controls were painted with transparent paint that did not affect their appearance. The number with intact wings at different times after the treatment was measured. The frequency distributions are not significantly different. From Silbergleid *et al.* (1980).

Age at capture (week)	Painted butterflies		Controls	
	n	%	*n*	%
0	81	83.5	88	90.
1	14	14.4	6	6.
2	2	2.1	2	2.
3	0	0	1	1.
4	0	0	0	0
5	0	0	0	0

construction engineering is used for shell thickness in a mollusk: the costs of buildi thicker shell have to be weighed against the benefits of reduced breakage, by v action or predators. This sort of research can be carried out at all levels, from the sin and qualitative through to sophisticated algebraic modeling.

Stage three is to test the hypothesis's predictions. Three main methods are availa One is simply to see whether the actual form of an organ (or whatever charact under investigation) matches the hypothetical prediction; if it does not, the hypoth is wrong somehow.

. . . by experiment, . . .

A second method is to do experiments. It is only useful if the organ, or behavior p tern, can be altered experimentally. Almost any hypothesis about adaptation will p dict that some specified form of an organ will enable its bearer to survive better t some other forms, but the alternatives are not always feasible. We cannot, for exam make an experimental pig with wings to see whether flight would be advantage When they are possible, experiments are a powerful means of testing ideas about ad tation. Animal coloration, for instance, has been studied in this way. Color pattern some butterfly species are believed to act as camouflage by "breaking up" the butterf outline. Silberglied *et al.* (1980), working at the Smithsonian Tropical Resea Institution at Panama, experimentally painted out the wing stripes of the butte *Anartia fatima*. The butterflies with their wing stripes painted out showed similar le of wing damage (which is produced by unsuccessful bird attacks) and survived equ as well as control butterflies (Table 10.1); the wing stripes, therefore, are not in f adaptations to increase survival. They may have some other signaling or reproduct function, though that would need to be tested by further experiments.

. . . and by the comparative method

The *comparative method* is the third method of studying adaptation. It can be use the hypothesis predicts that some kinds of species should have different forms of adaptation from other kinds of species. Darwin's classic study of the relation betw sexual dimorphism and mating system is an example we shall discuss below. So

hypotheses predict that different kinds of species will have different adaptations, others do not. Darwin's theory of sexual dimorphism does; but, for example, an optical engineer's model of how the eye should be designed might specify just a single best design, with the implication that all animals with eyes should have that design. The comparative method would in that case be inapplicable.

In summary, the three main methods of studying adaptation are to compare the predicted form of an organ with what is observed in nature (and perhaps also to measure the fitness of different forms of organism), to alter the organ experimentally, and to compare the form of an organ in different kinds of species.

10.7 Adaptations in nature are not perfect

Natural selection has brought into existence creatures that are in many respects marvellously well designed. The designs, however, are generally imperfect, and for a number of reasons. We shall look at several reasons in this chapter. In Chapter 11 we shall see another reason: that it may not be possible for an adaptation to be simultaneously perfect at all levels of organization. For example, birth control may be good for the population but not the individual. Most of the familiar examples of adaptation benefit the organism. They will therefore be (at best) imperfect at other levels, such as the genic, cellular, and group levels. However, we can still ask whether organismal adaptations are perfect even for the organism.

The quality of adaptation will progressively improve for as long as there is genetic variation to work on. If some genetic variants in the population produce a better adaptation than others, natural selection will increase their frequency. Although this process must always operate in the direction of improvement, it has never reached the final state of perfection. As Maynard Smith (1978) remarked, "if there were no constraints on what is possible, the best phenotype would live for ever, would be impregnable to predators, would lay eggs at an infinite rate, and so on." What are the constraints that prevent this kind of perfection from evolving?

10.7.1 *Adaptations may be imperfect because of time lags*

Fruits coevolve with animals

Many flowering plants produce fruits, in order to induce animals to act as dispersal agents. The fruits of different species are adapted in various ways to the particular animals they make use of. They must be attractive to the relevant animal, but also protect the seed from destruction by the animal's digestive system; they also must remain in the animal's gut for about the right amount of time to be dispersed an appropriate distance from the parent and then be properly deposited, which can be achieved by laxatives in the fruit. Many details are known about the ways in which individual fruits are adapted to the habits and physiology of the animal species that disperse them. Over evolutionary time, plants presumably have adapted the form of their fruits to whatever animals are around, and when the fauna changes, the plants will evolve (or rather coevolve — see Chapter 22), in time, to produce a new set of adapted fruits.

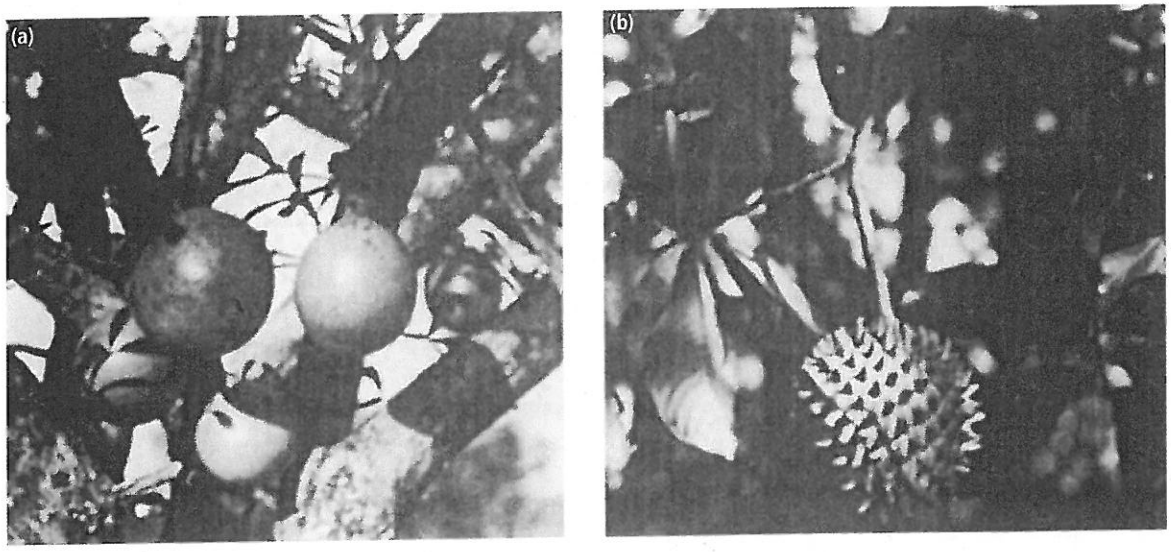

Figure 10.6
The fruits of (a) *Crescentia alata* (Bignoniaceae) and (b) *Annona purpurea* (Annonaceae) are two examples of fruits that were probably eaten by large herbivores that recently went extinct. The larger fruits in (a) are about 8 in (20 cm) long; the fruit in (b) is nearer 12 in (30 cm) long. Both trees were photographed in Santa Rosa National Park, Costa Rica. (Photos courtesy of Dan Janzen.)

But some fruits appear adaptively out of date . . .

Natural selection, however, takes time, and there will be a period after a major change in the fauna during which the adaptations of fruits will be out of date, and adapted to an earlier form of dispersal agent. Janzen & Martin (1982) have argued that the fruits of many trees in the tropical forests of Central America are "neotropical anachronisms" (see Figure 17.2, p. 495, for the geographic term neotropical). The fruits are anachronistically adapted to an extinct fauna of large herbivores (Figure 10.6).

Until about 10,000 years ago North and Central America had a fauna of large herbivores comparable in scale to that of Africa in recent times. Just as Africa has elephants, giraffes, and hippopotamuses, in Central America there were giant ground sloths, a giant extinct bear, a large extinct species of horse, mammoths, and a group of large relatives of mastodons called gomphotheres. These mammals now are all gone, but the species of trees that they used to walk beneath still remain. In the tropical forests of Costa Rica, some trees still drop large and hard fruits in great quantities. It accumulates, and much of it rots, at the base of the trees, and those that are moved by small mammals such as agoutis are not moved far. Here is how Janzen & Martin (1982) describe the fruiting of the large forest palm *Scheelea rostrata*: "in a month as many as 5000 fruits accumulate below each fruit-bearing *Scheelea*-palm. The first fruits to fall are picked up by agoutis, peccaries, and other animals that are soon satiated. . . . The bulk of the seeds perish directly below the parent." The fruits seem overprotected with their hard external coverings, they are produced in excessive quantities, and they are not adapted for dispersal by small animals like agoutis. It looks like a case of maladaptation: "a poor adjustment of seed crop size to dispersal guild." However, the fruits make

. . . as if they are adapted for a recently extinct megafauna

sense if they are anachronistic adaptations to the large herbivores that have so recently gone extinct. The large size would have been appropriate for a gomphothere, and the hard external cover would have protected the seeds from the gomphothere's powerful crushing teeth. Ten thousand years has not been long enough for the trees to evolve fruits appropriate to the more modestly sized mammals that now dwell among them.

The principle illustrated by the fruits of these Central American plants is a general one. Adaptations will often be imperfect because evolution takes time. The environments of all species change more or less continually because of the evolutionary fortunes of the species they compete, and cooperate, with. Each species has to evolve to keep up with these events, but at any one time they will lag some distance behind the optimal adaptation to their environment. Adaptation will be imperfect when natural selection cannot operate as fast as the environment of a species changes. (Box 22.1, p. 624, contains further discussion of fruit coevolution.)

10.7.2 *Genetic constraints may cause imperfect adaptation*

When the heterozygote at a locus has a higher fitness than either homozygote, the population evolves to an equilibrium at which all three genotypes are present (Section 5.12, p. 123). A proportion of the individuals in the population must therefore have the deleterious homozygous genotypes. This is an example of a *genetic constraint*. It arises because the heterozygotes cannot, under Mendelian inheritance, produce purely heterozygous offspring: they cannot "breed true." In so far as heterozygous advantage exists, some members of natural populations will be imperfectly adapted. The importance of heterozygous advantage is controversial, but there are undoubted examples such as sickle cell hemoglobin, which is indeed a practical manifestation of imperfect adaptation due to genetic constraint.

50% of European crested newt offspring die, because of a genetic peculiarity

The balanced lethal system of the European crested newt *Triturus cristatus* is a more dramatic example. Members of the species have 12 pairs of chromosomes, numbered from 1 to 12, 1 being the longest and 12 the shortest. Macgregor & Horner (1980) found that all individual crested newts of both sexes are "heteromorphic" for chromosome 1: an individual's two copies of chromosome 1 are visibly different under the microscope. They named the two types of chromosome 1, 1A, and 1B (the same two types are found in every individual). Meiosis, they found, is normal so that an individual produces equal numbers of gametes with 1A chromosomes as with 1B chromosomes. There is also little, if any, recombination between the two chromosomes.

The puzzle is why there are no chromosomally homomorphic newts, with either two 1A or two 1B chromosomes. Macgregor and Horner carried out breeding experiments, in which they crossed two normal individuals, and counted the proportion of eggs that survived. In every case, approximately half the offspring died during development. It is almost certainly the homomorphic individuals that die off, leaving only the heteromorphs.

The reason why half the offspring die is as follows. The adult population has two types of chromosome, each with a frequency of one-half. If we write the frequency of the 1A chromosome as p and of the 1B chromosome as q, $p = q = {}^1/_2$. By normal Mendelian segregation, and the Hardy–Weinberg principle, the proportion of homozygotes (or homomorphs) is $p^2 + q^2 = {}^1/_2$. In each generation, therefore, the

heterozygous newts mate together and produce half homozygous offspring and half heterozygous offspring, and then all the homozygotes die. The system looks incredibly inefficient, because half the reproductive effort of the newts each generation is wasted; but the same sort of inefficiency exists, to some extent, at any genetic locus with heterozygous advantage. If in humans a new hemoglobin arose that was resistant to malaria and viable in double dose, or in the crested newt a new chromosome 1 arose that was viable as a homomorph, it should spread through the population. Presumably the inefficiency remains only because no such mutations have arisen.

Heterozygous advantage may lead to gene duplication

Could a system with heterozygous advantage easily evolve into a pure breeding genotype with the same phenotypic effect? It probably could by gene duplication (Section 2.5, p. 30). Imagine that the relevant hemoglobin gene duplicated in an Hb^{+}/Hb^{s} individual, to become $Hb^{+}Hb^{+}/Hb^{s}Hb^{s}$. Genetic recombination could then produce a $Hb^{+}Hb^{s}$ chromosome, and that chromosome should be able to achieve anything that an Hb^{+}/Hb^{s} heterozygote can. The chromosome would also breed true, once it had been fixed. We might expect therefore that the existing Hb^{+}/Hb^{s} system would evolve to a pure $Hb^{+}Hb^{+}/Hb^{s}Hb^{s}$ system. Some "dosage compensation" might be needed after the gene had duplicated, but that should be no difficulty because regulatory devices are common in the genome. The apparent ease of this evolutionary escape from heterozygous advantage and segregational load is one possible explanation for the (apparent) rarity of heterozygous advantage. However that may be, the existence of some cases of heterozygous advantage suggests that natural populations can be imperfectly adapted because a superior mutation has not arisen.

10.7.3 *Developmental constraints may cause adaptive imperfection*

Developmental systems influence the course of evolution

A nine-penned discussion (Maynard Smith *et al.* 1985) of *developmental constraints* gave the following definition: "a developmental constraint is a bias on the production of variant phenotypes or a limitation on phenotypic variability caused by the structure, character, composition, or dynamics of the developmental system." The idea is that different groups of living things that evolved distinct developmental mechanisms, and that the way an organism develops will influence the kinds of mutation it is likely to generate. A plant, for example, may be likely to mutate to a new form with more branches than would a vertebrate, because it is easier to produce that kind of change in the development of a plant (indeed it is not even clear what a new "branch" would mean in the vertebrate — perhaps it might be extra legs, or having two heads). The rates of different kinds of mutation — or of "production of variant phenotypes" in the quoted definition — therefore differs between plants and vertebrates.

Developmental constraints can arise for a number of reasons. *Pleiotropy* is an example. A gene may influence the phenotype of more than one part of the body. A trivial instance would be that genes influencing the length of the left leg probably also influence the length of the right leg. The growth of legs probably takes place through a growth mechanism controlling both legs. This mechanism does not have to be inevitable for a constraint to exist. Perhaps some rare mutants do affect the length only of the right leg. A developmental constraint exists whenever there is a tendency for mutants (in this example) to affect both legs, and the tendency is due to the action of some developmental mechanism.

Pleiotropy exists because there is not a one-to-one relation between the parts of an organism that a gene influences and the parts of an organism that we recognize as characters. The genes divide up the body in a different way from the human observer. Genes influence developmental processes, and a change in development will often change more than one part of the phenotype. Much the same reasoning lies behind a second sort of developmental constraint. New mutations often disrupt the development of the organism. A new mutant, with an advantageous effect, may also disrupt other parts of the phenotype and these disruptions will probably be disadvantageous; but if the mutant has a net positive effect on fitness, natural selection will favor it. In some cases, the disruption can be measured by the degree of asymmetry in the form of the organism. In a species with bilateral symmetry, any deviation from that symmetry in an individual is a measure of how well regulated its development was. Mutations can therefore cause *developmental asymmetry*.

New mutations may disrupt development . . .

The Australian sheep blowfly *Lucilia cuprina* provides an example. It is a pest, and farmers spray it with insecticides. The flies, as we would expect (Section 5.8, p. 115), soon respond by evolving resistance. This evolutionary pattern has been repeated with a series of insecticides and resistance genotypes in the flies, and McKenzie has studied a number of cases. When the resistance mutation first appears, it produces developmental asymmetry as a by-product. Presumably, the disruption of development is deleterious, though not so deleterious that the mutation is selected against. The advantage in insecticide resistance more than makes up for a little developmental disruption. The mutation therefore increases in frequency. Selection will then start to act at other loci, to favor genes there that reduce the new mutation's deleterious side effects while maintaining its advantageous main effect. That is, selection will make the new mutation fit in with the blowfly's developmental mechanism. The genes at the other loci that restore symmetric development, while preserving the insecticide resistance, are called *modifier genes*, and the type of selection is called *canalizing selection*. Over time, in the sheep blowfly, the resistance mutation was modified such that it no longer disrupted development (Figure 10.7).

. . . but selection over time reduces the disruptive effect

McKenzie was able to show that the modification was caused by genes at loci other than the mutation-carrying locus. (This is important because, just as there is selection at other loci to reduce the deleterious side effects of the mutation, so selection at that locus will favor other mutations that can produce insecticide resistance without harmful side effects.) It is probably common, given the extent of genetic interaction in development, for new mutations to disrupt the existing developmental pattern. Canalizing selection, to restore developmental regulation with the new mutation, is therefore likely to be an important evolutionary process.

Another sort of developmental constraint can be seen in the "quantum" growth mechanism of arthropods. Arthropods grow by molting their exoskeleton and then growing a new, larger one. They do not grow while the exoskeleton is hard. The arthropod growth curve shows a series of jumps, often with a fairly constant size ratio of 1.2–1.3 before and after the molt. Now, there are various models of how body size can be adaptive: body size, for example, influences thermoregulation, competitive power, and what food can be taken. But none of these factors can plausibly explain the jumps in the arthropod growth curve. If, for example, the body size of an arthropod was adapted to the size of food items it fed on, it would hardly be likely that the distribution of sizes of food items in its environment set up a selection pressure for quantum

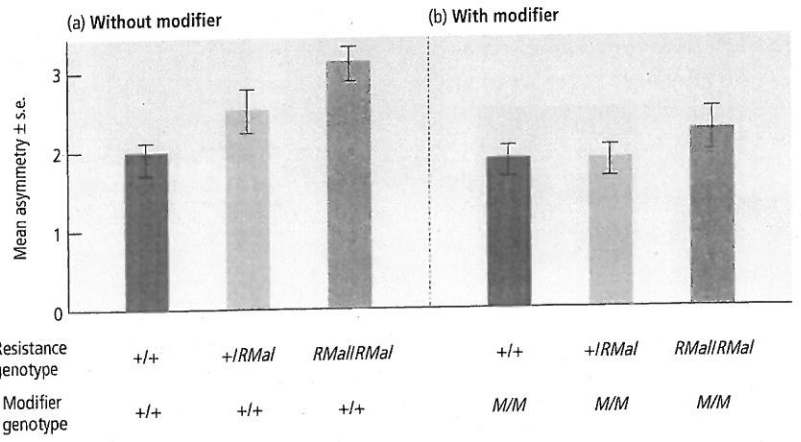

Figure 10.7
Developmental asymmetry in genotypes of the Australian sheep blowfly (*Lucilia cuprina*) that are, or are not, resistant to the insecticide malathion. (a) Developmental asymmetry in genotypes when the resistant gene *RMal* first appeared, soon after malathion was first used. + is the original, non-resistant genotype. *RMal* disrupts development, producing greater average asymmetry; and is selectively disadvantageous in the absence of malathion. (b) Developmental asymmetry of *RMal* flies after modifiers (*M*) have evolved to reduce the developmental disruption; it is now reduced near to the level of the original +/+ flies, and in the absence of malathion *RMal* has little selective disadvantage or is neutral relative to +. The sample size is 50 flies for each genotype. Redrawn, by permission of the publisher, from McKenzie & O'Farrell (1993).

growth. The explanation for the quantum jumps is a developmental constraint: growth, by molting, is dangerous and to grow with a smooth curve would require frequent risky molts. It is better to molt more rarely and grow in jumps.

Developmental constraints have been suggested as an alternative explanation to natural selection for two main natural phenomena. One is the persistence of fossil species for long periods of time without showing any change in form (Section 21.5, p. 606). The other is the variety of forms to be found in the world. We can imagine plotting a *morphospace* for a particular set of phenotypes and then filling in the areas that are and are not represented in nature.

Raup's analysis of shell shapes is an elegant example. Raup found that shell shapes could be described in terms of three main variables: translation rate, expansion rate, and distance of generating curve from the coiling axis (Figure 10.8). Any shell can be represented as a point in a three-dimensional space, and Raup plotted the regions in this space that are occupied by living shells (Figure 10.9).

A morphospace for shells shows all the shell forms that could possibly exist

Large parts of the shell morphospace in Figure 10.9 are not occupied. There are two general hypotheses to explain why these forms do not exist: natural selection and constraint. If natural selection is responsible, the empty parts of the morphospace are regions of maladaptation. When these shell types arise as mutations, they are selected

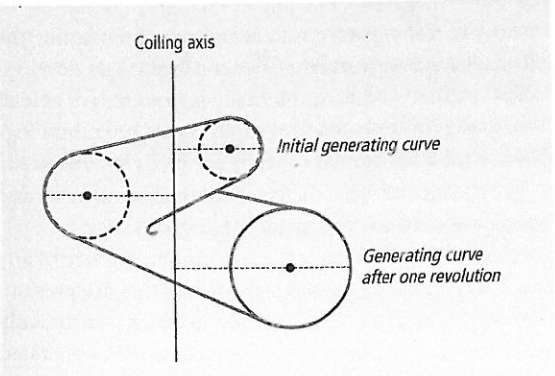

Figure 10.8
The shape of a shell can be described by three numbers. The translation rate (T) describes the rate at which the coil moves down the coiling axis: $T = 0$ for a flat planispiral shell, and is an increasingly positive number for increasingly elongated shells. The expansion rate (W) describes the rate at which the shell size increases; it can be measured by the ratio of the diameter of the shell at equivalent points in successive revolutions; $W = 2$ in the figure. The distance from the coiling axis (D) describes the tightness of the coil; it is the distance between the shell and the coiling axis, and in the figure it is half the diameter of the shell. See Figure 10.9 for many theoretically possible shell shapes with different values of T, W, and D. Redrawn, by permission of the publisher, from Raup (1966).

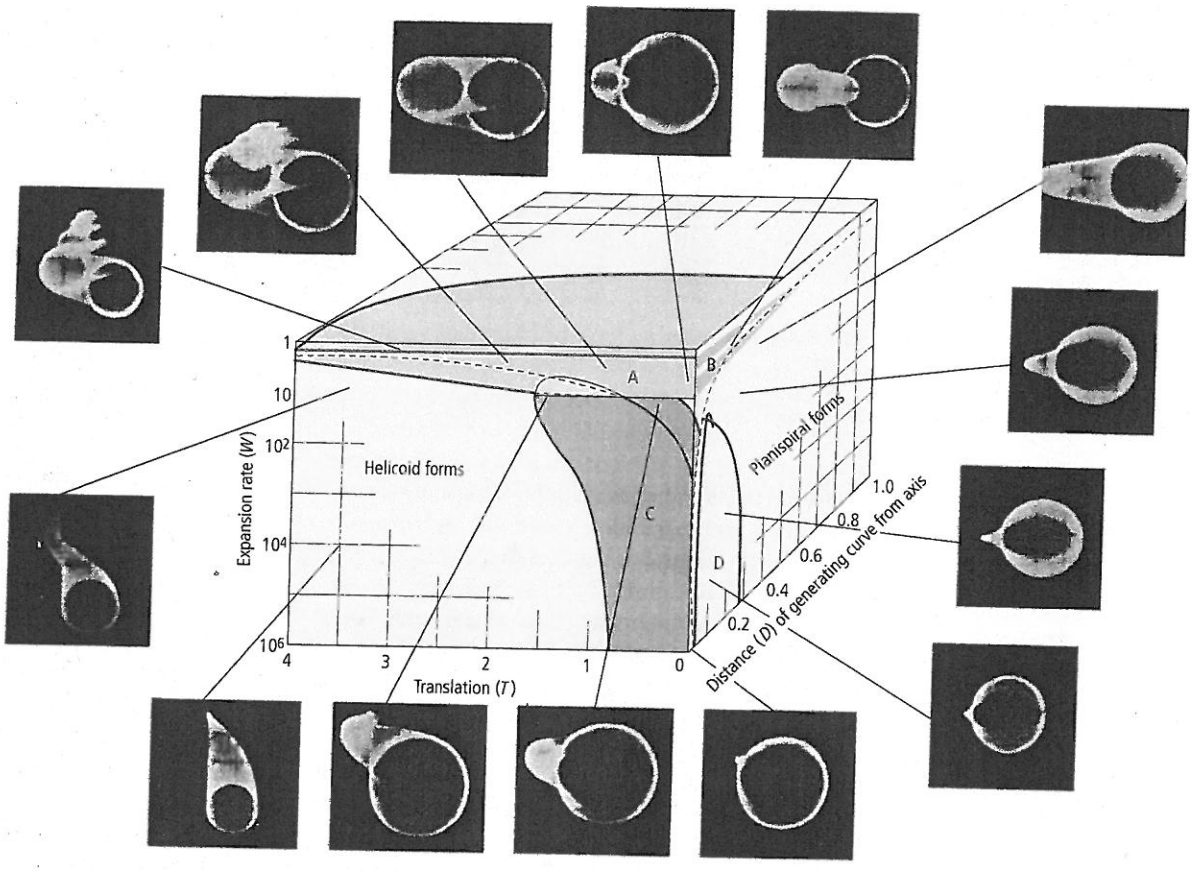

Figure 10.9
The three-dimensional cube describes a set of possible shell shapes. Around the outside of the figure, 14 possible shell shapes are illustrated as drawn by a computer. Only four regions in the cube are actually occupied by natural species: A, B, C, and D. All other regions in the cube represent theoretically possible but naturally unrealized shell shapes. The space is called a morphospace. reprinted, by permission of the publisher, from Raup (1966).

against and eliminated. Alternatively, the empty parts could be regions of constraint: the mutations to produce these shells have never occurred. If the constraint was developmental, it would mean that for some reason it is developmentally impossible (or at least unlikely) for these kinds of shells to grow. The non-existent shells would be embryological analogies for animals that disobey the law of gravity — they are shells that break the (unknown) laws of embryology. The absence of these shells would then be no more due to natural selection than is the absence of animals that break the law of gravity.

Constraint and selection can be alternatives . . .

Just as natural selection and constraint are hypotheses to explain the absence of any form from nature, so they can both hypothetically explain the forms that are present. Faced with any form of organism, we can ask whether it exists because it is the only form that organism possibly could have (constraint), or whether selection has operated

in the past among many genetic variants and the form we now observe was the one that was favored. If the form of an organism is the only one possible, an analysis that treated it as an adaptation would be misdirected. In some cases we can be more certain that variation is strongly constrained than in others. If the constraint is the law of gravity, adaptation is a fanciful hypothesis; but if the constraint is a conjectural piece of embryology, adaptation is much more worth investigating.

. . . that can be tested between . . .

How can we test between selection and constraint? Maynard Smith and his eight coauthors listed four general possibilities: adaptive prediction, direct measures of selection, heritability of characters, and cross-species evidence.

. . . by adaptive prediction . . .

The first test is the use of adaptive prediction. If a theory of shell adaptation predicted accurately and successfully the relation between shell form and environment — which forms should be present, and which absent, in various conditions — then, in the absence of an equally exact embryological theory, that would count in favor of adaptation and against developmental constraint. Conversely, a successful, exact embryological theory would be preferred to an empty adaptive theory.

. . . by measurements of selection . . .

The second test is a direct measure of selection. In the case of the shell morphospace, this would mean somehow making the naturally non-existent shells experimentally, and testing how selection then worked on them (Section 10.6). We then find out by observation whether there is negative selection against these forms.

Thirdly, we can measure the character's heritability. If a constraint is preventing mutation in a character, it should not be genetically variable. Genetic variability can be measured, and the constraint hypothesis will be refuted for any character that shows significant heritability. As it happens, this kind of evidence suggests that the gaps in the shell morphospace are not caused by developmental constraint. The heritability of a number of shell properties has been measured, and significant genetic variation found. Shell shape, therefore, is unconstrained to some extent.

. . . and by comparative evidence

Finally, cross-species evidence may be useful. It has particularly been used for pleiotropic developmental constraints. When more than one character is measured, and the values for the two characters in different organisms are plotted against each other, a relation is nearly always found. (This is true whether the different organisms are all in the same species, or from different species.) The graphs have been plotted most often for body size together with another character, and the relations are then called *allometric* (Darwin referred to it as the "correlation of growth"). Allometric relations are found almost whenever two aspects of size are plotted against each other graphically. A graph of brain size against body size for various species of vertebrates, for example, shows a positive relation. Graphs like these are two-dimensioned morphospaces, and are analogous to Raup's more sophisticated analysis for shells.

The observed distribution of points might, once again, be due either to adaptation or to constraint. It might be adaptive for an animal with a large body to have a large brain. Or it might make no difference what size an animal's brain is, and changes in brain size would simply be the correlated consequences of changes in body size (or vice versa). Mutations altering one of the characters would in that case be constrained also to alter the other. Huxley was an influential early student of allometry, and he liked to explain allometric relations by the hypothesis of constraint: "whenever we find [allometric relationships], we are justified in concluding that the *relative size* of the horn, mandible, or other organ is automatically determined as a secondary result of a single common

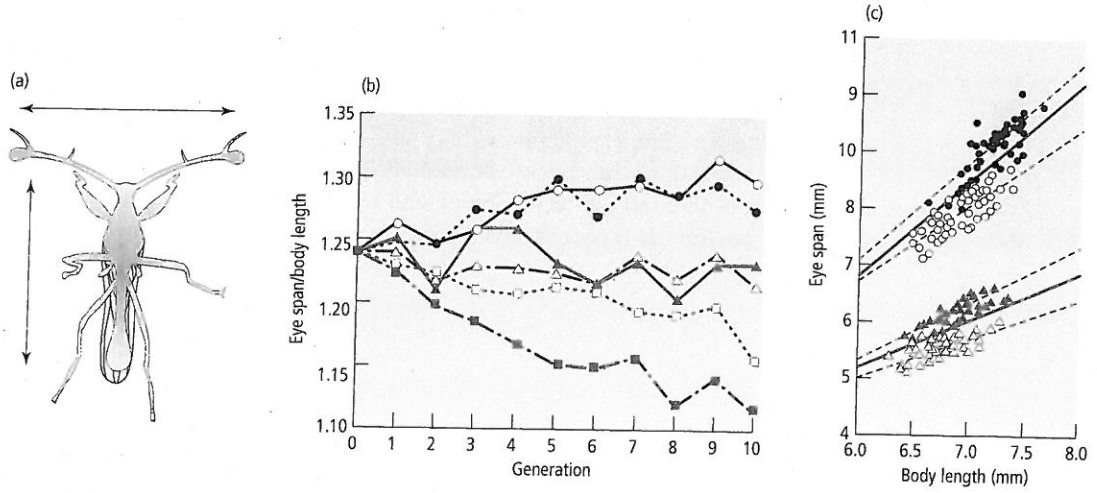

Figure 10.10
Artificial selection to alter the allometric shape of the stalk-eyed Malaysian fly *Cyrtodiopsis dalmanni*. (a) A silhouette of a fly, with arrows to indicate how eye span and body length were measured. (b) Results of one set of experiments on males. Circles are experimental lines in which males with high ratios of eye span to body length were selected to breed; squares are experimental lines in which males with low ratios of eye span to body length were selected to breed; and triangles are unselected control lines. Two replicates were done in each condition and they are distinguished by whether or not the symbol is filled in. (c) Another illustration of the allometric change; there are four sets of points. The top two (circles) are for males; the bottom two (triangles) for females. The filled in symbols are individuals of the high line after 10 generations of selection for increased relative eye span; the open symbols are individuals of the low line after 10 generations of selection for decreased relative eye span; and the dashed lines indicate the allometry in the unselected control lines. The male points correspond to replicate 1 (open circle) in (b). Note the response to selection, showing allometric relations are changeable, with the important change being in the slope of the lines in (c), which is more easily visible as a change in the ratio in (b). (0.25 in ≈ 6 mm.) Redrawn, by permission of the publisher, from Wilkinson (1993).

growth-mechanism, and *therefore is not of adaptive significance*. This provides us with a large new list of non-adaptive specific and generic characters" (Huxley 1932).

Allometric relations have been treated as non-adaptive

Some kinds of evidence are more persuasive than others. Allometric relations, in particular, are not strong evidence of developmental constraint. We can use the third kind of evidence (genetic variability) to see whether allometric relations are embryologically inevitable, or whether they can be altered by selection. Whenever anyone has looked, allometric relations have been found to be as malleable as any other character.

Figure 10.10 illustrates an artificial selection experiment by Wilkinson (1993) on the weird Malaysian fly *Cyrtodiopsis dalmanni*. These flies have their eyes at the ends of long eye stalks (Figure 10.10a, and Plate 5, between pp. 68 and 69). The eye stalks are particularly elongated in males and the character probably evolved by sexual selection. The important point here is that body and eye-stalk lengths are found to be correlated when they are measured in a number of individuals (Figure 10.10c). The ratio of eye span to body length in the natural population was 1.24 (yes, that is not a misprint: the eye stalks really are longer than the entire length of the body!). Wilkinson selected for increases or decreases in eye span relative to body length in two experimental lines and was able to alter the allometric relation in both directions (Figure 10.10). The

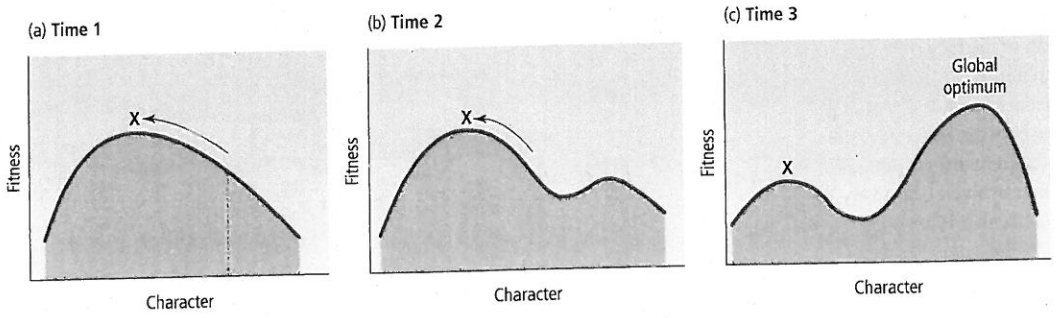

Figure 10.11
A historic change in adaptive topography has left a species stranded on a local peak. (a) Initially, there is a single optimum state for a character, and the population (X) evolves to that peak. (b) As the environment changes through time, the adaptive topography changes. The species has now reached the optimum. (c) The topography has changed, and a new global peak has arisen. The species is stuck at the local peak, because evolution to the global peak would traverse a valley: natural selection does not favor evolution toward the global peak.

allometric relation, therefore, is not a fixed law of embryonic development. Results like Wilkinson's suggest that allometric relations will have been tuned by natural selection in the past, to establish a favorable shape in each species.

In conclusion, not much is known about how embryology constrains mutation, but the general idea is plausible. The way an organism develops will influence the mutations that can arise in some of its characters. The interesting problems begin when we try to move from this general claim to an exact demonstration in a real case. The attempts so far, as in the example of allometry, have not been finally convincing. In particular cases, we can test between the alternatives of selection and constraint.

10.7.4 *Historic constraints may cause adaptive imperfection*

A population may be stuck at a local optimum

Evolution by natural selection proceeds in small, local steps and each change has to be advantageous in the short term. Unlike a human designer, natural selection cannot favor disadvantageous changes now in the knowledge that they will ultimately work out for the best. As Wright emphasized in his shifting balance model (Section 8.13, p. 216), natural selection may climb to a local optimum, where the population may be trapped because no small change is advantageous, though a large change could be. As we saw, selection itself (when considered in a fully multidimensioned context), or neutral drift, may lead the population away from local peaks; but it also may not. Some natural populations now may be imperfectly adapted because the accidents of history pointed their ancestors in what would later become the wrong direction (Figure 10.11).

The recurrent laryngeal nerve provides an amazing example. The laryngeal nerve is, anatomically, the fourth vagus nerve, one of the cranial nerves. These nerves first evolved in fish-like ancestors. As Figure 10.12a shows, successive branches of the vagus nerve pass, in fish, behind the successive arterial arches that run through the gills. Each nerve takes a direct route from the brain to the gills. During evolution, the gill arches have been transformed; the sixth gill arch has evolved in mammals into the ductus arteriosus, which is anatomically near to the heart. The recurrent laryngeal nerve still

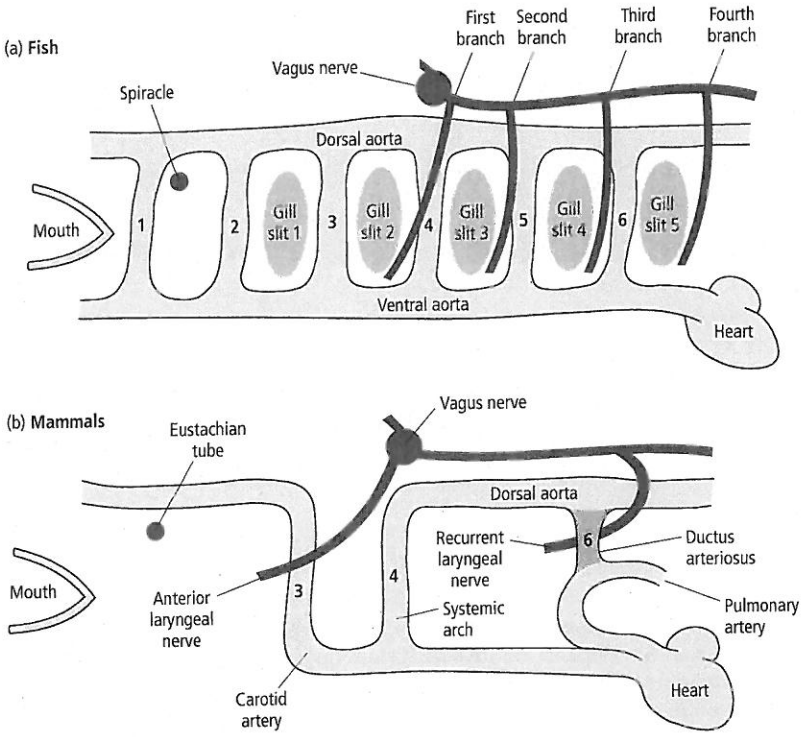

Figure 10.12
Evolution of the recurrent laryngeal nerve. (a) In fish, the vagus nerve sends direct branches between successive gill arches. (b) In mammals, the gill arches have evolved into a very different circulatory system. The descendant nerve of the fish's fourth vagus now passes from the brain, down to the heart (in the thorax) and back up to the larynx. Redrawn, by permission of the publisher, from Strickberger (1990), modified from de Beer (1971).

follows the route behind the (now highly modified) "gill arch": in a modern mammal, therefore, the nerve passes from the brain, down the neck, round the dorsal aorta, and back up to the larynx (Figure 10.12b).

The recurrent laryngeal nerve is probably maladapted in giraffes

In humans, the detour looks absurd, but is only a distance of a foot or two. In modern giraffes, the nerve makes the same detour, but it passes all the way down and up the full length of the giraffe's neck. The detour is almost certainly unnecessary and probably imposes a cost on the giraffe (because it has to grow more nerve than necessary and signals sent down the nerve will take more time and energy). Ancestrally, the direct route for the nerve was to pass posterior to the aorta; but as the neck lengthened in the giraffe's evolutionary lineage the nerve was led on a detour of increasing absurdity. If a mutant arose in which the nerve went directly from brain to larynx, it would probably be favored (though the mutation would be unlikely if it required a major embryologic reorganization); the imperfection persists because such a mutation has not arisen (or it arose and was lost by chance). The fault arose because natural selection operates in the short term, with each step taking place as a modification of what is already present. This process can easily lead to imperfections due to historic constraint — though most will not be as dramatic as the giraffe's recurrent laryngeal nerve.

A similar historic contingency may produce not actual imperfection, but differences between populations or species that are not adaptively significant. In an adaptive topography with several adaptive peaks, there may be more than one of similar height. The giraffe's laryngeal nerve looks like a case in which a local peak is clearly lower than

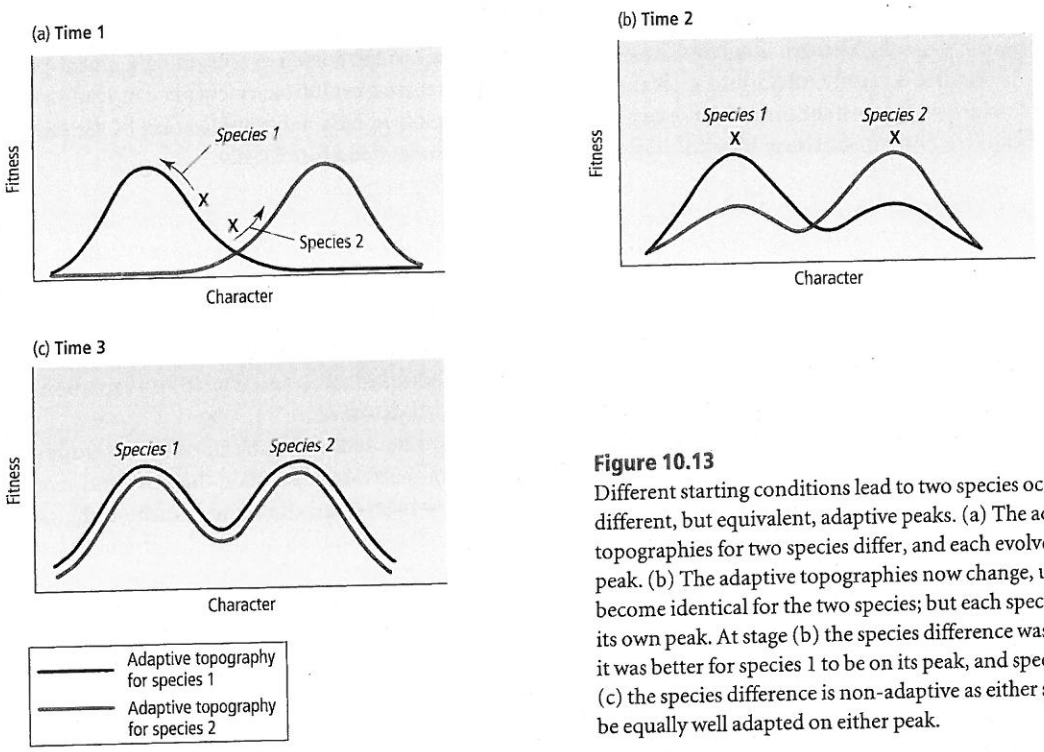

Figure 10.13
Different starting conditions lead to two species occupying different, but equivalent, adaptive peaks. (a) The adaptive topographies for two species differ, and each evolves to its own peak. (b) The adaptive topographies now change, until (c) they become identical for the two species; but each species remains on its own peak. At stage (b) the species difference was adaptive, thus it was better for species 1 to be on its peak, and species 2 on its. By (c) the species difference is non-adaptive as either species would be equally well adapted on either peak.

Species could have non-adaptive differences

the global peak, and it is therefore recognizably an imperfect adaptation. If there were several peaks of similar height, one would not be recognizably inferior to the others. Imagine now that the ancestors of a number of different populations started out near different future peaks. If they then experienced the same external force of selection, each one would still evolve to its nearest peak. The different populations would then evolve different adaptations. But they have evolved different adaptations because of their different starting conditions, not because they have adapted to different environments (Figure 10.13).

Kangaroos and gazelles may be an example

Kangaroos and placental herbivores such as gazelles are possible examples. The two forms are ecologically analogous, but have different methods of locomotion. Kangaroo hopping is no better or worse for moving than running on four legs. The lineage leading to kangaroos improved one method of moving, while that leading to gazelles concentrated on another. The difference is probably mainly a historic accident. If the argument is right, the distant ancestors of kangaroos faced different selective conditions from those of gazelles. The adaptations fixed in those ancestors then influenced subsequent evolution such that now, even though kangaroos and gazelles occupy similar ecological niches, the mutations influencing locomotion that are favored in the two groups are completely different.

The example illustrates a different idea from the giraffe's recurrent laryngeal nerve. Neither kangaroo nor gazelle is claimed to be imperfectly adapted; it is only the difference between the two that may be a historic accident. In the giraffe lineage, a similar kind of historic accident has generated an actual imperfection in its laryngeal nerve.

Whether historic accident leads to imperfection, or a neutral difference between lineages, depends on whether a global peak stays during evolution as a global peak or evolves into a local peak. In either case, past evolutionary events can lead to the establishment of forms that cannot be explained by a naive application of the theory of natural selection. Adaptation has to be understood historically.

10.7.5 *An organism's design may be a trade-off between different adaptive needs*

Many organs are adapted to perform more than one function and their adaptations for each are a compromise. If an organ is studied in isolation, as if it were an adaptation for only one of its functions, it may appear poorly designed.

Mouth design is a trade-off between feeding and eating

Consider how the mouth is used for feeding and breathing in different groups of tetrapods (amphibians, reptiles, birds, mammals). In mammals, the nose and mouth are separated by a secondary palate, and the animal can chew and breathe at the same time. The earliest tetrapods, some modern reptiles, and all modern amphibians, lack a secondary palate and have only a limited ability to eat and breathe simultaneously. A boa constrictor, for example, has to stop breathing while it goes through the complex motions of swallowing its prey — a process that can take hours. The mouth of any species that cannot breathe while it is feeding may, if it is judged only as an adaptation for feeding, appear inefficient compared with the mammalian system; the snake's mouth is a compromised adaptation for feeding. Of the reptilian groups, only crocodiles have a full secondary palate like mammals (it is presumably useful in crocodiles as it enables them to breathe air through the nose while the mouth is under water), and reptilian feeding systems can be understood as compromised in varying degrees by the need to breathe.

Trade-offs do not only exist in organ systems. In behavior, an animal has to allocate its time between different activities, and the time allocated to foraging (for example) might be compromised by the need to spend time on other demands. Trade-offs exist over the whole lifetime too: an individual's life history of survival and reproduction from birth to death is a trade-off between reproduction early in life and reproduction later on. At any one time, an animal may appear to be producing less offspring than it could, but that does not mean it is poorly adapted as it may be conserving its energies for extra reproduction later.

In summary, the adaptations of organisms are a set of trade-offs between multiple functions, multiple activities, and the possibilities of the present and future. If a character is viewed in isolation it will often seem poorly adapted; but the correct standard for assessing an adaptation is its contribution to the organism's fitness in all the functions it is employed in, throughout the whole of the organism's life.

10.7.6 *Conclusion: constraints on adaptation*

Evolutionary biologists are concerned to understand both why different species have different adaptations and how adaptations function within each species. They use

different methods to analyze adaptive differences between species and adaptations within a species. As we have looked at the sources of adaptive imperfection, we have seen some that produce adaptively insignificant difference between species and others that produce imperfect adaptation within one species. Let us finish by summarizing how the various kinds of imperfection could upset the methods of analyzing adaptation (Section 10.6).

Imperfect adaptations may or may not cause problems for the methods of studying adaptation

The comparative method could be misled by cases of adaptively insignificant differences between species. If the different forms of the adaptation are selectively neutral, or are equivalent locally adaptive peaks that different species evolved by historic accident, then attempts to correlate the differences with ecological circumstances should be unsuccessful. However, the fact that an adaptation can have several equivalently good forms does not disturb the study of the character by itself. The possibility of multiple adaptive forms should emerge from the analysis. If an enzyme has an optimal form, then it is no less an optimal form if 100 different amino acid sequences can realize it in practice. The problem for studies of particular adaptations within a species comes from the other source of imperfection. If the perfect form of the character has not arisen for reasons of history, embryology, or the genetic system, or because the environment has changed recently, then the character itself will be imperfectly adapted. If we try to predict the form of the character by an analysis of optimal adaptation, the prediction will be wrong.

What should the investigator do when a prediction turns out wrong? An analysis purely in terms of adaptation may produce spurious results. Any particular character could have evolved as an adaptation for any of a large number of reasons. Body size, for example, may be adaptive for thermoregulation, storing food, subduing prey, fighting other members of the same species, or other factors. If we assume that body size is an adaptation, we begin research by picking on one factor, such as thermoregulation, build a model relating thermoregulation to body size, and see whether the model predicted body size correctly. If the model fails, we could move on to another factor, such as diet. We build a model relating diet to body size, and see if that predicts body size any better. If this model fails, we could move on to a third factor . . . and so on. This method, however, if carried far enough, will almost inevitably find a factor that "predicts" body size correctly. Eventually, by chance, a relation will be found if enough other factors are studied, even if body size is a neutral character.

The methods are foolproof if the character under study is adaptive

The solution to the problem can be stated in a conceptually valid, but not always practically useful, form. The methods of studying adaptation work well *if we are studying an adaptation.* If the character under study is an adaptation then it must exist because of natural selection. We are right to persist in looking for the particular reason why natural selection favors it. If body size is an adaptation, there will be an adaptive model for it that is correct. However, if the character (or different forms of it) is not favored by natural selection, the method breaks down. Methods of studying adaptation should therefore be confined to characters that are adaptive, which in practice they mainly are. Adaptation can be a self-evident property of nature, and it would be absurd to claim that no properties of living things are adaptive. While research concentrates on obvious adaptations, it should be philosophically non-controversial.

However, that leaves plenty of room for controversy. Biologists do not all agree on how widespread, and how perfect, adaptations are in nature. Some biologists believe

that natural selection has fine tuned the details, and established the main forms, of organic diversity. Others think that the main forms may be historic accidents and the fine details due to random drift. Not surprisingly, the evolutionary biologists who study adaptation tend to be among the former and those who criticize it among the latter. But this difference of opinion is not about the fundamental coherence of the methods; it is about the range of their application. The controversy is unlikely to disappear in the absence of an objective, universally applicable criterion by which we can recognize which characters are adaptations. That brings us back to the problem of defining adaptation.

10.8 How can we recognize adaptations?

10.8.1 *The function of an organ should be distinguished from the effects it may have*

Obeying the law of gravity is not an adaptation

A character of an organism can have beneficial effects that are not strictly speaking adaptive. Some consequences follow from the laws of physics and chemistry without any need for shaping by natural selection. Here is an example discussed by Williams (1966).

> Consider a flying fish that has just left the water to undertake an aerial flight. It is clear that there is a physiological necessity for it to return to the water very soon; it cannot long survive in the air. It is, moreover, a matter of common observation that an aerial glide normally terminates with a return to the sea. Is this the result of a mechanism for getting the fish back into water? Certainly not; we need not invoke the principle of adaptation here. The purely physical principle of gravitation adequately explains why the fish, having gone up, eventually comes down.

The flying fish is not adapted to obey the law of gravity. When evolutionary biologists seek to understand how a character is adaptive, they consider the likely reproductive success of mutant, altered forms of the character. We can imagine many changes in the shape of the flying fish, but none of them will prevent it from returning to the sea. Even though returning to the sea is a "biological necessity," natural selection in the past has not acted between some types of fish that did return to the sea and some types that did not, with the former surviving and reproducing better.

A thought experiment about alternative forms of a character is only sensible if the alternatives are plausible. Fish that disobey gravity are not. Imagining alternative forms of a character is not absurd, but it can be taken to absurd extremes. In real cases, the alternatives are usually plausible and may even be known to exist. For example, postulating a melanic form of the peppered moth is not absurd, because it can be seen in nature.

In addition, not all the beneficial consequences of a character are properly called adaptations. A character is an adaptation in so far as natural selection is maintaining its form in modern populations. Beneficial consequences that are independent of natural selection are not adaptations. The point is obvious in practice, but must be borne in mind in conceptual discussion.

10.8.2 *Adaptations can be defined by engineering design or reproductive fitness*

The "design" of an eye for seeing is evidence the eye is an adaptation

We can distinguish between concepts of adaptation that define it in terms of inherent design of a character and those that look at its reproductive consequen The vertebrate eye is a good example to explain the "design" concept. Almost every would accept that the eye is an adaptation. We could recognize that it is adaptec describing its inherent design. From the principles of optical physics, we can tell the eye is correctly shaped to form optical images. Likewise, the heart is designe pump blood and the skeleton to support muscles. On the "design" concept, we recog adaptations as characters that are, on some appropriate engineering principle, fitte life in the environment of the species.

Adaptation has also been defined in terms of fitness measurements

Alternatively, we could define adaptations using measurements of reproductive s cess. If a character is an adaptation, then natural selection will work against gen alternatives. Natural selection will act against mutant forms of the eye that proc inferior images. Reeve & Sherman (1993) define an adaptation as that form of a cha ter, among a set of variants, that has the highest fitness.

The two concepts — the "design" and "fitness" concepts of adaptation — are clo related. A well designed form of an organ such as the eye will also have high fitr Both concepts are concerned with much the same underlying facts. However, they h different strengths and weaknesses. One strength of defining adaptation by meas ments of fitness is that it is objective and unambiguous. A mutant version of a chara either will or will not spread.

The two concepts have strengths and weaknesses

One weakness of the "fitness" concept is that it cannot always be used. Even character that does exist in many variant forms, it takes a lot of work to measure re ductive success in all the variants. Moreover, some characters do not vary in an e measurable way. The vertebrate eye is undoubtedly an adaptation, but nobody has correlated variation in its optical properties with survival and reproductive succe third problem is that a character could still be adaptive even if its relation with re ductive success was statistically undetectable. Natural selection can theoretically v on a character over millions of years and produce major changes through selec coefficients of 0.001 or less. It would be practically impossible to detect this am of selection in a modern population with the normal resources of an evolutio biologist. Forces that are important in evolution can in some cases be impossib study directly because they are so small. A direct measurement of reproductive su is most likely to demonstrate that a character is adaptive if the selection coefficie large; but these will tend to be the "obvious" characters in any case. The method wi less useful for characters whose adaptive status is controversial.

The strength of the design concept is that it is widely applicable. We can study character to see whether it is designed for some purpose. The weakness of the conc that it can be ambiguous. For example, the brain is surely an adaptation. How brain size might be 15 in^3 (250 cm^3) in one species and 18 in^3 (300 cm^3) in anc species. Is the difference between the two species adaptive? The design criterion a may not tell us the answer.

We might make an analogy with the uncertainty in the definition of "desig human fabrications. If we were to travel round the world and guess which objects

The recognition of adaptations can be uncertain in some cases

brought about by human design, we would see many obvious cases, such as architecture and engineered objects, and many non-obvious cases, such as heaps of earth. However, earth could have been heaped up for a special purpose, such as for a burial mound, or it could have just accumulated there by natural accident. We cannot always tell which cause operated just by looking at the result. The two causes are objectively distinct, but the distinction is historic: either the heaps of earth were constructed by human agency or they were not. However, the history is unobservable, and when we have to make the distinction purely using modern observable evidence, there will be difficult border-line cases. We should not, therefore, expect the distinction between designed and non-designed entities to always be clear in either the case of natural adaptation or of human fabrications.

Likewise, body coloration may be a simple adaptation, brought about by natural selection, or it may be non-adaptive and brought about by chance, as may be the case for the red color of the sediment-dwelling worm *Tubifex* (visual factors are not important in the sediment at the bottom of the water column). Again, either natural selection is favoring the body coloration or it is not; but if we try to decide whether it is just from looking at the character, the answer may not be clear. We have a clear theoretical concept of what an adaptation is, but that concept implies that adaptation cannot have a universal, foolproof, practical definition.

Summary

1 Three theories have been put forward to explain the existence of adaptation: supernatural creation, Lamarckism, and natural selection. Only natural selection works as a scientific theory.

2 Natural selection is not the only process that causes evolution, but is the only process causing adaptation.

3 Natural selection, at least in principle, can explain all known adaptations. Examples of coadaptation and useless incipient stages have been suggested but they can be reconciled with the theory of natural selection. The vertebrate eye could have evolved rapidly by small advantageous steps.

4 Some new organs (and new genes) evolve by continuous modification of a previously existing organ (or gene), while the function is constant. Others evolve by continuous modification, but with a change in function. Yet others evolve when previously existing but separate parts are combined.

5 Fisher proposed a model in which adaptation evolves in many small genetic steps. His model contrasts with Goldschmidt's, in which adaptations evolve by sudden macromutations. Fisher's model is being modified theoretically, and tested experimentally.

6 Adaptations may be imperfect because of time lags: a species may be adapted to a past environment because it takes time for natural selection to operate.

7 Adaptations are imperfect because the mutations that would enable perfect adaptation have not arisen. The imperfections of living things are due to genetic, developmental, and historic constraints, and to trade-offs between competing demands.

8 For particular characters, adaptation and constraint can be alternative explanations. Likewise, differences in the form of a character between species may be due to adaptation to different conditions or to constraint. Forms that are not found in nature may be absent because they are selected against or because a constraint renders them impossible.

9 Adaptation and constraint can be tested between by several methods: by the use of predictions from a

hypothesis of adaptation or constraint, by direct measures of selection, by seeing whether the character is variable and whether the variation is heritable and can be altered by artificial selection, and by examining comparative trends.

10 The methods of analyzing adaptation are valid when applied to adaptive characters and interspecific trends; they might be misleading for non-adaptive characters and trends.

11 Not all the effects of an organ will have evolved as adaptations by natural selection. Some will be inevitable consequences of the laws of physics.

12 Biologists disagree about how exact, and how widespread, adaptation is in nature.

13 There are criteria to distinguish adaptive from non-adaptive characters. Measurement of selection provides an objective criterion, but is not always practical. The inherent engineering design of a character is not always an objective criterion, but is widely applicable. The two criteria are closely related.

Further reading

Williams (1966) is a classic work on adaptation. Gould & Lewontin (1979) is an influential paper that criticizes the way adaptation has often been studied; Cain (1964) argues the opposite. Pigliucci & Kaplan (2000) look at 20 years of discussion about Gould & Lewontin (1979). Lewontin (2000) and Gould (2002b) variously update their viewpoints. Reeve & Sherman (1993) is a stimulating paper about adaptation. Dawkins (1982, 1986, 1996) argues that only natural selection can explain adaptation; the 1986 and 1996 books are written for a wide audience. Dennett (1995) is also written for a broad audience and discusses several of the topics covered in this chapter.

Allen *et al.* (1998) have compiled an anthology of classic papers about adaptation. My evolution anthology contains a section of extracts about adaptation (Ridley 1997) and Rose & Lauder (1996) have edited a multiauthor volume on the topic.

The natural theologian's argument from design was philosophically undermined by Hume in his *Dialogues Concerning Natural Religion*, which are in print in various paperback editions and (unlike some of Hume's other philosophical writings) readily intelligible. I include the passage in Ridley (1997). However, Hume's abstract argument did not convince people and it was Darwin's mechanistic theory of natural selection that historically toppled that long tradition of thought. See Simpson (1944, 1953) on orthogenesis.

Dawkins (1996) includes a popular account of Nilsson & Pelger's (1994) paper about eye evolution. Land & Nilsson (2002) is a book about animal eyes. Nitecki (1990) is a multiauthor book about evolutionary innovations. On feathers, see Prum & Brush (2002) and their references. On preadaptation in general, see also the popular essay by Gould (1977b, chapter 12). Gerhart & Kirschner (1997) discuss the lactose example.

On the genetics of adaptation, Leigh (1987) includes an account of Fisher's argument. Travisano (2001) discusses the emerging research program with microbial experimental systems.

The methods of studying adaptation are discussed (in addition to the multiauthor volumes referred to above) by Orzack & Sober (1994), Harvey & Pagel (1991), Parker & Maynard Smith (1990), Maynard Smith (1978), and Rudwick (1964). For the

experimental method, see the special issue of *American Naturalist*, a supplement to vol. 154 (July 1999).

On constraints, Antonovics & van Tienderen (1991) look at terminology. Barton & Partridge (2000) look at the topic in general. On "ghost" adaptations like the neotropical fruit, see the popular book by Barlow (2000). Byers (1997) is an example discussing the social behavior of the American pronghorn and Macgregor (1991) reviews the remarkable genetic constraint in the crested newt and refers to earlier work.

On developmental constraint, Maynard Smith *et al.* (1985) and Gould (2002b) are major reviews. McKenzie & Batterham (1994) and McKenzie (1996) discuss the insecticide resistance example (see also the further reading in Chapter 5, p. 135). Antibiotic resistance in microbes is a related topic. Levin *et al.* (2000) discuss how compensatory mutations that reduce the harmful side effects of the initial resistance mutations may influence the persistence of antibiotic resistance. The arguments are related to those in Box 5.2 (p. 119). On developmental stability in general, see Lens *et al.* (2002). Harvey & Pagel (1991) contains an account of, and references to, recent work on allometry. Chapter 9 has further references for canalizing selection. Chapter 20 looks at evolutionary development, which probably provides the concepts for future studies of developmental constraint. Galis *et al.* (2001) discuss the special case of constraints on digit numbers.

Certain human genes confer resistance to disease, but are otherwise disadvantageous. These genes probably illustrate constraints due to history (they evolved recently) and to trade-offs (disease resistance is so important that other adaptations are compromised). Schliekman *et al.* (2001) give some calculations for three such genes: $CCR5^{-}$ (resistance to HIV), hemoglobin S, and $\Delta 32$ (resistance to bubonic plague).

On definition, see the references already given to Williams (1966) and Reeve & Sherman (1993). I have extracted them, along with another good discussion by Grafen, in Ridley (1997). A further distinction is between historic and non-historic definitions. Gould has argued that only characters that retain a constant function should be called adaptations. See Gould (2002b) for a thorough recent statement of his view, and Reeve & Sherman (1993) for problems with it.