PHILOSOPHY OF BIOLOGY

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Sociobiology and the Extension of Evolutionary Theory

Sociobiology is a research program that seeks to use evolutionary theory to account for significant social, psychological, and behavioral characteristics in various species. Understood in this way, sociobiology did not begin with the publication in 1975 of E. O. Wilson's controversial book, *Sociobiology: The New Synthesis.* The evolution of behavior has always been a subject matter for Darwinian theory to address.

What separates sociobiology from its predecessors is its use of the vocabulary of contemporary evolutionary theory. Wilson announced that the principal problem for sociobiology is the evolution of altruism. This focus, plus the reluctance of many (but not all) sociobiologists to indulge in group selection hypotheses, is distinctive. Sociobiology is not just a research program interested in the evolution of behavior; its characteristic outlook is adaptationist, with strong emphasis on the hypothesis of *individual* adaptation.

The initial furor that arose around Wilson's book mainly concerned his last chapter, in which he applied sociobiological ideas to human mind and culture. He was criticized for producing an ideological document and charged with misusing scientific ideas to justify the political *status quo.* Sociobiology also was criticized for being unfalsifiable; sociobiologists were accused of inventing just-so stories that were not and perhaps could not be rigorously tested (Allen *et al.* 1976).

Some of these criticisms don't merit separate treatment here. My views about the charge of unfalsifiability should be clear from Chapters 2 and 4. Sociobiology, like adaptationism, is a research program; research programs do not stand or fall with the success of any one specific model.

At the same time, it is quite true that some popular formulations of sociobiological ideas have drawn grand conclusions from very slender evidence. In Chapter 4, I emphasized the importance of carefully specifying the *proposition* that an adaptation-

ist explanation is intended to address. When a sociobiologist seeks to explain why human beings are xenophobic or aggressive or easy to indoctrinate (Wilson 1975, 1978), the first question should be: Which fact about behavior are we actually discussing? Is it the fact that human beings are *sometimes* xenophobic, that they *always* are, or that they display the trait in some circumstances but not in others? The first problem is fairly trivial, while the second is illusory; human beings are not *always* xenophobic. As is the case for adaptationist explanations generally, well-posed problems should not be too easy (Section 5.5).

Just as human sociobiology cannot be rejected on the grounds that some single sociobiological explanation is defective, the program cannot be vindicated by appealing to the simple fact that the human mind/brain is the product of evolution. What is undeniable is that theories of human behavior must be *consistent* with the facts of evolution; so, too, must they be consistent with the fact that the human body is made of matter. However, it does not follow from this that either evolutionary biology or physics can tell us anything interesting about human behavior. In Section 7.5, I will examine an idea that runs contrary to the sociobiological research program—that the human mind/brain, though a product of evolution, has given rise to behaviors that cannot be understood in purely evolutionary terms.

My own view is that there is no "magic bullet" that shows that sociobiology is and must remain bankrupt, nor any that shows that it must succeed. Any discussion of the adequacy of sociobiological models inevitably must take the models one by one and deal with details (Kitcher 1985). Obviously, a chapter in a small book like the present one does not offer space enough to carry out that task. In any event, I'm not going to try to develop any full-scale estimate of the promise of sociobiology. My interest lies in a few broad philosophical themes that have been important in the sociobiological debate.

7.1 Biological Determinism

Evolution by natural selection requires that phenotypic differences be heritable. For example, selection for running speed in a population of zebras will lead average running speed to increase only if faster-than-average parents tend to have faster-than-average offspring (Section 1.4). What could produce this correlation between parental and offspring phenotypes? The standard evolutionary assumption is that there are genetic differences among parents that account for differences in running speed. Because offspring inherit their genes from their parents, faster-than-average parents tend to have faster-than-average offspring.

This basic scenario remains unchanged when a sociobiologist seeks to explain some sophisticated behavioral characteristic by postulating that it is the result of evolution by natural selection. As mentioned earlier, Wilson (1975) suggested that human beings are xenophobic, easy to indoctrinate, and aggressive and that these behavioral traits evolved because there was selection for them. For this to be true, an ancestral population must be postulated in which there is variation for the phenotype in ques-

tion. Individuals must vary in their degree of xenophobia, and those who are more xenophobic must be fitter than those who are less so. In addition, the trait must be heritable. A gene (or gene complex) for xenophobia must be postulated.

Such explanations are often criticized on the ground that there is no evidence for the existence of genes "for" the behavior in question. Even if the evidential point is correct, whether one views it as a decisive objection depends on how much of the rest of evolutionary theory one is prepared to jettison as well. Fisher (1930) constructed his model of sex ratio evolution (Box 1.3) without any evidence that there are genes for sex ratio. The same holds true for virtually all phenotypic models of evolution. Parker's (1978) optimality model of dung fly copulation time (Section 5.5) did not provide any evidence that there is a gene for copulation time, but that did not stop many evolutionary biologists from taking it seriously. It isn't that discovering the genetic mechanism would be *irrelevant* to the explanation; rather, such a discovery does not appear to be *necessary*, strictly speaking, for the explanation to merit serious consideration.

Even so, it is worth considering what it means to talk about a "gene for xenophobia" and also to consider more generally what the genetic assumptions are to which sociobiology is committed. We may begin with an assessment due to Gould (1980b, p. 91):

> There is no gene "for" such unambiguous bits of morphology as your left kneecap or your fingernail. Bodies cannot be atomized into parts, each constructed by an individual gene. Hundreds of genes contribute to the building of most body parts and their action is channeled through a kaleidoscopic series of environmental influences: embryonic and postnatal, internal and external.

"Beanbag genetics" is a pejorative label for the idea that there is a one-to-one mapping between genes and phenotypes. Gould's point is that beanbag genetics is false. But sociobiologists, in spite of the fact that they often talk about a "gene for *X*," are not committed to beanbag genetics. They can happily agree that "hundreds of genes" contribute to the phenotypes they discuss.

What does it mean to say that a gene (or complex of genes) is "for" a given phenotype? A gene *for* phenotype *X* presumably is a gene that *causes* phenotype *X*. But what does this causal claim amount to? Dawkins (1982a, p. 12) offers the following proposal:

> If, then, it were true that the possession of a *Y* chromosome had a causal influence on, say, musical ability or fondness for knitting, what would that mean? It would mean that, in some specified population and in some specified environment, an observer in possession of information about an individual's sex would be able to make a statistically more accurate prediction as to the person's musical ability than an observer ignorant of the person's sex. The emphasis is on the word "statistically," and let us throw in an "other things being equal" for good measure. The observer might be provided with some additional information, say on the person's education or upbringing, which would lead him to revise, or even reverse, his prediction based on sex. If females are statistically more likely than males to enjoy knitting, this does not mean that all females enjoy knitting, nor even that a majority do.

Let us formulate Dawkins's idea more explicitly. If we wish to say whether being female (F) causes one to like knitting (K), we first must specify a population and an environment. So let us consider the population of human beings alive now, and let the environment be the range of environments that people currently inhabit. I assume that Dawkins does not insist that the individuals considered must live in exactly the same environment since this would make it impossible to advance causal claims about the real world. Given these specifications, I take it that Dawkins's proposal is that "F causes K" means that $P(K \mid F) > P(K)$; this inequality is equivalent to $P(K \mid F) > P(K \mid \text{not-}F)$.

The trouble with this proposal is that it equates causation with correlation. The fact that women knit more often than men does not mean that being female is a positive causal factor for knitting. In just the same way, it may be true that drops in barometer readings are correlated with storms, but that does not mean that barometer drops cause storms (see Box 3.3).

To apply this point to the issue of what "gene for X" means, consider the fact that there are genetic differences between people living in Finland and people living in Korea. Suppose gene g occurs in 20 percent of the people in Finland but in 75 percent of the people in Korea. If I sample an individual at random from the combined population of these two countries and find that this individual has gene g, I have evidence that this person speaks Korean rather than Finnish. But from this it would be absurd to conclude that g is a gene for speaking Korean. The gene may simply be a gene for blood type; the frequencies of blood types in the two countries may be different.

There is no gene for speaking Korean. However, this does not mean that the population of Korean speakers has the same genetic profile as the population of Finnish speakers. What it means is that two people, *were they placed in exactly the same environment,* would end up speaking the same language despite whatever genetic differences they may have.

This idea can be represented schematically as follows. Suppose that everyone in the two populations has either genotype $G1$ or genotype $G2$. Suppose further that everyone is exposed to either Finnish or to Korean during early life. In principle, there are four "treatment combinations." The phenotypes that result from these four gene/environment combinations are listed as entries in the following 2×2 table:

		Environment	
		Subject is exposed to	
		Finnish	Korean
Genotype	$G1$	speaks Finnish	speaks Korean
	$G2$	speaks Finnish	speaks Korean

In this example, what genotype you possess *makes no difference to the language you speak.* Of course, an individual can't speak a language without having genes; an organism won't develop at all if it has no genes. However, when we ask whether genes causally contribute to the development of some phenotype, we usually have in mind a difference between one genotype and another; the contrast between having genes of some sort and having no genes at all is not the relevant comparison.

In this 2 × 2 table, most of the individuals are either in the upper-left or the lower-right corner. People who grow up hearing Finnish tend to have genotype *G*1, and individuals who grow up hearing Korean tend to have genotype *G*2. That is, in this case, there is a *gene/environment correlation.* This correlation allows us to predict what language people speak either by knowing their environment or by knowing their genotype. Your genotype can be a good predictor of the language you speak, even though your genotype has no causal impact on what language you speak.

I have just run through some of the basic ideas that biologists now use to understand the distinction between *nature* and *nurture.* It is a truism that every phenotype an organism possesses is the result of a causal process in which genetic and environmental factors interact. But given that these two sorts of causes play a role in the ontogeny of an individual, how are we to say which "contributed more" or was "more important"? Consider a phenotype like the height of a corn plant. If the plant is 6 feet tall, how are we to tell whether the plant's genes or its environment was the more important cause of its height? If the genes built 5 feet of the plant and the environment added the remaining 1 foot, we could say that the environment contributed more. But genes and environment do not work separately in this way (Lewontin 1974). How, then, are we to compare the importance of the two causal factors?

The fundamental insight of the modern understanding of this issue is that it must involve *variation in a population.* We don't ask whether genes or environment mattered more in the development of a single corn plant. Rather, we take a field of corn plants in which there is variation in height. We then ask how much of that variation can be explained by genetic variation and how much by variation in the environment.

The basic statistical idea used in this enterprise is called *the analysis of variance* (ANOVA). Again for simplicity, consider a field of corn plants in which every plant has either genotype *G*1 or *G*2 and every plant receives either one unit of water (*W*1) or two (*W*2). Suppose the four treatment cells contain the same numbers of plants and that the average heights within the cells are as follows:

		Environment	
		*W*1	*W*2
Gene	*G*1	4	5
	*G*2	2	3

In this case, shifting from *G*2 to *G*1 increases the phenotype by two units, regardless of whether the plants receive one unit of water or two. It also is true that shifting from one unit of water to two increases height by a single unit, regardless of whether the plants have genotype *G*1 or genotype *G*2. In this example, there is a *positive main effect due to genes* and a *positive main effect due to environment;* changing each makes a difference in the resulting phenotype. In addition, note that the genetic main effect is larger than the environmental main effect. Changing a plant's genes (so to speak) makes more of a difference to its height than changing its environment.

By rearranging the numbers in the above 2 × 2 table, you can construct a data set that would imply that the environmental main effect is greater than the genetic main effect. You also can describe data in which one or both of the main effects is zero. I leave these as exercises for the reader.

In the previous 2 × 2 table, influences are purely additive. Shifting from *G*2 to *G*1 means "adding" two units of height, regardless of which environment a plant inhabits; shifting from *W*1 to *W*2 means "adding" one unit of height, regardless of which genotype a plant possesses. The following data set is not additive; it involves a *gene/environment interaction:*

		Environment	
		*W*1	*W*2
Gene	*G*1	4	7
	*G*2	2	3

In this case, how much difference an increase in water makes depends on the plant's genotype. Symmetrically, it also is true that how much difference a change in genotype makes depends on the plant's environment. But as before, the main effects are calculated by determining how much difference *on average* a change in genes or a change in environment brings about in the resulting phenotype.

We now can clarify what it means to say that genes are more (or less) important than environment for explaining the variation of some phenotype in some population. This merely means that the genetic main effect is greater (or less) than the environmental main effect. There is a gene (or a gene complex) "for" some phenotype in a given population precisely when the variation of that phenotype possesses a genetic main effect.

One consequence of this idea is that a trait does not have to be purely "nature" (= genetic) or purely "nurture" (= environmental). *To say that genes influence some phenotype does not mean that the environment has no influence.* That genes make a difference does not mean that the environment makes no difference.

Another consequence is that it is meaningless to say that genes are more important than environment (or to advance the opposite claim) for a phenotype that does

not vary. If every human being has a head, then one cannot say that genes are more important than environment in shaping this phenotype among human beings.

Even though there is no genetic main effect for the phenotype just mentioned, this does not mean that genes play no role in the ontogenetic processes in which individuals develop heads. Again, it is essential to bear in mind that "genetic main effect" has to do with whether different genes tend to produce different phenotypes. If all genes produce the same phenotype (i.e., the trait is totally *canalized*), there is no genetic main effect.

Consider another trait that is (virtually) universal within our species: Practically every human being can speak a language. Many linguists talk about an "innate language capacity," which all human beings are said to share. What could this mean, if the trait does not vary? To make sense of this idea, we must embed the human population, within which the trait is universal, in a larger population. For example, let us consider human beings together with chickens. Some individuals in this superpopulation speak a language while others do not. How do we explain this pattern of variation? Is it merely that human beings and chickens grow up in different environments? Or do genetic differences play a role?

Unfortunately, we face, at the outset, the problem of gene/environment correlation. Human beings are genetically different from chickens, but it also is true that they live in different environments. To identify the respective contributions of genes and environment, we must break this correlation, or, since ethical considerations prevent us from doing this, we must try to figure out what would happen if the correlation were broken. Just as in the example about Korean and Finnish, we need to fill in all four cells in the following 2×2 table:

	Environment	
	Exposed to a human language	Not exposed to a human language
Human genes	Yes	No
Chicken genes	No	No

The four entries describe whether the individual will speak a language. In this example, the contributions of genes and environment are entirely symmetrical. Having the right genes is essential, but so, too, is living in the right environment.

Apportioning causal responsibility between genes and environment depends on the set of genes and the range of environments considered. Consider, for example, the genetic disease known as PKU syndrome (phenylketonuria). Individuals with two copies of the recessive gene (call it *"p"*) cannot digest phenylalanine. If their diet contains phenylalanine, they will develop a severe retardation. However, *pp* homozygotes will develop quite normally if their diet is carefully controlled.

Let us consider PKU syndrome both before and after these facts about its dietary control were discovered. Before the discovery, pretty much everyone ate diets that

contained phenylalanine. In this case, the explanation of why some individuals ended up with PKU syndrome while others did not was entirely genetic. However, once the diet of *pp* homozygotes was restricted, the causal profile of PKU syndrome changed. Today, it is true that both genes *and* environment make a difference; the syndrome now is no more genetic than it is environmental (Burian 1983).

A simpler example illustrates the same point. Suppose that a set of genetically different corn plants are raised in the same environment; differences in height then must be due solely to genetic differences. But if you take that same set of corn plants and raise them in a variety of environments, the environmental main effect now may be nonzero—indeed, it may even be larger than the genetic main effect. *Whether a phenotype is mainly genetic is not an intrinsic feature that it has but is relative to a range of environments* (Lewontin 1974).

The question "Do genes matter more than environment?" is meaningless. This query must be relativized to a phenotype. Which language you speak is determined by your environment, but your eye color is determined by your genes. In addition to specifying the phenotype in question, one also must fix the range of genes and environments one wishes to consider. A trait can be mainly genetic in one range of environments but fail to be so in another.

Given this account of what it means to talk about a gene (or genes) for X (where X is some phenotype), I now want to consider what sociobiology presupposes about the issue of genetic causation. Sociobiologists sometimes discuss traits that they take to be universal (or nearly so) within the species of interest. At other times, they discuss traits that show within-species variation. Let us take these two cases in turn.

I have already mentioned that evolution by natural selection requires that the evolving trait be heritable. We now must see that the heritability of an evolving trait itself evolves. The fact that a trait must be heritable while it is evolving does not mean that it must remain heritable after it has finished evolving.

Consider a simplified scenario for the evolution of the opposable thumb. There was an ancestral population in which some individuals had opposable thumbs while others did not, and this phenotypic difference reflected genetic differences between the two classes of individuals. Selection then caused opposable thumbs to increase in frequency; eventually, the trait went to fixation. At this point, the gene(s) for an opposable thumb also became fixed.

Even though the frequency of the relevant genes reached 100 percent of the population, there is nevertheless some variation in whether people have opposable thumbs. For example, people sometimes lose their thumbs in industrial accidents. For this reason, it could easily be true that present-day variation for the phenotype of having an opposable thumb is mainly environmental and nongenetic. So when a sociobiologist posits a gene for X by way of explaining why the X phenotype evolved to fixation, the genetic main effect that this demands must have existed ancestrally. It need not exist today.

Although such explanations involve no commitment to the existence of present-day genetic variation, suppose we found that such genetic variation exists. Does this

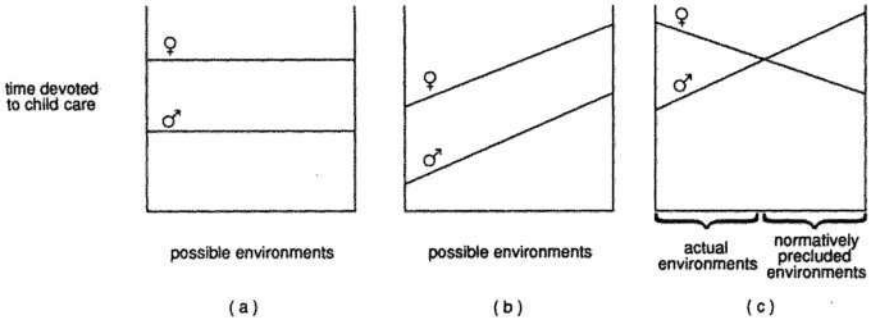

FIGURE 7.1 Three possible relationships between the average amounts of time that women and men devote to parental care.

automatically lend credence to a selective explanation? Here, we must be careful. If a trait is said to have evolved because of the strong selective advantage it provides, we should be puzzled as to why genetic variation for the trait still exists. It is not uncommon for sociobiologists to simultaneously say that a phenotype (like intelligence) was shaped by selection and to claim that the trait now has an important genetic component. Far from fitting together harmoniously, there is a dissonance between these two claims that we must learn to hear.

Now let us consider traits that sociobiologists think presently show within-species variation. One prominent example is the matter of behavioral differences between the sexes. Sociobiologists sometimes suggest that men are more promiscuous than women (and that women are more "coy" than men) and that evolutionary theory explains why. They also have commented on differences in patterns of child care, discussing why women stay home with the kids more than men do.

Let us focus on parental care. Suppose that women, on average, take care of their children more than men do in each of the various environments that human beings have inhabited to date. This difference between the sexes might obey three different patterns, depicted in Figure 7.1.

In part (a) of the figure, men and women differ in the average amount of time each spends on parental care. Note that the average amount of time spent by each sex is not affected by the environment. In part (b), the amount of time *is* influenced by changes in the environment, although the difference between the sexes is not.

Part (a) represents a stronger form of "biological determinism" than part (b). It is a curious terminological fact that "biological determinism" is so often used to mean *genetic* determinism (as if environmental causes like nutrition were not "biological"). Part (a) says that the absolute value *and* the relation between the sexes cannot be

modified by the environment; part (b) says that changing the environment can modify the absolute value for each sex but not the relation between the sexes.

Sociobiology is not committed to the ideas depicted in either part (a) or part (b). An evolutionary explanation of behavioral differences between the sexes does not have to maintain that there is *no possible environment* in which this difference would be erased or reversed. Sociobiologists often maintain that it would be very hard to eliminate certain behavioral differences between the sexes (Wilson 1975). For example, it might be necessary to completely overhaul the pattern of child care that boys and girls experience. Perhaps biological parents would have to be replaced by child-rearing experts who are trained by the state to behave in certain ways. In this radically altered environment, girls and boys might grow up to be parents who provide equal amounts of parental care. A sociobiologist might argue that this arrangement, though not impossible, would be undesirable. The new arrangement would require sacrificing values that many hold dear (Kitcher 1985).

This third possibility is depicted in Figure 7.1(c). In this arrangement, the environment affects not just the absolute amount of child care but whether women provide more of it than men. Although part (a) and part (b) represent versions of biological (i.e., genetic) determinism, part (c) cannot be interpreted in this way.

In all three figures, the behavioral difference between the sexes within the range of *actual* environments is said to have a nonenvironmental cause. If the *x*-axis represents all environmental causes, then the genetic difference between men and women (presumably, the fact that women are usually *XX* and men are usually *XY*) is said to have explanatory relevance.

As became clear in discussing Dawkins's knitting example, it is important not to be misled by gene-environment correlations. The fact that *XX* individuals, on average, provide more child care than *XY* individuals does not, by itself, entail that *XX* is a genetic configuration that codes for greater child care. Only if we control for environmental causes and *still* find that there is a genetic main effect can we conclude that this behavioral difference between the sexes has a genetic cause.

Sociobiologists generally have favored the hypothesis that important behavioral differences between the sexes have a significant genetic component. Selection has favored different behaviors in the two sexes. *Within women,* selection has favored one set of behaviors; *within men,* it has favored a different set. Of course, this hypothesis does not exclude what is obviously true—that some men provide more parental care than others and that there is variation among women for the trait as well. The hypothesis attempts to account for variation *between* the sexes, not *within* them. Variation *within* the sexes may be mainly environmental, but variation *between* the sexes, so the selectionist explanation implies, will have a significant genetic component.

Although many sociobiologists are inclined to explain this pattern of variation in genetic terms, it is not an inevitable commitment of sociobiological theorizing that all within-species variation must be explained in this way. A useful example of why this isn't intrinsic to the research program is provided by the work of Richard

Alexander (1979). Alexander is interested in explaining within-species variation. For example, he addresses the question of why some societies but not others follow the kinship system known as the avunculate. In this arrangement, men provide more care for their sisters' children than for the children of their spouses. Alexander suggests that this kinship system occurs in societies in which men are very uncertain about paternity. If women are sufficiently promiscuous, a man will probably have more genes in common with his sister's children than with the children of his wife. Thus, a man within such a society maximizes his reproductive success (advances his "genetic self-interest") by helping his sister's children, rather than helping his wife's.

I don't want to address the empirical issues of whether this explanation is correct. My point is that Alexander is not asserting that societies that follow the avunculate differ genetically from societies that do not. According to Alexander, human beings have a genetic endowment that allows them enormous behavioral flexibility. The human genotype has evolved so that individuals adjust their behaviors in a way that maximizes fitness. People in different societies behave differently not because they are genetically different but because they live in different environments. The avunculate maximizes fitness in some environments but not in others.

In a curious way, Alexander is a "radical environmentalist" with respect to within-species variation. Far from wishing to explain behavioral differences as "in our genes," he holds that behavioral variation is to be explained environmentally. This is about as far from a commitment to biological (i.e., genetic) determinism as one can get.

7.2 Does Sociobiology Have an Ideological Function?

Critics have seen sociobiology as the latest installment in a long line of biological ideas, stretching from the social Darwinism popular at the end of the nineteenth century through the IQ testing movement around the period of World War I to Nazi "racial biology," and to the debate about race and IQ in the 1960s (Chorover 1980; Lewontin, Rose, and Kamin 1984). Sociobiology, like its predecessors in this lineage, is charged with being ideological.

What might this charge of "ideology" mean? Several distinctions are needed. First, it might be claimed that individual authors or the people who determine which ideas are disseminated or the general readership of these views are motivated by ideological considerations. Second, there is the issue of *how much* of a role ideological considerations play in this three-step process of creation, dissemination, and acceptance. An extreme version of the ideology thesis might claim that there is not a shred of scientific evidence in support of sociobiological ideas, so the ideas are formulated, disseminated, and accepted for entirely nonscientific reasons. A less extreme thesis would be that the degree of conviction that people have with respect to these ideas far outruns the evidence actually at hand; what should be regarded as speculation gets interpreted as established truth.

Box 7.1 The Ought-Implies-Can Principle

Sociobiology has been criticized for defending the political *status quo.* If sociobiology entailed a strong thesis of biological determinism, the charge would make sense. If existing inequalities between the sexes or among the races or among social classes were biologically unalterable, then this fact would undercut criticisms of existing social arrangements. It would be hopelessly utopian to criticize society for arrangements that cannot be changed.

The argument just stated makes use of the *Ought-Implies-Can Principle: If a person ought to do X, then it must be possible for the person to do X.* If you *cannot* save a drowning person (e.g., because you cannot swim or have no access to a life preserver), then it is false that you *ought* to save that person: You cannot be criticized for not doing the impossible. Likewise, if our biology makes it impossible for us to eliminate certain inequalities, then it is false that we ought to eliminate those inequalities.

If the Ought-Implies-Can Principle is correct, then scientific results can entail that various ought-statements are false. Does this entailment relation contradict Hume's thesis (Section 7.4) about the relation of is-statements and ought-statements?

In all its guises, the ideology thesis is a thesis of *bias.* It claims that something influences the production/dissemination/acceptance process besides evidence; that extra something is the goal of advancing a political agenda. The ideology thesis does not entail that individual sociobiologists have been biased—the mindset of individual authors pertains to the production of sociobiological ideas, not to their subsequent dissemination or acceptance by a larger community. Suppose the mass media were biased in favor of publicizing scientific ideas that could be interpreted as justifying the political *status quo.* If sociobiological ideas were disseminated because they could be so interpreted, then sociobiology would perform an ideological function *even if no individual sociobiologist departed from reasonable norms of scientific objectivity.* Perfectly objective scientific findings can be put to ideological use.

I am here putting to work an idea explored in Section 3.7 concerning what it means to ascribe a function to something. What does it mean to say that the heart has the function of pumping blood but not of making noise? One suggestion is that the functional statement makes a claim about why the heart is there: Hearts persist because they pump blood, not because they make noise. In ascribing an ideological function to sociobiology, critics are making a claim about why such ideas persist.

Understood in this way, this functional claim is not obviously true. An empirical argument is needed to show that some part of the production/dissemination/acceptance process is biased and that the bias is due to the goal of advancing some political agenda. Glib statements about the "bias" of the mass media notwithstanding, it is no small task to muster evidence for claims of this sort.

Take a quite different and possibly simpler functional explanation of the persistence of an idea. Malinowski (1922) wanted to account for why South Sea Islanders

have elaborate rituals surrounding deep-sea fishing but none connected with fishing in fresh water. His explanation was that deep-sea fishing is far more dangerous than fishing in fresh water and that rituals evolved in connection with the former because they reduce fear.

To test Malinowski's conjecture, at least two hypotheses would have to be investigated. The first—that the rituals actually do reduce fear—might be investigated by an experiment in which some individuals are exposed to the rituals while others are not. We would like to know whether the first group is less fearful than the second. If deep-sea rituals really do reduce fear, the next question would be whether the rituals persist *because* they have this effect. In this connection, we would like to know if the rituals would persist even if they did not reduce fear. It is possible, after all, that rituals promote group solidarity and persist for this reason, quite apart from their effect on fear. If other rituals persist for reasons having nothing to do with fear reduction, this makes it less than transparent that deep-sea fishing rituals persist because they reduce fear. Perhaps the experiment to consider here would be to make deep-sea fishing quite safe. Would the rituals then wither away?

To document the claim that sociobiology has an ideological function, a similar pair of questions must be posed. Do sociobiological ideas really convince people that existing inequalities are legitimate and inevitable? This question is not settled simply by looking at what sociobiologists *say.* The issue is what impact various lectures, books, and articles have on their audience. Do people who read sociobiology accept the political *status quo* more than the members of some control group do? This is not obvious, but it may be true.

If sociobiological ideas do have this consequence, the second step would be to determine whether sociobiological ideas persist *because* they have this effect. Would such theorizing continue if it ceased to be understood as justifying the *status quo?* As in the case of deep-sea fishing rituals, the answer is not obvious. It may be that sociobiological theorizing is driven by a dynamic of scientific investigation that would propel the research program even if no one interpreted it as having political implications—after all, the evolution of behavior is an enticing problem area for biologists. Perhaps some sociobiological ideas persist for purely scientific reasons.

I said before that the question of whether sociobiology has an ideological function may be more complicated than the question of whether deep-sea fishing rituals have the function of reducing fear. One reason is that sociobiology is not a single idea; it is a web of various ideas, loosely connected with each other but elaborately connected with diverse elements in the rest of biology. It is possible that some themes in sociobiology persist for ideological reasons while others stay afloat for wholly scientific reasons. Just as there is no simple and global answer to the question of whether sociobiology is *true,* so there is no simple and global answer to the question of whether sociobiology functions to justify existing political arrangements. A serious investigation of either issue must proceed piecemeal.

Critics charge that sociobiology is ideology, not science. Sociobiologists protest that their own motives are scientific and that it is the critics themselves (some of

them Marxists) who are ideologically motivated. All this mudslinging aside, there is an issue here about the sociology of ideas that is worth considering seriously. It is no great shock to the scientific temperament to consider the possibility that *religious* ideas may persist for reasons having nothing to do with their truth. South Sea Islanders perform rituals to appease the gods; the rituals persist but not because there are gods who answer the Islanders' prayers. When this style of explanation is applied to the content of science, it is more difficult for scientists to approach it objectively. Yet, it is a possibility deserving of scientific scrutiny that some scientific ideas persist for reasons other than their evidential warrant. One should not accept this suggestion glibly, but neither should it be dismissed out of hand.

7.3 Anthropomorphism Versus Linguistic Puritanism

The next criticism of sociobiology I want to consider is aimed at suggestions like the following one, which was put forward by David Barash in his book *The Whispering Within* (1979, pp. 54, 55):

> Some people may bridle at the notion of rape in animals, but the term seems entirely appropriate when we examine what happens. Among ducks, for example, pairs typically form early in the breeding system, and the two mates engage in elaborate and predictable exchanges of behavior. When this rite finally culminates in mounting, both male and female are clearly in agreement. But sometimes strange males surprise a mated female and attempt to force an immediate copulation, without engaging in any of the normal courtship ritual and despite her obvious and vigorous protest. If that's not rape, it is certainly very much like it.
>
> Rape in humans is by no means as simple, influenced as it is by an extremely complex overlay of cultural attitudes. Nevertheless, mallard rape and bluebird adultery may have a degree of relevance to human behavior. Perhaps human rapists, in their own criminally misguided way, are doing the best they can to maximize their fitness. If so, they are not that different from the sexually excluded bachelor mallards. Another point: Whether they like to admit it or not, many human males are stimulated by the idea of rape. This does not make them rapists, but it does give them something else in common with mallards. And another point: During the India-Pakistan war over Bangladesh, many thousands of Hindu women were raped by Pakistani soldiers. A major problem that these women faced was rejection by husband and family. A cultural pattern, of course, but one coinciding clearly with biology.

Critics maintain that three errors occur in this and similar sociobiological accounts. First, there is *anthropomorphism:* A term ("rape") designed for application to human beings is extended to other species. Second, there is uncritical *adaptationism:* An explanation is invented for the mallard behavior that is not well supported by evidence. Third, the adaptationist explanation of the trait in ducks is read back into our own species.

The middle criticism I will not address here; I want to focus on the first and third. Why is it a mistake to think that a human behavior is "the same" as a trait found in some nonhuman species? And why should it be a mistake to think that the explanation of a trait found in a nonhuman species also applies to the human case?

To address these questions, I'll shift to another example, which is one of sociobiology's favorites—the existence of incest avoidance. Virtually all human cultures restrict or prohibit individuals from reproducing with close relatives. True, the pharaohs of ancient Egypt engaged in brother/sister marriages, but this is very much the exception rather than the rule. The sociobiological explanation of incest avoidance is that inbreeding increases the probability that offspring will have two copies of deleterious recessive genes. In consequence, natural selection has caused us to outbreed.

The sociobiological explanation just sketched applies to humans and non-humans alike. Yet, the explanation does not deny that human beings are unique; human beings avoid the behavior in part because they have an incest *taboo.* A taboo is a socially institutionalized system of beliefs and values. Human beings, unlike other organisms, avoid incest (to the extent they do) because of the beliefs and values that they have.

So the sociobiological account of incest avoidance says that human beings are unique in one respect but not in another. From an evolutionary point of view, we avoid incest for the same reason that other species do. However, the proximate mechanism that leads individual human beings to avoid inbreeding differs from the one that leads members of other species to do so.

This idea is represented in Figure 7.2. Consider some insect species *X* that has little inbreeding because individuals disperse from the nest before mating at random. Although human beings and species *X* avoid inbreeding for the same evolutionary reason, the proximate mechanisms are different. Here, we are using Mayr's (1961) distinction between proximate and ultimate explanation (Section 1.2). When sociobiologists explain incest avoidance in human beings by appealing to the selective advantage of outbreeding, they are not describing what goes on in the minds of human beings. They are attempting to describe evolutionary causes, not psychological (proximate) mechanisms.

It follows that the question "Why do human beings avoid incest?" can be addressed at two levels of analysis. One might try to answer it by discussing human psychology, or one might try to provide an evolutionary explanation. This is not to endorse what sociobiologists say about incest. My point is that *the psychological and the evolutionary answers are not in conflict.*

This idea has considerable relevance in evaluating Barash's explanation of rape in human beings. It is sometimes claimed that (human) rape should be regarded as an act of violence, not as a sexual act. The thought here is that rapists want to exercise power over their victims; it isn't sexual desire that drives the rapist but the desire to intimidate, humiliate, and punish (Brownmiller 1975). I will not try to assess whether this is a correct hypothesis about the psychology of rape. The point to rec-

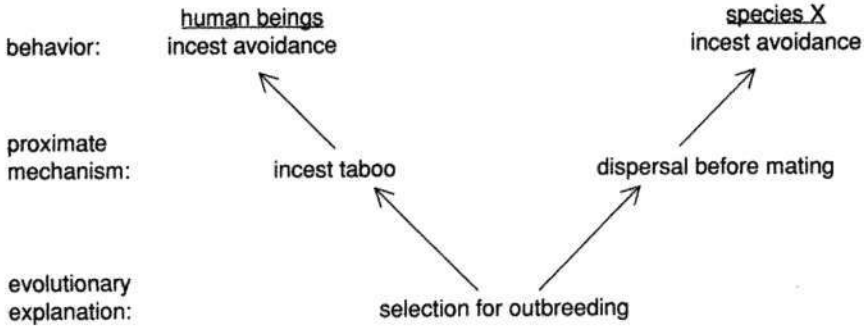

FIGURE 7.2 Even if human beings avoid mating with close relatives for the same *evolutionary* reason that the members of species *X* do, it does not follow that the behavior is under the control of the same *proximate mechanism* in the two species.

ognize is that it is quite compatible with the sociobiological explanation. According to Barash, rape evolved because of the reproductive advantage it provided for rapists; this says nothing, *per se,* about the psychological motives that make rapists do what they do.

This observation does not resolve our initial question about whether rape should be defined broadly so that it applies to nonhuman organisms or narrowly so that it is uniquely human. The distinction of proximate from evolutionary explanations shows that whichever way we define the behavior, both psychological and evolutionary questions can be posed about why rape occurs. Let us now turn to the issue of broad versus narrow definition.

Although choices of terminology may appear arbitrary, they often reflect assumptions about how research problems should be organized. If rape is defined as "forced reproduction," the term gathers together some behaviors but not others. Likewise, if it is defined as "an act of sexual violence motivated by the desire to exercise power," some behaviors but not others are gathered together.

According to ordinary usage, rape can occur without reproduction. It can involve oral or anal heterosexual acts and also coerced homosexual activity. The definition of rape as "forced reproduction," however, will not count such acts as rapes. Barash wants to find a common explanation of forced reproductive activity in humans and mallards, but this way of formulating the problem involves no special obligation to have the same explanation also cover nonreproductive behavior. By the same token, those who define rape as "an act of sexual violence motivated by the desire to exercise power" will want to provide a common explanation for coerced heterosexual inter-

Box 7.2 Incest

The sociobiological explanation of incest avoidance predicts that incest will be rare. But how rare is it? That depends on how "incest" is defined. If it is defined narrowly to mean reproduction between close relatives, we get one answer, but if it is defined more broadly to mean sexual contact between close relatives, we get another. Given how common sexual abuse of children is, perhaps the problem to address is not why incest is so rare but why it is so common.

Even if we opt for the narrower definition of incest, the question remains of how rare incest must be for the sociobiological explanation to be accepted. Can we shrug off a nonzero rate of reproduction among close relatives as compatible with the evolutionary model? How much incest would there have to be for us to conclude that the evolutionary explanation has been disconfirmed? Presumably, there is no threshold value.

Perhaps the comparative approach (Section 4.5) is more promising. Let us discover how much reproduction among close relatives there is within a variety of species, our own included. Then let us address the problem of explaining this pattern of variation. The simple idea that inbreeding is deleterious will not be sufficient. In some species, brother/sister mating is routine; in others, it is rare. If we jettison the simple question "Why do human beings avoid incest?" and substitute a comparative problem in its stead, our task becomes more difficult but also more interesting.

course and the sexual acts just mentioned. But they will feel no special obligation to have an explanation of rape also apply to the behavior of mallards. Each choice of terminology brings one set of acts to the foreground, demanding a common explanation and consigns another set of acts to the background, comprising an unrelated explanatory problem.

These conflicting taxonomies of behavior reflect the difference between what "sex" means to evolutionary biologists and what it means in ordinary language. For the evolutionary biologist, sex is a distinctive mode of reproduction found in many plants and animals. In ordinary language, sexual activity includes but is not limited to reproductive activity. An important part of what makes an act "sexual," in this vernacular sense, is the intentions of the actors.

It is by no means obvious that all or even most sexual activity, in the vernacular sense of that term, should be understood in terms of natural selection. Human mind and culture have given sexuality an amazingly complicated elaboration. To understand sexual behavior in terms of its relation to reproduction may be no more promising than understanding food customs in terms of their contribution to nutrition. Just as there is more to eating than survival, so there is more to sex than reproduction.

When Barash suggests that "rape" is a trait found in both human beings and in mallard ducks, he is saying that the explanation of the trait in one species has something significant in common with its occurrence in the other. We can use the con-

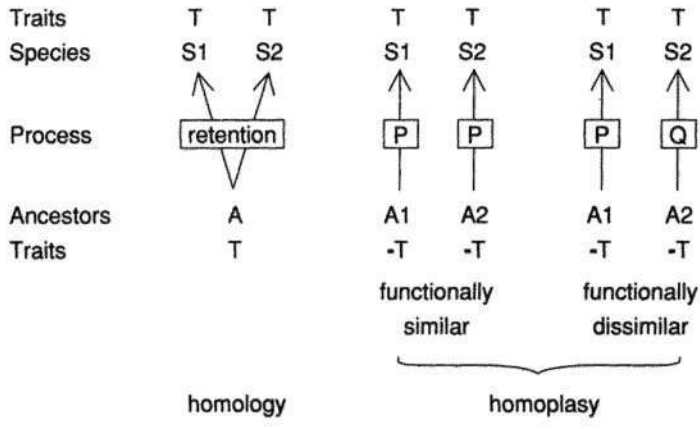

FIGURE 7.3 Three scenarios for the evolution of a similarity between species *S*1 and *S*2. They can share trait *T* as a homology, as a functionally similar homoplasy, or as a functionally dissimilar homoplasy.

cepts of *homology* and *homoplasy,* discussed in Chapter 6, to map out some of the options. Homologies, recall, are similarities inherited unmodified from a common ancestor. Homoplasies are similarities that evolved independently in the two lineages. Consider two species S1 and S2 that both exhibit some trait *T.* Figure 7.3 depicts three possible explanations of this similarity.

To illustrate the difference between what I am calling functionally similar and functionally dissimilar homoplasies, consider two examples. Wings in birds and wings in insects are homoplasies, but they evolved for very similar functional reasons. In both lineages, wings evolved because there was selection for flying and wings facilitate flight. Consider, in contrast, the fact that lizards and ferns are both green. This similar coloration is not inherited from a common ancestor; in addition, the reason the color evolved in one lineage has nothing functionally in common with the reason it evolved in the other. The occurrence of wings in birds and insects is a *functionally similar homoplasy;* the occurrence of greenness in lizards and ferns is a *functionally dissimilar homoplasy.*

When Barash applies the term "rape" to both human beings and mallard ducks, his point in using the common term is to suggest that the behaviors are either homologous *or* functionally similar. What is excluded by this sociobiological idea is that the apparent similarity between the behaviors is superficial and ultimately misleading.

Sociobiologists and their critics will agree that "greenness" in lizards and ferns is a functionally dissimilar homoplasy. Other traits are more controversial. "Rape" is the example I have discussed so far, but the same question arises in connection with other sociobiological explanations. For example, Wilson (1978) suggests that homosexuality in human beings evolved for the same reason that sterile castes evolved in the social insects. Sterile workers help their siblings to reproduce. The suggestion is that homosexuals do not reproduce but indirectly lever their genes (including "genes for homosexuality") into the next generation by helping heterosexual family members with child care.

The term "homosexuality" requires clarification. Once this is supplied, it is important to see what testable consequences follow from Wilson's proposal. For example, does his hypothesis predict that every family should contain homosexual offspring (just as every nest in a species of social insects contains sterile workers)? In addition, the hypothesis seems to predict that species in which there is more parental care should contain more "homosexual activity" than species in which there is less.

When critics of sociobiology object to the application of terms like "rape" to nonhuman organisms, sociobiologists often reply that the critics are trying to limit terminology for no good reason. After all, "selection" used to be a term that implied conscious choice, but Darwin saw the point of using the term in a "larger and metaphorical sense." Critics charge sociobiology with anthropomorphism; sociobiologists charge their critics with linguistic puritanism. These charges and countercharges easily suggest that the dispute involved here is not substantive. After all, it is up to us how we define our terms, and surely there is no serious issue about which definition is "really" correct (see the discussion of definitions in Box 1.1). But to dismiss the dispute about terminology in this way is to miss the substantive question that underlies it. The real problem is homology and functionally similar homoplasy, on the one hand, versus functionally dissimilar homoplasy, on the other.

7.4 Ethics

Sociobiologists have addressed two very different classes of questions about ethics. The first concerns why we believe the ethical statements that we do. If there are ethical beliefs that are held in all human cultures, then evolution may help to explain why these beliefs are universal. And values that vary from culture to culture also have been addressed by sociobiologists, for example via the hypothesis, favored by Alexander (1979, 1987), that human beings adjust their behavior to maximize fitness. Neither of these enterprises can be rejected *a priori;* everything depends on the extent to which specific hypotheses are supported by specific data.

In addressing the problem of explaining morality, it is important to break the phenomenon we call "morality" into pieces. Rather than asking whether "morality" is the product of natural selection, we should focus on some specific *proposition* about morality. Even if evolution helps explain why human societies possess moral codes, it is a separate question whether evolution helps explain the specific contents of those

codes (Ayala 1987). Perhaps there is a simple evolutionary explanation for why no society demands universal infanticide. On the other hand, it is not at all clear that evolutionary theory helps explain why opinion about the morality of slavery changed so dramatically in Europe during the nineteenth century. Rather than look for some sweeping global "explanation of morality," it is better to proceed piecemeal.

The second kind of question about ethics that sociobiologists have addressed is of an entirely different sort. Sometimes, the claim is advanced that evolutionary theory can tell us what our ethical obligations are. At other times, it is argued that the facts of evolution show that ethics is an illusion: although evolution leads us to *believe* that there is a difference between right and wrong, there really is no such thing (Ruse and Wilson 1986; Ruse 1986). In both instances, evolutionary theory is thought to tell us which ethical statements (if any) are true. It is this kind of project that I want to discuss now.

A common but by no means universal opinion among scientists is that *all facts are scientific facts.* Since ethical statements—statements about what is right or wrong—are not part of the subject matter of any science, it follows that there are no ethical facts. The idea is that in science, there are opinions *and* facts; in ethics, there is only opinion.

Let us say that a statement describes something subjective if its truth depends on what some subject believes; a statement describes something objective, on the other hand, if its truth or falsity is independent of what anyone believes. "People believe that the Rockies are in North America" describes something subjective. "The Rockies are in North America," on the other hand, describes something objective. When people study geography, there is both a subjective and an objective side; there are opinions about geography, but in addition, there are objective geographical facts.

Many people now believe that slavery is wrong. This statement describes something subjective. Is there, in addition to this widespread belief, a fact regarding the issue of whether slavery really is wrong? *Ethical subjectivism,* as I will use the term, maintains that there are no objective facts in ethics. In ethics, there is opinion and nothing else.

According to subjectivism, neither of the following statements is true:

Murder is always wrong.

Murder is sometimes permissible.

Naively, it might seem that one or the other of these statements must be true. Subjectivists disagree. According to them, no ethical statement is objectively true. Hume (1739, pp. 468–469) can be viewed as endorsing subjectivism in the following passage from his *Treatise of Human Nature:*

> Morality [does not consist] in any matter of fact, which can be discover'd by the understanding. . . . Take any action allow'd to be vicious: Wilful murder, for instance. Exam-

> ine it in all lights, and see if you can find that matter of fact, or real existence, which you call vice. In whichever way you take it, you find only certain passions, motives, volitions, and thoughts. There is no other matter of fact in the case. The vice entirely escapes you, as long as you consider the object. You never find it, til you turn your reflexion into your own breast, and find a sentiment of disapprobation, which arises in you, towards this action. . . . It lies in yourself, not in the object.

For Hume, the whole of ethics is to be found in the subject's feelings about murder; there is not, in addition, an objective fact about whether murder really is wrong.

Ethical realism conflicts with ethical subjectivism. Realism says that in ethics, there are facts as well as opinions. Besides the way willful murder may make you feel, there is, in addition, the question of whether the action really is wrong. Realism does not maintain that it is always obvious which actions are right and which are wrong—realists know that uncertainty and disagreement surround many ethical issues. However, for the realist, there are truths in ethics that are independent of anyone's opinion.

This book is not the place to attempt a full treatment of the dispute between subjectivism and realism. However, I do want to discuss two arguments that attempt to show that ethical subjectivism is true. I will suggest that neither of these arguments is convincing.

The first has its provenance in a logical distinction that Hume drew between what I will call *is-statements* and *ought-statements.* An *is-statement* describes what is the case without making any moral judgment about whether this situation is good or bad. An *ought-statement,* on the other hand, makes a moral judgment about the moral characteristics (rightness, wrongness, etc.) that some action or class of actions has. For example, "Thousands of people are killed by handguns every year in the United States" is an is-statement; "It is wrong that handguns are unregulated" is an ought-statement.

Hume defended the thesis that ought-statements cannot be deduced from exclusively is-statements. For example, he would regard the following argument as deductively invalid:

> Torturing people for fun causes great suffering.
>
> ---
>
> Torturing people for fun is wrong.

The conclusion does not follow deductively from the premisses. However, if we supply an additional premiss, the argument can be made deductively valid:

> Torturing people for fun causes great suffering.
> It is wrong to cause great suffering.
>
> ---
>
> Torturing people for fun is wrong.

Notice that this second argument, unlike the first, has an ought-statement as one of its premisses. Hume's thesis says that *a deductively valid argument for an ought-conclusion must have at least one ought-premiss.*

The term "naturalistic fallacy" is sometimes applied to any attempt to deduce ought-statements from exclusively is-premisses. The terminology is a bit misleading: It was G. E. Moore in *Principia Ethica* (1903) who invented the idea of a "naturalistic fallacy," and his idea differs from the one just described. However, since most people discussing evolutionary ethics tend to use Moore's label to name Hume's insight, I will follow them here. Hume's thesis is that the naturalistic fallacy is, indeed, a fallacy: You can't deduce an *ought* from an *is.*

Hume's thesis, by itself, does not entail subjectivism. However, the thesis plays a role in the following argument for subjectivism:

(S1) (1) Ought-statements cannot be deduced validly from exclusively is-premisses.
(2) If ought-statements cannot be deduced validly from exclusively is-premisses, then no ought-statements are true.

No ought-statements are true.

Premiss (1) is Hume's thesis. Premiss (2), which is needed to reach the subjectivist conclusion, is a *reductionist assumption.* It says that for an ought-statement to be true, it must reduce to (be deducible from) exclusively is-premisses.

My doubts about argument (*S*1) center on premiss (2). Why should the fact that ethics cannot be deduced from purely *is*-propositions show that no ethical statements are true? Why can't ethical statements be true though irreducible? It is worth noting that Hume's thesis concerns *deductive* arguments. Theories about unobservable entities cannot be deduced from premisses that are strictly about observables, but this provides no reason to think that theories about unobservables are always untrue.

There is another lesson that we can extract from Hume's thesis. When biological premisses are used to argue for some ethical conclusion, there must be ethical assumptions in the background. When these assumptions are flushed into the open, the arguments sometimes look quite implausible. For example, Wilson (1980, p. 69) points out that homosexual behavior is found in nature and thus is as "fully 'natural' as heterosexual behavior." Can we conclude from this that there is nothing immoral about homosexuality? We can, provided that we are prepared to append some further premiss of an ethical sort (for example, that all "natural" behaviors are morally permissible). More recently, the same ethical conclusion has been said to flow from the hypothesis that there may be a genetic component to homosexuality. Although I am fully in sympathy with the ethical conclusion, I think these arguments on its behalf are defective. Surely there are traits found in nature (and traits that have a genetic component) that are morally objectionable. Homophobia is a bad thing, but these are bad arguments against it.

I now want to consider a second argument for ethical subjectivism. It asserts that ethical beliefs cannot be true because the beliefs we have about right and wrong are merely the product of evolution. An alternative formulation of this idea would be that subjectivism must be true because our ethical views are produced by the socialization we experience in early life. These two ideas may be combined as follows:

(S2) We believe the ethical statements we do because of our evolution and because of facts about our socialization.

No ethical statement is true.

Philosophers are often quick to criticize such arguments for committing the so-called *genetic fallacy*. "Genetic" here has nothing to do with chromosomes; rather, a genetic argument describes the genesis (origin) of a belief and attempts to extract some conclusion about the belief's truth or plausibility.

The dim view that many philosophers take of genetic arguments reflects a standard philosophical distinction between the *context of discovery* and the *context of justification*. This distinction, emphasized by the logician Gottlob Frege, was widely embraced by the positivists. Hempel (1965b) tells the story of the chemist Kekulé, who worked on the problem of determining the structure of benzene. After a long day at the lab, he found himself gazing wearily at a fire. He hallucinated a pair of whirling snakes, which grabbed each other's tails and formed a circle. Kekulé, in a flash of creative insight, came up with the idea of the benzene ring.

The fact that Kekulé arrived at this idea while hallucinating does not settle whether benzene really has a ring structure. It is for psychologists to describe the context of discovery—the idiosyncratic psychological processes that led Kekulé to his insight. After he came up with this idea, he was able to do experiments and muster evidence. This latter set of considerations concerns the logic of justification.

I agree that one can't *deduce* that Kekulé's hypothesis was true or false just from the fact that the idea first occurred to him in a dream. But it is a mistake to overinterpret this point. I suggest that there can be perfectly reasonable genetic arguments. These will be *non*deductive in form.

Consider an example. Suppose I walk into my introduction to philosophy class one day with the idea that I will decide how many people are in the room by drawing a slip of paper from an urn. In the urn are a hundred such slips, each with a different number written on it. I reach in the urn, draw a slip that says "78," and announce that I believe that exactly 78 people are present.

Surely it is reasonable to conclude that my belief is probably incorrect. This conclusion is justified because of the process that led me to this belief. If so, the following is a perfectly sensible genetic argument:

Sober decided that there are 78 people in the room by drawing the number 78 at random from an urn.

p ═══════

It isn't true that there are 78 people in the room.

I have drawn a double line between premiss and conclusion to indicate that the argument is not supposed to be deductively valid. The *p* next to the double line represents the probability that the premiss confers on the conclusion. I claim that *p* is high in this argument.

It is quite true that one cannot *deduce* that a proposition is false just from a description of how someone came to believe it. But I see no reason to think that the context of discovery never provides any evidence at all about whether a belief is true (or plausible). If this is right, we must be careful to distinguish two different formulations of what the genetic fallacy is supposed to involve:

(1) Conclusions about the truth of a proposition cannot be *deduced validly* from premisses that describe how someone came to believe the proposition.
(2) Conclusions about the truth of a proposition cannot be *inferred* from premisses that describe how someone came to believe the proposition.

I think that (1) is true but (2) is false. Inference encompasses more than deductive inference. I conclude that argument (S2) for ethical subjectivism cannot be dismissed simply with the remark that it commits "the genetic fallacy."

The genetic argument concerning how I arrived at my belief about the number of people in the room is convincing. Why? Because *what caused me to reach the belief had nothing whatever to do with whether the belief is true.* When this *independence relation* obtains, the genetic argument shows that the belief is implausible. In contrast, when a *dependence relation* obtains, the description of the belief's genesis can lead to the conclusion that the belief is probably correct.

As an example of how genetic arguments can show that what you believe is probably true, consider my colleague Rebos, who decided that there are 104 people in her philosophy class by carefully counting the people present. I take it that the premiss in the following argument confers a high probability on the conclusion:

Rebos carefully counted the people in her class and consequently believed that 104 people were present.

p ═══════

104 people were present in Rebos's class.

When Rebos did her methodical counting, the thing that caused her to believe that there were 104 people present was *not* independent of how many people actually were there. Because the process of belief formation was influenced in the right way

by how many people really were in the room, we are prepared to agree that a description of the context of *discovery* provides a *justification* of the resulting belief.

Let us turn now to the argument for ethical subjectivism summarized by (S2). As the comparison of Sober and Rebos shows, (S2) is incomplete. We need to add some premiss about how the process by which we arrive at our moral beliefs is related to which moral beliefs (if any) are true. Suppose we were to agree with the following thesis:

(A) The processes that determine what moral beliefs people have are entirely independent of which moral statements (if any) are true.

This proposition, if true, would support the following conclusion: *The moral beliefs we currently have are probably untrue.*

The first thing to notice about this conclusion is that it does *not* say that ethical subjectivism is correct. It says that our *current* moral beliefs are probably untrue, not that *all* ethical statements are untrue. Here, we have an important difference between (S2) and the quite legitimate genetic arguments about Sober and Rebos. Clearly, a genetic argument might make plausible the thesis that the ethical statements we happen to believe are untrue. But I do not see how it can show that *no* ethical statements are true.

The next thing to notice about the argument for subjectivism concerns assumption (A). To decide whether (A) is true, we would need to describe (1) the processes that lead people to arrive at their ethical beliefs and (2) the facts about the world, if any, that make those beliefs true or false. We then would have to show that (1) and (2) are entirely independent of each other, as (A) asserts.

Argument (S2) provides a very brief answer to (1)—it cites "evolution" and "socialization." With respect to problem (2), the argument says nothing at all. Of course, if subjectivism were correct, there would be no ethical facts to make ethical beliefs true. But to *assume* that subjectivism is true in the context of this argument begs the question.

Because (S2) says only a little about (1) and nothing at all about (2), I suggest that it is impossible to tell from this argument whether (A) is correct. After all, lots of our beliefs stem either from evolution or from socialization. Mathematical beliefs are of this sort, but that doesn't show that no mathematical statement is true (Kitcher 1985). I conclude that (S2) is a weak argument for ethical subjectivism.

It is not implausible to think that many of our current ethical beliefs are confused. I am inclined to think that morality is one of the last frontiers that human knowledge can aspire to cross. Even harder than the problem of understanding the secrets of the atom, of cosmology, and of genetics is the question of how we ought to lead our lives. This question is harder for us to come to grips with because it is clouded with self-deception: We have a powerful interest in not staring moral issues squarely in the face. No wonder it has taken humanity so long to traverse so modest a distance. Moral beliefs generated by superstition and prejudice probably *are* untrue.

Moral beliefs with this sort of pedigree deserve to be undermined by genetic arguments. However, from this critique of some elements of existing morality, one cannot conclude that subjectivism about ethics is correct.

7.5 Models of Cultural Evolution

At present, there is considerable interest and controversy surrounding the application of biological ideas within the social sciences. Sociobiology is the best known of these enterprises. Various philosophical issues raised by sociobiology have been discussed in this chapter. In the present section, I want to discuss a less well-known movement within biology—one that strives to extend evolutionary ideas to social scientific phenomena but not in the way envisioned by sociobiology. Cavalli-Sforza and Feldman (1981) and Boyd and Richerson (1985) have proposed several models of cultural evolution. These authors have distanced themselves from the mistakes they see in sociobiology. In particular, their goal is to describe how cultural traits can evolve for reasons that have nothing to do with the consequences the traits have for survival and reproductive success. In a very real sense, their models describe how mind and culture can play an irreducible and autonomous role in cultural change.

In order to clarify how these models differ from ideas put forward in sociobiology, it will be useful to describe some simple ways in which models of natural selection can differ. As will become clear, I will be using "selection" and other terms more broadly than is customary in evolutionary theorizing.

Given a set of objects that exhibit variation, what will it take for that ensemble to evolve by natural selection? Here, I use "evolve" to mean that the frequency of some characteristic in the population changes. Two ingredients are crucial. The first is that the objects differ with respect to some characteristic that makes a difference in their abilities to survive and reproduce. Then there must be some way to ensure that offspring resemble their parents. The first of these ingredients is differential *fitness;* the second is *heritability* (Lewontin 1970).

In most standard models of natural selection, offspring resemble their parents because a genetic mode of transmission is in place. And traits differ in fitness because some organisms have more babies than others. It may seem odd to say that "having babies" is one way to measure fitness and that passing on genes is one way to ensure heritability, as if there could be others. My reason for saying this will soon become clear.

One way—the most straightforward way—to apply evolutionary ideas to human behavior is to claim that some psychological or cultural characteristic became common in our species by a selection process of the kind just described. This is essentially the pattern of explanation used by Wilson (1975).

The second form that a selection process can take retains the idea that fitness is measured by how many babies an organism produces, but it drops the idea that the relevant phenotypes are genetically transmitted. For example, if characteristics are transmitted because children imitate their parents, a selection process can occur without the mediation of genes.

The incest taboo provides a hypothetical example of how this might happen. Suppose that incest avoidance is advantageous because individuals with the trait have more viable offspring than individuals without it. If offspring *learn* whether to be incest avoiders from their parents, the frequency of the trait in the population may evolve. This could occur without there being any genetic differences between those who avoid incest and those who do not (Colwell and King 1983).

In this second kind of selection model, mind and culture displace one but not the other of the ingredients found in models of the first type. In the first sort of model, a genetic mode of transmission works side by side with a concept of fitness defined in terms of reproductive output—what I have called "having babies." In the second, reproductive output is retained as the measure of fitness, but the genetic mode of transmission is replaced by a psychological one. Learning can provide the requisite heritability just as much as genes.

The third pattern for applying the idea of natural selection abandons both of the ingredients present in the first. The mode of transmission is not genetic, and fitness is not measured by how many babies an organism has. According to this pattern, individuals acquire their ideas because they are exposed to the ideas of their parents, of their peers, and of their parents' generation; transmission patterns may be vertical, horizontal, and oblique. An individual exposed to this mix of ideas need not give them all equal credence. Some may be more attractive than others. If so, the frequency of ideas in the population may evolve. Notice that there is no need for organisms to differ in their survivorship or degree of reproductive success in this case. Some ideas catch on while others become passé. In this third sort of selection model, ideas spread the way a contagion spreads.

The *theory of the firm* in economics (discussed in Hirshliefer 1977) is an example of this third type of selection model. Suppose one wishes to explain why businesses behave as profit maximizers. One hypothesis might be that individual managers are rational and economically well informed; they intelligently adjust their behavior to cope with market conditions. Call this the learning hypothesis. An alternative hypothesis is that managers are not especially rational but that inefficient firms go bankrupt and thereby disappear from the market. This second hypothesis posits a selection process of type three. The mode of transmission is not genetic; a business sticks to the same market strategy out of inertia (not because the genes of managers are passed along to their successors). In addition, biological fitness does not play a role. Firms survive differentially, but this does not require individual managers to die or have babies.

Another example of type three models may be found in some versions of *evolutionary epistemology.* Popper (1973) suggests that scientific theories compete with each other in a struggle for existence. Better theories spread through the population of researchers; inferior ones exit from the scene. Other models in evolutionary epistemology are structured similarly (Toulmin 1972; Campbell 1974; Hull 1988).

The three forms that a selection model can take are summarized in Figure 7.4. "Learning" here should be taken broadly; it doesn't require anything very cognitive

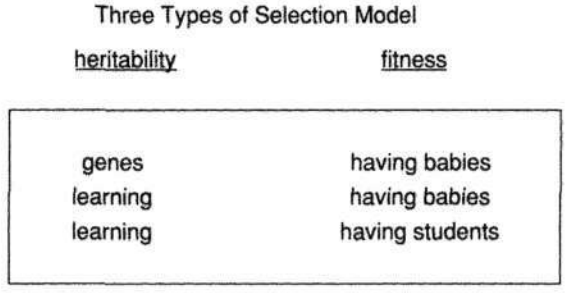

FIGURE 7.4 Selection processes of type I are standard in discussions of "biological" evolution; those of type III underlie discussions of "cultural" evolution. Those of type II are, so to speak, intermediate.

but can simply involve imitation. The same goes for "having students"—all that is involved is successful influence mediated by learning.

The parallelism between types I and III is instructive. In type I processes, individuals produce different numbers of babies in virtue of the phenotypes they have (which are transmitted genetically); in type III, individuals produce different numbers of students in virtue of the phenotypes they have (which are transmitted by learning).

Selection models of cultural characteristics that are of either type I or type II can properly be said to provide a "biological" treatment of the traits in question. Models of type III, on the other hand, do not propose biological explanations at all. In type III models, the mode of transmission and the reason for differential survival and replication of ideas may have an entirely autonomous cultural basis.

This threefold division is, of course, consistent with the existence of models that combine two or more of these sorts of process. My taxonomy describes "pure types," so to speak, whereas it is often interesting to consider models in which various pure types are mixed. This is frequently the case in the examples developed by Cavalli-Sforza and Feldman (1981) and by Boyd and Richerson (1985), one of which I'll now describe.

In the nineteenth century, Western societies exhibited an interesting demographic change, one that had three stages. First, oscillations in death rates due to epidemics and famines became both less frequent and less extreme. Then, overall mortality rates began to decline. The third part of the demographic transition was a dramatic decline in birthrates.

Why did fertility decline? From a narrowly Darwinian point of view, this change is puzzling. A characteristic that *increases* the number of viable and fertile offspring

will spread under natural selection, at least when that process is conceptualized by a type I model. Cavalli-Sforza and Feldman are not tempted to appeal to the theory of optimal clutch size developed by Lack (1954), according to which a parent can augment the number of offspring surviving to adulthood by having fewer babies. This Darwinian option is not plausible since women in nineteenth-century Western Europe could have had more viable offspring than they did in fact. People were not caught in the bind that Lack attributed to his birds.

The trait of having fewer children entails a reduction in biological fitness. The trait spread *in spite of* its biological fitness, not *because* of it. In Italy, women changed from having about five children, on average, to having about two. The new trait was far less fit than the old one it displaced.

Cavalli-Sforza and Feldman focus on the problem of explaining how the new custom spread. One possible explanation is that women in all social strata gradually and simultaneously reduced their fertilities. A second possibility is that two dramatically different traits were in competition and that the displacement of one by the other cascaded from higher social classes to lower ones. The first hypothesis, which posits a gradual spread of innovation, says that fertilities declined from 5 to 4.8 to 4.5 and so on, with this process occurring simultaneously across all classes. The second hypothesis says that the trait of having five children competed with the trait of having two and that the novel character was well on its way to displacing the more traditional one among educated people before the same process began among less educated people. There is evidence favoring the second pattern, at least in some parts of Europe.

Cavalli-Sforza and Feldman emphasize that this demographic change could not have taken place if traits were passed down solely from mothers to daughters. This point holds true whether fertility is genetically transmitted or learned. A woman with the new trait will pass it along to fewer offspring than a woman with the old one, if a daughter is influenced just by her mother.

What the process requires is some mixture of horizontal and oblique transmission. That is, a woman's reproductive behavior must be influenced by her peers and by her mother's contemporaries. However, it will not do for a woman to adopt the behavior she finds represented on average in the group that influences her. A woman must find small family size more attractive than large family size even when very few of her peers possess the novel characteristic. In other words, there must be a "transmission bias" in favor of the new trait.

Having a small family was more attractive than having a large one, even though the former trait had a lower Darwinian fitness than the latter. Cavalli-Sforza and Feldman show how the greater attractiveness of small family size can be modeled by ideas drawn from evolutionary theory. However, when these biological ideas are transposed into a cultural setting, one is talking about cultural fitness, not biological fitness. The model they construct of the demographic transition combines two selection processes. When fitness is defined in terms of having babies, there is selection *against* having a small family. When fitness is defined in terms of the psychological attractiveness of an idea, there is selection *favoring* a reduction in family size. Cavalli-

Sforza and Feldman show how the cultural process can overwhelm the biological one; given that the trait is sufficiently attractive (and their models have the virtue of giving this idea quantitative meaning), the trait can evolve in spite of its Darwinian disutility.

What are we to make of the research program in which models like this one are developed? Biologists interested in culture are often struck by the absence of viable general theories in the social sciences. All of biology is united by the theory of biological evolution (Section 1.2). Perhaps progress in the social sciences is impeded because there is no general theory of cultural evolution.

The analogies between cultural and genetic change are palpable. And at least some of the disanalogies can be taken into account when the biological models are transposed. For example, we know that biological variation is "undirected"; mutations do not occur because they would be beneficial. In contrast, ideas are *not* invented at random. Individuals often create new ideas—in science, for example—precisely because they would be useful. Another and related disanalogy concerns the genotype/phenotype distinction and the idea that there is no "inheritance of acquired characteristics" (Section 4.4). These principles may have no ready analogs in cultural transmission.

These disanalogies between genetic and cultural change do not show that it is pointless or impossible to write models of cultural evolution that draw on the mathematical resources of population biology. These and other structural differences between biological and cultural evolution can easily be taken into account in models of cultural change.

Another reservation that has been voiced about models of cultural evolution is that they atomize cultural characteristics. Having two children rather than five is a characteristic that is abstracted from a rich and interconnected network of traits. The worry is that by singling out a trait for theoretical treatment, we lose sight of the context that gives that trait cultural meaning.

It is worth realizing that precisely the same question has been raised about biological evolution itself. If you wish to understand the population frequency of sickle-cell anemia, for example, you cannot ignore the fact that the trait is connected with resistance to malaria. In both cultural and biological evolution, it is a mistake to think that each trait evolves independently of all the others. The lesson here is that individual traits should be understood in terms of their relationship to each other.

Although the criticisms I have reviewed so far do not seem very powerful, one rather simple fact about these models suggests that they may be of limited utility in the social sciences. Insofar as these models describe culture, they describe systems of cultural transmission and the evolutionary consequences of such systems. *Given* that the idea of having two children was more attractive than the idea of having five and *given* the horizontal and oblique transmission systems then in place, we can see why the demographic transition took place. But as Cavalli-Sforza and Feldman recognize, their model does not describe *why* educated women in nineteenth-century Italy came to prefer having smaller families, nor *why* patterns adopted in higher classes

cascaded down to lower ones. The model describes the *consequences* of an idea's attractiveness, not the *cause* of its being attractive (a distinction introduced in Section 1.6). Historians, on the other hand, will see the real challenge to be the identification of causes.

Models of cultural transmission describe the *quantitative* consequences of systems of cultural influence. Social scientists inevitably make *qualitative* assumptions about the consequences of these systems. If these qualitative assumptions are wrong in important cases and these mistakes actually undermine the plausibility of various historical explanations, social scientists will have reason to take an interest in these models of cultural evolution. But if the qualitative assumptions are correct, historians will have little incentive to take the details of these models into account.

The distinction between source and consequence also applies to some ideas in evolutionary epistemology, including evolutionary models of scientific change. Despite various disanalogies between genes and ideas, the thought that the mix of ideas in a scientific community evolves by a process of "selective-retention" has considerable plausibility (Campbell 1974; Hull 1988; see also Dawkins's 1976 remarks about "memes"). However, the question then arises of what makes one scientific idea "fitter" than another.

Historians of science address this question, though not in this language, when they consider "internalist" and "externalist" explanations of scientific change. Does one idea supplant another because it is better confirmed by observations? Or do scientific ideas come and go because of their ideological utility, their metaphysical palatability, or the power and influence of the people who promulgate them? Clearly, different episodes of scientific change may have different kinds of explanation, and a given change may itself be driven by a plurality of causes. Evolutionary models of scientific change inevitably lead back to these standard problems about *why* scientific ideas change. It seems harmless to agree that fitter theories spread; the question is what makes a theory fitter.

Although general models of the consequences of cultural evolution are no substitute for an understanding of the sources of differences in cultural fitness, there is something important that these models have achieved. A persistent theme in debates about sociobiology is the relative "importance" that should be accorded to biology and culture. I place the term "importance" in quotation marks because it cries out for clarification. What does it mean to compare the "strength" or "power" of biological and cultural influences?

One virtue of these models of cultural evolution is that they describe culture and biology within a common framework, so that their relative contributions to an outcome are rendered commensurable. What becomes clear in these models is that in comparing the importance of biology and culture, *time is of the essence.* Culture is often a more powerful determiner of change than biological evolution because cultural changes occur *faster.* When biological fitness is calibrated in terms of having babies, its basic temporal unit is the span of a generation. Think how many replication events can occur in that temporal interval when the reproducing entities are ideas

that jump from head to head. Ideas spread so fast that they can swamp the slower (and hence weaker) impact of biological natural selection.

This point recapitulates a theme introduced in Section 4.3. In the evolution of biological altruism, group selection pushes in one direction, but individual selection pushes in another. Whether altruism evolves depends on the relative strengths of these selection pressures. But what does "strength" mean in this context? If offspring exactly resemble their parents, the strength of selection is measured by the expected amount of change *per unit time.* If altruism is to evolve, differential survival and reproduction at the group level (i.e., the extinction of old groups and the formation of new ones) must happen *fast enough.*

There is a vague idea about the relation of biology and culture that models of cultural evolution help lay to rest. This is the idea that the science of biology is "deeper" than the social sciences, not just in the sense that it has developed further but in the sense that it investigates more important causes. The inclination is to think that if Darwinian selection favors one trait but cultural influences favor another, the deeper influence of biology must overwhelm the more superficial influence of culture. Cavalli-Sforza and Feldman and Boyd and Richerson deserve credit for showing why this common opinion rests on a confusion.

The conclusion to be drawn here is not that cultural selection *must* overwhelm biological selection when the two conflict but that this *can* happen. Again, the similarity with the conflict between individual and group selection is worth remembering. When two selection processes oppose each other, which "wins" is a contingent matter. The fact that a reduction of family size occurred in nineteenth-century Italy says nothing about what will be true of other traits in other circumstances. The human brain *can* throw a monkey wrench into an adaptationist approach to human behavior. Whether it does so is to be settled on a case-by-case basis.

It is a standard idea in evolutionary theory that an organ will have characteristics that are not part of the causal explanation of why it evolved (Section 4.2). The heart makes noise, but that is not why the heart evolved—it evolved because it pumps blood. Making noise is a *side effect;* it is evolutionary *spin-off* (Section 3.7). We must not lose sight of this distinction when we consider the human mind/brain. Although the organ evolved because of *some* of the traits it has, this should not lead us to expect that *every* behavior produced by the human mind/brain is adaptive. The brain presumably has many side effects; it generates thoughts and feelings that have nothing to do with why it evolved.

Both brains and hearts have features that are adaptations and features that are evolutionary side effects. But to this similarity we must add a fundamental difference. When my heart acquires some characteristic (e.g., a reduced circulation), there is no mechanism in place that causes that feature to spread to other hearts. In contrast, a thought—even one that is neutral or deleterious with respect to my survival and reproduction—is something that may spread beyond the confines of the single brain in which it originates. Brains are linked to each other by networks of mutual influence; it is these networks that allow ideas that occur in one head to influence ideas

that occur in others. This is an arrangement that our brains have effected but our hearts have not.

The idea that cultural evolution can swamp biological evolution does not imply that standard processes of biological evolution no longer operate in our species. Individuals still live and die differentially, and differential mortality often has a genetic component. This biological process is not *erased* by the advent of mind and culture; it remains in place but is joined by a second selection process that is made possible by the human mind.

It is quite true that biological evolution produced the brain and that the brain is what causes us to behave as we do. However, it does not follow from this that the brain plays the role of a passive proximate mechanism, simply implementing whatever behaviors happen to confer a Darwinian advantage. Biological selection produced the brain, but the brain has set into motion a powerful process that can counteract the pressures of biological selection. The mind is more than a device for generating the behaviors that biological selection has favored. It is the basis of a selection process of its own, defined by its own measures of fitness and heritability. Natural selection has given birth to a selection process that has floated free.

Suggestions for Further Reading

Caplan (1978) brings together some of the initial salvos in the sociobiology debate, as well as some earlier documents. Kitcher (1985) develops detailed criticisms of what he terms "pop sociobiology" but has positive things to say about other work on the evolution of behavior. Richards (1987) focuses mainly on the history of nineteenth-century evolutionary accounts of mind and behavior. Ruse (1986) argues that evolutionary theory can throw considerable light on traditional philosophical problems about knowledge and values.

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