



# 2

## Fundamental Concepts in Behavioural Ecology

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### 2.1 Introduction: what is 'behavioural ecology'?

Throughout history, the study of living creatures in their natural environment has been a source of fascination for people. From Aristotle to Darwin, the diversity, complexity, and sheer exuberance of animal behaviour has never ceased to astonish and to defy complete understanding. Indeed, the reasoning and logic behind certain behaviours seem at first to elude us. For instance, how does one rationally explain the sexual cannibalism of the praying mantis, or the sterility of the working caste within ant societies, or even the parasitic reproductive strategy of the common cuckoo (*Cuculus canorus*)? Individuals of the last species do not construct a nest, but rather lay their eggs in the nests of other bird species, often of smaller size. The newly hatched cuckoo rapidly excludes the eggs and young of its 'adoptive parents', monopolizing their attention and care. We cannot help but wonder what drives individuals of the parasitized species to accept the heavy cost of the young cuckoo rather than seek to raise their own offspring properly?

Certain behaviours are so remarkable that they appear *unnatural* to some observers. The infanticide in lions is one such behaviour. In this social species, groups of up to a dozen individuals are composed of adult females, their cubs, their immature daughters,

and one to six males. The females within a group are closely related, having usually grown up within a pride that has existed for multiple generations. Females typically remain within the same group for a long period of time. However, this is not so for male lions. They are excluded from their natal group as soon as they are independent of their mother, and subsequently form coalitions with their brothers or with unrelated young males. If a coalition recruits a sufficient number of young males, they can eventually take over a pride of females after forcefully evicting the adult males of the group. These power reversals lead to many spontaneous abortions in females, undoubtedly caused by the stress involved with such situations. Females for which pregnancy is too advanced for abortion will give birth, but the new dominant males quickly kill the cubs, particularly the males. Because it reduces the reproductive output of females, infanticide may at first appear to be an unusual behaviour.

Behaviours such as these seem paradoxical unless considered within their evolutionary framework which, as we shall see, can reveal the logic behind them. The interpretation of behaviour within this framework characterizes the approach of behavioural ecology.

### 2.1.1 An evolutionary approach to behaviour

Behavioural ecology seeks to explore the relationships between behaviour, ecology, and evolution. Here, behaviour is considered as the collection of decisive processes by which individuals adjust their state and situation according to variations in their environment, both abiotic and biotic.

**Decision** making does not necessarily refer to elaborate cognitive processes but simply to the fact that an animal is regularly confronted with multiple alternatives, each differing in their consequences in terms of individual survival and reproduction. Other more restrictive or mechanistic definitions of behaviour exist (see, for example, Manning 1979). The advantage of the above functional definition is that it emphasizes the crucial role of behaviour in adaptation and therefore in evolution.

The goal of behavioural ecology is to understand how behaviour results **from a combination** of the evolutionary history of a species, recent or current events occurring at the population level, and characteristics particular to individuals and to the conditions in which they developed. It also involves using our knowledge of biological evolution to construct an analytical framework of behaviour and to identify the various factors, both internal and external, that induce or constrain the expression of behaviour. Behavioural ecology belongs to **neo-Darwinism**, an evolutionary movement based on a modification of Darwin's ideas in light of discoveries on the nature of heredity. The field of **sociobiology** also uses this evolutionary approach to behaviour. However, the differences between sociobiology and behavioural ecology are subtle. They both use the same **hypothetico-deductive approach**, and in fact differ only in their focus of study. Sociobiologists are primarily concerned with the interactions between individuals within animal groups or societies, whereas behavioural ecologists are interested in all aspects of behaviour. Sociobiology is thus a subset of

behavioural ecology (Krebs and Davies 1981; Krebs 1985), and accordingly there is no strict advantage to regarding it as a separate field.

The questions typically asked in behavioural ecology are numerous and diverse. Why does a given predator focus its energy on a particular type of prey while another shows much greater diversity? Why do males provide parental care in one species and not in another that is closely related? Why do certain individuals within an animal society have the ability to reproduce whereas others are limited to sterile auxiliary roles? Why does the song of a certain passerine species differ from one individual to the next? The questions we ask can be even more specific, and expressed in quantitative terms. Why does the European starling (*Sturnus vulgaris*) bring its young a maximum of six food items per visit to the nest? Why does mating last on average 7 hours 40 minutes in a particular species of mite?

To answer these questions, behavioural ecologists often favour a utilitarian approach to behaviour. The underlying question, whether explicit or implicit, is then the value of a behavioural trait for the survival and/or reproduction of individuals, and for the replication of their **genotype** above others, as per the process of **natural selection** (Grafen 1984; Dawkins 1989). The number of food items brought back to the nest by a starling is then understood as a balance between the maximization of food brought to the nest and the costs in energy and travel time involving the distance between the nest and the food source. The presence of sterile individuals is interpreted with respect to the help they bring to the reproductive individuals to which they are closely related. However, if evolution by natural selection is indeed an optimizing process (Endler 1986; Dawkins 1986), it follows that the possibilities are never infinite (Jacob 1981). Throughout evolutionary time, various random events have demarcated areas within which species can evolve towards new forms and functions. With respect to this, the goal of behavioural ecology is also to establish the extent to which an observed behavioural trait results from the historical limitations that have guided its evolution. Behavioural ecologists must generally keep themselves from

hastily drawing the conclusion that the behaviour they study is the **direct** result of a selective process. This situation is illustrated in the following example.

### 2.1.2 Size homogamy in gammarids

Gammarids (genus *Gammarus*) are aquatic amphipod crustaceans distributed across the globe. An important characteristic of their reproductive behaviour is precopulatory **mate guarding**, or **amplexus**, in which males compete for the possession of females by guarding them before copulation. As in all crustaceans, amphipods regularly undergo moult, and it is only at that moment that females can mate. The time lapse between two consecutive moults can spread over several weeks depending on water temperature, an interval during which females cannot be inseminated. By contrast, males are always able to reproduce, apart from the time they are themselves moulting. Although there are equal proportions of males and females within natural populations, the **operational sex ratio** (i.e. the relative proportions of males and females available for mating at any moment) is strongly male-biased, resulting in strong competition among males for access to the few moulting females capable of being inseminated. Male gammarids have the ability to detect the hormonal status of females, and when a male encounters a female nearing her moult, he may choose to attach himself to her back, holding on to her with his gnathopods, two pairs of hypertrophied appendages, thus forming an amplexus. This nuptial ride persists until the female moults, after which the male that was guarding her fertilizes her eggs.

When collecting gammarids in amplexus, it is generally evident that individuals do not pair randomly. There exists a positive relationship between the size of the male and female within a pair (Figure 2.1). Larger males tend to attach themselves to larger females, while smaller males typically hold on to smaller females. This size-based association between males and females is referred to as size **homogamy**.

How do we interpret such a phenomenon? First, a selective, utilitarian interpretation. We know that

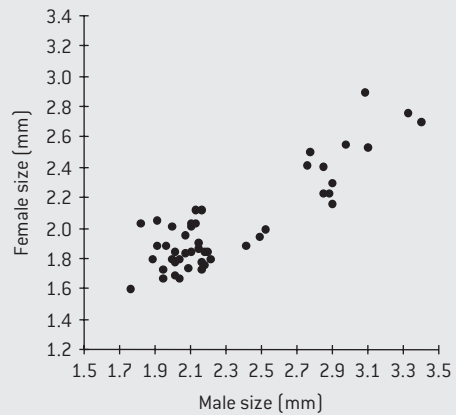


Figure 2.1 **Size homogamy (body length at the fourth coxal plate) in gammarids, *Gammarus pulex*, in amplexus**

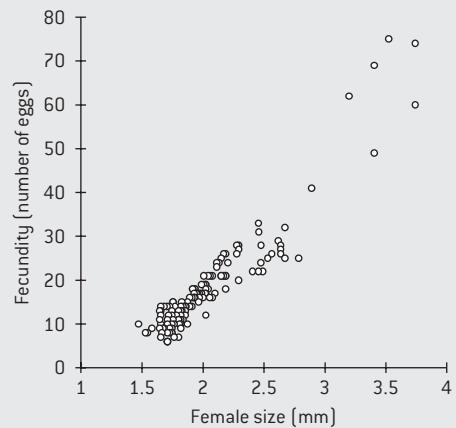


Figure 2.2 **Fecundity (number of eggs) according to size in female gammarids, *Gammarus pulex***

the number of eggs a female can lay increases exponentially with her size (Figure 2.2). For a male, therefore, a small size difference between two females can mean a large difference in the number of eggs fertilized. On an evolutionary scale, males that choose to pair with larger females will have a greater number of descendants. Moreover, larger males can also dominate smaller ones and can thus dislodge them from females onto which they have amplexed.

If this is so, then size homogamy can result from larger males controlling access to the most coveted, larger females, ensuring themselves a greater reproductive output. Smaller males would then be limited by their competition with larger males and could only attach themselves to smaller females (those left available by large males). According to this interpretation, it is the size-linked variation in female fecundity (which on an evolutionary scale favours males that pair themselves with large females) that creates a competition between males of different sizes, thus producing the observed homogamy.

This utilitarian explanation is consistent, and integrates many aspects of the behaviour and physiology of the species. However, we should not conclude that this interpretation is correct without considering some alternatives. Various alternative explanations, seemingly just as consistent, can be proposed. To begin with, it is necessary to confirm that individuals of different sizes do indeed occupy the same habitats and so can encounter each other at the same frequencies. For example, the presence of gammarid predators could lead to non-uniform size distribution of individuals as a function of the particle size of the substrate: large individuals of both sexes would benefit from occupying habitats with larger particles, as this would allow them to hide within the gaps and crevices in the substrate and escape predators. This heterogeneous size distribution could therefore create the observed size homogamy through the effects of unequal probabilities of encounter.

Another alternative explanation might be that the energetic cost of amplexus for males is proportional to the size of the female they carry. Only larger males could bear the energetic costs of pairing up with larger females. Smaller males would therefore have no interest in pairing up with larger females because they are literally too expensive for them. A final possibility is that morphological limitations prevent large males from attaching themselves effectively to small females, and conversely prevent small males from attaching to large females.

It is important to note that these various alternatives hypotheses are not mutually exclusive and it is possible that they have a cumulative effect to

produce the observed pattern. Before assuming the primary importance of competition between males for large females, we must consider the significance of all the above explanations (and any others that we might imagine; Bollache *et al.* 2000). A valuable approach to deciding which interpretation carries the most weight is to search for situations in which the many alternatives make very different predictions, which would allow us to differentiate between them. This simple example illustrates the precautionary measures that must always be taken when interpreting behavioural phenomena: **it is essential to rule out alternative explanations before raising our confidence that a given interpretation is true.** This is a fundamental principle behind any scientific process.

## 2.2 Behavioural ecology: an evolutionary perspective

### 2.2.1 What is evolution?

Behavioural ecology provides an evolutionary perspective on behaviour. It is appropriate then to define exactly and explicitly what we mean by evolution. The word evolution invokes, in its literal sense, a series of progressive transformations. In its common usage, the word has at least four different meanings: (1) a fact, (2) a transformation, (3) a particular course, and (4) an improvement. Although the first three meanings are valid, the fourth is not to be used in the context of evolutionary biology. Accordingly, the word evolution refers to the now well-established fact (Box 2.1) that species have progressively transformed over time. Taken in this sense, the word evolution incorporates the first three meanings seen in current use. It represents a history, that of life on earth.

Today, evolution is no longer just a theory, but a fact (see Box 2.1). Apart from some creationist religious movements, very few people challenge the reality of evolution. When we speak of the 'theory of evolution', we use 'theory' in its scientific sense to mean an organized body of knowledge and not its everyday

### Box 2.1 Empirical evidence for evolution

Since the time of Jean-Baptiste de Lamarck (1809), the empirical evidence for evolution is varied and stems from diverse domains of biology. The following is a summary of certain important arguments, each independently consistent with the evolution of species.

(1) Fossils show that ancient life forms are radically different from contemporary species. It can even be difficult at times to find present-day representatives for many fossil groups that lived tens or hundreds of millions of years ago. This is a result of two key processes: extinction and radiation. Most organisms that existed in the past are extinct today, whereas there also exists at present a great number of different forms, some of which did not exist in the distant past.

(2) There is a strong resemblance between the early stages of embryonic development within different members of a taxonomic group. For instance, vertebrate embryos pass through initial stages that resemble the adult forms of ancestral groups. In this manner, the embryos of mammals (considered derived forms) possess vestigial gill arches during early developmental stages, similar to those of fish (considered ancestral forms). In these ancestral species, development is complete at stages that for mammals correspond to the very beginnings of development. This is evidence that mammals arose from fish, because their development retains the traces of their ancestral history; evolutionary processes can only build on what already exists.

(3) The field of comparative anatomy has revealed remarkable similarities between the anatomical structures of related groups. This notable likeness can be seen when comparing the bones of our arms with those of a bird's wing, the forelimbs of a horse, a lizard or a frog,

or the anterior fins of certain fish. The similarities between these homologous organs strongly suggest that they are related through common descent, and that they gradually transformed over evolutionary time.

(4) Functionally complex organs are no longer used in some groups, indicating their abandonment over the course of evolution. For example, in bed bugs (heteropterans), females have well-developed copulatory organs, yet fertilization occurs directly across the abdominal wall (Stutt and Siva-Jothy 2001). Moreover, whales and snakes possess a vestigial pelvic girdle as well as rudimentary hind limbs. Such vestigial structures have no apparent function, and are just evolutionary leftovers inherited from their quadruped ancestors. How else can we explain their existence if not by evolution?

(5) The discipline of molecular genetics has shown that there lies an intriguing unity behind the enormous diversity of life. This dualism between external diversity and structural and functional unity is found at various levels. The same processes regulate the metabolism of creatures as diverse as snails, frogs and bacteria. The same genetic code allows the transmission of information from generation to generation in plants as well as in animals. The incredible diversity in form and colour seen in birds rests upon the same internal organization for all members of this group. The explanations behind this unity become obvious when we realize that these forms are all derived from more or less ancient common ancestors.

(6) For thousands of years, man has practised artificial selection on domesticated species. By the simple process of artificial selection for more suitable forms, man has, for example, created incredibly diverse dog breeds. This

demonstrates the enormous potential for the diversification of life from a common root. This type of evidence has been demonstrated in the laboratory an uncountable number of times.

(7) Biogeography has shown that portions of the earth's crust that have long been isolated tend to have life forms that resemble ancient fossilized forms found on neighbouring continents. For instance, Australia is home to mam-

mal groups that are closely phylogenetically related to mammals that existed in Eurasia at the beginning of mammal radiation, about 100 million years ago. Equally, New Caledonia is today the only place where one can find gymnosperm groups that have been extinct in the rest of the world for a long time.

All those phenomena, and many more, offer factual evidence for the reality of evolution.

meaning of an uncertain hypothesis. Evolution as such comprises several co-existing theories that give various degrees of significance to the different processes affecting biological evolution. The recognition of evolution as a fact then creates a framework in which the assorted theories explaining it can be placed. Scientific progress regularly allows for the subtle modification of evolutionists' viewpoints about the prevalence of each of the known evolutionary processes. A thorough understanding of biological evolution can only be attained by integrating the findings of such diverse fields as palaeontology, ecology, taxonomy, behavioural ecology, population dynamics, evolutionary physiology, population and quantitative genetics, and even developmental biology.

### 2.2.2 The logic of evolution: information, replicators, and vehicles

#### *Evolution*

In the absence of any knowledge in the field of genetics, the study of evolution was for a long time based on the study of morphological changes in organisms over time, principally by comparing fossilized forms among themselves and with extant forms.

Today, our concept of evolution integrates observed transformations with changes that occur over time in the frequency of different genes or **replicators**, which have

the ability to self-replicate and hence ensure their existence over time. In other words, evolution is the process by which the frequencies of genes change over time.

#### *Genotype and phenotype*

Before proceeding, it is essential to define two fundamental concepts: genotype and **phenotype**.

The **genome** corresponds to all of the genetic information carried by an individual (Mader 2004), although the same word is sometimes used to refer to a particular collection of genes forming a functional unit (genome of species, mitochondrial genome, genome inherited from an ancestral species in a polyploid individual, etc.). Because of the infinite combinations of genes and alleles that are possible, two individuals cannot possess exactly the same genome, apart from identical twins or clones. It is particularly important to understand the difference that exists between the two related words genome and genotype. In its strict sense, as defined by population geneticists, the genotype corresponds to the allelic composition of the gene locus or loci studied in an individual. In that sense, the genotype refers only to a class of individuals and then two different individuals can

belong to the same genotype or not depending on which locus (or loci) is (are) considered. The phenotype, on the other hand, is the collection of an organism's characteristics that result from the interaction between its genome and the **environment** in which it developed. In population genetics, however, the term phenotype refers in the narrow sense to only a subset of the characteristics of an organism, i.e. those dependent on a particular locus or on any other specific portion of the genotype. Depending on the gene or genes considered, two individuals can in that sense belong to the same phenotype or to two different phenotypes. We will consider the links between these two concepts in further detail in Chapter 5 and at the start of Chapter 6.

In behavioural ecology, we generally consider categories of individuals that share a particular characteristic (referred to as a strategy). Thus we tend to use a narrow definition of the term phenotype. We speak for example of the 'dispersing' phenotype to denote individuals that have moved between territories during their life, as opposed to the 'resident' phenotype that denotes individuals that remained on the same territory throughout their life.

*From genes . . .*

At this point, it is appropriate to emphasize that the word gene has a double meaning (Dawkins 1989, Haig 1997). In one sense, a gene refers to a material structure, a group of atoms organized into a particular sequence of deoxyribonucleic acid (DNA). In another sense, however, a gene refers to a more abstract concept, that of the **information** that is carried by that particular DNA sequence and is used to produce a protein. We therefore speak of **material** genes to refer to the many material copies of the same **informational gene** that exist in the same or different organisms. Ultimately, it is the information carried by the structure of DNA that persists across genera-

tions, and not the structure itself (Dawkins 1989). A gene is consequently a unit of information whose material support is a DNA sequence. We will come back to the question of information in biology in more detail in Chapter 4.

*. . . to avatars*

We use the term 'avatar' to designate the material forms produced by information (Gilddon and Gouyon 1989). Individuals are but 'avatars', that is to say by-products of genes (Dawkins 1982, Gilddon and Gouyon 1989). The word 'avatar' is taken from the Hindu religion, where it refers to the material forms taken by the god Vishnu during his visits to earth. Information is the sole target of selection, and avatars are merely its vehicle. Hence, an avatar is the entity under selection, but it is the self-replicating information it contains that is the absolute target of selection.

The characteristics of organisms can be seen as strategies put in place by genes to survive and replicate, while individuals are their **vehicles**. An individual exists only as a temporary vehicle, or a machine created by genes to help the genes survive and replicate (Dawkins 1976). Another way of putting it is that individuals are genes dressed in an elaborate external phenotype (Wittenberger 1981). When viewed in this manner, the gene reveals itself as the true unit of selection. As the famous evolutionist from the United States Georges Williams said in his now classic 1966 book on selection, 'genotypes are mortal', whereas 'genes are potentially immortal'. Indeed, a genotype disappears upon the death of its associated organism, but also during the process of meiosis, which allows only the genes to endure not their combination. Because of recombination during meiosis, the lifespan of genes is outstandingly longer than that of genotypes. The complex structures (cells, organs, organisms, societies) and behaviour that have evolved over the course of natural selection can be considered as adaptations for the benefit of genetic information, that is for the good of genes (replicators) rather than individuals (vehicles). This notion remains compatible with the fact that every

individual is an organized whole, within which the fate of a given gene is linked to the degree of cooperation or coordination between itself and other genes (Haig 1997).

### 2.2.3 Phenotype, genotype, and reaction norm

Natural selection can be analysed at two levels. The first is the level at which the individuals of the same species within a population are sorted. This sorting takes place as a function of the characteristics that vary from one individual to the next and that differentially affect their survival and reproduction. The second is the level at which the frequency of different alleles changes between two consecutive generations as a consequence of selection. This is the **response to selection**. There is a strong dividing line between these two levels which can be made further explicit through the notion of the **uncoupling of genotype and phenotype**. This notion uses several different concepts, many of them developed in quantitative genetics. We will limit ourselves here to the fundamental principles (but see Roff (1992, 1997), Stearns (1992), and Futuyma (1998) for a more complete discussion).

#### 2.2.3.1 Heredity and heritability

The phenotypic differences between the individuals of a population can be of genetic and/or environmental origin. Specifically, phenotypic variation can result from differences in the genetic information contained in the fertilized eggs from which these individuals originated, or from differences between the environments in which they developed (Cockburn 1991). In most cases, individual variation in a given trait results concurrently from genetic and environmental effects. We can thus divide the phenotypic variance of a trait ( $V_P$ ) according to the following formula:

$$V_P = V_G + V_E + V_{G \times E} \quad (2.1)$$

where  $V_G$  and  $V_E$  correspond to genetic variance and environmental variance, respectively, and  $V_{G \times E}$  cor-

responds to the variance that is due to the interaction between the genotype and the environment.

The total genetic variance of a character controlled by multiple genes can also be separated into numerous components. A part of genetic variance is from the specific interactions that occur within a given individual, including variance from the dominance of alleles at the same locus ( $V_D$ ) and from **epistatic** interactions between alleles from different loci ( $V_I$ ). The resemblance between parents and their offspring is determined by the additive effects of the alleles at each locus and at the different loci involved (called additive genetic variance,  $V_{AG}$ ).

$$V_G = V_{AG} + V_D + V_I \quad (2.2)$$

When  $V_{AG}$  approaches a value of zero,  $V_D$  and  $V_I$  also approach zero. If there is no additive variance, there cannot be variance due to dominance because it is the same for all individuals; nor, for the same reason, can there be variance due to epistatic interactions between loci.

It is the additive variance alone that allows for a response to selection. The **heritability** of a trait is therefore defined as the ratio of the additive variance and the phenotypic variance ( $V_{AG}/V_P$ ), usually represented by the term  $h^2$  (Falconer 1981). Heritability is then the portion ( $V_{AG}$ ) of the differences between individuals ( $V_P$ ) that is transmitted to descendants. Stated otherwise, heritability is the 'heredity of differences'. The problems stemming from measuring heritability are discussed in Box 2.2 and later in Chapter 20.

Because heritability is the proportion of additive genetic variance contributing to phenotypic variance, it indicates whether or not there is any genetic variation in a specific sample on which natural selection can act. However, even when there is appreciable genetic variation among individuals, heritability can be low because of substantial environmental variation. In a symmetric way, heritability can be high

### Box 2.2 How is heritability measured?

In practice, completely eliminating the environmental component of phenotypic variance is not an easy task. To do so, all environmental factors that could potentially act upon phenotypic variance must be controlled. One manner of solving this problem is to conduct experiments in which selection can be simulated and observe how it affects variation in a trait in the next generation. Suppose that we are studying the heritability of a trait that shows continuous variation in size across a population (Figure 2.3a, solid line). We can simulate selection by having, for instance, only individuals showing a larger size of the trait in question reproduce (Figure 2.3a, dotted line). The difference between the average value of the entire population ( $T_{mt}$  in Figure 2.3a) and the average value of the reproducing sample ( $T_{ms}$  in Figure 2.3a) is the

selection pressure  $S$  exerted in our experiment. We can then raise the descendants under standard conditions to remove environmental effects on phenotypic variance as much as possible. Upon measuring the average value of the trait in the descendent generation ( $T_{mF1}$ ), we can calculate the difference  $R = T_{mt} - T_{mF1}$ , which represents the selective response that we have created (Figure 2.3b). The ratio ( $R/S$ ) measures the proportion of the selection pressure that is transmitted to descendants, the heritability of the trait in question.

However, there are numerous situations in which such controlled experiments are not possible. In these cases, we can use the resemblance between parents and their offspring. Unfortunately, such measures can be misleading as this resemblance may be due to

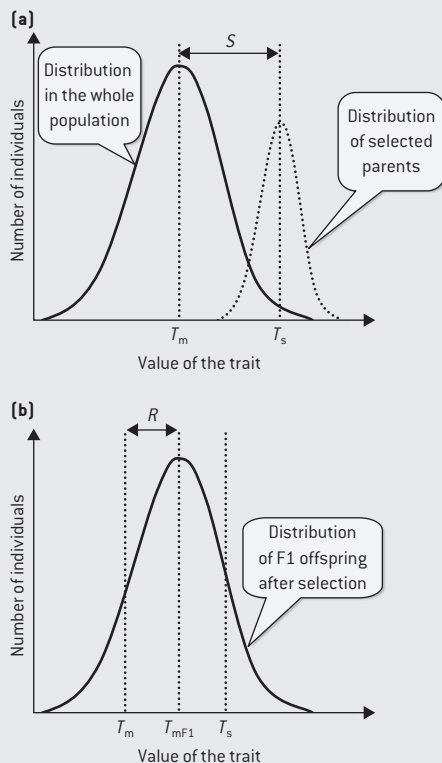


Figure 2.3 Selection experiment to measure the heritability of a character

- We are interested in the heritability of a trait showing continuous variation ( $x$  axis). The distribution of this trait in the population prior to selection is represented by the solid line. The trait has an average value of  $T_{mt}$  in the total population. In the parental generation, the experimenter only allows individuals with a large size of the trait (on average  $T_{ms}$ ) to reproduce. By doing so, the experimenter exerts a selection pressure that can be measured by the difference  $S$  between the average value of the trait in the whole population and the average value in the selected population.
- In the F1 descendant generation, the average value of the trait ( $T_{mF1}$ ) allows us to estimate the response to selection,  $R$ .  $R$  can vary between zero and  $S$ . The ratio  $R/S$  provides a measure of heritability, the portion of the differences between parents that is transmitted to the next generation.

**maternal effects**, environmental differences that cannot be controlled because they are linked to the parents, or to social (cultural) influences. This is especially the case when parents help raise and care for their young: for example, larger mothers might provide more food to their young (perhaps because they are dominant and can thus access better resources), which would lead to descendants having a larger average size, regardless of genetic variance. In such a situation one would find significant heritability of body size that would in fact result from parental effects, not necessarily from genetic variation among families. One way to avoid this problem is to make reciprocal transfers by exchanging half the descendants between two families. Such cross-fostering experiments allow us to distinguish, at least in part, maternal effects (being raised within a same family) from effects due to genetic variance (being born from the same parents but raised by different parents). However, the resemblance between siblings from a same litter can be due to dominance ( $V_D$ ) or **epistatic** interactions ( $V_I$ ), which are likely to be similar between siblings. The best way to avoid

these problems is to work with half-siblings that share the same father but different mothers. The resemblance between half-siblings would then be due to genes from the father (see Falconer (1981) and Roff (1997) for a discussion on heritability estimation methods).

Another possible complication is when the effects of the environment and genotype are not additive, but rather there exist interactions between the phenotype and the environment. This is the case for myopia in humans. For a century in several societies, males with myopia have preserved their capacity to survive and reproduce successfully because their poor sight was compensated for by the use of glasses. In addition, strongly myopic individuals have benefited from exemptions from military duty during recent conflicts, which further increases this capacity relative to other individuals exposed to the dangers of war. This situation is unique to the modern world. In a hunter-gatherer society, myopic individuals are at a strong disadvantage. Readers who are interested may read Chapter 20 which further develops on heritability a major evolutionary concept.

despite moderate genetic variation, if there is little variation in the environment. It is therefore important to remember that heritability is always defined with regard to a specific sample with a specific genetic composition and environmental context. Therefore, any heritability estimate is specific to a given sample, and heritability estimates cannot be directly compared between samples not having the same genetic or environmental composition (Vitzthum 2003). This may explain why heritability may change over time within a population (Charmantier *et al.* 2006).

### 2.2.3.2 Phenotypic variance and reaction norm

The division of phenotypic variance into separate components assumes that the two types of effects,

genetic and environmental, operate in an **additive** fashion. To illustrate this phenomenon, let us consider the fictitious example presented in Table 2.1. In this example, French gammarids are larger than Irish gammarids (Table 2.1a), and gammarids raised at 18 °C are larger than those raised at 10 °C. These two effects (geographic origin and temperature) express themselves in an additive manner, in the sense that the effect of elevated temperature is the same for both populations (11.9 units in the population from France and 12.1 units in the population from Ireland) and the size difference between the two populations remains about the same at both temperatures (10.1 units at 10 °C and 9.9 units at 18 °C). The fact that temperature and geographic origin are additive indicates that the joint effect of the genotype and the

a. Size at sexual maturity of the amphipod <i>Gammarus pulex</i> (arbitrary units)		
Origin of the population	Temperature during development	
	10 °C	18 °C
France	125.1	137.0
Ireland	115.0	127.1
b. Growth between 2 and 5 months of age in two lineages of rat as a function of dietary quality (arbitrary units)		
	Enriched diet	Poor diet
	Lineage A	55.2
Lineage B	48.6	42.3

Table 2.1 **Genotype–environment interaction**

environment is equal to the sum of each of these effects taken separately. In such condition, the term  $V_{G \times E}$  in Equation (2.1) is zero.

Let us now consider the case of the growth of two rat lineages as a function of environmental quality (Table 2.1b). The growth of lineage A is better than that of lineage B when nutritional conditions are good, but the opposite is observed when nutritional conditions are poor. The effects of the genotype and the environment are no longer additive in this case: there is a **genotype–environment interaction**. In such a case, the term  $V_{G \times E}$  in Equation (2.1) is not equal to zero.

Depending on the environment in which it develops, a same genotype can produce different phenotypes. The term **norm of reaction** is used to designate all the phenotypes that can originate from a given initial genotype.

It is possible to depict graphically the additive and interaction forms of genetic and environmental effects in relation to the concept of reaction norms. When the two effects are additive, the reaction norms of the two genotypes (which correspond to the variation of the phenotypes expressed across an environmental gradient) follow parallel trajectories

(Figure 2.4a). In the opposite situation, when an interaction exists, the two trajectories are no longer parallel (Figure 2.4b).

### 2.2.3.3 Phenotypic variance and phenotypic plasticity

If  $R$  is not different from zero (Figure 2.3), the trait in question is not heritable. However, this does not imply that the trait is not hereditary, that it is not genetically coded. It only means that differences between individuals are not caused by differences in the genes coding for this trait ( $V_{AG}$  is not different from zero). Stated otherwise, there is no longer any genetic variation in the genes responsible for the expression of the trait. A simple example to illustrate the difference between heritability and heredity is the number of eyes in vertebrates. This character is not heritable because it has lost all genetic variation. Of course, this does not mean that eye number is not genetically controlled.

If a trait is not heritable, what causes the observable variation in the trait within the population? According to Equation (2.1), when additive variance is zero, all phenotypic variance can be attributed to environmental variance. Differences between individuals are then caused solely by the differential effects of the environment during development.

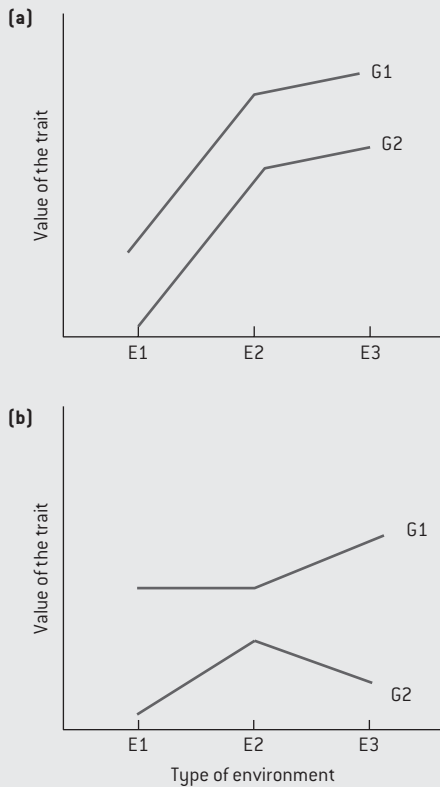


Figure 2.4 **Reaction norms for two genotypes (G1 and G2) in three environments (E1, E2, and E3)**

The curves joining the values of the trait along the gradient of environments represent the reaction norm of the study trait.

- a) When the environmental and genetic components of phenotypic variance are additive, the reactions norms are more or less parallel.
- b) When there are interactions between the genotype and the environment, the reaction norms are no longer parallel.

The environmental variance  $V_E$  brings about what is called **phenotypic plasticity**, the capacity of a given genotype to produce different phenotypes according to the environment in which an individual develops. Equation (2.1) can now be approximately reformulated by stating

that phenotypic variance results from heritability and phenotypic plasticity. In Chapter 20 we will decompose the term  $V_E$  into its various components. This will allow us to better understand the various components of phenotypic plasticity.

Phenotypic plasticity can either be adaptive or can simply reflect physiological effects that have no adaptive significance. It is favoured by natural selection when it allows individuals from a population to adapt efficiently to different environments that they might encounter. However, there are certain costs linked to the maintenance of the sensory and regulatory machinery necessary to ensure this flexibility (DeWitt *et al.* 1998), such that phenotypic plasticity remains limited.

#### 2.2.3.4 Phenotypic plasticity and adaptation

The adaptive significance of phenotypic plasticity can be clearly demonstrated by looking at organisms that produce clones. For instance, water fleas, freshwater cladoceran crustaceans, have asexual reproductive phases during which many individuals possessing the same genotype are produced. However, the members of a given clone can develop an extension over the head (called rostrum) of variable sizes (Figure 2.5) depending on the surrounding conditions.

In a detailed study on the water flea *Daphnia cucullata*, Anurag A. Agrawal and colleagues (1999) analysed the environmental factors influencing the development of the rostrum as well as its adaptive significance. First, they showed that if development took place in the presence of predators placed in enclosures such that they could not interact with the water fleas, adult water fleas had rostrums almost twice as long as individuals of the same clone raised in the absence of predators. These results were similar for two different predator types, the cladoceran *Leptodora kindtii* (average rostrum length of controls  $15.53 \pm 0.35$  versus  $29.71 \pm 0.49$  for individuals raised in the presence of predators,  $t = 23.72$ , degrees of freedom (df) = 303,  $P < 0.001$ ), and the dipteran *Chaoborus flavicans* (average rostrum length of controls

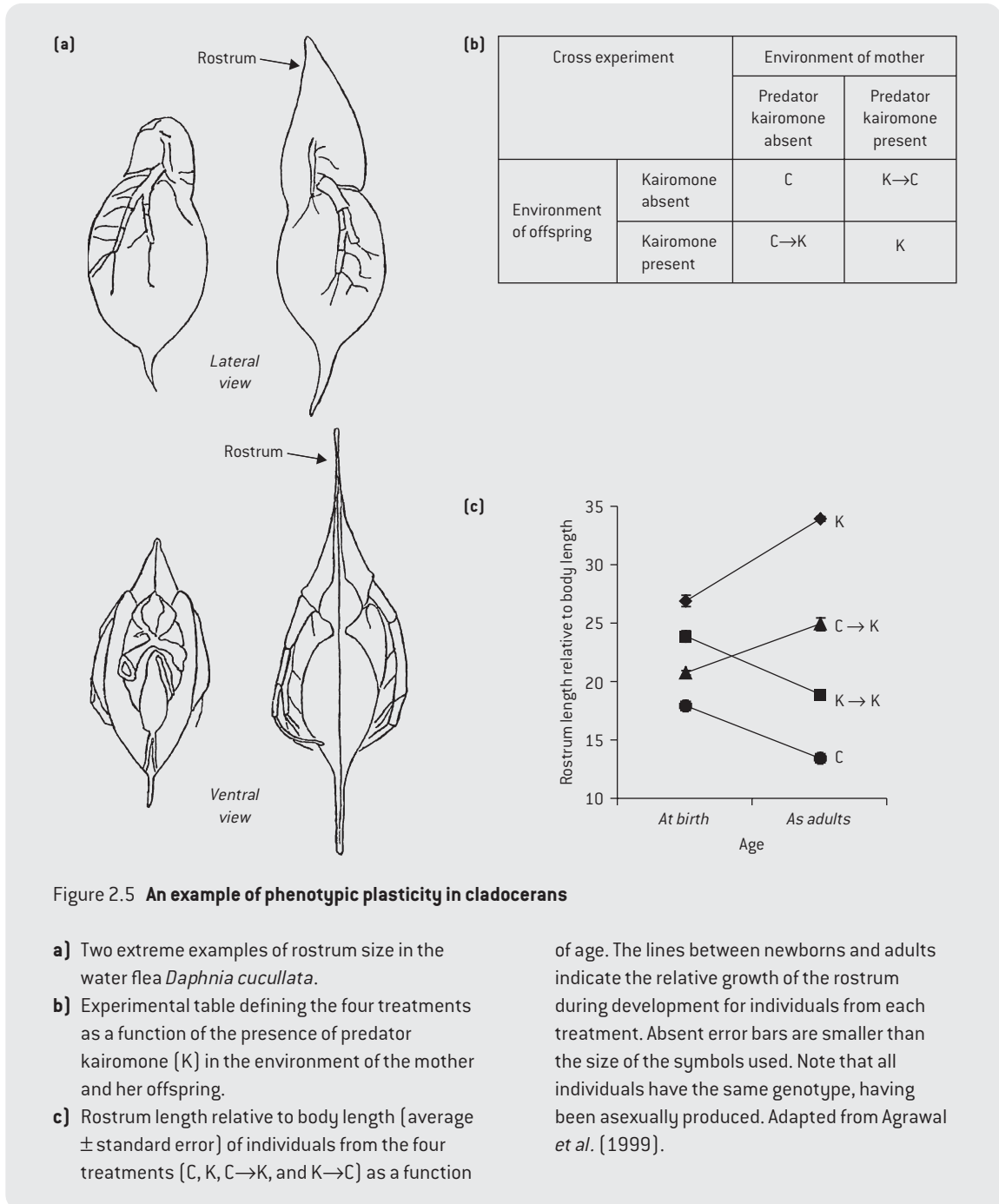


Figure 2.5 An example of phenotypic plasticity in cladocerans

- a)** Two extreme examples of rostrum size in the water flea *Daphnia cucullata*.
- b)** Experimental table defining the four treatments as a function of the presence of predator kairomone (K) in the environment of the mother and her offspring.
- c)** Rostrum length relative to body length (average  $\pm$  standard error) of individuals from the four treatments (C, K, C→K, and K→K) as a function

of age. The lines between newborns and adults indicate the relative growth of the rostrum during development for individuals from each treatment. Absent error bars are smaller than the size of the symbols used. Note that all individuals have the same genotype, having been asexually produced. Adapted from Agrawal *et al.* (1999).

$13.93 \pm 0.15$  versus  $27.88 \pm 0.28$  for individuals raised in the presence of predators,  $t = 44.29$ ,  $df = 470$ ,  $P < 0.001$ ). This first experiment demonstrates that it is indeed predators that influence rostrum development, and that water fleas recognize the presence of predators through chemical substances that the latter

release in the environment. Subsequently, when water fleas with and without rostrums were placed in contact with predators, it was shown that possessing a rostrum dramatically reduced the chances of being captured by a predator, indicating that the rostrum undeniably has an adaptive function.

Agrawal *et al.* (1999) then studied the mechanism leading to rostrum development by controlling the environment in which the mother and offspring develop (Figure 2.5b). At birth, young water fleas born from mothers having experienced an environment with predators had a larger rostrum than those born from mothers having lived in a predator-free environment (Figure 2.5c: comparison of treatments K and K→C to treatments C and C→K at birth). This demonstrates the existence of a maternal effect, because the environment experienced by the mother influences the state of newborns. The mother transmitted to her offspring a certain amount of information about the environment in which they are likely to develop. Similar to what was observed in the initial experiment, the presence of predators also triggered rostrum growth (Figure 2.5c: comparison of treatments K and C→K to treatments C and K→C). Individuals that developed in the presence of predators invested more in rostrum growth than individuals that developed in the absence of predators. Furthermore, once water fleas reached the adult stage, the maternal effects were still evident. For example, adults from treatment C→K had a smaller rostrum than adults from treatment K, despite the fact that in both cases, development took place in the presence of predators. Thus the difference observed between the two groups was entirely due to the environment in which the mothers had experienced. Furthermore, in this same experiment, Agrawal *et al.* (1999) also demonstrated the possible existence of a **grand maternal effect**. Hence, the causes and effects of phenotypic plasticity are multiple and complex.

#### 2.2.4 Genotypic and individual fitness

Another central concept in evolutionary biology is that of **genotypic fitness**, which can represent both an estimation and a prediction of the rate of natural selection. Let us consider the process of natural selection when it implies genetic heredity. Certain genotypes could be more successful than others, and it is possible to demonstrate this by studying a natural population (Endler 1986; Bell 1997).

Genotypes that replicate more effectively across successive generations are said to have a greater genotypic fitness. This concept is applicable to a group (or class) of individuals defined according to the allele (or alleles) they possess at a specific locus under consideration (or multiple loci considered simultaneously). **It thus designates the relative success of an allele or a combination of alleles across two generations. Thus, it is the capacity of an allele or a combination of alleles to change in frequency within the population across generations.** This success is a direct result of the differential survival and reproduction of individuals in the population that possess this allele or combination of alleles. Taken in this sense, the concept of genotypic fitness only makes sense when referring to classes of individuals, and it is not appropriate to speak of the genotypic fitness of a single individual. This strict definition of genotypic fitness is that adopted by population geneticists.

In practice, behavioural ecologists do not necessarily know the genetic origins of the traits they are studying. Their main concern is rather to determine the selective forces acting upon the character under study. To do so, it is more appropriate to measure at the individual scale the survival and reproductive consequences of variation (natural and experimentally induced) in the trait. Individual **fitness is the ability of a phenotype to produce mature descendants relative to other phenotypes in the same population at the same time.**

Measures of individual fitness (or simply fitness) are implicitly substituted for measures of genotypic fitness in most studies. It corresponds then to the average **demographic success** of a phenotype relative to the success of other phenotypes present in the

population. The quantification of individual fitness can be, depending on the objectives of the study, limited to a short period in the life of an individual (winter survival, the number of offspring produced in a single reproductive event) or, ideally, according to the total reproductive success of an individual calculated over its entire lifetime. Individual fitness designates the **success of a trait within one generation**. The fundamental questions of how to measure individual fitness will be developed in Chapter 5.

The concepts of genotypic and individual fitness are only meaningful when considered within a single population. It makes no sense to compare these values between genotypes or between individuals belonging to different populations or living at different time periods.

## 2.2.5 Evolution, natural selection, and adaptation

### 2.2.5.1 What is natural selection?

Behavioural traits, in the same manner as all other types of characters observed in living organisms, are the result of the history of species and populations, just like the organization of galaxies or the features of today's mountain ranges stem from past events. However, there is an important distinction between the organic and inorganic worlds. Living organisms have evolved over time because of, in large part, the process of selection (Endler 1986; Dawkins 1986, 1989). When the characteristics of organisms are beneficial to the survival and reproduction of individuals within a stable environment, copies of the allelic forms of the genes that are responsible for these characteristics multiply and spread throughout the population, which has the effect of increasing the frequency of these allelic forms in the population over time. This differential multiplication of genes according to their beneficial effects on the survival and reproduction of their vehicles within a given environment is the process of natural selection.

Since Darwin (1859), natural selection has played a pivotal role in the development of the modern synthesis of biological evolution (Fisher

1930; Williams 1966; Dawkins 1982; Endler 1986; Bell 1997). Despite this, its definition is often vague and imprecise, and reference to it still raises controversy. It is therefore necessary to define carefully and accurately what natural selection is, as well as explain its applications in the study of animal behaviour.

#### Definition

Natural selection is a process (Endler 1986), a group of related events linked through a chain of causality. There are certain requirements for natural selection, each independent of the other, and when these requirements are simultaneously met, the effects of selection ensue. There are three conditions that must necessarily be fulfilled to trigger natural selection.

- (a) There is **variation** among individuals in a particular trait.
- (b) There is a **consistent relationship** between this trait and the ability of individuals possessing the trait to survive (for example the ability to evade predators) and/or reproduce (for example the ability to produce viable offspring). In other words, there exists a consistent relationship between this trait and individual fitness. This is what is known as the **selection pressure**.
- (c) Variation in the trait can be passed on to the next generation, independent of effects related to the fact that successive generations may develop within the same environment. In other words, the trait must be **heritable** (or more generally transmittable; see **transmittability** in Chapter 20).

When these three conditions are met simultaneously, foreseeable effects will automatically be produced within one generation (1) and between two successive generations (2):

1. The frequency distribution of the trait will vary **predictably** among age classes or among different life cycle stages, beyond differences linked to the ontogeny of the trait (with the provision that environmental conditions remain stable throughout the life cycle); at the genetic level, the frequencies of the alleles coding for the trait will vary **predictably** over time within the same cohort.
2. If the population is not at **equilibrium**<sup>1</sup>, the distribution of the trait within a generation will differ **predictably** from the distribution within the parental generation, beyond the effects produced by conditions (a) and (c) alone; at the genetic level, the frequencies of the alleles coding for the trait will change **predictably** from one generation to the next.

This is one of the major points of evolution by natural selection. If conditions (a), (b), and (c) are fulfilled and if the environment remains stable between successive generations, it is possible to **predict** the direction in which evolution will take place between two generations. This does not mean that selection has a predetermined, organized goal, but rather that it is always the phenotype that produces the greatest number of descendants that increases within the population, until eventually replacing representatives of all other phenotypes. It is possible to distinguish between different modes of selection according to their effects on the mean and variance of a particular character (Box 2.3).

In sum, natural selection is a process in which individuals are sorted out according to their ability to survive and reproduce. The immediate response to selection is observable only at the level of changes

<sup>1</sup> By definition, when a population is at equilibrium, the same frequency distribution for a trait persists in each generation. The population is then not evolving. This can be the result of natural selection, or of its association with other antagonistic forces. If a population is not at equilibrium, evolution of the trait can occur. It is important to note that equilibrium is not an intrinsic property of a population and cannot be judged unless in relation to a given trait.

in allelic frequencies. The gene therefore represents the **unit of selection**. Natural selection has been demonstrated within a wide variety of organisms (see Endler 1986; Bell 1997), and numerous studies have shown that its impact can be rapid and large (see, for example, Malhotra and Thorpe 1991).

#### 2.2.5.2 Evolution, natural selection, and genetic drift

##### *Selection does not always lead to evolution*

If only conditions (a) and (b) of those described above are fulfilled, a selection pressure still exists in that only individuals in which the trait takes a certain value are better able to survive and reproduce. But in the absence of heritability of the trait, there is no selective response and the gene frequencies within the population will not change from one generation to the next. A selection pressure working on a trait is consequently not sufficient to cause evolution of the trait. For that matter, **evolution does not always imply natural selection**.

##### *Genetic drift: evolution does not necessarily imply selection*

Evolution can take place in a random manner by **genetic drift**, or by chance events such as large natural disasters, or the displacement of certain individuals. Thus, **evolution and selection are not interchangeable terms** (Sober 1984). Natural selection is one of the possible causes of biological evolution, but it is not the only one.

Genetic drift **corresponds to the random sampling of alleles between generations**. Genetic drift is a stochastic process. It emphasizes the random nature of transmitting alleles from one generation to the next given that only a fraction of all possible zygotes become mature adults. Because a pair of diploid sexually reproducing parents only have a limited number of offspring, not all of the parent's alleles will be passed on to their progeny because of chance assortment of chromosomes at meiosis. In a large population this will not have much effect in each generation because the random nature of the process will tend to average out. However, in a small

### Box 2.3 Effects of selection on the mean and variance of a trait

Selection can occur according to different modes. To keep matters simple, we will consider only two essential modes: **stabilizing selection** and **directional selection** (there is a third mode, **diversifying selection**, which leads to bimodality in the distribution of a trait by symmetrically favouring its extreme values). Let us consider a continuous trait that is normally distributed within a population and whose variance is equal to the mean (this type of distribution is typical of morphological characters: size, weight, number of segments, etc.). Under stabilizing selection (Figure 2.6), the genotype representing the average value of the character has the greatest

fitness. Genotypes responsible for the other more extreme values of the character have a lower fitness, and more so the further they are from the mean value. This mode of selection results in a narrowing of the distribution about the mean, which remains unchanged across two generations, although the variance is reduced. Under directional selection (Figure 2.6), the genotypes that represent one of the two extremes of the distribution are favoured. The mean will then change in this direction between two generations, and the variance may also be reduced.

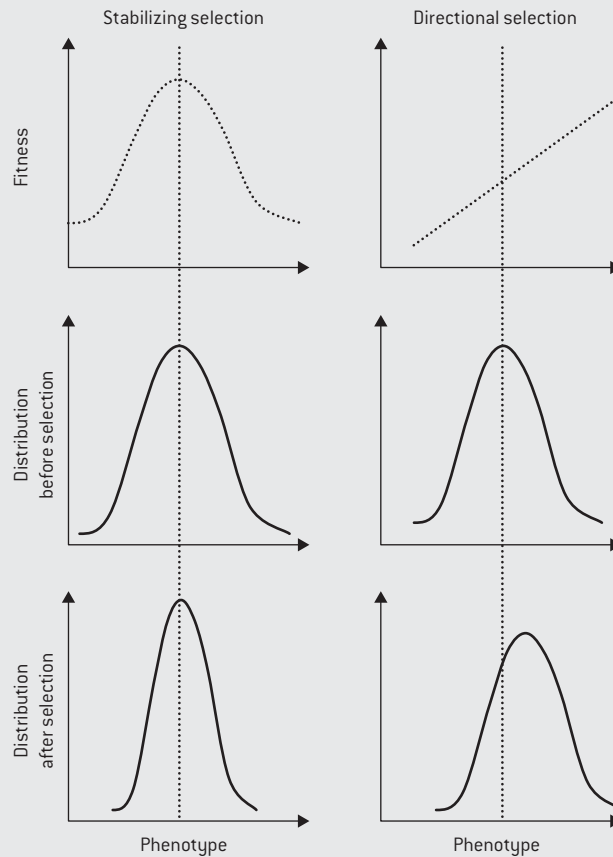


Figure 2.6 **Stabilizing and directional selection**

population the stochastic effect can be significant and lead to more lasting changes in allele frequencies between generations. The importance of genetic drift therefore depends critically on the size of the population. Actually, the exact probabilities that determine the duplication of alleles during sexual reproduction are only seen within populations of infinite size. Because all natural populations are of finite size, genetic drift always has a more or less moderate effect depending on the size of the population and the extent of other operating evolutionary forces (selection, dispersal). Hence, genetic drift and selection work concurrently within small populations. The essential difference between natural selection and genetic drift is found however in the conditions in which they take place. Variation in a trait does not have to have survival and/or reproductive consequences (condition (b) of selection) for genetic drift to occur.

#### *Natural selection versus genetic drift*

The only difference between natural selection and genetic drift is in the condition (b) of how the various phenotypes are selected. In genetic drift there is no link with fitness. It is just a random drawing of some individuals from the population. Thus, because of its random nature, genetic drift can affect the frequency of one allele either positively or negatively at each generation, such that it cannot be responsible for directional and consistent changes in allele frequencies over several generations.

If this is indeed the case, why do behavioural ecologists consider natural selection of such central importance? Natural selection is by far the most important force in biological evolution because only natural selection can explain the evolution of complex structures such as bird or insect wings, eyes, etc. It is indeed highly unlikely that such structures evolved for other reasons than the fact they do increase fitness. The argument supporting this position has been included in many well-known works (Dawkins 1982, 1986, 1989; Dupré 1987; Bell 1997), and we encourage the curious reader to explore them. We will confine ourselves here to

the essential aspects. The privileged status that is bestowed upon natural selection simply results from its central role in understanding the logic behind the hierarchical organization of life. The complex traits that are observed today did not appear 'as if' following a simple mutation. They are more likely the result of a series of cumulative changes, where each intermediate step was better adapted to the environment than the preceding one (see Dawkins 1986). This phenomenon cannot be explained in terms of genetic drift, only through natural selection. Natural selection is at the basis of how organisms **adapt** to their surroundings.

#### 2.2.5.3 *Selection and adaptation*

The adaptive state of organisms is a narrow connection between the form taken by the organs that fulfil various functions and the environmental conditions in which these functions must be fulfilled. This connection is especially evident when species that are distantly related but living in the same environment have striking resemblances. This is referred to as evolutionary **convergence**. A typical example of convergence is the remarkable resemblance between the external appearances of marine mammals and fish, despite the fact that the origins of the two groups are separated by over one hundred million years. Previously attributed to divine intervention, adaptation is today considered the result of the cumulative effects of natural selection in the past.

**Adaptation** is a historical concept (Sober 1984). A trait is considered an adaptation if it was fixed (or stabilized) within a population following a selection event.

#### **a** *Two meanings to the word 'adaptation'*

However, certain authors use the same word to describe any trait whose frequency in a population is increasing through natural selection. The term adaptation then has a certain ambiguity to it. It can simultaneously describe two different aspects of evolution, which need to be clearly distinguished:

**adaptation can refer to the final result of a completed process or a process currently underway.** The study of adaptive states or the degree of adaptation applies to the ‘finished product’ of a selection event. At this stage, the trait is fixed within the population or its frequency is stable. When a trait is fixed, there is no longer any genetic variation in this trait and its heritability is zero (hence the expectation that traits that contribute much to fitness should have low heritability (Falconer 1981). The study of allelic frequency changes is consequently not applicable to describe the degree of adaptation. Conversely, the study of selection in progress concerns the process itself. The continuation of the process necessarily implies that the trait maintains a certain degree of heritability over time. It then becomes possible to detect changes in allelic frequencies as long as the genetic origins of the trait have been identified.

#### *Four mechanisms of adaptation*

Overall, **the adaptive state of an organism can be produced by four important mechanisms**, all quite different (Gould and Lewontin 1979; Laland *et al.* 2000). First, the degree of adaptation can be the result of the Darwinian processes of mutation/selection as described above. Next, the processes of physiological and behavioural development allow organisms to shape themselves to the conditions prevalent during their development (through phenotypic plasticity; see Chapters 5 and 6 for further details). Of course, the adaptations themselves are not passed on, but the ability of organisms to display such plasticity is genetically transmitted. The third mechanism relates to cultural adaptation, which has become transmittable through the learning process (see Section 2.2.7 and Chapter 20). The fourth mechanism is rarely accounted for despite the fact that it plays a major role in human adaptation. It is ‘niche construction’ (Laland *et al.* 2000), the fact that organisms constantly modify their own environment, which in turns affect their fitness. In species where offspring often inherit their parents’ territory, niche construction may strongly affect the fitness of descendants over many

generations and such variation in environment becomes transmitted across many generations. In other words it becomes transmittable and thus open to selection. We will discuss such issues in Chapter 20.

#### 2.2.6 Inclusive fitness

The use of individual fitness to characterize genotypic fitness, while justifiable within a limited framework, can lead under certain circumstances to erroneous conclusions. This is especially the case in the field of social behaviour. Individual fitness considers the consequences of an organism’s behaviour only in light of the propagation of its genes **as a result of the survival and reproduction of that individual**. We have seen, however, that the individual is not the unit of selection (see Section 2.2.2). Therefore, it is appropriate to measure the success of a behavioural trait by estimating the consequences of this behaviour for the genes that are involved in its expression. This means considering not only the consequences related to the survival and reproduction of the individual exhibiting the behaviour under study but also to consequences for other individuals that are affected by this trait. This leads us to the introduction of the concept of **inclusive fitness** (Hamilton 1964a, b). This central concept in behavioural ecology originates from sociobiology where it served to replace group selection as a better account for the evolution of altruistic behaviour among related individuals (see Box 2.4).

##### 2.2.6.1 A major but subtle concept

Despite its importance the concept was not always well understood (see Wilson (1975) and Barash (1982), two ‘stars’ of sociobiology!), and so it is imperative to explain the logic behind it clearly (Maynard-Smith 1982; Grafen 1984; Creel 1990).

When formulating his ideas on inclusive fitness, Hamilton drew attention to the fact that a gene responsible for a particular social behaviour could be selected for or against depending on the effects of the

**Box 2.4 Group selection: fact or fiction?**

In the early 1960s, many ethologists thought that certain phenotypic traits that reduced the survival and reproductive success of individuals that bore them could still be selected for if these traits otherwise increased the long-term stability and survival of the individual's group or the species. Selection it was therefore assumed could also operate **at the group level**. This idea was most clearly articulated by a Scottish ecologist Vero Wynne-Edwards (1962) in his work entitled *Animal Dispersion in Relation to Social Behaviour* (see also Wynne-Edwards 1986).

In the absence of any regulatory force, the growth rate of a population is exponential. The expansion of a population is however limited by the maximum number of individuals that the environment can sustain (called the **carrying capacity**). According to Wynne-Edwards, animals naturally tend to avoid overexploiting their environment, especially their food resources. This was achieved through altruistic behaviour in which certain individuals postponed or sacrificed their own reproduction in order to avoid overpopulation that would be fatal to all. Wynne-Edwards also proposed that certain social displays of species served to allow individuals to evaluate their population size and hence adjust their reproduction according to their perception of resource availability. In support of his theory, Wynne-Edwards cited numerous examples suggesting that animals do not always realise their full reproductive potential, and even actively regulate it. For example, in certain species, individuals do not immediately reproduce upon reaching sexual maturity, but rather defer reproduction for no apparent benefit. When a species has a distinct social structure, it is

common for subordinate individuals to not reproduce at all. In certain cases, infanticide even occurs. According to Wynne-Edwards, these trends and events supported the existence of population autoregulation by individual behaviour.

This idea was strongly criticized and refuted by Williams (1966) in his work *Adaptation and Natural Selection*. Williams gave the example of a population in which each individual was genetically predisposed to limit its own reproduction. If a single individual that was less genetically inclined to sacrifice its own reproduction entered the picture, it would undoubtedly leave more descendants than the other members of the population. This individual's 'abnormal' behaviour would spread across successive generations, until it was the only strategy within the entire population, even if in the end the whole population crashed because of it. Natural selection is a **blind** cumulative process that simply cannot think ahead. The examples of reduced individual reproduction interpreted by Wynne-Edwards as the self-restraint are now interpreted as the result of social constraints related to competition between individuals. Today, despite some efforts to revitalize group selection (see Borrello 2005), the individual and gene level selection point of view defended by Williams and Dawkins still dominates scientific thought. The reader interested in thinking about different levels of selection is recommended to consult Keller (1999). For those interested in reading about selection at levels above that of the individual we also suggest the papers of this theory's strongest adherent David S. Wilson (1997) as a good place to start.

behaviour on individuals other than the immediate progeny of its vehicle, as long as there is a certain amount of genetic similarity by descent linking the vehicle to the other individuals. So for Hamilton the fitness of a trait should **include** its bearer's fitness but also the fitness of all individuals likely to be bearing the same gene and to have been affected by the behaviour of the bearer. As a result, an early but erroneous way of thinking of inclusive fitness has been to consider it as the total number of direct descendants plus the number of direct descendants of genetically similar individuals, where the latter value is corrected for the coefficient of genetic similarity between the vehicle and the other individuals. If we ponder this definition for a moment, it becomes clear that inclusive fitness would then always tend towards infinity because each individual has a certain degree of genetic relatedness by descent, albeit minimal, with a great number of individuals in the population. Reduced to this definition, the concept of inclusive fitness would be of very little interest and of no utility.

*Hamilton's first definition . . .*

The correct interpretation of inclusive fitness is both more complex and more subtle (Hamilton 1964a, b; Grafen 1984; Creel 1990; Bourke and Franks 1995). In his initial article, Hamilton (1964a, b) defines it as:

'The personal fitness which an individual actually expresses in its production of adult offspring . . . stripped of all components which can be considered as due to the individual's social environment, leaving the fitness which he would express if not exposed to any of the harms or benefits of that environment, . . . then augmented by certain fractions of the quantities of harm and benefit which the individual himself causes to the fitnesses of his neighbours. The fractions in question are simply the coefficients of relationship appropriate to the neighbours whom he affects.'

What Hamilton calls the 'social environment' corresponds to the portion of the environment comprising an individual's interactions with its

neighbours. Neighbours can have a positive or a negative influence on a given individual's progeny, by either facilitating the individual's reproduction and/or the survival of its descendants, or conversely by hindering and limiting the reproductive potential of that individual through their behaviour. Likewise, an individual can have a positive or a negative effect on the reproduction of its neighbours by its actions. Within a given population, it is possible to calculate the total number of additional **direct** descendants imparted by the help of neighbours, divided by the total number of individuals (reproducers and helpers) in the population. This quantity corresponds to the average amount of help *per capita* within the population. Equally, it is possible to calculate the average amount of hindrance in the population, which would correspond to the total number of **direct** descendants lost due to the behaviour of neighbours divided by the total number of individuals.

*. . . was later improved*

However, Creel (1990) pointed out the necessity to replace a portion of Hamilton's original definition: '*all components which can be considered as due to the individual's social environment*' should be replaced by the term '**the per capita average effect (average amount of help – average amount of hindrance) due to the social environment of the individual**'. The importance of the adjustment brought by Creel can be illustrated using a simple example (Bourke and Franks 1995). Let us consider a population of animals in which there are two types of individual: reproducers and helpers. Individuals in the first category can only reproduce if they obtain help from conspecific helpers from the second category. Although the genetic basis of helping behaviour can be found in individuals from both categories, it is only expressed in certain individuals, the helpers (a similar situation is seen in certain species of social Hymenoptera, analysed in detail in Chapter 15). Then, according to Hamilton's original definition, the inclusive fitness of all reproducers would be zero. In fact, all the progeny of a reproductive individual is due entirely to the assistance of helpers, and that

same reproducer does not contribute whatsoever to the reproductive success of others. This unreasonable result is corrected in the modified definition proposed by Creel, where only the **average** effect of one individual on the reproductive success of others is removed. It follows that in such a population, the inclusive fitness of reproducers ultimately depends on the help they manage to obtain. An individual that obtains more help than average would have a positive inclusive fitness once the average amount of help per reproductive individual was removed. The same reasoning can be applied to helpers. Because they are sterile by definition, their inclusive fitness depends directly on the amount of help they provide to genetically similar individuals. If this amount is great enough (or if help is predominantly given to the most genetically similar individuals), the genetic origin of helping behaviour will spread throughout the population. If helpers do not favour reproducers according to their degree of genetic similarity, the gene will not spread.

### 2.2.6.2 Kin selection

How exactly do individuals evaluate the degree of genetic similarity between themselves and their neighbours? This has been a notorious problem since Dawkins (1976) proposed his 'green beard effect' metaphor. Let us suppose that a gene appears in a population that causes the two following effects (we know that a gene can have more than one effect; see Chapter 5): its owners all have a green beard and behave in an altruistic manner towards every other individual with a green beard. Let us also assume that it is impossible for a mutant to cheat by having a green beard but not displaying the altruistic behaviour. This situation is highly improbable, but if by chance it did occur the consequences would be clear: the gene would inevitably spread throughout the population.

Of course, nobody believes (not even Richard Dawkins) that the green beard effect could be commonly observed in its literal form. However, some evidence for green-beard gene have actually been found in nature, most specifically in the red fire ant (*Solenopsis invicta*, Keller and Ross 1998) and in

the social amoeba *Dictyostelium discoideum* (Queller *et al.* 2003). Most of the time, however, individuals distinguish genetically similar individuals through less specific signs than a 'green beard'. Kinship and familiarity are examples of such signs. A brother, a sister, or more generally 'those that were raised with me' represent an indicator of genetic similarity. Furthermore, two recent independent theoretical studies have shown that social influences can play the role of a green beard effect. First, Hochberg *et al.* (2003) with a simple model showed that the evolution of social discrimination causes the congealing of phenotypically similar individuals into different, spatially distinct tribes (or **cultures**, see Chapter 20). They also showed that such a result was only obtained with altruistic and selfish behaviour, not with spiteful and mutualistic behaviour. Second, Jansen and van Baalen (2006) showed that if the green beard and altruism effects are caused by loosely coupled separate genes, altruism is facilitated through beard chromodynamics in which many beard colours cooccur. In fact, both of these models showed that 'culture' may play the role of such a green beard effect (we will come back to the question of the evolution of culture in Chapter 20).

Natural selection preserving altruistic behaviour directed towards kin is aptly called **kin selection**.

### 2.2.6.3 Hamilton's rule

The ideas proposed by Hamilton (1964a, b) simplified the calculations necessary to establish the conditions under which certain alleles spread within populations. Let us consider some social behaviour that involves two individuals, the **actor** (that performs the social behaviour) and the **recipient** (towards whom the social act is directed). **Hamilton's rule** involves three terms:  $c$ , the degree of modification to the actor's fitness (considered a cost to the actor);  $b$ , the degree of modification to the recipient's fitness (considered a benefit to the recipient); and  $r$ , the degree of genetic similarity between the actor and the recipient. This degree of genetic similarity is

mathematically defined as a regression coefficient (see Bourke and Franks (1995, pp. 14–17) for a detailed explanation of the mathematical definition of the degree of genetic similarity; see also Chapters 13 and 15). Hamilton's rule is expressed by the formula:

$$br - c > 0$$

This rule is only valid under certain conditions. It is especially important to verify that costs and benefits are additive. An individual that is helped  $x$  times and that helps  $y$  times should in total experience a change of  $xb - yc$  to its number of descendants.

The interest in Hamilton's rule lies in a large part in its simplicity. Indeed, the rule is easier to apply than the concept of inclusive fitness. In practice, it is convenient to use the coefficient of relatedness between the interacting individuals for the value of  $r$ . However, applying the rule to real situations requires following several well-determined steps. It is especially imperative to specify which behavioural alternatives are under study and to evaluate properly all of their consequences. For instance, one could study the following alternatives: a, to reproduce *versus* b, not to reproduce in order to help one or many relatives reproduce themselves. If the animal chooses option b, then the cost suffered is the number of offspring it would have produced had it chosen option a. The benefits are more difficult to calculate because they require estimating the difference between the recipient's reproductive success when the actor plays a and b. Various examples applying Hamilton's rule are presented in Chapter 15 in relation to the study of the evolution of **cooperation**.

## 2.3 Conclusion

When evaluating the purpose or function of a behaviour it is crucial to keep in mind several of the basic concepts discussed in this chapter. Although animal behaviour may appear to defy logic at first, the application of the evolutionary framework often makes the biological reasons behind the behaviour quite clear. An evolutionary perspective applied at the correct level of selection can often turn an apparently damaging behaviour into one that is evolutionarily profitable because it helps transmit the vehicle's genes to the next generation. We will see many such examples in this book. As the great geneticist and evolutionary biologist Dobzhansky (1973) said, '*nothing in biology makes sense except in the light of evolution*'. It is therefore important to keep in mind that the most obvious explanation is not always the correct one.

However, it is also important to emphasize that variation in behaviour may also involve learning that contributes to phenotypic plasticity. Furthermore, learning from others may lead to the transmission of behaviour across generations. Although the transmission of the variation in such learned behaviours may not necessarily involve genetic variation, the genetic basis of an individual's flexibility and capacity to learn is likely to involve genetic variation. The transmission across generation of non-genetic, yet Transmittable, phenotypic variation that involves learning is usually called 'cultural transmission'. The importance of such culture in the evolution of animal behaviour is addressed in Chapter 20.

## >> Further reading

> For a complete discussion of the genetics behind evolution:

**Sober, E.** 1984. *The Nature of Selection. Evolutionary Theory in Philosophical Focus*. MIT Press, Harvard.

> For a discussion of how genes drive evolution:

**Dawkins, R.** 1989. *The Selfish Gene*, 2nd edn. Oxford University Press, Oxford.

> For a complete overview of natural selection and how it can be detected in natural populations:

**Endler, J.A.** 1986. *Natural Selection in the Wild*. Princeton University Press, Princeton.

> For a case study about the genetic component of phenotypic variance and adaptation to the local environment:

**Tracy, C.R.** 1999. Differences in body size among chuckwalla (*Sauromalus obesus*) populations. *Ecology* 80: 259–271.

> For a case study of natural selection in response to environmental conditions:

**Grant, B.R. & Grant, P.R.** 1989. Natural selection in a population of Darwin's finches. *The American Naturalist* 133: 377–393.

> For a case study demonstrating heritability measurements of morphological traits:

**Boag, P.T.** 1983. The heritability of external morphology in Darwin's ground finches (*Geospiza*) on Isla Daphne Major, Galapagos. *Evolution* 37: 877–894.

> For a review of reaction norms and phenotypic plasticity:

**Stearns, S.C.** 1989. The evolutionary significance of phenotypic plasticity. *BioScience* 39: 436–445.

**West-Eberhard, M.J.** 1989. Phenotypic plasticity and the origins of diversity. *Annual Review of Ecology and Systematics* 20: 249–278.

> For a review of cooperative breeding in mammals integrating inclusive fitness and kin selection:

**Jennions, M.D. & Macdonald, D.W.** 1994. Cooperative breeding in mammals. *Trends in Ecology and Evolution* 9: 89–93.

> For a comprehensive look at kin selection in insects:

**Queller, D.C. & Strassmann, J.E.** 1998. Kin selection and social insects. *BioScience* 48: 165–175.

## >> Questions

1. Explain why the terms 'heredity' and 'heritability' are not synonymous.
2. How do genotype–environment interactions affect the phenotypic variance of a trait?
3. In what type of environment is phenotypic plasticity most likely to be favoured by selection?
4. What is the difference between genotypic fitness and individual fitness? Which concept is more useful to the field of behavioural ecology, and why?
5. What exactly is natural selection, and what conditions are necessary for it to occur?
6. Why can genetic drift alone not explain the adaptations of organisms to their environment?
7. How do the concepts of inclusive fitness and group selection differentially explain altruistic behaviour? Which concept has more validity?
8. What was the major problem with the initial interpretation of Hamilton's (1964a, b) definition of inclusive fitness? What did Creel (1990) propose to improve this definition?
9. How are the concepts of inclusive fitness and kin selection related?
10. Meerkats (*Suricata suricatta*) are cooperatively breeding mammals that live in packs consisting of two to three family units, each with a single breeding pair. Other pack members forego their own reproduction in order to assist the breeders. Design an experiment that tests if meerkat group behaviour follows Hamilton's rule.
11. You are studying a population of a granivorous passerine bird species that shows variation in beak size. Propose a detailed experimental design that would allow you to measure the heritability of beak size in this population.
12. Using the concepts you learned in this chapter, construct a logical argument defending that it is genes, and not individuals, that are the units of selection.