

## Chapter 8

### The Role of Genes in Natural Selection

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The whole purpose of our search for a ‘unit of selection’ is to discover a suitable actor to play the leading role in our metaphors of purpose. We look at an adaptation and want to say, ‘It is for the good of . . .’. Our quest is for the right way to complete that sentence.

— Dawkins, *The Extended Phenotype*

Darwin’s theory of natural selection is extraordinarily simple, but the natural world to which it applies is extraordinarily complex. Herein lies the difficulty that is the subject of the next two chapters.

The theory is that whenever individuals reproduce at different rates according to their inherited and randomly mutated properties then those individuals will evolve to become adapted to their environments. But nature does not come ready-carved into individuals. Ecosystems spread and compete with others. Species compete within ecosystems. Species are divided into sub-populations and colonies. Organisms are made of many reproducing cells. Cells contain many components that have their own replicating DNA. And each DNA string contains many replicating sequences. So which of these individuals does the theory apply to? What is the correct way to complete Dawkins’ sentence? There are two possible answers.

The first answer is that of Darwin, Wallace, Mayr, Dobzhansky, D.S. Wilson, Gould, and Sober, and was generalised and given canonical form by Lewontin (1970). Sterelny and Kitcher (1988) describe this as a ‘pluralist’ description of evolution since it makes no *a priori* claim about what those individuals are; they may be genes, organisms, cells within an multi-cellular body, groups of individual, species, or even multi-specied ecosystems (Wilson, 1976)(Goodnight, 1990). All of these units can be selected according to the traits they inherit, and so can be candidates to complete Dawkins’ sentence.

The second answer is that of Fisher, Haldane, Wright, Williams, E.O. Wilson, Hamilton, and Dawkins, who insist that the bottom line of evolution is how much it changes gene frequencies and, through these, how much it changes phenotypes. These phenotypic effects can be expressed at many different levels, and in many different ways; but it is only the effects on gene frequencies that matter. Therefore genes are the ultimate beneficiaries of adaptations.

The debate between pluralism and gene-centrism in evolution is a paradigm case of the more

general debate over levels of organisation in nature. The pluralist explains evolution in terms of selection operating at many levels independently, whilst the gene-centrist reduces evolution at higher levels to the differential replication of genes. In the next two chapters I apply the general arguments of chapters 2 and 3 to this debate. My aim is to provide a synthesis of the two traditions and to show that not only are both necessary in order to understand evolution but, strictly speaking, neither makes sense without the other.

This debate is important for two reasons. The first is the implications that it has for our understanding of biological evolution — and this will be the main focus of the next two chapters. But this debate also has implications for the way that we apply Darwin’s theory to *social* evolution. And this will be the subject of chapter 10

### 8.1 Evolution and Mendelian Inheritance

What are the conditions necessary for evolution by natural selection? Are proteins and nucleic acid necessary, or could evolution occur in entities made of, say, silicon polymers? Intuitively there seems to be no reason why carbon is the only possible substrate. Is metabolism necessary, or could evolution occur in entities that are more inert? Again there seems to be no obvious reason why not. Are genes necessary, or could evolution occur in entities in which there were no discrete and universal genetic code? It is here that intuitions start to differ.

According to Lewontin, a change in gene frequencies is not a necessary part of the definition of evolution by natural selection. He argues that evolution occurs whenever we have the differential reproduction of individuals according to inherited and randomly mutated traits, and this definition is silent on the nature of the mechanism underlying this process: “No particular mechanism of inheritance is specified, but only a correlation in fitness between parent and offspring” (1970, p1). Dennett describes evolution, characterised in this way, as an *algorithmic* process, and a defining characteristic of an algorithmic process is that it is ‘substrate neutral’; i.e. it is one in which

the power of the procedure is due to its *logical* structure, not the causal powers of the materials used in the instantiation, just so long as those causal powers permit the prescribed steps to be followed exactly. (1995, p51)

Now there is no *a priori* reason why evolution by natural selection cannot occur in substrates other than proteins and nucleic acid, any more than cognition requires neurons or life requires carbon chains — all these phenomena are ‘multiply realizable’ (Kim, 1992). If we use this weak sense of substrate neutrality then Dennett’s claim is definitely true. However the issue becomes more complex when we try to define exactly what constitutes ‘following the prescribed steps’ of natural selection.

Darwin was able to characterise how the force of natural selection works in a remarkably simple way: if the organisms in a population that possess one characteristic are better able to survive and reproduce than the organisms with the alternative characteristic, and if [this characteristic] is passed on from parent to offspring, then the proportion of individuals with that characteristic will increase. ... Of course, some clarification is required of what it means to say that a trait is “inherited.” But as a first approximation, the conditional claim *if* there is heritable variation in fitness, *then* there will be evolution (if nothing interferes) seems to be remarkably straightforward. (Sober, 1985, p27)

Unfortunately the theory of natural selection became a little less straightforward as soon as we ‘clarify’ what inheritance means.

The first point in ‘clarifying’ inheritance is that natural selection as an account of the origin of species depends on discrete genetic, i.e. *Mendelian*, inheritance. If the inheritance and development of traits is not discrete then the result is not the origin of species, just blending, as Darwin was fully aware. It is to Thomas Huxley’s credit that, even whilst he acted as ‘Darwin’s Bulldog’ in his defence of the fact of evolution, he remained sceptical whether natural selection was its mechanism. This was because the discrete inheritance necessary for the formation of species had not been empirically demonstrated to his satisfaction — despite Darwin spending half a life-time trying to evolve distinct species through artificial selection (Desmond & Moore, 1992, p510-11). It was largely for this reason that natural selection was not generally accepted as the dominant force in evolution until it was unified with Mendelian genetics by Fisher (1930), Haldane (1932) and Wright (1931). But Dennett (1996) points out that Mendelism was a ‘triumph for substrate neutrality’ since neither Mendel, nor the early population geneticists, had any idea what the material realisation of these units of heredity was.

Mendel saved Darwin; but what saved Mendel? Until the discovery of the structure of DNA in 1953 Mendel’s genes were *abstracta*: theoretical entities defined solely in terms of observed patterns of inherited traits. Therefore their status as legitimate scientific entities rested entirely on their empirical usefulness. However the empirical success of Mendelian genetics as an explanation of the distribution of traits depends on finding traits that are discretely encoded in genes whose effects on phenotypes and fitness are relatively robust, and so fit our simple models. Mendel was extremely fortunate in his choice of organism and trait since, in coloration in peas, he happened upon a trait that met these conditions. However most traits are not due to single genes with full penetrance and complete expressivity, and so do not always follow the clear patterns of Mendelism. If Mendel had started by enquiring into, say, the height of his peas then it is unlikely that he would have found the clear patterns that allowed him to formulate his theory, since height depends on many different genes and is very sensitive to environmental changes. Mendelian analysis can not, in general, be used to determine the genotype of an individual organism for such quantitative traits (Griffith & Suzuki, 1993, p827).

Until 1953 Mendelian genetics was not a true theory (in the sense of section 3.3), but an as-yet unfalsified hypothesis. Until 1953 Mendel’s patterns of inheritance were always at risk of being exposed as nothing more than empirically useful descriptions, like Copernicus’ epicycles. But once Mendel’s *abstracta* were transformed into the *illata* of DNA then, even if counter-examples to Mendel’s laws were discovered, his laws of inheritance (and hence also Darwin’s theory of natural selection) would not be threatened. For example, Mendel’s Second law states that, in a diploid organism with haploid gametes, genes at different loci will be transmitted independently; thus if  $A,a$  and  $B,b$  are pairs of alleles at different loci, and if an organism is heterozygous at both loci, then the probabilities that a gamete will receive any of the four possible genetic combinations  $AB, Ab, aB, ab$ , are all equal. But this is not true in general (Kitcher, 1984). For example, alleles on the same chromosome will tend to be transmitted together. Only genes on non-homologous chromosomes assort independently. But because we have some understanding of how Mendel’s genes may be arranged on a chromosome, and of the mechanism underlying meiosis, we do not

dismiss Mendel as a result.

A similar argument applies to the case of the non-inheritance of acquired characteristics — the postulate that marked modern evolutionary theory's break from Lamarck. After 1953 Crick re-christened this postulate as the Central Dogma of molecular biology, which states that information cannot pass from protein to DNA but only the reverse; i.e. changes to the phenotype incurred during development will have no effect on the DNA passed on to subsequent generations through the germ-line. However it is interesting to note that Crick called this the central *dogma* of molecular biology, rather than the central *theorem*: it was a claim about how inheritance worked, not a known fact<sup>1</sup>. It is only over the last 40 years that have we slowly discovered the extent to which the Central Dogma is true. Indeed, there are some isolated cases in which it is *not* strictly true, but this does not threaten the Central Dogma — or Darwin. Because we now understand the biochemistry of how DNA is replicated the Central Dogma can now be regarded as a true theory about how genetic inheritance works, rather than a purely empirical generalisation.

Thus Lewontin's conditions are not quite sufficient to guarantee evolution by natural selection. It is not enough that traits are inherited, but this inheritance must be *via* discrete units that are unaffected by the resulting phenotype: i.e. genes. Lewontin's conditions are couched in terms of patterns of inheritance observable in the traits of organisms but evolution requires that, not only does the substrate produce these patterns, but the substrate produces these patterns *in the right way*. And the 'right way' includes discrete genes that are replicated independently of phenotypic properties. Inheritance cannot be characterised purely at the level of the traits of individuals. This problem manifests itself when we try to determine whether a particular trait is *heritable*.

## 8.2 Nature and Nurture

Inheritance, loosely speaking, is a similarity between parents and children. However this similarity can be caused in two different ways: either because something is 'passed down' from parents to children, or because they share a common environment. Only the first form of inheritance produces natural selection, but all biological traits are the product of developmental processes that involve both. Therefore if we want to determine whether a trait will be amenable to adaptation then we must find some way of determining the contribution of genetic, rather than environmental, inheritance. This contribution is normally quantified as the *heritability* of the trait, defined as the proportion of total variance in the trait that is due to genetic, rather than environmental, variance.

There are two sets of problems in determining heritability. The first are methodological. The standard technique for measuring heritability is to raise a variety of strains in a variety of environments and then calculate their relative effects on the trait. But this technique cannot distinguish differences that are due to genetic factors from those that are due to the maternal environment provided by the uterus or the cytoplasmic environment of the zygote. For example babies born to poor, badly educated, mothers have lower birth-weight and shorter concentration spans than those born to the rich and well educated (Gonzales, 1985). Therefore, even if those children are subsequently adopted and raised in random distribution of environments — or if those children are given a common educational environment — and we subsequently find that there is a strong correlation between, say, the IQ of the birth-mother and that of the children, we still do not know

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<sup>1</sup>Thanks to John Maynard Smith, personal communication, for this point.

if this correlation is due to genetic or maternal-environmental effects (Roubertoux et al., 1990). The environment starts to affect a child well before birth, therefore environmental and genetic effects can only be experimentally separated by transplanting embryos between uteruses, or chromosomes between zygotes. Such experiments demonstrate two things. The first is that uterine and chromosomal environments do indeed have effects that would ordinarily be classed as genetic in those organisms, such as humans, where such techniques are not possible (Nosten, 1989)(Nosten & Roubertoux, 1988)(Carlier & Roubertoux, 1984) (Carlier et al., 1991). The second is that, in an operational sense, we cannot determine the heritability of a trait from the observed properties of the whole organism, but must also be able to isolate the genetic material and manipulate it directly. If there were no discrete genetic material then it would be impossible to conclusively determine the heritability of a trait using this method, and hence whether selection for it will produce adaptation.

The alternative method of determining heritability is to measure the response to selection directly: if the distribution of a trait in a population responds to selection, then that trait must be heritable<sup>2</sup>. But although this is the method typically used in artificial selection experiments (Orgel, 1979), it cannot be used as a way of defining sufficient conditions for evolution since if we claim that evolution occurs when there is the differential reproduction of heritable traits, but define a trait as heritable when it evolves in response to selection, then we are left with a tautology. We need a method for determining whether a trait will respond to selection that does not reduce to observing that, in fact, it does.

The problems of separating the contributions of environmental and genetic factors are compounded when investigating human behavioural traits, since ethical considerations forbid us from manipulating environments and/or genetic material directly. For example Plomin *et al* (1977)(1985) discuss how an individual may actively or passively construct their own environment according to some genetic predisposition. Teachers, for example, may give a particularly bright child a more enriched educational environment, whilst the peers and carers of a sociable child may well reciprocate their affection. The only way to separate the effects of environmental and genetic factors in these situations would be to give a sub-population of children purposely impoverished environments, and this is not ethically possible: we would have to deliberately ignore their demands for stimulation or affection. The same confusion occurs when genetic and environmental factors are correlated for other reasons. For example in many adoption studies there is considerable selective placement, in which children are placed in homes similar to the parental environment (Loehlin, 1979). This again has the effect of confusing environmental and genetic effects.

These methodological problems in determining heritability reflect an underlying conceptual problem. The purpose of the concept of heritability is to measure the relative contributions of genetic and environmental factors to a trait. However all traits are *completely* dependent on both environment and genes. Both are 100% necessary, so does it make any sense to compare their relative contributions (Anastasi, 1958)? But although this truism applies to individuals it does not apply to differences *between* individuals in a population: if we consider a population of genetically identical individuals then the differences between them must be due to environmental factors; and if individuals are raised in the same environment then differences must be due to genetic factors. But in order to quantify heritability it is necessary to compare the contributions of each factor

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<sup>2</sup>Though in section 8.4 I discuss examples of traits that *are* heritable but still do not respond to selection.

when the two effects are combined. This is done using analysis of variance (ANOVA) techniques:

Suppose we wanted to measure the heritability of the yield of wheat by raising two different strains under two fertilisation regimes<sup>3</sup>. The analysis of variance represents the causal role of these two factors by showing how the average yield of plants on a plot departs from (1) the average yield of all plants, (2) from the average yield of all plants that received as much fertiliser as those in the plot in question did, and (3) from the average yield of plants of the same strain. Suppose that the yield followed this pattern:

|                  | Strain S1 | Strain S2 | Marginal Average |
|------------------|-----------|-----------|------------------|
| Fertilisation F1 | 1         | 3         | 2                |
| Fertilisation F2 | 3         | 5         | 4                |
| Marginal Average | 2         | 4         | <b>3</b>         |

In this case differences in strain and environment have equal effects on yield, and so its heritability will be 50%. In another case the respective yields may be

|                  | Strain S1 | Strain S2 | Marginal Average |
|------------------|-----------|-----------|------------------|
| Fertilisation F1 | 1         | 5         | 3                |
| Fertilisation F2 | 3         | 7         | 5                |
| Marginal Average | 2         | 6         | <b>4</b>         |

in which case genetic factors have a greater effect, and so the heritability of the trait for this population in this range of environments will be greater than 50%. Moreover, in both of these cases, the combined effects of the two factors is a linear sum of the separate contributions. In the second case strain *S2* produces, on average, a yield 2 units greater than the overall mean; *F2* promotes yield by 1 unit; and the combination of *F2* and *S2* yields 3 more units than mean. However, suppose the yield is

|                  | Strain S1 | Strain S2 | Marginal Average |
|------------------|-----------|-----------|------------------|
| Fertilisation F1 | 1         | 3         | 2                |
| Fertilisation F2 | 5         | 9         | 7                |
| Marginal Average | 3         | 6         | <b>4.5</b>       |

Here the marginal yield of *F2* is 2.5, that of *S2* is 1.5, but the combined effect of *F2* and *S2* is 4.5. The extra half a unit of yield is not due to environmental effects, nor to genetic factors, but to the interaction of them both. In other cases there may be no 'main' effects at all, with all variance due to interaction. For example, one environment may increase the yield of one strain but decrease the yield of another:

|                  | Strain S1 | Strain S2 | Marginal Average |
|------------------|-----------|-----------|------------------|
| Fertilisation F1 | 1         | 5         | 3                |
| Fertilisation F2 | 5         | 1         | 3                |
| Marginal Average | 3         | 3         | <b>3</b>         |

In this case particular fertilisers or strains have no net effects at all. This is not a purely artificial

<sup>3</sup>This example has the same structure as human adopted twin experiments. For 'strain' read 'monozygotic twins', and for 'fertilisation regime' read 'home environment'.

example. For example Levins and Lewontin (1985) discuss two mutations in *Drosophila*, known as *Ultrabar* and *Infrabar*, which affect the number of facets in the adult's eye. *Infrabar* produces less facets than *Ultrabar* if the fly develops in an environment at 15°C, but more at 30°C. The marginal mean effects of mutations and environmental factors are very small — since they will be positive in one case and negative in another — but the combined effect is considerable. In such cases heritability gives us no useful guide as to the response of the trait to selection.

ANOVA techniques account for interaction by regarding the value of a trait as a sum of the contribution due to genetic factors, that due to environmental factors, and a third term that describes the effects of their interaction. But heritability only measures the relationship between the first two parts. This much is uncontroversial, the issue is whether it matters. Plomin argues that it does not, for two reasons.

The first reason is that Plomin claims that, as a matter of fact, the interaction effects in, for example, human behavioural traits tend to be quite small (1988, ch9). However Wahlsten (1990) points out that this is more likely to be due to the fact that ANOVA is poor at detecting interaction effects in small population sizes, rather than their absence — a problem that is more likely to occur in human adoption studies than in research on laboratory animals:

Interaction has been evaluated in studies of human IQ and usually none is seen (Plomin et al., 1977) (Plomin & DeFries, 1983) (Plomin, 1986). Generalisations have then been made that heredity and environment are truly additive, and sophisticated path models have been developed to partition variance and covariance under the assumption that interaction is negligible (Heath, 1985) (Henderson, 1982) (Phillips, Fulker, & Rose, 1987) (Plomin et al., 1985). On the other hand, an immense collection of well-controlled laboratory studies of animals has provided abundant evidence of significant and illuminating interactions between heredity and environment. At the 1987 Behavioural Genetics Association meeting in Minneapolis, the concurrent sessions on human and animal studies were like two separate worlds in terms of attitudes towards interaction. Many human geneticists dismissed interaction and cited heritability estimates with great confidence, while most of those studying mice, rats, and fruit flies documented once case of interaction after another and expressed skepticism about heritability coefficients. (Wahlsten, 1990, p112)

If the assumption of zero interaction is made and ANOVA techniques used uncritically then serious errors can occur. For example Schönemann (1997) notes that adoption experiments typically give heritabilities for human IQ of around 60–80%, which is higher than that of traits that can be more precisely measured in larger, and better controlled populations, such as body fat in pigs, or length of wool in sheep (30–50%); whilst the same measures used on such spurious human traits as wearing sunglasses after dark can give heritabilities of over 100%. Indeed, according to the same data, religious preference is more heritable than race!

However Plomin also argues that ANOVA-type heritability coefficients are meaningful even if interaction effects are present since heritability only measures the relationship between the main (additive) effects. 'Main effects and interactions are independent — mean effects of  $G$  and  $E$  are not invalidated by the presence of  $G \times E$  interactions' (1990). However the existence of additive main effects is an *assumption* of the ANOVA technique, and their statistical significance in accounting for the distribution of the trait is no guarantee that they accurately reflect the underlying relationship between nature and nurture. For example, suppose that a trait were the arithmetic

product of  $G$  and  $E$  (i.e.  $Y = G.E$ ). An ANOVA on this data may still find some additive component, but this would solely be an artefact of the method. Wahlsten (1990) (following Nabi) shows the problems that would result if the same method were used to analyse, for example, gravity. We know that the *actual* law is

$$F = \frac{Gm_1m_2}{d^2} \quad (8.1)$$

But suppose that we analysed the effects of mass ( $m_2$ ) and distance ( $d$ ) on the force ( $F$ ) measured on an object by a fixed mass ( $m_1$ ).

The experimenter's conclusions from the ANOVA would be that both mass and distance are important for the force, although the internal factor (mass) is rather more important and accounts for more variance than the external factor (distance), and mass and distance are additive because the interaction term is not even close to significance. He might even proclaim a simplified law of gravitation,  $F = \mu + m + d$ . ...

It makes no sense to say that a person's weight depends more on body size than planet of residence, but this is precisely what ANOVA techniques try to measure. And there is no *a priori* reason why the interaction between genes and environment in the development of an organism will be simpler than that between mass and distance in producing gravity — indeed most of the evidence from developmental genetics points to more complexity, not less (Pritchard, 1986). Therefore it is similarly meaningless to say that a trait depends more on nature than on nurture. The analysis of variance can often succeed in hammering complex interactions into additive holes, but we should not confuse the result of this procedure with the original shape. Heritability coefficients give us a distorted picture of the relationship between genes and environment in the process of inheritance, but what is the alternative?

### 8.3 Inheritance and Mechanism

What do we mean by an 'inherited' trait, i.e. one that is amenable to adaptation by selection? Consider a lineage of black ravens. Their blackness may be due to genetic factors, environmental factors (such as a peculiarity in the local diet) or a combination of both. We can observe that the trait has, in fact, been inherited in the past but the question is whether it is *inheritable*, i.e. whether this trend will continue into the future. Inheritability is thus a *dispositional* property, like solubility; and just as the solubility cannot be equated with the fact of dissolving in water, so inheritability cannot be equated with the fact of having similar offspring. We need to identify some property of the offspring of the lineage that gives us some reason to believe that it will share the trait of its ancestors. We need some *justification* for an induction from past to future ravens. In section 3.5 I argued that such inductions are *not* justified simply by noting instances of black ravens, but only by explaining the observed connection between ravenhood and blackness through an understanding of the developmental processes connecting the wild-type genome of *Corvus corax* to feather pigment production. This provides good grounds for believing that all organisms that carry those genes would be black.

Inheritability is an implicitly causal property: it is not just a correlation between traits of parents and children, but rather implies that something is passed on from parents to children that

*causes* that trait to re-occur. If we uncover such a process then a trait will still be properly classified as inheritable even if that correlation may be disrupted by an environmental factor. Ariew (1997), for example, cites the example of opposable thumbs, which seem to be a paradigm case of an inherited adapted trait. However almost all modern humans who lack thumbs are the victims of thalidomide, and other environmental factors; therefore the variation of the trait in the population that is due to genetic effects, and hence the heritability of the trait, is very low. The reason why we are confident that thumbs are amenable to adaption is not due to heritability measures, but because we have a good idea of how they develop. In particular we know that pentadactyl limbs are the result of highly canalised processes that are (normally) very robust against environmental disruption, and so will re-appear in the lineage as soon as the environmental factor is removed. This understanding then allows us to disregard cases in which the trait fails to be passed on: thumbs are inheritable even though they have low heritability.

Of course, methodologically speaking, it is very difficult to understand the complex developmental processes that link genes to phenotypic traits; and statistical heritability experiments are a good way of identifying particular mutations worthy of further investigation. However we should not confuse the method of investigation with the object of investigation. Fisher himself — the inventor of ANOVA — warned against ‘the so-called coefficient of heritability, which I regard as one of those unfortunate short-cuts which have emerged in biometry for lack of a more thorough analysis of the data (1951, p217). And Bateson concludes that ‘analyses of statistical interaction should be the starting point of attempts to understand how developmental processes work and should not be treated as ends in themselves (1987, p2). If the environment plays no role in the development of a trait, or if its contribution is strictly separable from that of genetic factors (i.e. the variances are additive), then we can use heritability to characterise inheritance. However this is the exception rather than the rule and, in general, determining inheritability involves isolating the genetic and developmental mechanisms underlying that inheritance.

The mechanism of this genetic inheritance need not be nuclear DNA. For example Sonneborn has shown that if the patterns of cilia on the surface of *Paramecia* are surgically altered then the same pattern will be inherited by descendants (1963); moreover this transmission is independent of changes in the nucleus (1970). However the inheritance in this case is still ‘genetic’ in the sense that it is mediated by a heritable unit analysable using the same formal logic and procedures developed for DNA-based heritable systems, even though the unit in this case is composed of microtubular protein assemblies and not DNA (Frankel, 1983)(Whittle, 1983).

The same point generalises to other cases of property inheritance. For example, suppose two rich families bestow their privileges on their offspring. The first give their children a large sum of money as soon as they reach majority, but then let them fend for themselves. The second ensure that their children go to the best schools and universities, inherit titles, and are introduced to all the right social networks, but the children are never given any money directly. Both will produce long dynasties of wealth and social power. However in the first case it is the wealth that is inherited, with privilege being the likely result given a particular social environment. In the second case it is the social position that is inherited, and wealth is the result that accrues. Inherent in the concept of inheritance is the idea that something is preserved through copying, even though its effects may be dependent on the environment. The two sets of children inherited different properties from their

parents, even though the results were the same. When determining which properties are inherited it is the mechanism of the copying process that matters, not the observed result.

In the case of natural selection, correlations between parents and offspring can be achieved in one of two ways. The first is through the mechanism of a shared environment. But in order for correlation to count as inheritance there must also be some element of this causal link that is in a strong sense independent of the developmental environment, i.e. there must be an entity possessed by both parents and children as a result of which the trait may, or may not, develop, depending on the environment. Moreover, if we are to use inheritance to *explain* an observed correlation between parents and children, and hence to explain the response of the trait to selection, then that entity must have an identity independently of its role in the development of that trait.

(Of course Mendel realised all this 100 years ago when he developed the notion of the recessive gene — i.e. a gene that is possessed by an organism, and may be passed on to its offspring, but which does not have phenotypic effects. The concept of the recessive gene only makes sense if its possession conditions are independent of its expression conditions, i.e. if we can determine whether an organism possesses a gene independently of whether any observable traits are affected. However the question here is not whether natural selection is, in fact, mediated by a genetic mechanism but whether such a mechanism is a necessary condition for natural selection to proceed.)

#### 8.4 Evolution and Development

A breed of cow that reliably gave birth to a high proportion of heifers compared to bullocks would be very profitable to farmers, but no amount of artificial selection has managed to produce one. Why not? The reason is that one more condition must be met if a trait is to respond to selection. It is not sufficient that there is a discrete genetic mechanism underlying a correlation between parents and offspring (i.e. that the trait is inheritable). It is also necessary that the *effect* of that genetic factor in the development of a trait must, to some extent, be independent of others. This is an aspect of development that Needham originally identified as ‘dissociability’:

In the development of an animal embryo, proceeding normally under optimum conditions, the fundamental processes are seen as constituting a perfectly integrated whole. They fit in with each other in such a way that the final product comes into being by means of a precise co-operation of reactions and events. But it seems to be a very important, if perhaps insufficiently appreciated, fact, that these fundamental processes are not separable only in thought; that on the contrary they can be dissociated experimentally or thrown out of gear with one another. This conception of out-of-gearishness still lacks a satisfactory name, but in the absence of better words, dissociability or disengagement will be used in what follows. It is already clear that embryonic growth can be stopped without abolishing embryonic respiration, and conversely, it is probable that growth or differentiation, under certain conditions, may proceed in the absence of the normal respiratory processes. There are many instances, again, where growth and differentiation are separable. It is as if either of these processes can be thrown out of gear at will, so that, although the mechanisms are still intact, one or other of them is acting as “layshaft” or, in engineering terms, is idling. (1933, p180-1)

If a trait is not dissociable from others then any mutation that is developmentally viable will, *ipso facto*, carry that trait. Therefore there will be no variation in that trait in the population. Even

if that trait is inheritable then it will still be impervious to adaptation by natural selection. Thus we find that no amount of selection succeeds in changing the symmetry of ocelli in *Drosophila* (Maynard Smith & Sondhi, 1960) or the sex ratios in diploid organisms (such as cattle) (Maynard Smith, 1978). Gould (1977, p234) has also used the effects of such developmental constraints to explain persistent stasis in evolution in which long-term selection fails to produce any appreciable change.

Developmental constraints can affect evolution in many ways, either by preventing any response to selection at all, or by preferring selection in one particular direction (Maynard Smith, 1985). The response of a population to selection is often likened to a ball rolling across a fitness landscape towards the valleys of optimal adaptation. However the effect of developmental mechanisms is to transform the ball into a complex polyhedron that will not necessarily roll straight down a slope but may prefer one direction to another, or even come to rest on one face.

Wagner and Altenberg (1996) discuss how such developmental constraints may be understood at the level of the genotype. They argue that a pre-condition for dissociability is the absence of ‘universal’ pleiotropy (Wright, 1968), in which every gene has an effect on every trait. If an organism is to respond to selection then its genotype must be divided into discrete ‘gene nets’ with partially independent developmental effects. If this is the case then ‘genetic change can occur in one of these gene nets without influencing the others, thereby much increasing its chance of being viable. The grouping leads to a limiting of pleiotropy and provides a way in which complex developing organisms can change in evolution’ (Bonner, 1988, p175).

If there is no genetic modularity then no amount of mutation and selection will produce reliable adaptation because selective pressures will not be able to ‘see’ — i.e. have an impact on the likelihood of replication of — individual genes. This problem can be pictured in terms of the topography of fitness landscapes. Pleiotropy increases the ruggedness of fitness landscapes since movement along every dimension in genotype space — i.e. mutation at every locus — will have an effect on the trait under selection, and hence the fitness of the phenotype. In such rugged landscapes selection will often result in populations becoming stuck on local maxima, and this is a significant practical problem in using artificial evolution as a multivariate optimisation technique (Goldberg, 1989). But if much of the genome is neutral with respect to the trait under selection then the population is less likely to be trapped and selection will be more likely to produce adaptation (Barnett, 1998) (Huynen, Stadler, & Fontana, 1996) (Kimura, 1983). Wagner and Altenberg empirically investigated the effects on evolution of genotype-phenotype maps with varying amounts on pleiotropy. They found that where development was insufficiently modular then ‘the Darwinian process of mutation, recombination and selection [was] not universally effective in improving complex systems’ (1996, p967).

Sometimes selection will not succeed in producing adaptation; and this failure can only be explained by understanding the developmental processes that produce a complete organism from an inherited genome. Dobzhansky argued that nothing in biology can be understood except in the light of evolution. But it is equally true that evolution can not be understood except in the light of developmental biology.

Genomes are not always modular, and development is not always dissociable. Pleiotropy may not be universal, but is still widespread. This fact underlies one part of Lewontin and Gould’s infamous critique of adaptationism in which they questioned the practice of

breaking an organism into unitary “traits” and proposing an adaptive story for each considered separately. Trade-offs among competing selective demands exert the only brake upon perfection; nonoptimality is thereby rendered as a result of adaptation as well. We criticise this approach and attempt to reassert a competing notion (long popular in continental Europe) that organisms must be analysed as integrated wholes, with *Bauplän* now so constrained by phyletic heritage, pathways of development, and general architecture that the constraints themselves become more interesting and more important in delimiting pathways of change than the selective force that may mediate change when it occurs. (1978, p581)

If the conditions for adaptation by natural selection are defined purely at the level of individuals, as Lewontin does, then the failure of selection to produce adaptation counts as a ‘hit’ against adaptationism. But if conditions on the mechanism of inheritance and development are included in that definition then we can account for those ‘failures’ — just as Einstein could account for the failure of Mercury to follow an elliptical orbit, and Crick and Watson could account for departures from Mendel’s second law. In this way Lewontin and Gould’s negative critique of adaptationism can be turned into a positive proposal for defining the conditions in which adaptation by natural selection occurs.

Selection for a trait will only produce adaptation to the extent that that trait is dissociable and modular; but just because a trait is dissociable does not imply that its development is independent of the rest of the organism. For example one of the most remarkable demonstrations of genetic modularity and developmental dissociability has been Halder *et al*’s (1995) success in getting complete physiological eyes to sprout on the wings, legs, and antennae of otherwise normal *Drosophila* by targeted mis-expression of the ‘eyeless’ gene. But even though the effects of ‘eyeless’ are highly modular, this does not imply that the same gene would produce a compound eye if were transcribed into, say, a mammal.

Lewontin and Gould challenged what they saw as the reductionist atomism of the adaptationist view of traits, and instead emphasised how organisms develop as whole integrated units. This is a healthy warning, but dissociability need not imply atomism. Modularity does not imply that particular genes, or gene nets, act alone, but only that changes within a gene net have limited effects on the development of other traits. Dissociability does not imply that organisms are composed of prior parts like mythological chimeras, but that those modular parts develop in, and are dependent on, the context of the whole organism. Dissociable traits are thus emergent products of whole organisms.

## 8.5 Conclusion

A trait will respond to natural selection only if it is inherited *via* a modular and discrete genetic mechanism, and develop dissociably. If these conditions are not met then no amount of selection will produce adaptation. Therefore a change in gene frequencies is a *necessary* condition for natural selection. But in the next chapter I will argue that a change in gene frequencies is not, in itself, a *sufficient* condition.