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1

The legacy of Lamarckism

Every thing possible to be
believed is an image of truth.
Blake: *Proverbs of Hell*

The idea that the inheritance of acquired characters plays an important role in evolution has been the subject of controversy for over a century. Enthusiasm for the idea, which is usually associated with the name of Lamarck, has sometimes led to charlatanism and fraud, while opposition to it has led to 'Lamarckist' being used as a term of abuse. Nowadays, biologists usually regard ideas about the inheritance of acquired characters as nothing more than an interesting part of the history of biology. Lamarckian evolution is rejected on the grounds that there is no evidence for it, no mechanism that can produce it, and no need for it in evolutionary theory. Some people go even further and argue that the inheritance of acquired characters is theoretically impossible—it is incompatible with what is known about genetics and development.

The aim of this book is to show that there are now well recognized mechanisms by which some acquired characters can be transmitted to the next generation, and that such characters have probably played a significant role in evolution. We want to make it clear right at the outset that although we argue that some types of Lamarckian evolution are possible, there is nothing in what we say that should be construed as being anti-Darwinian.[1] We are firm believers in the power and importance of natural selection. What we do maintain, however, is that some new inherited variations are not quite as random as is generally assumed, but arise as a direct, and sometimes directed, response to environmental challenge, and that the effects of such induced variations deserve more recognition in evolutionary theory.

In this chapter we want to look at some of the reasons given for rejecting the idea that acquired characters can be inherited, and show why we think they are wrong. The objections to Lamarckism are based partly on the limited evidence for the inheritance of acquired characters, but also on prejudice and conservatism, and on a view of heredity that is no longer appropriate. During the past fifty years there has been a gradual narrowing of the concept of heredity. Although this was probably important and necessary for the development of genetics as a discipline, it is now a

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The aim of this book is to show that there are now well recognized mechanisms by which some acquired characters can be transmitted to the next generation, and that such characters have probably played a significant role in evolution. We want to make it clear right at the outset that although we argue that some types of Lamarckian evolution are possible, there is nothing in what we say that should be construed as being anti-Darwinian.[1] We are firm believers in the power and importance of natural selection. What we do maintain, however, is that some new inherited variations are not quite as random as is generally assumed, but arise as a direct, and sometimes directed, response to environmental challenge, and that the effects of such induced variations deserve more recognition in evolutionary theory.

In this chapter we want to look at some of the reasons given for rejecting the idea that acquired characters can be inherited, and show why we think they are wrong. The objections to Lamarckism are based partly on the limited evidence for the inheritance of acquired characters, but also on prejudice and conservatism, and on a view of heredity that is no longer appropriate. During the past fifty years there has been a gradual narrowing of the concept of heredity. Although this was probably important and necessary for the development of genetics as a discipline, it is now a

handicap to evolutionary thinking. The trend needs to be reversed, because there is more to heredity than DNA, and DNA is not just a passive information carrier, it is also a response system.

Before discussing Lamarckism and the objections to it, it is necessary to look at Lamarck's ideas and at what people have meant and mean today when they talk about the inheritance of acquired characters. One of the difficulties in discussing the subject is that Lamarck's ideas have themselves been the subject of cultural 'Lamarckian' inheritance. Terms such as 'Lamarckism', 'acquired character' and 'Lamarckian evolution' have undergone changes in meaning as they have been used and modified to fit the interests and biases of those using them. As we shall show later in this chapter, this is still the situation today: recent discussions of the inheritance of bacterial adaptations have shown that there is no general consensus about what would constitute evidence for the inheritance of acquired characters.

Lamarck's Lamarckism

Jean-Baptiste Lamarck (1744–1829) was the first consistent evolutionist.[2] His evolutionary theory was a network of different ideas, some old and some relatively new. They were discussed most fully in his book *Philosophie zoologique* published in 1809, fifty years before Charles Darwin published *On the origin of species by means of natural selection*.

Lamarck believed that the natural laws operating on living matter are the same as those operating on non-living matter. The difference between living and non-living is a consequence of the way in which the material is organized. Under certain special conditions, which allow the action of what were known in Lamarck's time as 'subtle fluids', inanimate matter can be reorganized in a way which changes it into living matter. According to Lamarck, the spontaneous generation of living organisms is a normal, and not uncommon, occurrence of both the past and the present. A certain combination of material constituents and the right environment produce a living organism which is a self-sustaining, growing, and self-complicating entity.

Once generated, simple life forms change. Lamarck thought that there are two reasons for change. One is an inevitable consequence of the organization of living matter, which differs from non-living matter because it acts as a whole. This whole is preserved, but also altered, by the movements of the fluids it contains. These movements divide, erode, etch out channels, and leave sediments in the soft parts of the body. Once the movements of fluids have established one set of structures, further movements build on and elaborate them to produce even more complex structures. Thus, according to Lamarck, the inherent tendency for a slow, gradual increase in complexity is a consequence of the basic properties of the living body. It occurs because the motions of the body fluids produce results that have a snowball effect.

If the self-complicating property of living organisms were the only factor causing change, the series from simple to complex forms would be progressive and linear. However, Lamarck believed that a second factor is involved. This is the ability of living organisms to react to their environment. Responses to the environment cause an alteration and diversion in the direction and pattern of the fluids in the body. The result is adaptive modifications of structure and function. Since environmental effects are accidental, the changes they induce cause a branching away from the main linear series. Both the inherent tendency for an increase in complexity, and the adaptive modifications, contribute to the transformation of organisms. Evolution is primarily progressive, but it is also divergent.

The linear nature of evolutionary history can be seen in Lamarck's famous diagram (reproduced in Fig. 1.1).[3] To modern eyes, which are used to phylogenetic trees, it seems upside down, since the 'lower' animals are at the top rather than at the bottom. Yet, in some ways, it is the same as modern phylogenetic trees, because the lineages that appear at the top are the most recent. According to Lamarck, since simple forms of life are transformed into more complex forms, and simple forms are created anew all the time, the simple organisms that exist today are the most recent in origin. Lamarck believed that there is no extinction in nature. The disappearance of species from the fossil record is due to their transformation into something else, not to their extinction. Lamarck's theory of transformation is illustrated in Fig. 1.2. It can be thought of as a series of escalators in which each lineage begins at the bottom with spontaneous generation, and continues up through the various forms as a result of the action of subtle fluids. Different lineages start at different times and progress upwards on parallel but independent escalators.

Lamarck's ideas about the nature of evolutionary adaptation and the mechanisms involved are summed up in the laws given in his *Philosophie zoologique*:

> First law
> In every animal which has not passed the limit of its development, a more frequent and continuous use of any organ gradually strengthens, develops and enlarges that organ, and gives it a power proportional to the length of time it has been so used; while the permanent disuse of any organ imperceptibly weakens and deteriorates it, and progressively diminishes its functional capacity, until it finally disappears.
>
> Second law
> All the acquisitions or losses wrought by nature on individuals, through the influence of the environment in which their race has long been placed, and hence through the influence of the predominant use or permanent disuse of any organ; all these are preserved by reproduction to the new individuals which arise, provided that the acquired modifications are common to both sexes, or at least to the individuals which produce the young. (Lamarck 1809, translated by Elliot 1914, p. 113)[4]

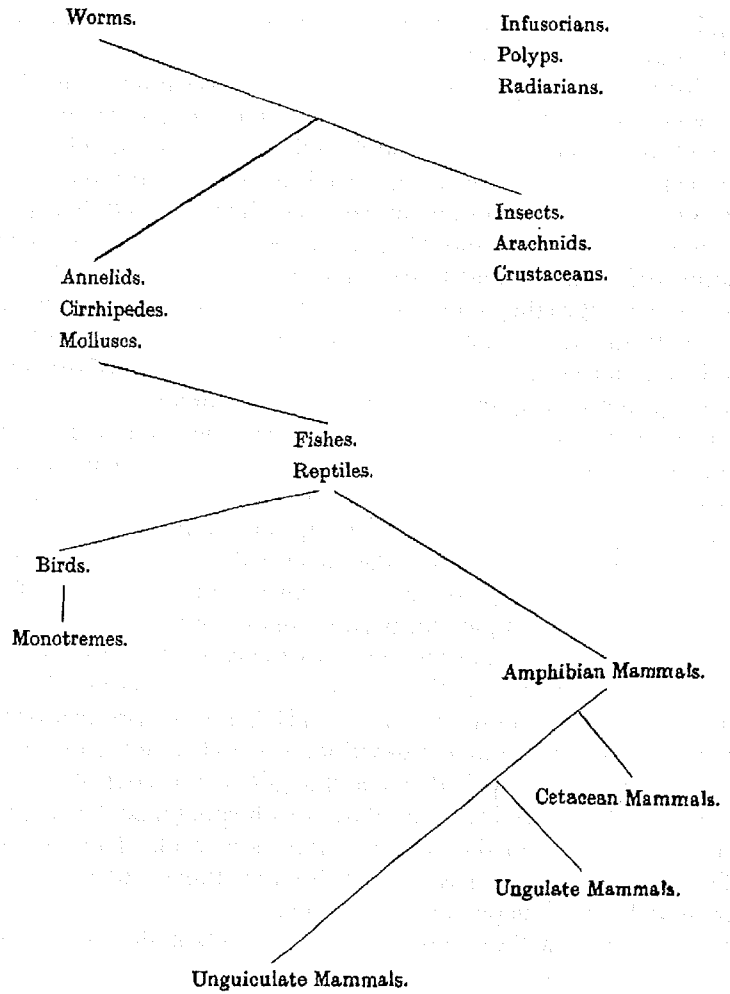


Fig. 1.1 Lamarck's figure 'showing the origin of the various animals'. (From the 1914 English translation of Lamarck's *Philosophie zoologique*, p. 179.)

The first law describes how the use and disuse of organs lead to structural modifications. Lamarck believed that the adaptive responses of animals to new environments are mediated by changes in behaviour; changes in morphology are consequences of changes in behaviour. The second law describes the evolutionary consequences of the first. It assumes that the acquired adaptive changes are inherited. The idea was not original, and Lamarck did not claim that it was. In Lamarck's day the notion that acquired characters could be inherited was almost universally accepted.[5] Lamarck believed that if environmental changes persist, acquired,

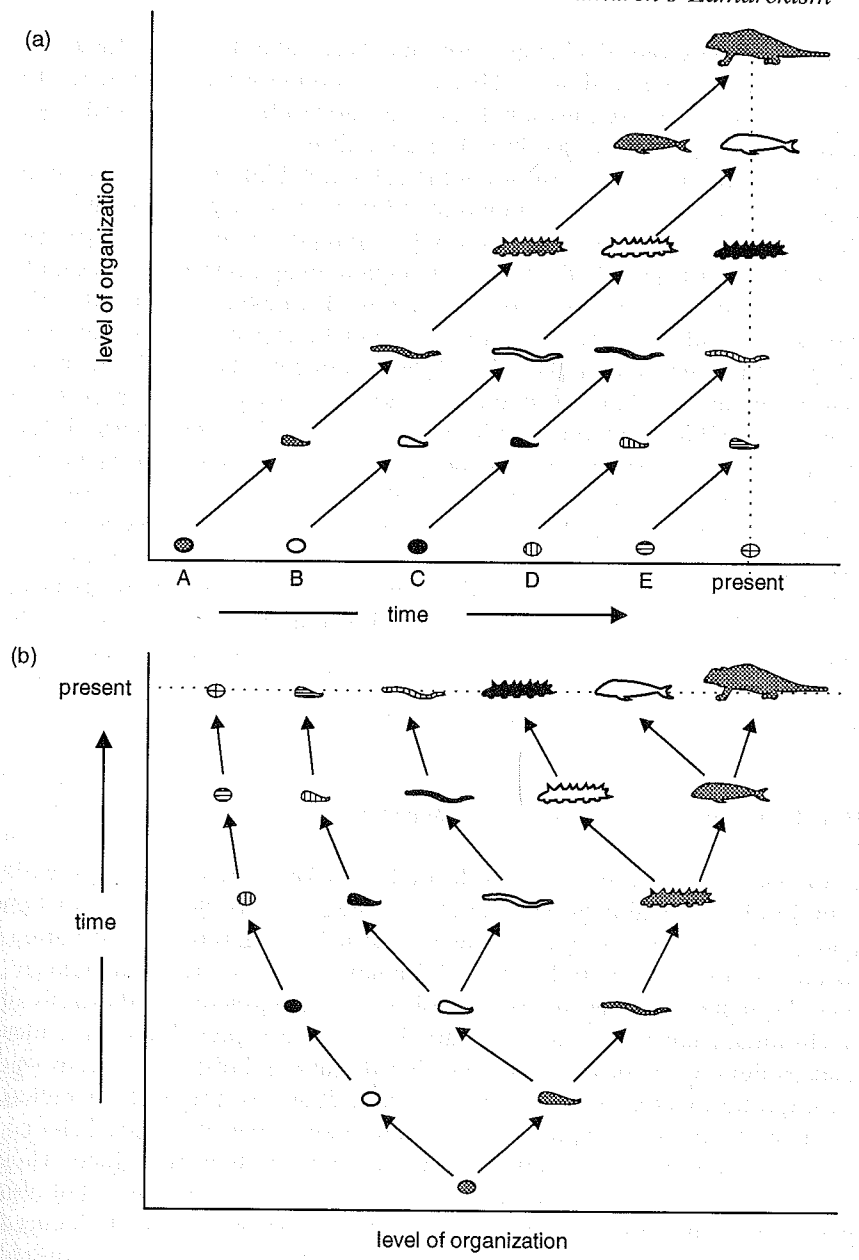


Fig. 1.2 A comparison of (a) Lamarck's theory of transformation and (b) a phylogenetic tree. (a) Lineages A–E begin with spontaneous generation, and pass through the same series of forms. The older lineages have reached more complex levels of organization. Differences in shading indicate that the forms are not identical in each lineage. (Loosely based on Bowler 1989, p. 85.) (b) A phylogenetic interpretation of the forms shown in (a), in which it is assumed there is no extinction.

adaptive, physiological changes are not only inherited, but they also accumulate over generations and become evolutionary adaptations. Evolutionary adaptations are thus simply an extension of the physiological adaptations that occur during the life of an organism.

The evolutionary origin of physiological adaptability itself did not concern Lamarck. For him it was inherent in the very essence and definition of life. Of course, by taking adaptability for granted, the explanatory power of his theory was greatly limited.[6] A complete evolutionary theory should be able to explain not only the evolution of adaptations, but also the evolution of adaptability. It is not sufficient to explain the evolutionary origin of the thick skin on the soles of the feet of a newborn child by saying that originally the skin thickened as an adaptive response to the pressure caused by walking, and this acquired character then became inherited. It is also necessary to explain how the adaptive response—the skin thickening that occurs in response to pressure and reduces the risk of injury—evolved.[7] Lamarck's theory had nothing to say about the evolution of the ability to adapt. Adaptability was taken for granted.

Lamarck's ideas about inherited changes were based on the typical, adaptive, developmental and physiological responses of the individual. He did not regard the variation between different individuals as important. According to Lamarck, all individuals have the same ability to respond adaptively, and all transmit the response to the next generation.

Post-Lamarck Lamarckism (neo-Lamarckism)

Lamarck's theories about the role of fluid movements in biology were found to be untenable and were soon abandoned. His ideas on extinction and spontaneous generation were also shown to be wrong and were forgotten. What eventually came to be termed 'Lamarckism', or 'Neo-Lamarckism', was the belief that the inheritance of acquired characters is the basis of evolutionary change. Many 'Lamarckists' also accepted Lamarck's idea that evolution was progressive, and that the use and disuse of organs was an important cause of change, but the inheritance of acquired characters became central to 'Lamarckism'. Frequently the types of acquired characters that could be inherited were very generally and loosely defined. They included not only characters that were changed by use or disuse, but also passively acquired mutilations, and characters that were directly induced by the external environment, without the behavioural mediation required by Lamarck's theory. Lamarck himself had explicitly rejected a direct effect of the environment on animal structures. He believed that new environmental conditions resulted in new activities and habits, and it was these changes in behaviour that caused changes in the body. Even in plants, which do not have 'behaviour', Lamarck stressed that the response

to the environment was mediated by its effect on *internal* activities, such as those associated with nutrition or transpiration.

Rather surprisingly, Lamarck, who seems to have had a theory about everything from mineralogy to meteorology, never developed a theory of inheritance. He suggested no mechanism that would allow acquired characters to be inherited. This was in spite of the fact that Buffon, with whom he had a close association, was well aware of the need for a theory of heredity, and had himself elaborated an idiosyncratic version of a theory that had existed since the time of Hippocrates.[8] The basic idea of this theory, and of most other theories of heredity at this time, was that all parts of the body sent small representative particles to the reproductive organs where they formed 'the germ', which gave rise to the next generation. It is an irony that one of the most famous exponents of this theory, the person who gave it the name by which it is commonly known, was none other than Charles Darwin. In the version elaborated by Darwin, the particles were christened 'gemmules' and the whole hypothesis 'pangenesis'. According to Darwin's and other versions of this theory, an environmentally modified part, or a part that had become modified as a consequence of use and disuse, liberated modified gemmules into the circulation. The modified gemmules reached the germ cells and eventually participated in the formation of the corresponding modified part in the offspring. In this way, acquired characters could be passed on to the next generation.

Ideas about the nature of inheritance changed at the turn of the century when, as Zirkle put it:

> the discovery of Mendel's forgotten work put the whole matter on a new basis and pangenesis came to the end of its 2300-year career. (Zirkle 1946, p. 145)

However, it was not the rediscovery of Mendel's work in 1900 that led to doubts about the role of the inheritance of acquired characters in evolutionary change. The debate about its role began long before then, and continued long after. One of the strongest challenges to the idea of the inheritance of acquired characters came from August Weismann in the 1880s, well before the rediscovery of Mendel's work. Until he was in his mid-forties, Weismann believed wholeheartedly in the inheritance of acquired characters, but once he had changed his mind, he became the most forceful opponent of the idea. He argued that there was no evidence for this type of inheritance: all of the reputed cases could be explained in other ways. Moreover, there were many adaptations, such as those of the sterile worker castes of social insects, which, even in theory, could not be acquired through Lamarckian mechanisms. The adaptations acquired by a sterile worker ant during its lifetime cannot be transmitted to the next generation. Most important of all, Weismann maintained, there was no realistic mechanism by which acquired characters could be inherited. Weismann dismissed the two types of theory that attempted to explain

how somatic changes could affect the germ line, saying that one had to assume:

> . . . either the presence of hypothetical tracks along which a modifying, though totally inconceivable, influence might be transferred to the germ-cells, or else the discharge of material particles from the modified organ, must take part in the formation of the germ-plasm. . . (Weismann 1893, p. 393)

He argued that both theories were not only unsupported by direct observation or experiment, they were also incompatible with physiological, cytological, and anatomical observations. Weismann believed:

> that *all permanent—i.e., hereditary—variations of the body proceed from primary modifications of the primary constituents of the germ*; and that neither injuries, functional hypertrophy and atrophy, structural variations due to the effect of temperature or nutrition, nor any other influence of environment on the body can be communicated to the germ-cells, and so become transmissible. (Weismann 1893, p. 395; Weismann's italics)

In other words, the germ line is unaffected by changes in the soma.

Weismann accepted Darwin's theory of evolution. According to Darwin's theory, the cause of evolution is natural selection acting on the inherited differences between individuals. Individuals with variations favourable for survival and reproduction leave most offspring, so gradually the favourable variations spread through the population. Darwin himself did not reject the idea that acquired characters could be inherited. Weismann did. Weismann believed that evolution through natural selection does not require the inheritance of acquired characters. The differences between the neo-Darwinian (Weismannian) idea of evolution through natural selection, and the Lamarckian idea of evolution through the inheritance of environmentally induced acquired characters, is shown in Fig. 1.3. Weismannian evolution operates through changes in populations; Lamarckian evolution operates through changes in individuals. It has been said that Lamarckism is an anti-Darwinian theory because it advocates directed variation: new environments elicit new, adaptive, heritable variation.[9] But Lamarckism is an anti-Darwinian theory not just because it advocates directed variations; it is anti-Darwinian also because it advocates identical directed variations in *all* the individuals of a population exposed to the new environment.

In spite of Weismann's powerfully and passionately argued case in favour of natural selection and against the inheritance of acquired characters, the debate between the neo-Lamarckians and neo-Darwinians continued until well into the twentieth century.[10] Some form of Lamarckism was generally accepted by most American and German palaeontologists who explained the evolutionary trends found in fossil series in Lamarckian terms.[11] Although their observations could not possibly provide support for a *mechanism* of evolutionary change, they claimed that they provided

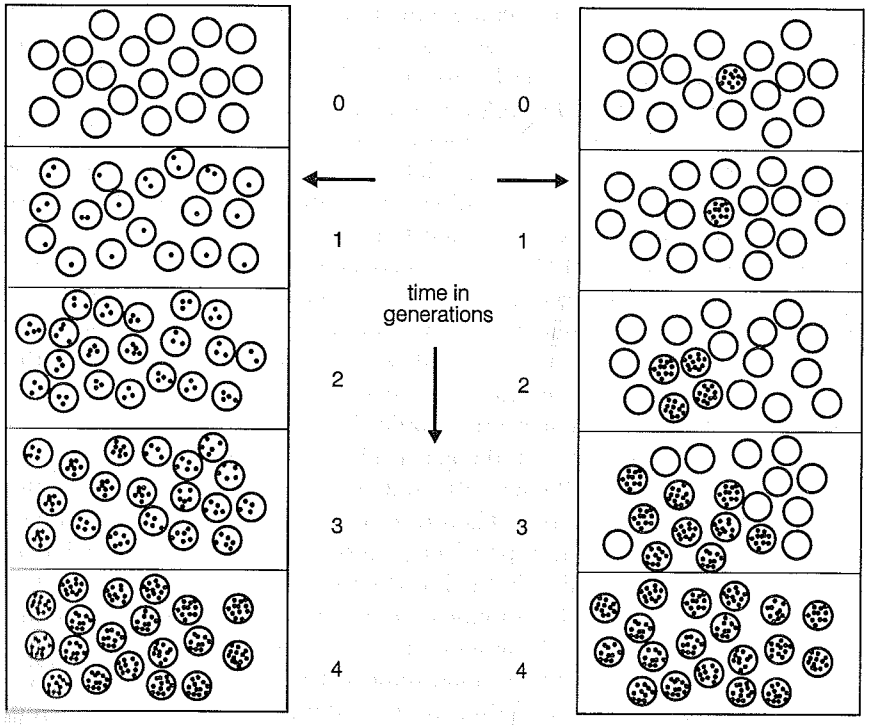


Fig. 1.3 The evolution of an adaptation to a new environment by (a) Lamarckian evolution and (b) Darwinian evolution. Individuals with the adaptation are represented by shaded circles, the intensity of shading indicating the degree of adaptation. At the time indicated by the horizontal arrows, the environment changes, and this change persists through subsequent generations. In Lamarckian evolution, all individuals acquire the adaptation, and it is gradually enhanced as individuals in subsequent generations continue to respond to the environment. In Darwinian evolution, by chance an individual has an appropriate adaptation that enables it to contribute proportionally more progeny to subsequent generations. (Loosely based on Medawar 1957, pp. 80–81.)

evidence for the inheritance of acquired characters. Hence Osborn, who was later influenced by Weismann's ideas and became more doubtful about the inheritance of acquired characters, was able to publish a paper in 1889 with the title 'The palaeontological evidence for the transmission of acquired characters'. For the palaeontologists, inherited changes brought about by the effects of use and disuse, or as a result of the direct influence of climatic factors, seemed a better explanation of the directional trends they found in their fossil series than did the selection of random variations suggested by the neo-Darwinists.

The neo-Lamarckists also could not accept that there was no direct causal relation between ontogenetic and phylogenetic characters. It seemed unreasonable to ascribe to coincidence the similarity between epidermal thickenings that develop during an animal's lifetime in response to rubbing, and the inherited epidermal thickenings that are present at birth in areas subject to pressure at a later date. Darwinian explanations of evolutionary changes that had no obvious adaptive value seemed even less reasonable and acceptable to the neo-Lamarckists. For example, Rensch (1983) described how, in 1929, he accepted a neo-Lamarckian rather than a neo-Darwinian explanation of geographic variations in size. He believed that size variation was due to direct climatic effects which, over many generations, became inherited. He followed the ideas of Cope who, at the turn of the century, had developed a theory of 'diplogenesis' to explain how environmental influences on somatic characters could be transmitted to the next generation through the germ cells.[12] This theory suggested that the change-producing influence affected the germ plasm as well as the somatic parts of the organism. Therefore, the effects could be transmitted to the next generation. This type of mechanism has also been referred to as 'parallel induction'. Parallel induction occurs when the characters of the offspring show that both the somatic and the germ line modifications in the parent were of a corresponding and equivalent type (see Fig. 1.4).

Weismann's definition of the inheritance of acquired characters was a very narrow one. He claimed that the inheritance of acquired characters can be said to occur only if first, the environmental change affects a somatic trait, and second, the modified soma itself (and *not* the environmental agent which affected it) induces a change in the germ plasm which in turn produces the same somatic modification in the following generation.[13] The phenomenon covered by this narrow definition is sometimes referred to as 'somatic induction'. Somatic induction occurs if the environment first modifies the soma, and this effect is then transmitted to the germ cells (Fig. 1.4).

A much broader interpretation of the concept of the inheritance of acquired characters was adopted by other evolutionary biologists.[14] For example, in 1909, in their book *Les théories de l'évolution*, Yves Delage and Marie Goldsmith criticized Weismann's restrictive view, and emphasized what a broad church Lamarckism is:

> Neo-Darwinism, which has found its most complete expression in Weismann's writings, constitutes a well-harmonized system of conceptions relative to the structure of living matter, ontogenesis, heredity, evolution of species, etc. Lamarckism on the other hand is not so much a system as a point of view, an attitude towards the main biological questions.
>
> Whatever theory emphasizes the influence of the environment and the direct adaptation of individuals to their environment, whatever theory gives to actual factors the precedence over predetermination can be designated as Lamarckian. (Delage and Goldsmith 1909, translated by Tridon 1912, pp. 244–245)

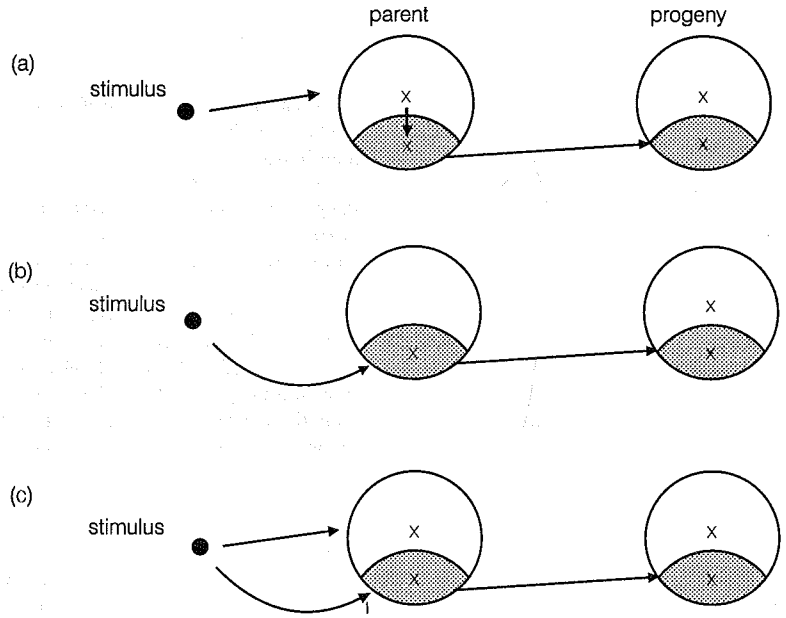


Fig. 1.4 Inherited environmental effects. Circles represent organisms with a soma (unshaded) and germ line (shaded); **X** is the effect of the environmental stimulus. (a) Somatic induction (a 'Lamarckian' mechanism): the stimulus produces an effect in the parental soma, which is transferred to the germ line and thence to the progeny. (b) Random or directed germ line variations (Weismann's mechanism): the germ line is affected directly, so the effect is passed to progeny. (c) Parallel induction: the stimulus acts on both the germ line and soma, so the effect is passed to the progeny. (Based on Fothergill 1952, p. 158.)

For people like Delage and Goldsmith, Lamarckian inheritance occurs whenever a stimulus-dependent character (i.e. a character whose appearance depends on a specific external stimulus) in one generation, becomes stimulus-independent in the following generations (i.e. appears whether or not the stimulus is present). Such a broad interpretation makes 'Lamarckism' and 'the inheritance of acquired characters' purely descriptive terms. No assumptions are made about the mechanisms that bring about the transition from stimulus-dependent to stimulus-independent adaptations. There is no restriction on the type of character involved, nor on the type of external stimuli. Many different phenomena can be described as Lamarckian. In Table 1.1 we have listed some examples of types of inheritance that have been called Lamarckian, and indicated the mechanisms suggested for the transmission of the acquired character. We shall return to many of these again and elaborate on them in later chapters.

Table 1.1 Different types and mechanisms of 'Lamarckian' inheritance

Affected cell types	Affected character	Response to the inducing agent	Mechanism leading to the character's inheritance	References
Somatic	Somatic	Phenotypic change in somatic cells	Pangenesis: transport of gemmules or other entities to germ cells	Hippocrates, Darwin, many others; see Zirkle (1946)
Somatic that can become germ line	Somatic	None—random somatic mutations	Somatic selection between cells followed by the selected mutant cells becoming gametes	Buss (1987), Klekowski (1988); see Chapter 2
Somatic	Somatic	Transcription of mRNA	Somatic selection followed by transfer of mRNA to the germ line where it is reverse transcribed into DNA and integrated into germ-cell DNA	Steele (1979); see Chapter 2
Somatic	Usually somatic, could be germ line specific	Phenotypic change (change in gene expression?)	Natural selection of the ability to respond to the stimulus	Baldwin (1896), Waddington (1942), Schmalhausen (1949); see Chapter 2
Somatic and germ line	Somatic	Parallel induction: phenotypic change in soma and corresponding mutation in germ line	Somatic change (e.g. hormonal) causes an identical genetic change in the germ line	Stempell, and others; see Rensch (1983)
Somatic and germ line	Somatic or germ line specific	Parallel induction: identical mutations in soma and germ line	Conventional genetic transmission	see Blacher (1982)
Somatic and germ line	Somatic or germ line specific	Parallel induction: identical heritable epigenetic changes in soma and germ line	Epigenetic inheritance through the germ line	Jablonka and Lamb (1989); see Chapter 6
Germ line (or unicellular organism)	Germ line	Directed mutation	Conventional genetic transmission	Weismann (1902), Cairns *et al.* (1988), Hall (1988); see Chapter 3
Germ line	Somatic or germ line	Change in chromatin structure	Epigenetic inheritance	Jablonka and Lamb (1989); see Chapters 4 and 6

Table 1.1 (*continued*)

Affected cell types	Affected character	Response to the inducing agent	Mechanism leading to the character's inheritance	References
Somatic (or unicellular organism)	Somatic	Change in architectural structures	Templating and self-propagation	Sonneborn (1964), Nanney (1968), and others; see Chapter 4
Somatic (or unicellular organism)	Somatic or germ line	Change in metabolic feedback	Self-sustaining metabolic loops	Delbrück (1949), Hinshelwood (1953); see Chapter 4

Definitions of 'Lamarckian' terms

Because of the confusion that has always surrounded the use of 'the inheritance of acquired characters', Mayr (1982*a*) has suggested that the term 'soft inheritance' should be used to cover all the different meanings that have been associated with the former concept. He defines soft inheritance as:

> Inheritance during which the genetic material is not constant from generation to generation but may be modified by the effects of the environment, by use or disuse, or other factors. (Mayr 1982*a*, p. 959)

Medawar made a useful distinction between two types of Lamarckism. He described the 'weak' form of Lamarckism in the following way:

> Modifications acquired in each member of a succession of individual lifetimes, as a result of recurrent responses to environmental stimuli, may eventually make their appearance in ontogeny even when the environmental stimuli are absent or are deliberately withheld . . . and the age of appearance of these modifications in ontogeny will eventually anticipate the age at which environmental stimuli could in any case have been responsible for them. (Medawar 1957, p. 83)

This weak form of Lamarckism has nothing to say about the mechanism underlying the inheritance of the acquired character. The 'strong' form does. The 'strong' form of Lamarckism requires that:

> The repeated induction of character-differences within the lifetimes of individuals of successive generations is accompanied by a genetic change in each individual, the change being such as eventually to reproduce the character-difference elicited by environmental stimuli even when those stimuli are withheld. (Medawar 1957, p. 91)

For the purposes of this book we define the inheritance of acquired characters in the following way. The inheritance of an acquired character has occurred if:

(1) the change in the character is induced by the environment;
(2) the induced change is specific and repeatable, although not necessarily adaptive;
(3) a specific change in hereditary information is involved;
(4) the change is transmitted to the next generation.

Essentially, this definition conforms with Mayr's definition of soft inheritance, but it avoids Mayr's phrase 'the genetic material', which is usually assumed to be synonymous with DNA. It therefore allows for the possibility that heritable information can be carried in ways other than in the sequence of bases in DNA. The definition also conforms with Medawar's definitions of both the weak and strong forms of Lamarckism, again providing that the word 'genetic' in Medawar's definition is not confined to classical DNA-based heredity. It is important to note that our definition, like those of Mayr and Medawar, is a definition of the inheritance of *acquired* characters, not of *required* characters.

Mendelian genetics and Lamarckian evolution

With the benefit of hindsight, the way in which the new science of genetics influenced ideas about evolution at the beginning of this century is strange.[15] At first many Mendelians claimed that their work showed that Darwin was wrong. They argued that mutations are much more important than natural selection in bringing about evolutionary change. On the other hand, many non-geneticists believed that the discontinuous characters studied by the geneticists are irrelevant in evolution. Natural selection works on continuous variation, which can be influenced by environmental factors. Many embryologists and physiologists thought that even if Mendelian factors in the nucleus are responsible for individual and racial characteristics, non-Mendelian hereditary factors located in the cytoplasm are responsible for the characters that determine the genus and species to which an animal belongs.[16] They believed that the pliable cytoplasm, which harbours these non-Mendelian factors, allows the inheritance of acquired characters. Initially, therefore, Mendelian genetics did not strengthen Darwinism, and did little to make non-geneticists doubt the possibility of the inheritance of acquired characters.[17]

It was not until the late 1930s that Mendelian genetics became integrated with evolutionary biology in a way that signalled the end for Lamarckian theories of evolution. In what is now known as 'The Modern Synthesis of Evolution', the Mendelian gene, a factor that is stable in heredity and immune from the influence of the environment, was accepted as the material basis of all evolutionary change. It was assumed that environmental effects on characters, which are so widespread and so striking, do not involve changes in the genotype; the phenotypic differences induced by the environment are not inherited.

The conceptual distinction between genotype and phenotype—between instructions and their implementation—was made by Johannsen in the first decade of this century.[18] It was of fundamental importance for the development of genetics, and also had important consequences for the development of evolutionary theory, because it was the concept of heredity that grew out of Johannsen's ideas that was incorporated into the Modern Synthesis of evolutionary biology in the 1930s and 1940s, and to a large extent remains with it. It is a very restricted notion of heredity, and it is this restricted view that has been the basis of many of the objections to the possibility that acquired characters can be inherited.

Johannsen's ideas were based both on the patterns of inheritance of Mendelian genes, and on his own work on pure lines of plants. Pure lines are strains produced by self-fertilization. Johannsen found that lines developed from different individuals had different characteristics. Although individuals within a line differed in appearance, the differences between them were not heritable. Selection was ineffective in pure lines.[19] Similar results were obtained by Jennings (1909) with *Paramecium*, in which pure lines derived from single individuals were found to differ in size, structure, and physiological characteristics. Although these characters were influenced by environmental conditions, the environmentally induced changes were not passed on.

In 1909, Jennings asked about the pure line idea 'Is it possibly of sufficient importance to deserve agitating a little before the American Society of Naturalists?' Clearly it was, because in the following year the Society held a symposium devoted to 'The Study of Pure Lines or Genotypes'.[20] Most papers read at this meeting supported Johannsen's ideas. Johannsen's own contribution was entitled 'The genotype conception of heredity'. In it Johannsen attempted to clarify the concept of heredity in biology. He insisted that biological heredity is not the transmission of characters, it is the transmission of what we would now say are the instructions for building characters. Johannsen distinguished between 'heredity', the passing on of 'potentialities', and 'transmission', a concept based on human practices such as the transfer of property or ideas from one person to another. He argued that in biology the physical transmission of the personal qualities of individuals to their progeny does not occur:

> The *personal qualities* of any individual organism do not at all cause the qualities of its offspring; but the qualities of both ancestor and descendant are in quite the same manner determined by the nature of the 'sexual substances'—i.e., the gametes—

from which they have developed. Personal qualities are then *the reactions of the gametes* joining to form a zygote; but the nature of the gametes is not determined by the personal qualities of the parents or ancestors in question. (Johannsen 1911, p. 130; Johannsen's italics)

The appearance of an individual depends on the inherited potentialities, which Johannsen called the 'genotype', and on the environment. The character, the end-product of the interaction between environment and genotype, Johannsen christened the 'phenotype'.[21] The unit of biological heredity, the Mendelian factor which Johannsen named 'gene', was not a material model or representation of the phenotype, but a unit of information. All individuals in a pure line are genotypically the same. Their heritable genotypes are unchanged by environmental factors, although the material realization of these genotypes may be.

Johannsen stressed the implications of his conceptual distinction between genotype and phenotype for the questions concerning the inheritance of acquired characters:

The principle of pure lines or, generally, pure culture, is of importance also for elucidating the celebrated question of the inheritance of 'acquired characters'. Mendelism and pure-line researches are here in the most beautiful accordance, both emphasizing the stability of genotypical constitution; the former operating with the *constituent unities*, the latter with the behavior of the *totality* of the genotypes in question. . . . as yet no experiment with genotypically homogeneous cultures has given any evidence for the Lamarckian view, the most extreme 'transmission'-conception ever issued. (Johannsen 1911, p. 141; Johannsen's italics)

Initially, Johannsen's experiments were also seen as evidence that Darwinian natural selection could not be the basis of evolutionary change. Selection, like environmental actions, had no effect on pure lines, so Johannsen concluded that mutation was more important than selection in bringing about evolutionary change.

In the long run, Johannsen's influence on evolutionary ideas in the first quarter of this century was probably of less significance than the influence his concept of heredity had on the development of the new science of genetics. According to Johannsen:

Heredity may then be defined as *the presence of identical genes in ancestors and descendants*, or, as Morgan says in full accordance with this definition: 'The word heredity stands for those properties of the germ-cells that find their expression in the developing and developed organism'. (Johannsen 1911, p. 159; Johannsen's italics)

It was this restricted concept of heredity that was adopted by the influential American geneticists. As genetics increased in importance and influence, so did this view of heredity.[22] The mechanisms of cellular inheritance—of the inheritance of determined and differentiated states during development

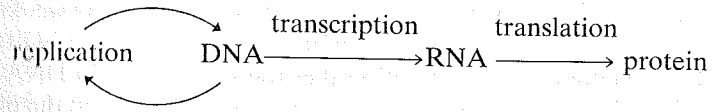
—were largely excluded from the study of heredity. Only nuclear genes were important, and these were immune from cytoplasmic and environmental effects. Sapp has summed up the situation in this way:

Ironically, Johannsen's genotype/phenotype distinction offered geneticists the conceptual space or route by which they could bypass the organization of the cell, regulation by the internal and external environment of the organism, and the temporal and orderly sequences during development. Although the genotype/phenotype distinction represented an implicit theoretical acknowledgement of the beginning and end of a production, in practice, Mendelian geneticists ignored developmental processes and the possible influence of extragenic conditions in the production of characters. (Sapp 1987, p. 49)

When in the late 1930s, after a quarter of a century of largely independent growth, disciplines such as biogeography, palaeontology, systematics, natural history, and genetics began to be integrated in the Modern Synthesis, the view of genetics brought to evolutionary studies was based on a narrow concept of heredity. It is this limited view of heredity that remains with much of evolutionary biology today.[23] There was little in the synthesis about development and differentiation.[24] It was the Mendelian hereditary unit, the gene, whose behaviour could be studied through transmission genetics, and whose frequency could be manipulated on paper by the population geneticists, that became the material basis of evolutionary change.

Molecular genetics and the inheritance of acquired characters

The 1940s and 1950s saw the growth of microbial and biochemical genetics, and the development of techniques for studying gene action. The gene was found to control the production of specific proteins. The molecular nature of the gene and the way in which it specifies proteins were quite rapidly unravelled: the genetic material is DNA, which carries the information for making proteins encoded in its base sequence; this information is first transcribed into RNA, and the RNA messages are then translated into the amino acid sequences of proteins. The *central dogma*[25] of molecular biology was established: information flow is unidirectional—it passes from DNA to proteins, but not in the reverse direction:



The environment can alter the instructions in DNA only accidentally. Proteins, and systems built from proteins, are highly sensitive to the environment, but since protein is not the hereditary material, modifications in protein structure and function cannot lead to inherited changes.

Molecular genetics has tended to reinforce the attitude to heredity adopted by the Mendelian geneticists. The genotype has become identified with DNA, the phenotype with proteins or the products of proteins. The genotype is regarded as a set of instructions, subject to only random changes; the phenotype is the result of the implementation of these instructions, and can be modified by the environment in an adaptive way. One of the most explicit articulations of this position has been made by Dawkins, who claimed:

> The inheritance of acquired characteristics not only *doesn't* happen: it *couldn't* happen in any life-form whose embryonic development is epigenetic rather than preformationistic. (Dawkins 1986, p. 298; Dawkins's italics)

Since embryonic development is not preformationistic—genes do not contain a description of the adult characters—Dawkins argued that if a character is modified by the environment, information about the change cannot, even in theory, be fed back into the genes. Dawkins pointed out that DNA is not, as is sometimes said, a blueprint; it is more like the recipe for a cake: a set of instructions for carrying out a process. The words of the recipe (the DNA sequences) do not correspond to crumbs of the cake (parts of the body); they represent stages in the process of making the cake. Consequently, a word difference leads to a whole-cake difference:

> 'Baking powder' does not correspond to any particular part of the cake: its influence affects the rising, and hence the final shape, of the whole cake. If 'baking powder' is deleted, or replaced by 'flour', the cake will not rise . . . There will be a reliable, identifiable difference between cakes baked according to the original version and the 'mutated' versions of the recipe, even though there is no particular 'bit' of any cake that corresponds to the words in question. This is a good analogy for what happens when a gene mutates. (Dawkins 1986, p. 297)

Using this cake analogy, Dawkins says of Lamarckian inheritance:

> We can no more imagine acquired characteristics being inherited than we can imagine the following. A cake has one slice cut out of it. A description of the alteration is now fed back into the recipe, and the recipe changes in such a way that the next cake baked according to the altered recipe comes out of the oven with one slice already neatly missing. (Dawkins 1986, p. 298)

This cake analogy is a clear and comprehensible expression of the current dogma about Lamarckian inheritance. It also exposes the weakness of that dogma. The theoretical impossibility it is meant to illustrate is an artefact of the analogy. The assumption that the instructions in DNA are isolated from their implementation, with no interactions between the products of the instructions—the protein or phenotype—and the DNA instructions themselves, is not valid. As we shall discuss in Chapter 3, there is evidence suggesting that there are mechanisms that enable the genome to sense an environmental change, respond to it, and transmit the response to

descendants. The recipe is not isolated from the cake, because it is contained in and is executed by the components of each crumb of the cake. The analogy is inadequate because it is based on a rigid distinction between the genotype and phenotype—between the instructions encoded in DNA and the result of the execution of these instructions.

There is a second invalid assumption in the cake analogy. It is that there is a single inheritance system. We want to suggest a rather different analogy, which reflects the fact that there are multiple inheritance systems. This analogy shows how the distinction between genotype and phenotype becomes blurred if information is transmitted in ways additional to that involving DNA base sequence. Instead of a cake and a recipe, consider a piece of music that is transmitted from generation to generation as a written score. If the score represents hereditary information in DNA, the phenotype is a specific interpretation of this score at a certain time by certain artists. The interpretation does not affect the score. However if there is another transmission system—recordings—through which a particular interpretation can be transmitted from generation to generation along with the written score, the situation is rather different. There can then be evolution of interpretations of the score, based on the influence that one interpretation has on subsequent interpretations, and that these have on still later ones, and so on. Both the phenotype (the present interpretation) and the genotype (the written score) influence subsequent interpretations.

We believe that this music analogy is more appropriate than the cake analogy because the old notion that the DNA of nuclear genes is the sole carrier of hereditary information is incorrect. Molecular biology, which was at first centred around genetics, has now spread to encompass many other disciplines, including embryology. Studies of growth and development are no longer divorced from studies of heredity. Through molecular studies of differentiation and cellular inheritance, it has become clear that information is transmitted in ways other than through the primary base sequence of DNA. As we shall show in later chapters, these additional systems can also transmit information between generations of organisms, and permit the inheritance of acquired characters.

The arguments against the inheritance of acquired characters just discussed stem from the conventional picture of the relation between genetics and development. There is another argument, an evolutionary argument, against the idea that acquired characters can be inherited. It is that most acquired characters are detrimental: they are the consequences of injury, disease and ageing.[26] Consequently, it is argued, mechanisms allowing the inheritance of such maladaptive changes should be strongly opposed by natural selection. Although this must be true, it has to be remembered that the same argument can be applied to mutations: they, too, are frequently maladaptive, yet no one doubts that they occur and are inherited, in spite

of often being counter-selected. As with other evolutionary features, including mutation frequencies, the extent to which acquired characters are inherited presumably depends on the relative costs and benefits of the presence or absence of the mechanisms involved. We shall consider this further in Chapter 8.

Why is evidence for Lamarckian inheritance so sparse?

If there are mechanisms through which acquired characters can be inherited, why has more than a century of study of heredity failed to reveal sufficient cases of 'Lamarckian inheritance' for it to have become an accepted part of evolutionary theory? We shall discuss this in some detail in Chapter 6, but for the present we want to suggest two types of reason for the apparent paucity of evidence for Lamarckian inheritance. The first is that usually people have looked in the wrong type of organism, in the wrong place, and for the wrong type of change. The second is that people have gone to great lengths to interpret all experiments and observations that might be taken as evidence for Lamarckian inheritance in non-Lamarckian terms.

Evidence for or against the inheritance of acquired characters is not to be found in the type of experiment carried out by Weismann in the last century. He cut off the tails of mice for 22 generations and showed that it had no effect on the tail length of the progeny.[27] Many generations of male and female circumcision, and the docking of sheep's tails, have shown the same thing: mutilations are not inherited. This type of experiment (which is the type illustrated by Dawkins's slice of cake analogy) would demonstrate Lamarckian inheritance only if information from the somatic parts of the adult mammalian body were transferred to germ cells. As far as we know it is not. An experiment that is more likely to demonstrate Lamarckian inheritance is one in which an induced change affects the whole organism, including the cells that produce the next generation. For example, cellular adaptations to an environmental change that affects all cell types in a species that reproduces by fragmentation are likely to be inherited. Unfortunately, the dominance of Mendelian genetics during the first half of this century meant that the study of heredity centred on organisms and characters that are unlikely to yield evidence of the inheritance of acquired characters very readily. The animals used were mainly mammals and insects, in which the germ line is segregated from the soma early in development. Most of the characters studied were stable adult features. Examples of irregular hereditary patterns were found, but as Sapp (1987) has documented, for many years the study of such oddities attracted few workers and little financial support. Studies of heredity were concentrated on nuclear genes and chromosomes, and their segregation in crosses between sexually reproducing organisms; organisms and characters that were not amenable to analysis by the techniques of transmission genetics were generally excluded.

To a large extent the concept of heredity adopted by the geneticists, and the methods and organisms they used to study it, have prevented cases of Lamarckian inheritance being found. Induced inherited changes are most likely to be detected, and are probably of more evolutionary importance, in plants, in some invertebrate groups other than insects, and in microorganisms (see Chapters 2 and 8). The inherited, environmentally induced changes that occur in organisms such as the mouse and *Drosophila* are probably small, causing only minor variations in the expression of genes, and are likely to have been explained away as the result of the action of 'modifiers', rather than being investigated seriously. As Lindegren (1949) described with reproach, even in *Neurospora*, mutations that were found to be unstable in inheritance were discarded as a matter of course.[28]

The reason for the paucity of examples of the inheritance of acquired variations may therefore be similar to that which, until relatively recently, pertained to the rarity of examples of jumping genes. For more than thirty years most people regarded jumping genes as an esoteric peculiarity of maize, a rarity of no fundamental importance for genetics. Yet now that their existence is taken for granted, and the right techniques are available, they are found almost everywhere.

In fact, and in contradiction to general belief, there are several well-recognized and quite well-understood examples of the inheritance of acquired characters. Most involve microorganisms; many involve changes in cytoplasmic DNA or cytoplasmic organelles. For example, if the chloroplasts of the protist *Euglena* are destroyed with streptomycin, the subsequent generations of their progeny lack chloroplasts; bacteria can acquire and transmit to their descendants plasmids from species that carry genes for drug resistance.[29] It is now quite generally accepted that some cell organelles such as mitochondria and chloroplasts were originally prokaryotic symbionts in eukaryotic cells. We do not intend to devote much space in this book to the inheritance of these and similar acquired characters, because they do not pose a theoretical problem for orthodox evolutionary theory. But the fact that they are not regarded as a problem is of interest, because it illustrates the general attitude to Lamarckian inheritance. Discussing examples such as those above, Fitch suggested that the reason why they are not regarded as a problem for Darwinism has nothing to do with genes or DNA *per se* because:

> Genes and DNA are the means of inheritance and both Darwinism and Lamarckism must incorporate these facts.
>
> The reason for there being little concern among Darwinists for this rather common inheritance of acquired characters is that 'the inheritance of acquired characters' is more of a slogan that captures a part of the Lamarckian spirit than a

statement of its basic distinctiveness from Darwinism. Rather, it seems to me, the crucial difference arises from the issue of cause and effect between the phenotype and the genotype. Clearly, each affects the other but we are in no danger of circularity because the question is whether an evolutionary novelty, arising at the level of the phenotype, can produce a genotype that assures the phenotype's continuance, or whether an evolutionary novelty, arising at the level of the genotype, can produce a phenotype that assures the genotype's continuance. In every one of the above examples of the inheritance of acquired characters, *it was the genetic material that changed first and was acquired*. . . . Only an overly narrow definition of the source of genetic variability, more narrow than anything Darwin could have seriously proposed, could require a modification of Darwinism . . . (Fitch 1982, p. 1137; Fitch's italics)

We agree with Fitch that 'the inheritance of acquired characters' is not well defined, and that the basic tenets of Darwinism are not shaken by the observations he discusses. What we find interesting is his reliance on the genotype-phenotype distinction to show why the observations have not been a problem for Darwinism. Fitch seems to have been saying that so long as the primary changes occur at the genotype level, the inheritance of acquired characters is compatible with Darwinism. Although this attitude is certainly compatible with Darwin's Darwinism, which embraced the inheritance of acquired characters, we doubt that it is readily acceptable to most contemporary Darwinians. The attempt to place observations such as acquired drug resistance within the Darwinian framework is strange for two reasons. First, as Fitch himself stressed, Darwinian evolution does not require all evolution to be Darwinian. Second, as Fitch also pointed out, molecular biology is revealing all sorts of strange genomic behaviour and genomic responses, which have to be incorporated into evolutionary theory. If the internal and external environments can direct genomic behaviour in the way it seems to, surely the genotype–phenotype distinction has become so blurred that it is no longer possible to say whether a response begins with the genotype or with the phenotype, and it is no longer useful to try to do so.

When considering the impact molecular biology would have on evolutionary theory, Fitch (1982) predicted that some organisms would be found to have mechanisms that increase the mutation rate specifically in those genes whose activity could be useful for survival. Evidence for such 'directed mutation' was soon found: some mutations in bacteria appear preferentially in the environmental conditions in which they are beneficial. This discovery came as a shock to most biologists, reared as they had been on the notion of random mutation. It led to a lively debate about the interpretation and significance of the experiments. We discuss this work in some depth in Chapter 3. What we want to highlight here is the emotional response to the possibility of directed mutation, and the way in which the debate revealed an extreme reluctance to admit the possibility of Lamarckian

evolution. For example, Lenski and his colleagues (1989) claimed that even if the existence of directed mutations in bacteria were proved beyond reasonable doubt, it would not constitute evidence of Lamarckian evolution, because Lamarck thought that the inheritance of acquired characters occurs through the effects of use and disuse. This is unreasonable. First of all, some explanations of directed mutation do in fact suggest that the mutations are induced as the result of a form of 'use', namely, gene expression. More importantly, as we discussed earlier, Lamarckism has not remained unchanged since Lamarck's time, any more than Darwinism has remained unmodified since the publication of *The origin of species*. Many scientists have considered themselves, and were considered by others, to be Lamarckians because of their belief in the direct effect of the environment on heritable qualities, not necessarily through use and disuse. It is confusing if, in order to avoid the stigma of Lamarckism, Lamarckian evolution is re-defined in restrictive terms.

The debate about directed mutation also illustrates another way of avoiding the stigma of Lamarckism, and reconciling the possibility of induced inherited variations with neo-Darwinian orthodoxy. This is to shift from thinking about selection of individuals, to thinking about selection within the individual. Some interpretations of the experiments showing directed mutation were based on selection occurring between variable intracellular DNAs, RNAs, etc. In this way, instead of the individual bacterium being the unit of selection, the unit of selection became the intracellular molecule. On this basis, although directed mutation looks Lamarckian when the unit of analysis is the individual bacterium, the adaptive response is really the result of Darwinian selection between accidental molecular variants *within* the bacterium. As Keller (1992) has noted, the issue is whether directed mutation can be described in the Darwinian language of chance and selection, or whether the Lamarckian language of purpose and choice has to be used. If the individual is the unit of analysis, it seems that Lamarckian language is appropriate, whereas if considered at the level of intracellular variations, Darwinian language is suitable.

Keller has shown how, through the choice of language and use of intracellular selection, directed mutation was brought within the Darwinian framework. Although more comfortable for Darwinians, the problem with this approach is that it makes no sense to change the level of analysis if Darwinian and Lamarckian explanations are to be usefully compared and assessed. Medawar recognized and emphasized this point many years ago when discussing a possible Lamarckian mechanism for gradual adaptation in bacterial cultures:

It may be assumed that there are alternative pathways of metabolism within each cell, i.e. alternative enzyme sequences or metabolic gearings, as there are, for

example, alternative pathways for the degradation of glucose. Such metabolic pathways may for a variety of reasons be so adjusted as to be mutually inhibitory, so that only one prevails in any one of a possible set of steady states. The inhibition of one such system therefore entails its replacement by another. In other words, as Hinshelwood (1946) has made clear, the Lamarckian transformation . . . may be Darwinian at the lower analytical level represented by the enzymic population or complex of intersecting metabolic pathways within the individual bacterial cell. Such a description would be pointless for any except explanatory purposes, but it shows that *no discussion of the rival interpretative powers of Darwinism and Lamarckism can have any useful outcome unless a certain analytical level is defined and adhered to*. Hereafter we shall be concerned with individual organisms as analytical units, for it is only in this context that the rivalry is of any moment. (Medawar 1957, p. 82; our italics)

Perhaps the foremost reason for the reluctance to accept Lamarckian interpretations is the feeling that by so doing, one is accepting purposeful evolutionary responses: that an organism has some indefinable properties that propel it towards some goal. How does the organism *know* how to change its genetic material according to new environmental specifications? Again, the debate on directed mutation yielded examples of how this problem can be circumvented. Bruce Wallace (1990) and Sydney Brenner (1992) both attempted to make the Lamarckian idea of environmentally directed mutation more palatable to Darwinians by treating it as an adaptive response, which has itself evolved through Darwinian selection of random variations. Brenner wrote of directed mutation in bacteria:

There may still be biologists who would like the phenomenon to have some trivial explanation and to go away. However, even if it stays, as seems likely, no radical alteration of our views is demanded. Bacteria spend a considerable part of their natural lives under nutritional constraints so that if there was a mechanism to promote adaptive mutation, the mechanism itself would have been selected for, and the apparent genetical intentionality of *E. coli* could have been brought about by the process of natural selection. (Brenner 1992, p. 168)

Wallace argued that, from an evolutionary point of view, it does not matter whether an adaptive response is brought about by a mechanism involving a DNA change such as the excision of a DNA segment, or by more conventional means such as the binding of a regulatory protein. Whatever the nature of the response, the ability to make the adaptive modifications is under genetic control, and the genetic programme underlying the response system must have evolved via Darwinian selection. According to Wallace, the important part of evolution is the evolution of the genetic programme, not of the specific manifestations of that programme. His argument emphasizes the point made by many previous critics of Lamarckism, that Lamarckian mechanisms of induced variation may explain the evolution of an adaptation, but they cannot explain the evolution of adaptability. Although the specific adaptive response may be

the result of directed mutation or other types of genomic response to the environment, the genetic machinery that underlies it must have evolved first by Darwinian mechanisms.

The evolution of systems that can lead to Lamarckian evolution is a fascinating subject, and we shall return to it in Chapters 7 and 8. We do not doubt that the basic mechanisms underlying the inheritance of acquired variations evolved in a Darwinian fashion by the selection of accidental variations. However, why should the evolutionary origin of Lamarckian inheritance systems be of any relevance when considering the effects these systems have once they are in place? By stating that the rules of the evolutionary game must have evolved via Darwinian selection, Wallace and Brenner may have helped make the idea of directed mutation more acceptable to Darwinists, but if the rules of the game are Lamarckian, their evolutionary origin is irrelevant to the way in which they dictate the course of evolution. Consider cultural inheritance. It is reasonable to assume that the cognitive mechanisms that allow the transmission of information among human beings were the result of Darwinian evolution, but once cognitive mechanisms such as long-term memory, and the ability to imitate and to teach appeared, they dictated the course of cultural evolution. The new rules of the game help to determine the course, the direction, and the rate of evolution. The same is true for the inheritance of acquired characters. The ability to acquire and transmit changes in some characters may have evolved through Darwinian evolution, but once the response and transmission systems had evolved, they constituted a new mechanism of evolution, operating alongside Darwinian evolution by means of natural selection of random genetic variations.

Through Darwinian selection, multiple inheritance systems have evolved, generating different types of heritable variation, which now play a role in evolutionary change. The transmission system on which we shall concentrate in this book is the Lamarckian inheritance system that operates in cell lineages. We are going to present a picture of heredity which, unfortunately, is not as neat and elegant as that of classical genetics, where the unaltered gene passes from generation to generation, immune to environmental influences. The non-DNA systems that we shall discuss are sometimes referred to as *epigenetic inheritance systems*, although the distinction between genetics and epigenetics, like the distinction between genotype and phenotype, has become rather blurred. Epigenetic inheritance systems are responsible for transmitting determined and differentiated states during ontogeny. The attitude of evolutionary biologists to this type of inheritance was summed up with characteristic honesty by John Maynard Smith in 1966:

The view generally taken by geneticists of differentiation, when it is not simply forgotten, is that the changes involved are too unstable to be dignified by the name 'genetic', or to be regarded as important in evolution. I tend to share this view, although I find it difficult to justify. (Maynard Smith 1966, p. 71)

The mechanisms underlying differentiation and the role of epigenetic inheritance in development are now beginning to be unravelled. We hope to show that the epigenetic inheritance systems, which are so important in development, are also important in evolution, and that evolution and development are far more directly intertwined than is usually supposed. When epigenetic systems are considered, the environment is more than a mere selective agent, it is also an inducer of specific heritable variations.

Summary

The ideas developed by Lamarck are very different from what is now commonly referred to as Lamarck's theory of evolution. 'Lamarckism' is generally considered to be more or less synonymous with 'the inheritance of acquired characters', but there is no agreement about exactly what this phrase means. For some people it is appropriate only if the characters that are acquired are adaptive, while for others any repeatable change induced in one generation and transmitted to the next can be regarded as an inherited acquired character. Since the 1940s, most people have doubted that Lamarckian evolution can occur at all, believing that changes in the phenotype cannot be transmitted to the genotype, and therefore that Lamarckian inheritance is impossible in principle. Belief in the one-way flow of hereditary information between genotype and phenotype was reinforced when the central dogma of molecular biology was formulated in the late 1950s, proclaiming as it did that information passes from DNA to proteins, but never in the reverse direction.

In recent years, molecular biology has shown that the genome is far more fluid and responsive to the environment than previously supposed. It has also shown that information can be transmitted to descendants in ways other than through the base sequence of DNA. Even so, most people still deny that Lamarckian evolution occurs. On the one hand, there are those who accept that the genome is a response system and not just a passive information carrier, but argue that because it is the genotype or DNA that is modified, the induced changes cannot be regarded as Lamarckian. On the other hand, there are those who recognize the existence of non-DNA inheritance systems (such as cultural inheritance), but argue that since only the genotype is inherited in a biological sense, the transmission of non-DNA variations does not qualify as inheritance and is therefore irrelevant in biological evolution. Critical evaluation of the role of the inheritance of acquired variations in evolutionary change is thus avoided by using arbitrary definitions of heredity and acquired variations.

Adhering to the idea that evolutionary change cannot be the result of the inheritance of environmentally induced changes is misguided. Not all adaptive changes are the result of Darwinian selection of random variations created by the shuffling of genes and rare chance mutations. The nature of different types of heritable variation is now beginning to receive closer attention, and there is a growing realization not only that some DNA variations can be environmentally induced, but also that there are non-DNA heritable variations that play a crucial part in development. The importance of the inheritance systems underlying the variations seen in development has only recently been recognized. Nevertheless, as we show in the next chapter, there was interest in epigenetics and development, and an awareness that the mechanisms underlying development needed to be integrated into evolutionary theory, long before molecular biology began to uncover the types of mechanism that may be involved.

PRIONS

Notes

1. We feel it necessary to stress our belief in Darwinian evolution because recent history has shown that any argument suggesting that Darwinian evolutionary theory should be modified or amended is liable to be used by Creationists as evidence that the theory of evolution is wrong. Like most Darwinians, we believe that Darwinian evolutionary theory is a flexible theory, quite capable of accommodating modifications and amendments.
2. See Mayr (1982*a*, p. 352). Other accounts of Lamarck's ideas and his place in the history of biology can be found in Blacher (1982), Bowler (1983), Jordanova (1984), and Oldroyd (1983).
3. Dr Peter McLaughlin has directed our attention to the fact that in the original French edition, Lamarck's figure, and the addition of which it is a part, appear at the very end of the book, and not immediately after Chapter 8 as in the English translation published in 1914. He suggests that this placement reflects the interpretation given to Lamarck's ideas in the post-Darwinian climate at the beginning of this century, when one of the main concerns was with phylogenetic trees.
4. In his *Histoire naturelle des animaux sans vertèbres* published in 1815, Lamarck gives four, rather than two, laws: the first law describes the inevitability of increase in size; the second and third describe how the need to cope with the environment leads to changes in organs through use and disuse; the fourth deals with the inheritance of the acquired character.
5. An account of the history of the idea that acquired characters can be inherited is given in Zirkle (1946).
6. The criticism that Lamarckism does not explain adaptability has been made many times, e.g. see Blacher (1982, pp. 154–155), and Dawkins (1986, p. 299).
7. The example of skin thickening and calluses has been a favourite with evolutionary biologists seeking to explain the relation between adaptation and adaptability, and we shall refer to it in other parts of this book. Darwin used the prenatal thickening of the sole of the human foot as an example of the inheritance of an acquired character (Darwin 1871, Vol. 1, Chapter 4).
8. Accounts of early theories of inheritance are given in Zirkle (1946), Blacher (1982), and Mayr (1982*a*).
9. For example, see Gould (1982, p. 381).

10. Detlefsen (1925) gives a contemporary account of Lamarckism in the 1920s.
11. Pfeifer (1965) and Rensch (1980) give accounts of American and German neo-Lamarckism in the early part of this century.
12. A detailed exposition of this theory is given in Cope (1904).
13. In later life Weismann accepted that environmentally induced characters could be inherited if they affected the germ line itself directly. He also accepted the idea of parallel induction—the parallel effect of the environment on both germ line and soma. Weismann did not consider these to be cases of the inheritance of acquired characters because there was no *transfer* of information from soma to germ line. These aspects of Weismann's theory are discussed more fully in Chapter 2.
14. See Hull (1984, p. xliii) for a discussion of how 'Nearly every type of hereditary phenomenon has been termed at one time or another Lamarckian'.
15. See Bowler (1988) Chapter 5 for a discussion of the impact of Mendel's findings on evolutionary biology. Falk (1994) gives an interesting discussion of the way in which de Vries, one of the 're-discoverers' of Mendel's laws, gave a secondary role to Mendelian characters in his theory of evolution.
16. See Sapp (1987, p. 17). Sapp's book gives a fascinating account of the history of the debate about the relative importance of the nucleus in inheritance and development.
17. One of the most important consequences of Mendelian genetics for theories about the inheritance of acquired characters was that it introduced new and strict methodological requirements for experiments designed to test them. It became clear that it was necessary to use pure lines in order to exclude the possibility that hidden recessives and altered epistatic interactions are responsible for newly acquired inherited characters.
18. Although Johannsen was at pains to disassociate himself from Weismann's ideas (e.g. see Johannsen 1923), in many ways his genotype–phenotype distinction follows naturally from Weismann's insistence on the difference between effects on the soma and those on the germ line (see Chapter 2).
19. In fact, as Provine (1971) has documented, almost immediately after publication in 1903, Johannsen's data were claimed by Pearson and Wheldon to be at variance with his conclusion that selection is ineffective in pure lines.
20. Jennings paper suggesting that the pure line idea was important was published in the *American Naturalist* 1910; papers presented at the symposium held in December 1910 are to be found in the *American Naturalist* of the following year.
21. Originally, Johannsen applied the terms phenotype and genotype to populations, rather than individuals. 'Genotype' was almost synonymous with 'pure line', and 'phenotype' referred to the range of variation in a pure line over several generations. However the terms were rapidly adopted for the genetic constitution (genotype) and appearance (phenotype) of individuals. For a discussion of the history of the genotype–phenotype distinction and its role in the history of biology see Churchill (1974) and Allen (1979).
22. For an interesting discussion of the route by which development became detached from genetics and the reasons for it, see Falk (1994). Harwood (1993) has stressed that in Germany genetics was not divorced from studies of development and evolution as it was in the USA, and the view of heredity adopted by most German geneticists was much broader.
23. Hull (1988) gives a modern version of biological inheritance which almost echoes Johannsen's: 'In biological evolution, inheritance counts as "Lamarckian" if adaptive changes in the phenotype of an organism were transmitted to the genetic material and thereafter inherited by the organism's progeny. Acquired characteristics must be *inherited*, not just transmitted'. (p. 37, Hull's italics). Hull makes the distinction between transmission and heredity in the same way, and indeed in the same context, as that made by Johannsen in his 1911 paper. He argues that the analogy between the inheritance of cultural products and the inheritance of genes is only metaphorical, and can be misleading, because genes transmit instructions, whereas usually in the cultural context one thinks of transmission of overt qualities.
24. For a discussion of the role of embryology in the evolutionary synthesis, see Hamburger (1980).
25. Crick (1958). The central dogma of 1958 did not explicitly include mRNA, and of course did not include the possibility of reverse transcription from mRNA to DNA. When reverse transcription was discovered in 1970, no conceptual change in the central dogma was necessary, because information still could not flow from proteins to DNA, from phenotype to genotype.
26. Lamarck was aware of this problem and rejected direct influences of the environment on hereditary characters. He stressed that only active responses to the environment would lead to heritable changes.
27. Weismann (1904, Vol. 2, p. 66). Weismann's experiment was not, of course, designed to refute Lamarck's version of the inheritance of acquired characters, which was based on use and disuse. Like Dawkins' cake analogy, the results of the experiment are relevant only to the primitive type of idea about the inheritance of acquired characters that originated long before Lamarck's time.
28. Lindegren wrote: 'The genetical data on which the modern conception of the gene is based are intensively selected data . . . The search for precisely segregating genes compels the selection of genetical material. In our own work on Neurospora we were unable to classify the progeny of over two-thirds of our matings'. (Lindegren 1949, Chapter 20, pp. 6–7)
29. For a more detailed discussion of these and similar examples, see Landman (1991).