1 Evolutionary approaches to behaviour

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Evolutionary theory was first applied to the behavioural sciences by Darwin (1871, 1872). Since then the field of evolutionary behavioural science has blossomed. The history of this field is detailed and well-rehearsed within the literature (e.g. Buss, 2008; Dennett, 1995) and it would not be possible to do it justice within the confines of a chapter. Instead, this chapter will introduce a particular perspective on evolutionary behavioural science in the hope that it will act as a stimulus to discussion and help the reader to unravel subsequent chapters in this volume. Inevitably, there is some historical detail in order to contextualise certain points, but this chapter is far from a complete history. I hope that historically minded readers will forgive this.

This volume is about evolutionary psychology, but I have chosen to write a chapter about evolutionary behavioural science. Psychology is, of course, a behavioural science and much activity within the discipline is solely about measuring behaviour. However, psychology is also interested in endocrine, neurological and cognitive mechanisms that all cause behaviour, and these mechanisms are discussed in computational or information-processing terms. These mechanisms can be said to mediate input–output relations, with the final outputs being behavioural. Evolutionary theory is used in order to discuss the functions of behaviour – what any given behaviour is designed to achieve – and to determine the functional limits for underlying psychological mechanisms. Evolutionary theory, then, gives us an ultimate explanation (Tinbergen, 1963) that helps us to develop accounts of the kinds of proximate mechanisms that constitute, in this case, the psychology of an organism; it does not tell us the detail of those mechanisms, which is a task left to the proximate methods of the discipline of psychology. This chapter discusses the breadth of this application, hence its focus on evolutionary behavioural science as a whole.

Evolutionary theory is a theory of design. Before moving on to discuss the process of evolution, it is important to clarify this key notion of design. Design is not confined to organic life, and an unnatural example will help to abstract the fundamental features of this concept. Think of a simple robot with the task of moving across a room whilst avoiding various obstacles (see Dickins and Dickins, 2008, for a fuller treatment of this example; and also Braitenberg, 1984). One possible design for such a robot would be based around a simple car chassis. It would have four wheels, two at the front and two at the back. The back wheels would be attached to a motor each, to power them independently. At the front of the robot, there would be two depression paddles. The left paddle would be connected to the back right motor; the right paddle would be connected to the back left motor. These connections would be inhibitory, such that depressing the left paddle would stop the right motor. As the left motor is still going, the robot would turn to the right (see Figure 1.1). In the same way, depressing the right paddle would cause the robot to turn left.[1] In this way, obstacles would be avoided and the robot could continue its travels across the room.

[1] There are obvious design flaws in this robot. First, if the robot hits an object straight on then both paddles could potentially be depressed and then both motors would stop. There are various ways around this, including angled paddles that will not make such a contact. Second, in a closed room the robot will eventually find a wall and begin a jerky kind of wall-following behaviour until it powers down. Again, more design is needed to resolve this and I leave it to the reader's imagination to improve the robot.

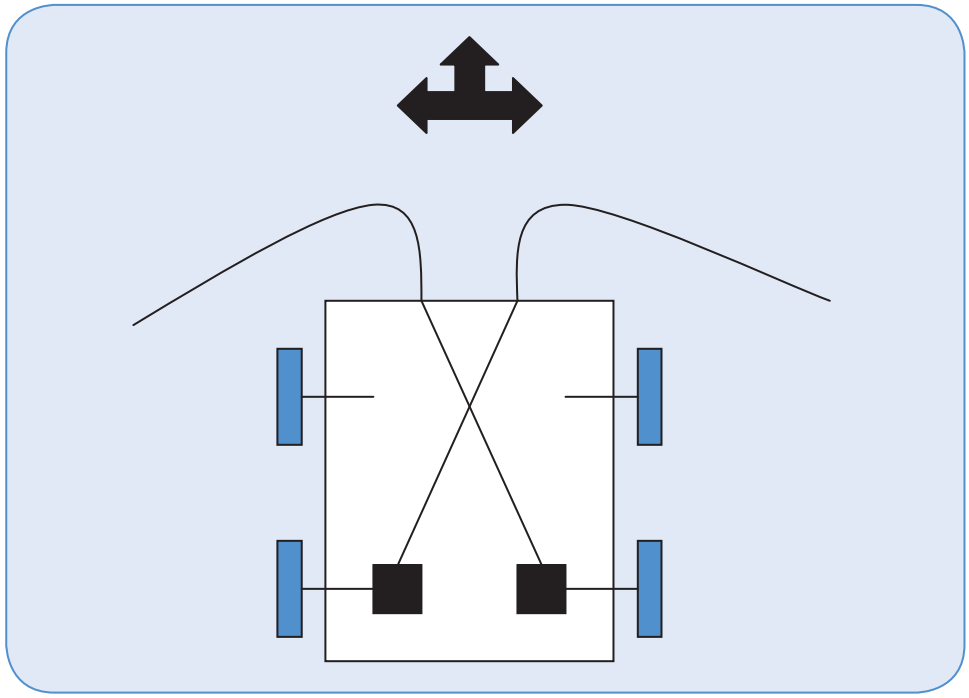

Figure 1.1. *A robot with two depression paddles at the front of its basic chassis design.*
These paddles are linked to rear motors such that when the left paddle is depressed it inhibits, or stops, the back right wheel, and when the front right paddle is depressed the back left wheel stops. In this way the robot can avoid most obstacles. This simple design is like those found in biological organisms in that it is a conditional architecture – if (P1: left sensor depressed) then (Q1: stop back right wheel).

The robot clearly has a function and has been designed, by an engineer, to meet this function. The depression paddles are an essential part of this design for they are the robot's only source of inputs from the outside world and, to this extent, they act as a sensory system. The only knowledge this robot has of the outside world, through which it has to move, is through these paddles. To this end, its only experience of the world is tactile and such inputs are the only inputs of any importance. The robot could be bathed in light and sound but such things will have no effect because the robot has not been designed to use them.

There is much discussion about the relationship between function and knowledge in the philosophical literature (e.g. see Millikan, 1993). I am using the term loosely in this chapter to imply a meeting of design and environment such that the robot, in this case, can deliver its functions. So, if a design provides a good fit to the environment the design embodies facts about that environment and in this sense represents something about the environment. This relates to the difference between environment and ecology that is mentioned later. I am not seeking to discuss notions of content, concepts, meta-representational states and all else associated with high-order cognition. However, the astute reader will realise that there is a relationship to be drawn out.

We can characterise the robot's design as a series of conditional rules, such that the robot's architecture embodies a number of possible decisions. Informally, we can say:

If (P1: the right paddle is depressed) then (Q1: turn left)
If (P2: the left paddle is depressed) then (Q2: turn right)

These conditional rules can readily be translated into more technical terminology that captures the actual mechanical actions made to move from P to Q. This may appear superfluous or, worse still, an exercise in artificial formalism. It is certainly a simple point that this robot can be described in this way. However, the point extends to all organisms and all biological systems. Think of a bat; bats navigate and hunt using echolocation. They emit pulses of sound and listen for a returning echo in order to locate the distance and direction of moths. Distance is easy enough to calculate as sound travels at a constant speed; so, simply timing the duration from utterance to received echo and then dividing it by two will give the distance. Bats have cognition that enables them to calculate the distance and, to some extent, you could argue that bat cognition encompasses this simple mathematical algorithm; it represents a truth about the world.

Bats determine direction of prey by calculating whether the echo reaches their right or left ear sooner. Folds within the ear help to determine the vertical positioning of the bat owing to the angle at which the sound hits the ear. Smaller objects reflect sound less intensely than larger ones and inbuilt knowledge of the sizes of suitable prey determines whether the bat will treat the echo as indicative of food. The sounds that bats emit are at very high frequencies, often too high for human ears. They are also at extremely loud volumes – indeed, if they were within the human frequency range but at the same volume, they would be damaging. This is clearly an issue for the bats. To counter this, they temporarily deafen themselves whilst emitting the sound, and then switch their hearing back on to receive the echo.

There is more to say about the biology of bat echolocation (see Altringham, 1999), but from this account you can see that we are dealing with systems that deliver outputs and that they are describable in terms of decision rules. So, for example, horizontal location during hunting can be characterised as follows:

If (P1: left ear input first) then (Q1: dip left wing and raise right wing)

Bats, just like robots, are clearly designed to process inputs and deliver outputs. When we discuss bats, we might refer to them as processing information about the outside world in order to navigate and hunt. That information is processed by conditional systems, and the relationship between the inputs and the processing systems captures truths about the world. Note that 'information' refers to the functional relationship between an input and a system. If the system is designed to take this input, designed to have a conditional response, then the input is informative. If not, then it is an empty input. Information is not something in the world to be captured; it is a consequence of design.

Bats, unlike robots, have no engineer, no designer who has intentionally built them to capture moths. To this end, we need an account of the design of organic life that does not rely upon any kind of agency: evolutionary theory delivers this.

A BRIEF INTRODUCTION TO EVOLUTIONARY THEORY

Darwin's interest was in the diversity and variation found in life. He noted that the various traits he observed in species appeared fit for purpose, as if they were designed to do a specific job, and he sought to explain this. His explanation was the theory of evolution through natural selection.[2]

The Darwinian view sees organisms as engaged in a struggle for life. They face numerous problems that can deleteriously affect their survival and reproductive chances. However, the heritable traits that organisms possess can help them to solve these problems and, if this happens, those traits can be passed onto their offspring. As traits vary between individuals, such that one individual may deliver a solution to a problem more effectively than the next, some organisms thrive relative to others. Those with a variant of a trait better suited to the struggle for life are more likely to survive and to reproduce than those with less-well-suited variants, and gradually this 'better' variant will come to dominate in the population because of its heritability. Just so long as the problem remains a stable aspect of the environment, this variant will reach fixation.

It is the problems of survival and reproduction that provide selection pressure for particular variants. Natural selection is, therefore, an economic consequence of the interaction between problems of survival and reproduction and heritable traits. Those traits that are selected can be termed 'adaptations'. Darwin's own formulation of the natural selection of adaptations can be distilled into a number of key principles. First, the principle that there is a struggle for life, an idea seeded by Darwin's reading of Malthus's (1798) *An Essay On the Principle of Population*, in which the consequences of geometric rates of reproduction relying upon finite, and at best gradually linearly increasing, resources, were discussed. Malthus's stark prediction was that population growth will exceed resources and will then be checked by famine, disease and war.[3] The second principle is that of trait variation. From these Malthusian catastrophes, some

[2] Darwin also established sexual selection. Sexual selection is a special form of natural selection in which the ecology of sexual reproduction establishes key selection pressures. Indeed, sexual selection is sometimes rephrased as sex-dependent selection (for recent discussions, see Carranza, 2009; Clutton-Brock, 2008).

[3] Malthusian tensions between population growth and resources are not regarded as the only form of struggle in modern times. More technically, what the Malthusian idea captured was the notion of differential survival and reproduction within a population.

individuals will emerge and continue their lives. The traits that enable them to survive can be passed on, which is the third principle of inheritance. Darwin draws the three principles together in the following passage of *The Origin* (1859/1985, pp. 169–170):

> If during the long course of ages and under varying conditions of life, organic beings vary at all in the several parts of their organisation, and I think this cannot be disputed; if there be, owing to the high geometrical powers of increase in each species, at some age, or year, a severe struggle for life, and this certainly cannot be disputed; then, considering the infinite complexity of the relations of all organic beings to each other and to their conditions of existence, causing an infinite diversity in structure, constitution, and habits, to be advantageous to them, I think it would be a most extraordinary fact if no variation ever had occurred useful to each being's own welfare, in the same way as so many variations have occurred useful to man. But if variations useful to any organic being do occur, assuredly individuals thus characterised will have the best chance of being preserved in the struggle for life; and from the strong principle of inheritance they will tend to produce offspring similarly characterised. This principle of preservation, I have called, for the sake of brevity, Natural Selection.

The Origin was published on 24 November 1859, and the impact of this elegant process of selection was immediately felt. Darwin had provided an explanation for design that required no hidden hand, no grand designer, and he demonstrated this through detailed and exhaustive natural history. What was lacking, however, was a theory of inheritance; how were traits passed on from generation to generation in a manner that would permit selection to do its work?

Many histories of Darwin (see Dennett, 1995) note that he had not read Mendel's paper on particulate inheritance published some two years before *The Origin*. One can only speculate as to how Darwin might have put this to use, although Darwin was not alone. Mendel's work on garden peas and other plants, and his notion of inheritance, was effectively ignored until the twentieth century, when chromosomes became the focus of attention for research on heredity (see Fox Keller, 2000). Then, in 1953, the structure and character of DNA was revealed, with the authors of that key paper laconically noting at the end that their discovery may have some impact upon theories of heredity (Watson and Crick, 1953). Genetics had come of age.

Genes code for proteins,[4] and in so doing build traits; genes vary in trait expression and genes are inherited. What is more, genes can become altered through mutation[5]

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[4] In fact, genes code for polypeptide chains (see Box 1.2) that in turn become proteins and enzymes (which are a subcategory of proteins) and some genes control the expression of other genes.

[5] I am focusing upon mutation in this chapter for ease of exposition. However, there are a number of random events that can lead to the emergence of new forms. For example, because of the random nature of the allocation of genes during gamete formation it is possible for genes (or alleles) to become over- or underrepresented within a population. Changes in relative gene frequencies as a consequence of such action are referred to as 'genetic drift'. Clearly, genetic drift can change the available traits within a population and this is like evolutionary change. However, it is worth noting that those genes are still subject to selection pressure. Recombination, founder effects and hybridisation can also introduce new forms (see Ridley, 2004, for a full discussion).

and in this way new trait variation can be introduced.[6] Monod (1972) famously described evolution as a process of chance and necessity. Mutation is the main aspect of evolution that is reliant upon chance. Once a beneficial mutation is produced, that gives some competitive advantage in the struggle for life, selection will 'take hold' of it and this blind process will take that trait to fixation within a population.

During the 1960s, much theoretical work made it clear that selection at the level of the gene best explained the main effects of evolution. Genes could be conceptualised as functionally eternal, replicating themselves through the generations just so long as natural selection permitted it. In this way, an adaptation was defined as any trait that causes its underlying genes to increase in relative frequency within the gene pool. Dawkins (1976) captured the essential logic at work in his book *The Selfish Gene*.[7] Genes were to be seen as self-interested, seeking their replication through the vehicles that carried them. If this replication is best served by building traits that make the organism bearing them selfish, then selection will see to it. Equally, if it is beneficial to build organisms that cooperate to attain resources, then genes for cooperation will be selected for. Nothing in selfish gene theory implies selfish behaviour in the organisms carrying those genes.

This theoretical innovation had fruitful consequences. First, it has been possible to separate biochemical accounts of molecular genetics from functional discussions where it is useful to talk of *genes for* a particular trait (Haig, 2006). Second, it allows genes to be seen as strategists, and their strategies, their traits, account for the changes in gene frequencies over time. In the final analysis, a complete biological science will provide the full biochemistry but evolutionary biology can productively move forward without this knowledge. These consequences were mathematically realised in the form of evolutionary game theory (Maynard-Smith, 1982). Traits, as competing strategies, could be modelled to see which would come to be stable over time, such that they could not be shifted by the arrival of a new mutant strategy, even if other strategies could survive in the population (so-called evolutionarily stable strategies; see Box 1.1).

The biochemistry of genes does have some theoretical impact. Genes are best understood as functionally described portions of DNA. Their function is to build polypeptide chains, that later become enzymes or proteins, or to control the expression of other genes. During protein synthesis, DNA unravels, the code is transcribed and translated through the action of various types of RNA, leading to the formation of a polypeptide chain (see Box 1.2), which is folded into a protein. In the now classical formulation, information is regarded as flowing in one direction only, from DNA to protein. There is no information flowing back from the protein, or any other intermediate stage in the process, to the DNA. In other words, DNA is unaffected by

[6] Most mutations are neutral in effect; the next most likely outcome is that the mutation is deleterious to the organism, and the least likely outcome is that the mutation is beneficial.

[7] Dawkins' book effectively summarises the canonical work of George C. Williams, William H. Hamilton, John Maynard Smith and Robert L. Trivers, as he openly acknowledges.

BOX 1.1. EVOLUTION AND GAMES

The Prisoner's Dilemma is one of the best-known games used in evolutionary theory. In its original incarnation, the game is set in a prison. Two prisoners are planning an escape, but the authorities become suspicious and decide to interrogate both prisoners separately. The authorities have no firm evidence, so they suggest a deal to each of the prisoners – if they reveal the escape plot, the authorities will treat them well and even reduce their sentence. However, if both prisoners stay quiet, their plot will be safe and they can potentially escape, even though the act of escape may carry risks. So, each prisoner has to decide whether to stay quiet and cooperate with the other, keeping their plan a secret, or to cheat on the other by revealing the plot. They have to weigh the costs and benefits. If one decides to remain quiet whilst the other talks to the authorities, they will be a sucker and get nothing for their efforts. If both decide to reveal the plot, then the authorities will punish them but not too heavily as the regulations reward realisation of wrongdoing even at this late stage.

The Prisoner's Dilemma is a game about cooperation, and the simple version above is about cooperation in a one-shot game where each player has only one strategic move that they can make. Cooperation is a trait seen in many species, and evolutionary biologists have been keen to understand how such behaviour can become a stable strategy within a population. Your intuitions, whilst reading the preceding paragraph, probably told you that the prisoner should defect and reveal the plot. Even if they both keep quiet, there are still risks associated with trying to escape, but a confession will secure either earlier release or at least minimal punishment.

Axelrod and Hamilton (1981) used the Prisoner's Dilemma to reveal the underlying game-theoretic dynamics in natural cooperation. Table 1.1 is a version of the original pay-off matrices used in their key paper. It represents the pay-offs for two possible one-shot game scenarios. In other words, each player has only one move to make, either cooperate or defect, and the matrix maps the consequences. The values in the matrix can be thought of as the fitness gain in each interaction for player A. Fitness could be measured directly in terms of offspring, or indirectly in terms of useful resources that will later enhance fitness.

Table 1.1. The Prisoner's Dilemma Game.

		Player B	
		Scenario 1	***Scenario 2***
		Cooperate	Defect
Player A	Cooperate	R = 3 Reward for mutual cooperation	S = 0 Sucker's pay-off
	Defect	T = 5 Temptation to defect	P = 1 Punishment for mutual defection

The pay-off to player A is shown with example values.

Source: Based on Axelrod and Hamilton (1981).

Scenario 1
If player A encounters B, who always cooperates and A cooperates too, then A increases her fitness by 3. But if A defects – that is cheats B – she increases her fitness by 5; if B always cooperates, it therefore is in A's interests to defect.

Scenario 2
If player B always defects and A cooperates, A's fitness is unchanged. If A defects, she increases her fitness by 1; if B defects, it benefits A to defect also.

The overall conclusion is that, whatever the other player's choice, it pays A to defect. This means that cooperation is not what is termed an Evolutionarily Stable Strategy (ESS). If there were a population of cooperators, a mutant defector would soon spread. Furthermore, this strategy would be the best response to itself and any other strategic move and therefore unbeatable. Defect strategies are an ESS, as a mutant cooperator will not spread. The values used are exemplars. However, using the notation in the matrix above (Table 1.1), the expression for the requisite conditions to maintain this ESS is:

$$T > R > P > S \text{ and } R > (S + T)/2$$

If the two individuals play only once, a one-shot game, then defection is always the best response. Defection is also the best policy if the number of interactions is known in advance. Once the last move is reached, an individual should defect (think of that last move as a one-shot) and this means defect will also be best on the penultimate move and so on all the way back to the very first. But, if the series of encounters goes on with no end in sight, or there is a possibility, however small (w), that the individuals will encounter one another again, then more complex encounters and strategies can emerge.

Axelrod (1984) tested this by running a computer contest in which various scientists played their strategies against one another, against themselves (unwittingly) and against random defector/cooperator strategies (with equal probability of cooperation or defection). The pay-offs were as in Table 1.1 and were iterated between pairs of contestants with w = 0.99654.

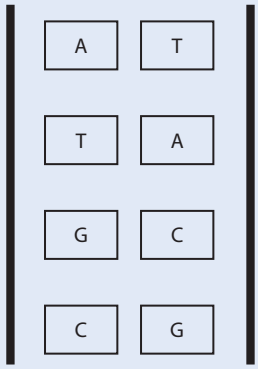
The winning strategy was the tit-for-tat strategy. This cooperated on its first move and thereafter did whatever its opponent did on the previous move. This means that tit-for-tat becomes a strategy of cooperation based on reciprocity. The strategy succeeded because it was punishing, and this discouraged defection, and it was forgiving after one punishment, which restored cooperation.

Tit-for-tat was an ESS provided w was large (Axelrod and Hamilton, 1981). It did not allow a mutant defector to take advantage. However, if an always-defect strategy emerged first this would win out, whatever the value of w, as a mutant tit-for-tat strategy would quickly get forced to the suckers pay-off where it would not get future compensation. So how could tit-for-tat ever get started as an ESS? This is still an open research question.

Whatever the answer to this question, it is the case that this strategy exists in natural populations. Wilkinson (1984) studied vampire bats (*Desmodus rotundus*) in Costa Rica that reciprocally feed one another. Individuals who did not secure a blood meal at night would beg for food from other bats and this situation could be reversed later, such that the benefactors later became altruists. Bats lose weight markedly with increasing time since their last meal. If a fed bat gave up just a small percentage of its meal to an unfed bat, this would benefit the unfed bat enormously in terms of staving off weight loss at very little cost to the fed bat. His cost-benefit asymmetry is essential to establishing tit-for-tat. Regurgitation occurred between close relatives but also between unrelated individuals who shared a roost. What is more, these bats were companions for many years, giving the opportunity for reciprocation. It could be that the bats are acting as if their neighbours are kin (Dunbar and Shultz, 2007).

BOX 1.2. DNA

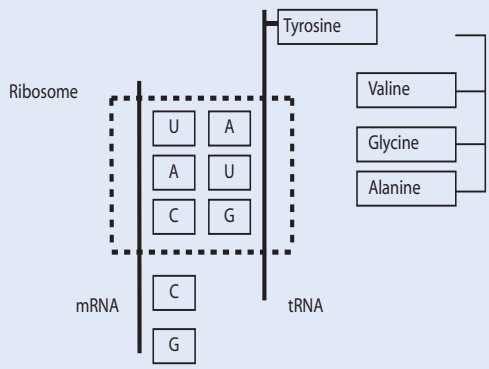
DNA, or deoxyribonucleic acid, is the molecule that forms our genetic material. DNA is found in the nucleus of the cell and has a double helix structure – two strands of sugar and phosphoric acid wind about each other and held apart by pairs of four bases: Adenine (A), Thymine (T), Guanine (G), and Cytosine (C). The structural properties of these bases mean that A always pairs with T and G always with C, as follows:



The base pair structuring allows DNA to replicate during cell division. The double helix unwinds into two strands, separating the base pairs and each strand then attracts the appropriate bases to construct its complement. Two complete helices are formed.

DNA encodes the various sequences of 20 amino acids that make up the many specific enzymes and proteins. RNA (ribonucleic acid) is also involved in this process. The message is decoded in two steps – *transcription* and *translation*. Information only goes from the DNA to the phenotype; it does not run the other way. This is the central dogma.

During transcription the sequence of base pairs on one strand of the double helix is copied to mRNA (messenger RNA). mRNA is single stranded and is formed in a similar fashion to DNA replication except that uracil substitutes for thymine such that A pairs with U. mRNA then leaves the cell nucleus, enters the cytoplasm and connects with the ribosomes, which are the location of protein formation. The next stage is translation in which the mRNA is translated into amino acid sequences. tRNA (transfer RNA) transfers amino acids to the ribosomes; each tRNA is specific to one amino acid. tRNA, with the attached amino acid, pairs up with the mRNA in a sequence dictated by the base sequence of the mRNA as the ribosome moves along the mRNA strand. Each of the 20 amino acids found in proteins is specified by a codon made up from three sequential mRNA bases. So, the mRNA triplet code GUA attracts tRNA with the complementary code CAU, and this tRNA transfers its attached amino acid, valine, which is then bonded to a growing polypeptide chain of amino acids. The next mRNA codon, say UAC, then attracts tRNA with the complementary codon AUG, which has the amino acid tyrosine attached and so on:



The sequence of amino acids determines the initial shape and function of the proteins.

the subsequent stages of the process. This is known as the 'central dogma of molecular biology' (Crick, 1970).

The central dogma effectively deals with a view often referred to as 'Lamarckian evolution': the idea of the inheritance of acquired characteristics as formulated by Jean-Baptiste Lamarck. If information only flows one way in order to synthesise a protein, in order to build a trait, then whatsoever happens to that trait later in terms of its development cannot affect the substrate of inheritance. So, the children of a weightlifter will not be born with bigger muscles as a consequence of their father's exercises. If he is successful at weightlifting, it is likely that he has genes that build muscles with a lot of developmental scope. His children may well inherit these genes, and if they exercise too they will resemble him in this trait, but they have only inherited the predisposition to develop muscle in a certain way.

Biological evolution relies upon genes, as we have seen, and the central dogma is a crucial aspect. For evolutionary change to occur, there must be new variation as a consequence of mutation and this variation needs to be selected. Other kinds of change can be seen over time, and one can even discuss other notions of inheritance. It could be argued that the children of the weightlifter have inherited his gym and this has led to their developing their muscles. Their children, too, inherit the gym and so on down the generations such that the characteristic of large muscles appears stable within the population. If the generations of muscle-building people are in some way favoured, perhaps because having big muscles allows more resources to be accessed through farming, then this family's descendants will increase in relative frequency. If this ability is absolutely crucial, then they could come to dominate the population.

Dickins and Dickins (2008) discuss this kind of change and draw a distinction between intrinsic and extrinsic inheritance. The children extrinsically inherit gyms, for gyms are not part of the children. Gene variants that code for relatively more muscle development in response to exercise are intrinsically inherited, for they are a part of the children. Indeed, the children are just the latest in a long line of vehicles that these particular replicators have been using. If the children inherited the gym but not the gene variants for muscle development, they would not thrive in the manner described. If the children inherited the gene variants but not the gym, they equally might not thrive, but they would have the potential to should circumstances change. Thus, there is a fundamental asymmetry at work here, which is captured by the central dogma. Extrinsic inheritance cannot alter the intrinsically inherited facts. There is no potential for true biological evolution in such systems.

The division between intrinsic and extrinsic inheritance is the key to evolutionary biology and, as we shall see, has implications for the behavioural sciences. Strict adherents to the central dogma of molecular genetics will see anything outside the genes, so any gene product or gene expression modifier, as extrinsic. The strategies that genes create are the cause of their selection, and genes effectively live or die by their strategies; but the strategies never cause the genes to change thereby altering the strategy for subsequent generations.

Plasticity and evolution

The preceding discussion may lead one to assume that evolutionary biologists believe that genomes contain all the responses to the ecology[8] that an organism will ever need. In some sense they do, as discussed in the opening comments of this chapter, but such a statement requires a qualification with regard to the granularity of the argument.

Genomes provide a capability to deal with a specific ecology, but that can mean anything from the production of rigid and fixed responses to the ability to learn. In 2001, when the first human genome was published, scientists were surprised that it consisted of between 20 and 25 000 functional genes (Venter *et al.*, 2001), not least because simpler organisms have comparable, if not larger, genomes. For example, the nematode worm (*Caenorhabditis elegans*) has a genome of 20 000 functional genes (*C. elegans* Sequencing Consortium, 1998) organised on six chromosomes. However, simpler organisms are arguably defined as simpler by dint of the restrictions upon their ecological niche. Nematode worms live in the soil in temperate environments and feed on bacteria found in decaying vegetation. They do far less, and exploit a much narrower band of resources. To this end, natural selection has built all (or many) of the solutions to the relevant ecological problems into the genome. By this definition of complexity, more complex organisms have greater ecological bandwidth as well as greater day-to-day variation in ecological change. The amount of information involved in broad bandwidth ecologies is large and would make it difficult for natural selection to incorporate into a genome whilst still maintaining coherent development. Indeed, for the most complex of organisms, those often termed 'generalists', such as rats and humans, this may be computationally intractable at the level of the genome. However, the solution has been to build mechanisms that essentially store information outside of the genome and permit learning. These include epigenetic mechanisms and nervous tissue, especially brains.

Psychologists are well aware that brains store many, many more bits of information than the human genome (Pinker, 1997). We will return to the proximate detail of psychology later in the chapter, but for now we should note that learning mechanisms, which take account of specific aspects of ecology, have been selected for. This allows for great flexibility and the overall generalist strategy that typifies humans, accounting for their wide dispersal across the planet.

Epigenetic mechanisms are increasingly drawing attention in evolutionary biology (e.g. Jablonka and Lamb, 2005). Epigenetics, as a discipline, is primarily concerned with the development of organisms. In this way, epigenetics contributes to discussions and theories about how final adult form is produced. It is not specifically an evolutionary

[8] The term 'environment' refers to everything that is outside of something else, what that something is surrounded by. The term 'ecology' refers to that aspect of the environment something is adapted to use. So, the human eye has been selected to deal with a portion of the electromagnetic spectrum and that portion defines its visual ecology. Birds use other portions of the same spectrum and thus inhabit different visual ecologies. The electromagnetic spectrum is part of a shared environment for avian species and humans. Therefore, the term 'ecology' is an evolutionary one, whereas 'environment' is not. Behavioural scientists who discuss environmental effects are most likely not evolutionarily minded.

theory, but rather the study of particular proximate mechanisms involved in development. However, a number of researchers think that epigenetic processes have implications for our understanding of evolution.

Epigenetics literally means 'above genetics', implying that there is something operating above the standard genetic inheritance that has been discussed so far in this chapter. More crucially, researchers in this field often refer to the notion of epigenetic inheritance, which, on the surface, can appear to be a Lamarckian process. The core idea is that the expression of genes can be affected by extrinsic factors and that sometimes these effects can be passed along generations without any changes to the DNA sequence that constitutes the genes in question. For example, Jablonka and Lamb (2005) note that liver cells, kidney cells, skin cells and so on all have identical DNA but, all things being equal, they continue to produce daughter cells of the same kind, so skin cells beget skin cells in spite of the potential in the DNA to do otherwise. Jablonka and Lamb (2005) see the extrinsic factors affecting gene expression as epigenetic inheritance systems. These include systems that alter RNA functions, and therefore transcription, systems that alter the chromatin packaging of DNA and systems that attach methyl groups to cytosine.

To the behavioural scientist, these epigenetic inheritance systems could appear to be of distant relevance to their topic. Crews (2008) refers to these concerns as 'molecular epigenetics', which he contrasts with molar epigenetics which emerged from the move to functionalism within psychology. Specifically, Crews discusses how comparative psychology became focused upon the interaction of genetic endowments and other biological systems with the environment, and how these interactions led to species-typical behaviours developing from conception to death. In so doing, Crews is also referring to the origin of epigenetics in debates about preformation, which began with Aristotle (see Depew and Weber, 1996).

Molar epigenetics, of course, relies on molecular effects at some point. Where functional psychologists are interested in particular environmental (and therefore ecological) effects upon development, they must assume that the environmental aspect of concern is, to all intents and purposes, acting as an input for the organism and affecting development. As can be seen from work on the effects of maternal care upon offspring stress response (see Box 1.3), it would seem that epigenetic mechanisms allow organisms to track key, yet changeable, aspects of the ecology across generations without irreversibly altering DNA. In times of risk, as in the maternal care example, increasing the sensitivity of stress response in one's offspring, as well as oneself, is useful. In this way, the epigenetic mechanisms are processing information and solving problems for the organism and, therefore, can be seen as candidate adaptations.

As extrinsic factors can lead, through epigenetic inheritance, to the introduction of different variants of form, and these variants can then be carried across generations at some increased frequency, it looks like an evolutionary change. But, by the definitions given above, this is not so. Given that there is no change to the DNA, the central dogma of molecular genetics remains intact. Gene-level selection has not been challenged and, as said, we can think of epigenetic mechanisms as possible adaptations and therefore as selected precisely to track specific kinds of ecological change

BOX 1.3. MATERNAL CARE AND EPIGENETICS

Champagne (2008) has reviewed the literature on maternal care and epigenetic inheritance. Much work has been conducted in rodent models, investigating the role of licking and grooming behaviour (henceforth grooming) in the development of pups. In mice, when pups are prevented from attaining the normal amount of interactions with their mothers it has been shown that those pups go on to nurse their own pups less and engage in less grooming behaviour toward them. In short, the maternal strategy is inherited. In Long-Evans rats the amount of grooming engaged in by mothers varies between individuals but is a stable individual trait; it is also inherited by their offspring such that daughters of low-frequency grooming mothers will in turn be low frequency grooming mothers to their offspring. Fostering studies indicate that this inheritance is not genetic but rather modified by behaviour. This would appear to be a candidate for molar epigenetic explanation and Champagne's overview is worth examining in detail:

A number of nuclei in the brain have been associated with the activation of maternal care but the medial preoptic area in the hypothalamus appears to be crucial. Female rats that are low on grooming and are lactating have relatively low oxytocin receptor binding in the medial preoptic area in contrast to high grooming females. Oxytocin is a neurohormone implicated in reducing social inhibition and facilitating affiliative and kin bonding. Champagne notes that experimentally introducing an oxytocin receptor binding antagonist reduces grooming in previously high grooming females.

There is a role for mesolimbic dopamine in grooming. Dopamine increases evenly prior to the onset of grooming in high grooming females. This is released in the nucleus acumbens. The amount of dopamine released directly predicts how long the mother will engage in grooming, and upon cessation the dopamine level returns to baseline. In those mothers who are low in grooming there is no substantial increase in dopamine activity and the behaviour is short-lived. The current hypothesis is that the hypothalamic oxytocin neurons mediate this mesolimbic response and cause the stable individual differences between mothers in terms of maternal care.

Grooming will affect stress response in pups. The hypothalamic-pituitary-adrenal axis (HPA-axis) delivers what is sometimes referred to as a passive stress response. Corticotropin-releasing factor (CRF) is synthesised, stored and released by neurons in the hypothalamus – specifically at the paraventricular nucleus (PVN). Neurons elsewhere in the brain, which respond to fear stimuli, stimulate these hypothalamic neurons to secrete CRF into a local blood vessel. This then travels a short distance to the pituitary gland, which contains CRF receptors, and this in turn stimulates the secretion of adrenocorticotrophic hormone (ACTH). ACTH is a classical hormone and is distributed throughout the entire blood stream, targeting the distant adrenal gland where ACTH stimulates a class of hormones known as 'corticosteroids'.

Corticosteroids are released from the outer layer of the adrenal gland. Corticosteroids are involved in responses to threat and help to mobilise energy by releasing stored glucose. More precisely they break down proteins (from muscles and other tissues) into amino acids that are then used in the liver to synthesise glucose at critical times. This energy is directed to key sites. Corticosteroids also cause the release of fatty acids, or lipids, which are another source of energy.

The release of corticosteroids from the adrenal gland sends an inhibitory signal back to the hippocampus, which in turn inhibits PVN activity. This negative feedback loop, when functioning properly, sees to it that just enough corticosteroid action is produced to remove the stressor or remove the organism from the stressful situation. If this malfunctions it can lead to a prolonged stress response in the absence of any stressor as well as neurotoxic effects.

Champagne (2008) notes that pups of mothers who are low in grooming have a particular HPA response. They maintain heightened adrenocorticotropin and corticosterone levels after exposure to stressors. These pups also have a reduction in hippocampal glucocorticoid receptor mRNA and an increase in hypothalamic CRF mRNA thereby making the maintenance of baseline corticosteroids problematic once a stressor is removed. Thus the stress response is prolonged and there is the potential for neurotoxic effects. Those pups that receive less grooming have relatively elevated corticosteroid levels after exposure to a stressor. It is known that this has behavioural effects such as a reduction in exploratory behaviour and an increase in inhibition. In short, it leads to behavioural depression.

The individual differences in hypothalamic activity associated with high and low grooming are found in the pups of high and low mothers such that the pups of low grooming mothers have relatively low oxytocin receptor binding in the postpartum period. The pups of low grooming mothers also display low oestrogen sensitivity. Oestrogen normally increases the expression of the oxytocin receptor gene, and at parturition pups are normally exposed to high oestrogen, which in turn should lead to increased oxytocin receptor binding. The insensitivity in the pups of low grooming mothers is like that found in mice that lack a functioning copy of the oestrogen receptor alpha, which is involved in affecting gene expression, thus causing a reduction in the ability of oestrogen affect upon that expression. In the offspring of low grooming mothers the expression of oestrogen receptor alpha is reduced relative to that of high grooming mothers, which potentially will affect the oxytocin activity in the medial preoptic area. What is more, fostering experiments have revealed that this alteration to gene expression is affected by postnatal maternal care – low-grooming mothers cause a reduction in the level of oestrogen receptor in the medial preoptic area of the hypothalamus. This has been linked to high levels of methylation at a number of sites in the promoter region of oestrogen receptor alpha.

Stressed mothers expose their *in utero* offspring to higher levels of glucocorticoids and the offspring in turn have increased levels of plasma corticosterone and CRF mRNA in the amygdala. This leads to behavioural depression as noted above. Normally high-grooming mothers that are experimentally stressed in the final week of pregnancy produce low-grooming behaviours of the sort associated with a reduction in hypothalamic oxytocin action. The relationship between stress exposure and low grooming remains unknown but the low grooming will induce the effects discussed.

In summary: Low maternal care affects the expression of a gene that is crucial to the development of the neurobiology of maternal care. In so doing, low maternal care is inherited. The affect of low maternal care is to alter the passive stress response in offspring and through the epigenetic inheritance of maternal care this response can also be passed along a number of generations. Methylation is not irreversible and there is evidence that postnatal behaviours, such as social enrichment, can switch the low maternal care lineage back to high maternal care by attenuating the HPA response. In this way, the organism can react to the environment and prepare its offspring for a similar future but not irreversibly so. This is a form of transgenerational learning, not an evolutionary change.

(Dickins and Dickins, 2007). Indeed, epigenetic mechanisms can be compared with brains as they are effectively designed to learn about specific aspects of the ecology and to respond to them. The flexibility of epigenetic learning relative to neural learning is unknown, and epigenetic mechanisms clearly affect brain development. To this end, epigenetic mechanisms can perhaps be referred to as a 'pre-brain'.

FITNESS, SOCIOBIOLOGY AND LIFE HISTORY THEORY

Fitness is a much used concept in evolutionary biology and it has at least five different senses (Dawkins, 1983). The sense of most relevance to this chapter is that of 'classical fitness', as Dawkins labels it, which originated within the discipline of ecology. Classical fitness (henceforth, fitness) is a property attributed to an individual organism and is 'often expressed as the product of survival and fecundity' (Dawkins, 1983, p. 183). Put crudely, the longer one survives and the more one reproduces, the fitter one is. Indeed, the empirical application of this notion is often referred to as 'baby-counting'. Fitness, then, captures the fundamental dynamic at the heart of Darwin's formulation of evolution through natural selection: traits only reach fixity through successful reproduction, and the more offspring an individual has, the more copies of its genes it gets into the gene pool. Dawkins (1983, p. 184) discusses the limitations of this concept and arrives at the following:

> Ideally we might count the relative number of descendants alive after some very large number of generations. But such an 'ideal' measure has the curious property that, if carried to its logical conclusion, it can take only two values; it is an all-or-none measure. If we look far enough into the future, either I shall have no descendants at all, or all persons alive will be my descendants . . . If I am descended from a particular individual male who lived a million years ago, it is virtually certain that you are descended from him too. The fitness of any particular long-dead individual, as measured in present day descendants, is either zero or total.

In spite of this caution, we can attribute fitness to an organism within a limited timeframe and in so doing use it to model the evolutionary success or failure of its behaviours. Moreover, we can discuss the strategies implemented by genes as more or less fit. Fitness has a central place in evolutionary behavioural science, as is evident from the early developments in sociobiology. Sociobiology was a term applied by Wilson (1975/2000) to an amalgamation of proximate biology, behavioural ecology, ethology and a number of other disciplines. Wilson had paid close attention to various theoretical innovations in evolutionary biology and noted the success of gene-level explanations. Wilson also understood that social behaviour was a fertile ground for the 'new' evolutionary explanations and in particular he focused upon the

problem of altruism. If genes were to be understood as self-interested replicators, then what could altruism have been selected for? Altruism is, by definition, an act that confers a benefit on another at some cost to oneself. In fitness terms, an individual altruistic act, understood in isolation, is bad news for the altruist and good news for the benefactor.

The initial explanations for altruism centred on kin selection. Given that relatives share some genetic variants[9] (or alleles) with you, an investment in a relative is a proportional investment in your own genes. The degree of relatedness is expressed as *r*. A full sibling has an *r* of 0.5, as they share 50% of their gene variants with you; a nephew or niece has an *r* of 0.25. It is the same for descendant kin. A child shares 50% of its gene variants with its parent, and a grandparent shares 25% of its gene variants with its grandchild. As genetic relatedness decreases, it might be predicted that the willingness to invest and the amount invested would also decrease. Haldane famously quipped that he would jump into a river to save two brothers or eight cousins, thereby capturing the logic of this argument. However, the real issue is when altruistic and, therefore, costly behaviour would emerge and stabilise within a population; that is, under what conditions would altruism benefit fitness? It was Hamilton (1964) who brought the mathematical precision of game theory to the notion, expressing the relationship between costs, benefits and relatedness as follows:

$$C_a < r_{ab} B_b$$

where *C* is the cost, *B* the benefit, *r* relatedness, *a* the donor and *b* the recipient. So, the cost to individual *a* has to be less than the product of the relatedness of *a* to individual *b* and the value of the benefit. So, if I am in the business of benefiting my brother at a cost of one unit (say £1) to me then the benefit must be worth more than two units (say £2.10) to him. For example:

$$C\,(\text{£1 from Tom}) < r(0.5)B(\text{£2.10 to Ben})$$

What this means is that altruistic behaviour will stabilise in a population whenever the cost of the act is offset by the indirect fitness benefits of assisting a relative. If this is not the case, then selection will not permit such altruism. Hamilton's rule captures the parameters of this relationship.

Kin selection is not a complete account of altruistic behaviour, for we see altruism between non-relatives. Hamilton and colleagues applied game theory here too, most

[9] My father, David Dickins, taught me a valuable pedagogical trick when I was younger. If you ask a group of students how much genetic commonality they have with chimps, they usually report a figure between 95 and 98%. Then, when you ask them how much genetic commonality they have with their brother or sister, they unflinchingly state that it is 50%. So, you conclude aloud, you have more in common with a chimp than your own sibling. This focuses the subsequent discussion. The students are conflating two uses of genetic commonality. On the one hand, we do have about 95% of the same *kinds* of genes as chimps, whilst on the other hand we have 50% of the exact same *variants* of genes as our siblings. This is a type-token distinction.

notably with the Prisoner's Dilemma (see Box 1.2 for details). As we have seen with this game, one-shot interactions favour defection but iterated games with an uncertain number of interactions spread across time allow for the emergence of a tit-for-tat strategy. This notion of incurring a cost for another individual's benefit, and in the future attaining a benefit from them, is referred to as 'reciprocal altruism' (Trivers, 1971).

As the vampire bat example demonstrates, reciprocal altruism is in essence a form of trade and it flourishes when each individual gains more in benefits than it cost them to be altruistic. Giving up a small portion of a blood meal to another is low cost to the donor but extremely significant to the future fitness of the benefactor. Given this asymmetry, it means that the future fitness benefits for the initial donor, when that individual is in need of a donation from the initial benefactor, far outweigh the costs first accrued. To this end, altruism will stabilise in the population, as it will be selected for. Ecologically, this is dependent upon a high degree of uncertainty over resource acquisition such that any bat could find itself unfed.

The problem of altruism firmly established evolutionary game theory as an essential tool for assessing the fitness of competing behavioural strategies. However, the fitness of successful strategies is understood in terms of the average pay-off over an extended period. This does not capture the dynamic nature of 'lived strategies' over the course of an organism's lifetime. This issue is addressed by life history theory, which has also emerged from the discipline of Ecology (Stearns, 1992).

The core assumption of life history theory is that organisms are fitness maximisers: they seek optimal fitness. Fitness maximisation is not a one-shot game. Organisms develop to adulthood, reproduce, grow old and die.[10] Across all of these stages, organisms have access to different kinds and differing levels of resources, owing to changes in local ecological conditions, which they can invest in their fitness and decisions have to be made about what, when and how much to invest. These decisions are the outcome of naturally selected strategies.

Strategically, organisms can either attempt to delay mortality, and thereby buy more time for reproduction, or alter the onset of fertility, where 'fertility' refers to the amount of reproduction rather than the biological capacity to reproduce. Given this, fitness maximisation is changeable across lifespan and can be characterised by a set of energetic trade-offs (Kaplan and Gangestad, 2005). These are current versus future reproduction, quantity versus the quality of offspring and mating versus parenting effort.

Work on clutch size in bird populations provides a good example of such trade-offs. Each egg represents a possible chick and therefore a possible reproducing adult. In this way, an egg is an investment in future fitness for the adult pair that produces it. Each egg also represents a number of costs: they are energetically, and hence

[10] Life history theory explains why organisms senesce and therefore die (Kirkwood, 1997). Put briefly, in order to maintain a body the organism has to invest energy. If this investment led to perfect maintenance, then any extra energetic investment would not alter maintenance and would be wasted. At this point it would make sense to redirect that investment to reproduction, thereby increasing fitness. Given this, it is in an organism's interests to let the body decay and allocate resources to reproduction instead. This is referred to as the 'disposable soma theory'.

nutritionally, expensive to produce; any chicks represent further energetic investment in terms of feeding, which has to be balanced against effort to feed oneself; the more eggs and chicks one has, the more such resources have to be divided. Resources are finite and differ between ecologies such that one would expect to see different decisions reached in different circumstances.

David Lack (1947) was perhaps the first to discuss the clutch size issue for birds in these terms. His initial hypothesis was that the number of chicks that survived would be in a linear and inverse relationship to the number of eggs in a clutch. So, as clutch size went up, chick survivorship went down. This would be a consequence of the finite nature of resources and the division across mouths.

Casual observation of wild birds would indicate that Lack's hypothesis was correct, and indeed a number of clutch size manipulation experiments have appeared to support him (Stearns, 1992). However, it appears that there are complexities that Lack overlooked. Boyce and Perrins (1987) reported on a 22-year study of 4489 clutches of great tits (*Parus major*) in an Oxfordshire woodland. They found that the average clutch size was 8.53 eggs, but the clutch size with the highest rate of chick survivability was 12. Furthermore, the results of clutch size manipulation experiments, where clutch sizes were increased by three, again showed 12 to yield the best survivorship. It would appear that the great tits could have laid more eggs, and this violates Lack's initial hypothesis.

Boyce and Perrins (1987) have revealed some assumptions at the heart of Lack's hypothesis. First, great tits have a lifespan of three years on average (though the oldest recorded, according to the British Trust for Ornithology, was almost 14 years), begin reproducing at one year and can potentially produce more than one brood in a season. So, great tits can produce somewhere between two and four clutches before death, and if predation and other risks are reduced, many more. This means that any investment in a current brood needs to be traded off against future investments. A current versus future trade-off is likely to alter the outcome of Lack's initial hypothesis and will lead to clutch size varying according to the resource richness of the ecology, the ability of the adults to capture these resources and their ability to begin a new brood in short order after the first has fledged. There is also the issue of the quality of the offspring. It is possible that, given resources, producing fewer eggs and fewer chicks will improve their quality and that they will in turn be more likely to successfully reproduce. So, there may be a quantity/quality trade-off to consider. Indeed, the initial measure of chick survivorship may not be the best measure of fitness, but perhaps the reproductive success of the parents' chicks is.

As these avian examples show, life history theory sees an organism's task as that of capturing energy and using it to maintain itself and to reproduce (Kaplan and Gangestad, 2005). This job leads to one of the key trade-offs, that between current and future reproduction, with only three possible outcomes: no current reproduction, some energy allocation in the present and some in the future, and total reproduction in the present followed by death. All of these outcomes are seen in natural populations.

A strategy of no current reproduction is rare and is generally a consequence of resource stress. In anorexic girls, poor nutrition will lead to cessation of menstruation

whilst the body directs what resources there are to maintain life (see Chan and Mantzoros, 2005); there are clearly mechanisms for switching fertility off. It is also worth noting that in economically stressed populations, such as those in Addis Ababa, Ethiopia, the fertility rate is declining and is now below replacement fertility (1.9[11]; Gurmu and Mace, 2008).

Pacific salmon (*Oncorhynchus spp.*) spawn in their natal river, with females producing thousands of eggs in a number of nest sites, which are then fertilised by the males. During their long journey from the ocean back to these freshwater rivers, the salmon deplete their fat stores and general somatic capital dramatically, with many adults not surviving the journey. Spawning marks the end of the adult life cycle and within a couple of days the adults die. This is a case of total current fertility.

Allocating energy across current and future reproduction is the typical pattern of fertility in humans, and many other species, including great tits. This strategy is not without variation between individuals, and a large part of this variation is the result of facultative or conditional responses to ecological conditions. The literature on teenage pregnancy provides an excellent case study of such effects.

Teenage pregnancy is generally defined as pregnancy under the age of 18 years. The United Kingdom has the highest rate of teenage pregnancy in Western Europe. In England, in 2007, 41.7 in every one thousand girls aged between 15 and 17 years had a conception and 50.6% of those conceptions were terminated.[12] Girls from low socioeconomic status backgrounds are at least four times more likely to have a teenage conception than girls from wealthier backgrounds, and poorer girls are far less likely to abort should they become pregnant (Lee *et al.*, 2004). Given that socioeconomic status is a key indicator of ecological conditions, the fact that there are distinct differences across these groups indicates a possible adapted fertility strategy. It is possible that the ecological conditions faced by these women are prompting them to favour current reproduction. To this end, we may wish to think about teenage pregnancy as the extreme end of a general early fertility strategy.

There are a number of theories about what it is that prompts females to early fertility. These theories assume that particular aspects of the environment inform developing females about a likely future. If these cues lead to the prediction that there is an uncertain or poor trajectory, development is hastened and these females enter into a fast life history. This will include growing smaller bodies, diverting resources to reproductive physiology and beginning sexual behaviours sooner (Hawkes, 1994; Hill, 1993). Such cues would appear to induce development.

A candidate developmental inducement is father absence. Belsky, Steinberg and Draper (1991) propose that attachment is related to judgements about possible futures. Specifically, they see puberty as the beginning of independence for an individual: they are readying themselves to be a sexual adult. According to Belsky *et al.*, any perception of risk in the current childhood environment is more likely

[11] A fertility rate of 1.9 means that every 100 mothers produce 190 children; replacement fertility is usually agreed to be 2.2.

[12] Statistics taken from the Teenage Pregnancy Unit; available online at http://www.everychildmatters.gov.uk/teenagepregnancy/.

to cause earlier puberty. So, what they found is daughters raised in a single-parent, father-absent family are more likely to reach menarche sooner than those raised by both a mother and father are. The reasoning behind this is that father absence suggests a lack of resources, such that an optimal decision would be to reproduce sooner rather than later whilst still healthy, and so on. Risks other than father absence will also lead to this effect.

Chisholm (1999) places this social and developmental psychological theory within the context of life history theory and interprets early puberty, as a consequence of exposure to risk, as a fitness maximising strategy. Various behavioural aspects of earlier sexual behaviour are clearly candidates for psychological decisions, albeit unconscious according to Belsky *et al.*, but menarche looks to be a physiological decision. Belsky *et al.* also note that risk is not necessarily the only factor. They point, for example, to clear heritable variation in onset of menarche, such that maternal age of menarche is a good predictor.

A number of studies (Cater and Coleman, 2006; Lee *et al.*, 2004) have also demonstrated conscious decision-making about fertility that indicates reasoning in terms of current versus future reproduction. It would seem that a number of teenage pregnancies are in fact planned and that many young women in high-risk, low-socioeconomic status positions realise that the best time for them to reproduce is now rather than later. In part, this is because they are young and as healthy as they are going to be, but it is also because they can call upon their own parents for support. All told, girls in poorer environments are more likely to allocate effort and resources to current reproduction (Dickins, 2006; Wilson and Daly, 1997). More recently, in an extension of the Belsky *et al.* argument, Nettle, Coall and Dickins (2010) have shown that lack of paternal involvement and low birth weight predict both intended and actual early fertility in a British population.

Life history theory enables scientists to make predictions about and explain population level patterns of behavioural and general trait variation. Understanding how organisms are likely to react to their ecology is clearly of enormous use and it helps to guide a subsequent set of questions about the proximate mechanisms that may deliver behaviour. Indeed, life history theory is fundamentally a black-box approach to the proximate mechanisms that lead to optimal fitness. There is no need to say or assume anything about these mechanisms when discussing the fitness benefits of particular behavioural strategies; but we can be assured that mechanisms do exist to deliver evolved strategic behaviour, and once we have evidence for maximisation this then becomes the focus of evolutionary psychology.

EVOLUTIONARY PSYCHOLOGY

In its simplest guise, evolutionary psychology is the application of evolutionary theory to psychology. Historically, evolutionary thinking led to the emergence of comparative psychology and many modern psychologists rely on animal models in

order to explain behaviour, often generalising their conclusions to humans. In recent years, however, evolutionary psychology has become strongly associated with a particular school of thought emerging from the laboratory of Leda Cosmides and John Tooby at the University of California at Santa Barbara. This school sought to provide proximate explanations of key findings from sociobiology at the cognitive level.

As we have seen, game theory has helped us to understand the selection parameters for cooperative altruism. Once cooperation emerges within a population, other strategies can co-exist including defection, or cheating. This does not mean that cheating can beat the strategy of cooperative altruism, for this is an evolutionarily stable strategy. But the existence of an evolutionarily stable strategy does not imply that there are no other strategies available, and it is possible to have more than one evolutionarily stable strategy within a population (for a discussion see Maynard-Smith, 1982).

Cheating can operate in a number of ways within a generally cooperative population. For example, sociopathy can be understood as an example of a cheating strategy embodied in a stable personality variable (Mealey, 1995; see also Nettle, 2006). Sociopathic individuals rely on cooperative first moves of individuals within the population and they defect. After a while, the reputation of a sociopath precedes them and they shift populations. In this way, sociopathy is a parasitic life history strategy and is also evolutionarily stable. Another and more common way in which cheating exists in populations is by happenstance. If we assume that individuals are interested in benefiting their own fitness, we can further assume that if they see an opportunity to defect and feel that they can get away with it, they will. Intuitively, you can predict that within large modern populations, such as our own, there are numerous occasions when we effectively have only one-shot interactions with people. If individuals encounter such an opportunity on the off-chance, then they are more likely to defect. Furthermore, such situations should be a rich hunting ground for confidence tricksters and will be subject to stringent laws, should the potential costs be significant.

I am reminded of an occasion whilst on holiday in France. My friend and I had travelled across to France from the United Kingdom in her car, which was a right-hand-drive vehicle. She was driving and I was in the passenger seat. As we entered a motorway, we approached the tollbooth, which was on my side. Fresh arrivals, we only had large denomination notes and I was sorting through them as we pulled up to the booth. The man in the booth said something fast in French and I saw ten francs come up on the display. I handed him a note and he said something else that I could not decode rapidly. At the same time, he hit the switch, the light went green, the barrier went up and my companion moved off – before I could receive any change. I only lost ten francs but I remember both of us being annoyed. The man had relied on my poor French, my unfamiliarity with the currency and my companion's focus upon the road to defect on what should be a cooperative strategy of maintaining France's highways. He also knew I would never see him again as I was clearly heading away from his neighbourhood. It was a perfect defection.

Our annoyance was not just at the loss of money but also at my gullibility. In discussion, it was felt that I should have made it clear I was expecting change and told my companion to wait until I received it. We both clearly felt that I should have been more sensitive to the possibility that we could be cheated in this situation; we both had a sense that a social contract had been violated.

Cosmides and Tooby argue that, given the possibility of being cheated in the manner just described, humans should have evolved psychological mechanisms that make them sensitive to situations where they may be cheated (Cosmides, 1989; Cosmides and Tooby, 1992). Put more colloquially, Cosmides and Tooby (1992) argue that humans should have psychological mechanisms for cheater detection. Such strategies would be selected for as they reduce costs and benefit fitness.

Cosmides and Tooby note that cooperation between individuals is a form of social contract, just as my companion and I did. Individual *a* agrees to do *x* for individual *b* on the understanding that at some point in the future *b* will do *y* for *a*. This is the logic of tit-for-tat: if you scratch my back, I'll scratch yours. This structure is that of a conditional rule. In order to be sensitive to cheating, one must be able to check that a conditional rule, a social contract, is being followed and not violated.

There is a large literature on people's ability to detect violations in conditional arguments and much of it centres upon the Wason Selection Task (WST). Wason (1966) developed a card selection task in order to see whether people will, when asked to check the veracity of a conditional rule, attempt to falsify or verify it. Wason's interest stemmed from Popperian notions about the demarcation of science from non-science and he was curious to see if falsification was a natural human ability. The basic task is presented in Figure 1.2a.

The WST consists of variants about a core problem that focuses upon four marked cards, only one side of which can be seen. In the original version, two of the cards had letters marking them, one vowel and one consonant, and two had numbers, one even and one odd. The task was to say which of the cards it was essential to turn over to check the rule 'If a card has a vowel on one side then it has an even number on the other'. The logical way to do this is to attempt to falsify the rule by turning over the vowel card and the odd-number card. If the vowel card has an odd number on the back, then the rule is falsified, if the odd-number card has a vowel on the back, the rule is again falsified. On average, less than 9% of people make the correct choice in this form of the WST (see Evans, Newstead and Byrne, 1993, for a discussion).

People seem poorly equipped for abstract conditional reasoning and much experimental effort has been spent trying to facilitate performance by making the problems less abstract. Cosmides (1989; Cosmides and Tooby, 1992) hypothesises that placing the WST within the context of a social contract should lead to significant facilitation, because human psychology should be biased towards this kind of reasoning, given

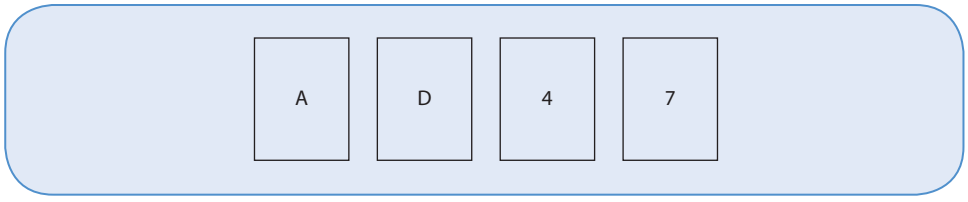


Figure 1.2a. *Wason Selection Task: Abstract Form.*

Task: Which of the cards is it essential to turn over to check the rule? If a card has a vowel on one side then it has an even number on the other.

Answer: A and 7.

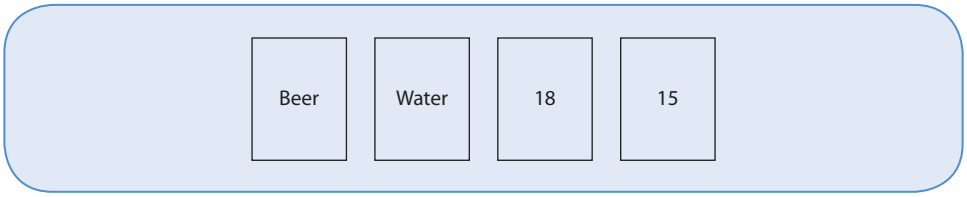


Figure 1.2b. *Wason Selection Task: Social Version.*

Task: You are a member of bar staff in a pub. Each of the cards above represents an individual customer in the bar. There is information about age on one side and chosen drink on another. Which of the cards is it essential to turn over to check the rule: if a person is drinking alcohol they must be 18 years or older.

Answer: Beer and 15.

Source: Based on Cosmides (1989).

the arguments above. To this end, a series of experiments was conducted in which social contracts were used (an example is given in Figure 1.2b) and, as predicted, performance was massively enhanced: on average people make the correct choice in the task and will uncover any potential defection. It would seem that this aspect of psychology is designed to take and process specific inputs in a specific way.

Game theory tells us of parameters for selection of particular traits. From this, we have been able to tell much about the nature of cooperation at the population level. The innovation of Cosmides and Tooby has been to realise that, for those strategies to stabilise, there must be specific psychology in place. In other words, they have sought to uncover the proximate psychological mechanisms that would prevent rampant cheating from threatening the cohesion of a cooperating group. Sociopaths move on because people eventually realise what they are doing and even if they are not punished they lose their opportunity to parasitise. Given the nature of our ecology, the uncertainty over quite how many interactions we are to have with others and uncertainty about the stability of resource acquisition our fitness is maximised through cooperation, on average. This effect has been replicated many times, across cultures, and there is some evidence for specific neurological sites of action (Stone *et al.*, 2002; Sugiyama, Tooby and Cosmides, 2002).

Cosmides and Tooby (1992) go further than simply demonstrating a social contract facilitation effect. They also use this set of experiments to make a number of theoretical claims about evolved psychology. First, they argue that the specificity of response in their experiments is evidence against the idea that cognition is a general processing system. Their work shows clear biases and clear obstacles to performance. Their experiments added support to the view, from cognitive psychology, that the mind is constituted by an undetermined number of domain-specific computational devices, or modules, that are sensitive to particular inputs and process them in specific ways (see Dickins, 2003, for a detailed discussion of this proposal; Samuels, 1998, 2000). What is more, Cosmides and Tooby (1992) argue that the modularity claim made sense from the perspective of natural selection. Selection resolves particular contingent problems for organisms at particular points in evolutionary time as those problems arise. This means that selection builds isolable and discrete traits

one by one. The mind will be structured in the same manner, with new psychological mechanisms, or modules, bolted on as and when selection pressures demand.

These latter arguments about cognitive organisation are secondary to the task of evolutionary behavioural science. Experimental manipulations of human participants, or subjects from other species for that matter, that can demonstrate the nuts and bolts of evolved conditional strategies are the main work of evolutionary psychology, and this area has thrived since the early work of Cosmides and Tooby, as the chapters in this book attest.

CONCLUSION

This chapter has not aimed to be exhaustive and has merely scratched the surface of evolutionary approaches to behaviour. What this chapter has done is to reinforce the purpose of applying evolutionary theory within the behavioural sciences, and that is to account for design. In so doing the basic mechanics of evolution have been described and the influence of gene-level thinking, notions of fitness and game theory have been shown at work in life history theory, the main theoretical framework of behavioural ecology and evolutionary psychology.

The kind of evolutionary psychology discussed can be seen as very narrow in its focus. Psychology, as a discipline, covers many areas such as social psychology, developmental psychology, health psychology, clinical psychology and so on. It is really only cognitive psychology that attempts to model the mind, and the proximate mechanisms that cause behaviour. The other subdisciplines are all behavioural, but also proximal, in their focus.

One of the key problems that the behavioural sciences face is the individuation of behaviours and subsequently of the mechanisms that cause them. The adoption of evolutionary theory resolves this issue. It will not inform scientists about the detail of mechanisms, but it will inform them about what the organism needs to achieve and, in so doing, will give information about the kinds of mechanisms the organism must have. To this end, the project of evolutionary psychology, as captured in this book, can be seen more broadly than in the previous section; indeed, it should be seen as an amalgamation of population-level modelling and proximate-level theorising and experimentation.

This broad approach sees organisms as calibrating to their ecology by capturing information. As said at the beginning of the chapter, those inputs that are informative are so simply because of the evolved design of the system using them. What genetics, epigenetics, life history theory and evolutionary psychology show us is that organisms are a hierarchy of calibrating, conditional mechanisms that afford facultative responding to key aspects of the environment at appropriate times. Systems that permit heritable change operate on a generational timescale and can help organisms to forecast the future (Bateson *et al.*, 2004); systems, such as proximate physiological and psychological responding, can afford day-to-day calibration, much as our robot can

avoid new obstacles when they are encountered and we are sensitive to new possibilities for defection. Natural selection has built the constraints to all these mechanisms and in so doing has captured facts about change and the parameters of change.

When discussing life history theory and early fertility, general population level distinctions were made using socioeconomic status as an index of ecological hardship and risk. The general argument was that females in harder, higher-risk ecologies would be more likely to opt for an early fertility strategy and this would be embodied within physiological development and behavioural decisions. The evolutionary reason for this is that early reproduction under these circumstances would maximise fitness as current risk is informative of future risk and, therefore, the accumulation of resources cannot be relied on. As this is a black-box approach, the nature of information processing was not discussed in very much detail, but this is clearly something that evolutionary behavioural science should address. How might we begin to do this?

First, the hypotheses of Belsky, Steinberg and Draper (1991) require further work. Is it the case that early menarche is indicative of early reproduction? It certainly affords the opportunity for early reproduction, but it is also possibly a side effect of other aspects of low socioeconomic ecologies. The recent work of Nettle *et al.* (2010) has begun to address this issue. Second, it is worth noting that it is not the case that the majority of girls in low socioeconomic status groups have teenage pregnancies; it is very much a minority. Given this, it would be fair to argue that teenage conceptions represent the extreme end of an early fertility strategy. What is it that is different about teenage girls in this minority? Psychology has a strong tradition of individual differences research,[13] and personality differences would be the place to start.

Nettle (2006) argues that the 'Big Five' personality traits of Extraversion, Neuroticism, Openness to Experience, Conscientiousness and Agreeableness capture heritable life history strategies. Central to his thesis is the idea that these strategies are held in the population through frequency-dependent selection. The earlier example of sociopathy is also a probable case of frequency-dependent selection (Mealey, 1995). As noted, sociopaths rely on the cooperative first moves of others and they then defect. One constraint on sociopaths is the number, or frequency, of sociopaths in the group. If the frequency of sociopaths is too high, there will be insufficient people to parasitise as too many will be waiting to defect and not cooperating. Because of this, sociopathy is capped within the population; it can never go to saturation, even though it is a candidate evolutionarily stable strategy. This is the essence of frequency-dependent selection. Nettle argues that the Big Five personality traits are held in the population in the same way and represent particular ecological niches.

Nettle's (2006) treatment of Neuroticism is pertinent to early fertility. According to Nettle, Neuroticism conveys particular advantages, including vigilance to dangers as well as increasing striving and competitive behaviours. There are, of course, costs, and these are typically stress and depression. Those girls who become teenage mothers may well be more sensitive to the risks in their ecology than others are,

[13] Research into individual differences has been a core component of psychology since Darwin, as scientists were keen to measure trait variation, because variation is a core component for natural selection, as we have seen.

and this may be what shifts them into very early fertility. Such sensitivities may well put females in low-socioeconomic environments ahead of the curve in terms of their fitness and one may predict a higher occurrence of Neuroticism within these populations more generally. What is more, there is an association in the literature between teenage pregnancy and depression (Azar *et al.*, 2007). This has been related to hypothalamic-pituitary-adrenal axis (HPA-axis) dysregulation, presenting the possibility for an endocrine explanation focusing upon cortisol function. Cortisol dysregulation is also associated with Neuroticism (McCleery and Goodwin, 2001; Oswald *et al.*, 2006). Such things will be genetically heritable, but there is every reason to investigate epigenetic possibilities too given the preceding discussion.

Cognitive psychological questions can also be investigated as we may predict differences in performance on reasoning about the future. This can be captured in terms of the consideration of future consequences (Strathman *et al.*, 1994), time preference and future discounting (Frederick, Loewenstein and O'Donoghue, 2002). There is also reason to look to genetic differences between early- and late-fertility females (Mendle *et al.*, 2009).

This discussion is merely presenting the reader with loose hypotheses. They will need much refining before a proper research programme can be undertaken and results collected. However, they serve to demonstrate how the evolutionary behavioural scientist can begin with population-level concerns, ask questions from a life history perspective and refine knowledge about the patterns of behaviour in and between groups. This work provides a basis for evolutionary psychologists with interests in proximate mechanisms to begin their investigations. Their enquiries are not too distant from those of other, non-evolutionary, psychologists, but they are informed by a theory that places the function and design of such mechanisms at its centre. If a psychological theory does not make sense in light of evolutionary biology then it is false.

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