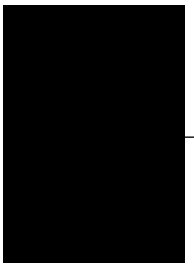

The Biological Mind

A Philosophical Introduction

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4 Nature and nurture

In *English Men of Science: Their Nature and Nurture*, Francis Galton (1874) tells us that, "nature is all that a man brings with himself into the world; nurture is every influence from without that affects him after his birth" (12). Galton was one of Charles Darwin's cousins. He was fascinated with the question of what's innate and acquired, because he wanted to structure society in such a way that those with supposedly "innate" or "hereditary" talents would increase in frequency. He was obsessed with building genealogies, and he did so for hundreds of famous families in a bid to prove that intellect, creativity, and even physical strength are faithfully passed down from parent to child (Galton 1892 [1869]). Although the "nature/nurture" dispute can be traced to ancient times, Galton managed to bring the problem to the forefront of biology. Since then, biologists and psychologists have debated the question of how much of the human personality is due to "nature" and how much is due to "nurture." Another way of putting the point, which I take to amount to the same thing, is in terms of what's "innate" and what's "acquired."

Some people reject the terms of the debate entirely. In fact, some philosophers, biologists, and psychologists have argued that the terms of the debate are so confused, or so poorly defined, that we should scrap it without looking back. I agree with this assessment. In this chapter, I'll tackle the idea of innateness. I agree with those biologists, philosophers, and psychologists who think the idea of innateness borders on meaninglessness, and that the distinction between innate and acquired is a bad way to think about development. Then, I'll discuss ways that evolutionary approaches to the mind could get on just fine without the idea of innateness. Specifically, it would be more fruitful to make a distinction between traits that are relatively "robust," and those that are relatively "plastic." The distinction between robustness and plasticity captures an important phenomenon but does so without presupposing the idea of innateness.

Others, however, have urged that we *reform* the distinction, instead of eliminating it. One position is that the "nature/nurture" dispute can be placed on a firmer scientific footing by rephrasing the distinction in terms of *genes* and *environment*. What parts of how we are – our personalities or behavior – are caused by our genes, and what parts are caused by our environments? For many, the distinction between genes and environment appears to be a modern, perhaps more scientific, way of approaching the nature/nurture territory. As I'll show in this chapter, the distinction between genes and environment, valuable as it is, is *not* a modern way of recasting the distinction between nature and nurture, or innate and acquired. That's because we have no simple way of dividing up causal responsibility for any particular trait between genes and environment. The answer is always, unequivocally and inextricably, "both." So we need to adopt a different framework to study development.

The problems that I'll discuss with respect to innateness are, to a greater or lesser extent, problems for the various evolutionary approaches to psychology that I discussed in the last chapter. For example, evolutionary psychologists tend to assume that the mental modules that they investigate are *innate*, or that the information for the developmental process that produces these modules is *genetically encoded*. The idea of innateness, however, is not just a problem for evolutionary psychology. The very *idea* of gene-culture evolution seems to assume that some parts of who we are, are caused by "culture," and other parts are caused by "genes," which seems tantamount to saying they're "innate." So, if we decide that we seriously want to rid ourselves of innateness, we have a lot of soul-searching to do.

Section 4.1 will take a critical look at the popular idea of *innateness*, and discuss why many biologists and philosophers think the idea is unscientific. Sections 4.2 through 4.4 will examine different ways that contemporary philosophers and biologists have tried to define "innateness," all of which have serious problems. Section 4.2 will consider the idea that an innate trait is just one that's "not learned." Section 4.3 will consider the idea that an innate trait is one that's genetically (rather than environmentally) caused. Section 4.4 considers the idea that a trait is innate if the information about the trait is somehow encoded in the genes. Section 4.5 will develop a more constructive approach to the mind, one that centers on the ideas of robustness and plasticity rather than innate and acquired.

4.1 WHAT IS INNATENESS?

The concept of innateness has been the subject of considerable controversy in twentieth-century biology and psychology. But attacks on the concept of innateness go back at least as far as the seventeenth century. The English philosopher, John Locke, believed that all knowledge emerges from experience. His critique of *innate ideas* was part of a broader assault on earlier philosophers such as Plato and Descartes who believed that some knowledge was "inborn." Locke did not merely reject the theory of innate ideas, but tried to show that the very notion was somehow nonsensical or incoherent.

Interestingly, Locke was not trying to attack the notion of innate *abilities* or *instincts*, such as the sucking reflex in infants. Some even think he made use of the idea freely (Keller 2010, 18). But it seems inconsistent to say that the notion of *innate abilities* is sensible and wise, but the

notion of *innate ideas* is nonsensical. After all, many advocates of innate ideas use the notion of an innate *ability* as a starting point for framing their definitions (Chomsky 1980, 33; Stich 1975, 6). If we seriously want to throw out the concept of innate ideas we may have to root it out at its source and throw out the idea of innate abilities, too.

Locke's line of attack was this: on any apparently reasonable definition of what the word "innate" means (as in, "innate ideas"), almost everything turns out to be "innate," or almost nothing does. But most people who believe there are innate ideas, believe that only *some* ideas are innate, and *some* ideas are acquired. Locke's analysis, if correct, would have the implication that those people don't really understand what they mean.

Consider one example (I'm going to improvise a bit on Locke's own arguments). Suppose I say that a trait is "innate" if I possess it at the moment of conception. Then almost nothing about me will be "innate." What I bring into the world with me is a genome, some cytoplasm, some organelles, and two cellular envelopes, an outer and an inner one. (There are also various relational facts about me, such as the city I'm born into, the parents I'm born to, my zodiac sign, and so on, but I'll ignore those for the time being.) Clearly, few of the facts about me, such as my eye color, skin color, sexual orientation, love of travel and philosophy, and so on, were traits that I literally possessed at conception. They took time to develop. So according to that definition, almost nothing interesting about me is innate.

But we can try another definition. Perhaps we can define an "innate ability" *not* as one that I possess at the moment of conception, but one that, at the moment of conception, *I'm capable of acquiring*. Under this definition, there are genuinely innate limits to what I can and can't do. I can never naturally grow webbed feet, but I can learn the alphabet. But Locke thought this makes every ability I ever acquire, such as my ability to speak English or wash dishes or memorize all the capitals of Europe, "innate." That's because, as a point of definition, if I eventually *do* acquire something, then I must have always been *capable* of acquiring it. Locke drove the point home succinctly: "… if the *capacity* of knowing be [the meaning of innate], all the truths a man ever comes to know, will, by this account, be every one of them innate" (Locke 1836 [1689], 10; emphasis mine).

One might object to Locke's line of reasoning here. One might argue, for example, that there is a real difference between *having* a capacity at the moment of conception, and *acquiring* a capacity later on in life. Fine. But anyone who takes this line has to spell out just what it is to have a capacity (or disposition), and what the difference is between having a capacity at conception versus acquiring a capacity later on (see Stich 1975 and Sober 1998 for discussion). (Consider: have I always had the capacity to write? Or did I acquire that capacity later in life? And if I acquired it later in life, when exactly did I get it?) I'm not saying it's impossible to spell all this out in a satisfying way. But it's not a trivial task. And to the extent that one doesn't know how to answer those questions, one doesn't know what it is to be innate.

The point here is *not* that "everything is *really* acquired," or that "humans are just a product of their environments," or that, "we're all blank slates at birth." That's just radical environmentalism, which is wrong. The point is to place the very distinction between innate and acquired into question. After all, suppose we decide that the "innate/acquired" distinction makes no sense. Not only would it be senseless to attribute any innate ideas or abilities to people, but it would also be senseless to claim that the human mind at birth is a "blank slate,"

and that everything we know, and everything we become, is a result of the impressions that rush in from the outer world. To reject the concept of innateness is to reject a flawed conceptual framework for thinking about people and their accomplishments.

4.2 INNATENESS AND LEARNING

About two hundred years after Locke, Francis Galton (Charles Darwin's cousin) resurrected the nature/nurture dispute. As Galton put the point, "Nature is all that a man brings with himself into the world; nurture is every influence from without that affects him after his birth" (see above). Unfortunately, Galton's work doesn't help us in coming any nearer to the meaning of "innate." For example, Galton's claim that innateness is, "all that a man brings with himself into the world," doesn't escape the kinds of problems that Locke raised. What I "bring with me" into the world is a bit of protoplasm, and what I become is the product of the interaction between that bit of protoplasm and everything else in the world.[1]

Although Galton's work did not help to clarify the *meaning* of "innate," it did help to build the modern science of genetics. Geneticists such as Karl Pearson and Walter Weldon enthusiastically carried his project forward and founded a school called "biometry". Though some of their assumptions were thrown out early in the twentieth century – most notably, that the genetic variation that is the source of evolution is fundamentally "continuous" rather than "discontinuous"[2] – figures like Weldon and Pearson transformed the early study of heredity by encouraging the use of sophisticated statistical techniques and the careful collection of large data sets. Galton also thought that studying identical twins would help us sharply distinguish between nature and nurture. At the very least, since identical twins share nearly all their genes, any differences between them should be due to environment and upbringing (Galton 1875).

Galton's provocative work triggered another round of debate about the very meaning of "innateness." Throughout the twentieth century, a steady stream of psychologists, social scientists, and biologists attempted to tear down the distinction, even as geneticists defended it. For example, the American psychologist Knight Dunlap, writing in 1919, exposed the "deplorable" misuse of the term "instinct": the very *idea* is "confused" and ambiguous, it leads to arbitrary classifications of behavior, and it's "capable of great abuse" (307). Although he was talking about "instinct" and not "innateness," his work ignited an "anti-instinct" crusade that soon came to target the idea of innateness as well (Boakes 1984, 217).

In the same vein, the Canadian psychologist Donald Hebb (1953) insisted that even asking the question of what is innate and what is acquired, "is a symptom of confusion" (43). Asking how much of a behavior is due to heredity and how much to environment is as meaningless as asking, "how much of the area of a field is due to its length, how much to its width." It's a kind of nonsensical question that has the superficial appearance of meaning. Hebb also argued that it's nearly impossible to exclude the effects of learning in the origin of behavior. Many of Hebb's arguments were developed by the American geneticist Richard Lewontin (1974) in a much more famous paper. The biologists Daniel Lehrman (1953) and, later, Patrick Bateson (1983), continued this line of attack. Philosophers associated with developmental systems theory also attacked the distinction (Oyama et al. 2001). This is not to say that people like Lewontin and

Lehrman thought that we should throw out the idea of innateness entirely. They just believed that the distinction is deeply problematic.

Not everybody was convinced by these sorts of critiques. One of the most famous defenders of the concept of innateness was Konrad Lorenz. In the 1960s, Lorenz wrote a small, influential book defending his position. He admitted that biologists were often unclear about what they meant by innateness, but said it could be scientifically defined. To say that a trait is "innate" is to say *the information about it is encoded in the genes*. This idea of defining innateness in terms of genetically encoded information redefined the nature/nurture dispute in the vocabulary of modern genetics.

One of the main concerns that Lorenz had was that if we got rid of the innate/acquired distinction, we would have no framework for analyzing behavior. "Commendable though semantic purism is," he noted, getting rid of the concept of innateness, "leaves us without a word denoting an indispensable concept … The obvious need for a term is a sure indication that a concept which corresponds to something very real does exist" (Lorenz 1965, 2). Simply stated, if we threw out the concept of innate, we'd just need to come up with another word to replace it. Isn't there *some* sense in which my preference for sugary drinks over bland drinks is innate, while my preference for Pepsi over Coke is acquired? If we got rid of the term "innate," we'd have to come up with some other term that does the job. The philosopher of biology William Wimsatt, though himself a critic of innateness, summarized this concern aptly: "for all of its checkered past, the distinction has permitted many insights which only the foolish would ignore" (Wimsatt 1986, 185).

Consider the case of what Lorenz famously called "fixed action patterns." These are relatively stable, stereotyped, complex adaptive behaviors that creatures exhibit with no obvious training or instruction. The male stickleback fish, for example, has a red belly and fights aggressively over territory. When it sees the red spot on other males, it goes into attack mode. In fact, it goes into attack mode when it sees pretty much anything with a red underside, such as a wax figure with a swab of red paint underneath. This is true even if a stickleback has never encountered any stickleback males before. Nobody needs to teach it the specific instructions, "if you see something that's red on the bottom, attack it." This suggests that the prerogative is somehow *inborn*, perhaps encoded in its genes.

Given its turbulent history, how might we, *today*, defend the idea of innateness – assuming we're inclined to defend it at all? There are two main routes that modern defenders of "innateness" tend to go.[3] The first is to rely on the idea of learning, and say that a trait is innate if *it's not learned*. This idea goes back to Plato, who thought that our most exalted concepts, such as justice and the divine, don't need to be "learned" but merely remembered. Some experience, of course, is required for stirring up these innate ideas from the depths of memory, but this is a qualitatively different process from learning. Echoing Plato, the philosopher Jerry Fodor says that concepts need only be "triggered" by experience, rather than "learned" (Fodor 1981; see Cowie 1999, ch. 4, for discussion). The second is to rely, as Lorenz did, on the idea of genetic causation or genetic information. Perhaps one should say that *a trait is innate if the information for it is encoded in our genes*. Both of them are flawed. In the next two sections, I'll focus on the idea of genetic causation and genetic information. Here, I will briefly discuss problems with defining an "innate" trait as simply one that is not "learned."

There are at least three main problems with defining "innate" as "not learned" (Bateson 1983, 53–55; Cowie 1999, 2009). The first, and most obvious, is that it only applies to things like ideas, beliefs, and behaviors, and not to physical characteristics like freckles or tattoos. At best it only illuminates a very limited portion of a much larger domain. Second, the very concept of *learning* is problematic. What do we mean when we say that something is "learned"? Typically we think of some kind of explicit instruction or modeling, say, from parent to child or teacher to student. More generally, we take it as anything we pick up from experience. But as the biological foundations of psychology become illuminated, we are gradually redefining learning in terms of brain activity. In fact, many neuroscientists equate learning with pretty much any activity-dependent neural change.[4] One consequence of these definitions is that for some neuroscientists, "learning" has become synonymous with almost all brain development, even prenatal! So this definition would make practically *all* psychological abilities a product of learning. We're right back to Locke's problem that even seemingly reasonable definitions of "innate" tend to balloon out to encompass everything.

Even friends of this kind of definition recognize that the line between the "learned" and the "unlearned" will move around as science progresses, and hence what counts as "innate" and "not innate" will move around as well. The philosopher Richard Samuels holds that something is "innate," in part, if, "it is postulated by some correct psychological theory," but, "no correct psychological explanation of its acquisition exists" (Samuels 2007, 25). In other words, he thinks a psychological ability is "innate" if psychologists recognize its existence but can't explain its origin (though perhaps neuroscience or biology can). This view is called "primitivism." He recognizes that his definition makes what's "innate" dependent on how we eventually decide to draw the line between psychology and biology (Samuels 2007, 36; Godfrey-Smith 2007). Anyone who has taken a neuropsychology class knows how elusive that line is. This doesn't defeat the concept of innate, but it suggests that it relies, in part, on human conventions.

A third problem is that, on pretty much any construal of "learning," it's almost impossible to exclude subtle effects of "learning" when we study the origin of a behavior. A traditional method for testing whether or not a behavior is "innate" is the *isolation experiment*. We attempt to isolate the creature from the sorts of environmental interactions from which it could learn the behavior, and then see whether it still exhibits this behavior. The problem here is that it's impossible to exclude all possible sources of interaction that have shaped the form of the behavior. For example, much of what we call "learning" begins *prenatally*. Mammals begin learning how to classify visual information in the womb as a result of spontaneous retinal activity. Is this "learned" or "unlearned"? It should probably be classified as "learned," though it would be easy to misclassify as "unlearned" if we ignore the learning that takes place in the womb. Chicks can peck at grain very shortly after hatching, suggesting that the behavior is "innate." Yet the series of muscle movements involved in pecking are practiced and perfected prenatally, as a result of the reverberations of the mother's heartbeat, suggesting that they are "learned" (Lehrman 1953, 134). Each stage of the chick's development is a result of the interaction between the previous stage of development and the signals that continually impinge upon it. Opponents of innateness fear that the concept of innateness could discourage detailed developmental research, since it treats certain aspects of behavior as "given," or as not

requiring any special explanation. This is certainly one of the concerns that led Griffiths (2002, 73) to call for abandoning the idea.

So much for defining "innate" in terms of not being learned. But perhaps we can define what it means for a trait to be "innate" in terms of being caused by our genes, or in terms of the information about the trait being encoded in our genes. After all, if anything is innate, then certainly things like eye color, hair color, or having thirty-two teeth, are innate. And if any information is encoded in our genes, then the same things are. In the next section we'll explore the prospect of defining innateness in terms of genetic causation.

4.3 GENES AND ENVIRONMENT

When we raise the question of what's innate and what's acquired, or what's nature and what's nurture, it's common for people to rephrase the question in terms of *genes* and *environment*. Which parts of how we are – our personalities or behavior – are caused by our genes, and which parts are caused by our environments? For many, the distinction between genes and environment is the safest route to tackling the old nature/nurture dispute. But the attempt to divide up psychological traits in any rigid or absolute way into parts that are "genetically caused" (or under "genetic control"), and into parts that are "environmentally caused" (or under "environmental control"), shares the same kinds of flaws as the attempt to divide up behavior in terms of innate and acquired.

There are at least two major ways that biologists think about the role of genes in evolution: as causal factors in the process of development from embryo to adult, and as sources of difference between individuals. As a consequence, we can adopt one of these two perspectives when we consider what it might mean to say that a trait is "innate." The first way of thinking about genes is more at home in developmental biology. Here, scientists think about genes as specific sequences of DNA that generate the proteins that regulate the way our bodies grow and function. The second way of thinking about genes is more at home in population genetics. Here, scientists mainly focus on the way a certain gene changes in its frequency in a population of individuals over time.

What this means is that there are at least two different ways of defining "innate," that mirror the two different standpoints for thinking about genes. From the first standpoint – that of the developmental biologist – to say that a trait is "innate" would mean, roughly, that a sequence of DNA exerts powerful control over the development of that trait. From the second standpoint – that of the population geneticist – to say that a trait is "innate" is to say that the differences between individuals with respect to that trait are strongly correlated with genetic variation rather than environmental variation. Perhaps it would not be too much of a stretch to say that the first definition is "causal" and the second is "statistical."

Let's adopt the perspective of the developmental biologist, who thinks of the gene as a cause of development. Perhaps we can simply say that a trait is "innate," so long as the trait is under rigid genetic control. For example, we might define a trait as being innate if it is closely associated with one or a very small number of loci (specific locations) on the chromosome (Sarkar 1998, 82). Yet there are two serious problems with this way of defining innateness.

First, why do we even divide up causes of development into precisely *these* two categories when we're trying to explain the emergence of a specific trait – the *genes* and everything *outside of them*? (A second, related, question is: what do we even mean by the word "gene"? – see Griffiths and Stotz [2013].) The genes – by which I will mean stretches of DNA coiled in our chromosomes – aren't the only biological units passed faithfully from parents to offspring. The mother's egg is filled with a viscous substance called cytoplasm. (The sperm has cytoplasm too; there's just much less of it.) When the sperm fertilizes the egg, there are chemical gradients in this cytoplasm that control which genes are turned on and which genes are turned off. Thus, the newly conceived embryo inherits not only its parents' DNA, but these cellular cytoplasmic gradients which tell the DNA how to function (Jablonka and Lamb 2005). But nobody says, "Roheni is aggressive because of some combination of her cellular cytoplasmic gradients and *everything outside of them*," even though logically it would be just as correct. Even the apparently innocent act of dividing the sources of behavior into "genes and environment" may tacitly bias the way we answer the question. Some philosophers of biology think that the gene is merely a *resource* that the cell uses to help make the proteins it needs to survive, rather than a kind of micromanager of the cell's doings (Moss 2003, 46; Sarkar 2005, 365–388; Keller 2010, 51). If that's right, then we probably should not give the genes the kind of starring role that we do when we describe development as an outcome of "genes and environment."

Second, even if we accept the legitimacy of this way of dividing up sources of development, figuring out *how much* the genes contribute to development and *how much* the environment contributes may be a senseless task. As Richard Lewontin put it, if Sally and Bill both lay bricks to make a wall, we can ask how much Sally contributed, and how much Bill contributed, by counting the number of bricks each laid. We can say things like, "Sally made 75 percent of the wall and Bill made 25 percent." But if Sally lays bricks and Bill lays mortar, it doesn't make sense to ask how much of the wall is due to Sally and how much due to Bill (Lewontin 1974, 402). This is the point Hebb made with his example of the width and length of a field. It is the same with genes and environment. The contributions they make are not only different, but "incommensurable," which means they can't be put on the same scale.

Elliott Sober (1988) provides a clear illustration. Consider the way that forces combine in Newton's physics. If one force hits an object from the east at 30 mph (miles per hour), and another hits it from the west at 40 mph, then the object will move west at 10 mph, in the absence of other forces. In this scenario, it makes perfect sense to ask what each force contributes independently of the other. For example, we can meaningfully ask questions like, "Which direction would the object move if the second force didn't exist?" Yet it doesn't make sense to think of genes and environment as separate forces that combine to explain a trait. We can't ask, "How tall would I be if I didn't have any genes?" or, "How much would I weigh if I didn't have an environment?" Hence, even in the context of developmental biology, it's very hard to spell out the idea of the gene as an agent of causation in any clear and precise way.[5]

As an alternative, we might try to define the idea of "innateness" from the perspective of population genetics. When population geneticists ask about genetic and environmental contributions to a trait, such as height, or weight, or eye color, or even schizophrenia, they're considering a certain population of individuals and they're asking to what extent the differences in, say, height amongst the members of this population are correlated with differences in genes,

and to what extent they're correlated with differences in environmental variables (such as food intake). The measure of the genetic component of variation for a trait is called the "heritability" of that trait (more specifically, "broad sense heritability" – see Sarkar 1998). Heritability is a statistical idea. It says nothing about the mechanics by which genes contribute to development, though as the biologist and philosopher Evelyn Fox Keller (2010) points out, it can be difficult for people to keep these ideas separate in their minds. Specifically, people tend to think that if a trait shows high heritability, then genes exert a lot of control over its development, though these two things are logically independent. At any rate, perhaps we should just say that a trait is "innate" if it has a high heritability, that is, if it scores high on this statistical measure.

There are at least three main problems with defining "innateness" in terms of heritability. One problem is that heritability depends heavily on the particular group we choose to sample, and the particular set of environments that we're focusing on. It also depends on the precise genetic composition of the population. For example, suppose we sample a population of healthy, affluent US residents. It is possible that the heritability of weight would be high – that is, that the differences between them with respect to weight are primarily correlated with genetic differences (though that's not necessarily the case). If we broaden our sample to include *both* affluent US residents, *and* people who are malnourished as a result of famine, the heritability of weight will almost certainly be much lower (since a greater proportion of the outward variation is due to environmental factors). Hence, the heritability of a trait is not a fixed property of that trait, but is relative to the group of individuals that are sampled and the environments sampled. The philosopher Ned Block, who's mainly known for his work in philosophy of mind, has a lucid paper on the pitfalls of heritability (Block 1995).

A second problem is that heritability only applies if the genetic and environmental components of variation are statistically independent of one another. But that doesn't always happen. Consider a fictional community in which taller children are given more food than shorter children. Then it's quite possible that genetic and environmental sources of variation for height differences will not be statistically independent. In this case, heritability does not apply.[6]

Finally, the notion of heritability certainly doesn't measure what most people intuitively have in mind when they think about what genes contribute to a trait. For example, consider the trait of walking on two legs. Certainly, our ability to walk on two legs is influenced by our genes. Typically, however, the only reason that people don't walk on two legs is because of illness or accident. Hence, most of the variation for the trait is due to the environment. As a consequence, the genetic component of variation for the trait of walking on two legs – its broad sense heritability – is zero, or close to zero, in most populations. But that doesn't mean genes don't contribute to it! In short, a high level of heritability doesn't indicate genetic causation, just as a low level of heritability doesn't indicate the absence of genetic causation. This was the fatal flaw underlying famous studies that attempted to use twins to estimate the heritability of intelligence and other personality traits: they committed the fallacy of making inferences about genetic causation from high heritability scores (Sarkar 1998, ch. 4).[7]

Here's the main issue: if we define "innateness" in terms of "heritability," we have to concede that what's innate is relative to the group that we're talking about and the specific environment they're placed in. But people usually think that what makes a trait "innate" is somehow independent of these sorts of extraneous factors. So we're at a standstill.

Incidentally, the fact that heritability is relative to the specific population and environment has consequences for bioethics, particularly genetic engineering or even genetic counseling (Tabery 2009). A study on the interaction of genes and environment in antisocial personality disorder illustrates the problem. The research team of neuroscientist Avshalom Caspi purported to show that antisocial personality disorder best correlates with a mix of genetic and environmental risk factors (Caspi et al. 2002). Their assumption was that a certain gene variant (*MAO-A* gene) regulates a certain brain enzyme (MAO-A) associated with impulse control. This crucial assumption has been called into question, though I'll still use their story for the sake of illustration (Fowler et al. 2007; Alia-Klein et al. 2008; see Longino 2013, 97, for discussion). People with *both* the gene variant *and* a rough childhood were very likely to have antisocial personality disorder. Having the gene alone is not enough. So far, this merely shows that genes and environments sometimes interact to produce mental disorders. However, here's the exciting part: people with the gene were *less likely* to develop antisocial personality disorder if they were raised in nurturing homes, than those without the gene in nurturing homes! The gene doesn't have a consistent effect across environments. In some environments, it "predisposes" one *toward* antisocial personality disorder; in others, it "predisposes" *against* antisocial personality disorder. We can't say that there's a "gene for" antisocial personality disorder in any absolute sense.

The fact that the supposed gene for antisocial personality disorder has different effects in different environments is an example of a general phenomenon called *phenotypic plasticity* (Pigliucci 2001). Because of phenotypic plasticity, some philosophers don't even like talking about a "gene for" this or that, as in a "gene for" eye color or "gene for" anxiety. But there's probably nothing wrong with talking about a "gene for" height, or a "gene for" eye color, or a "gene for" anxiety, so long as it's clear that all we mean is that this particular stretch of DNA makes a difference to height, or eye color, or mood, *in this particular group and set of environments*. This is sometimes called the notion of gene as "difference maker" (Sterelny and Kitcher 1988; Sterelny and Griffiths 1999, ch. 4). According to this idea, to say that there's a "gene for," say, obesity, is to say that there are segments of DNA that make a difference to obesity, relative to certain groups and environments, and relative to alternative segments of DNA. This isn't particularly problematic, since the notion is explicitly understood in a context-and-environment-dependent way.

A good way of describing and displaying the interaction of genetic and environmental components of variation is through the use of *norms of reaction* (Sarkar 1999). These are graphs that display the different phenotypes that arise when a given genotype is placed in different environments. In other words, the norm of reaction displays information about phenotypic plasticity. This conveys precisely the kind of information that geneticists are interested in, but avoids the appearance that we can meaningfully talk about a "genetic" component of variation in some non-relative way.

4.4 INNATENESS AND GENETIC INFORMATION

As I indicated in the last chapter, some have tried to define "innateness" *not* in terms of genetic causation, but in terms of genetic *information* – that is, the information that's encoded in the

genes. As I see it, there are two main problems with this approach. First, it inherits all the problems of trying to define genetic causation; secondly, it inherits the problem of the "indeterminacy of content," a problem that will be developed in more detail in Section 7.4.

The idea here is that a trait is "innate" if *the information about that trait is encoded in our genes*. I have brown eyes because my genome encodes information about brown eyes. This was the way that Konrad Lorenz himself tried to define innateness. In other words, he agreed that *traits* per se are not "innate," but only the information fixed in the genome as a result of natural selection. He thought that isolation experiments could sharply distinguish between the "information" acquired by the genome over an evolutionary span of time, and the "information" acquired by the individual organism as a result of learning about its environment. He insisted that the two sources of information don't blur together in an unanalyzable way. Moreover, he took it for granted that though the genome encoded this information, an environment was required to "realize" it or bring it into being: "No biologist in his right senses will forget that the blueprint contained in the genome requires innumerable environmental factors in order to be realized" (Lorenz 1965, 37).

According to this way of speaking, the genes – certain sequences of DNA coiled in one's chromosomes – are like coded messages. They're something like the sequence of dots and dashes in the Morse code, or a computer program that runs on instructions "encoded" in a computer language like C++ or Java. This way of thinking about DNA, as containing coded instructions about a phenotype, is attractive, no doubt in part because scientists often do talk about the DNA as a kind of "code." One of the great achievements of molecular biology in the 1960s was cracking the "genetic code," that is, discovering that certain segments of DNA typically correspond to certain amino acids, the building blocks of proteins.

But is this way of speaking anything more than a metaphor? Unlike a computer program, DNA has no author – it's a product of random drift and natural selection. Moreover, unlike a set of instructions, there is nobody to "read" them off. The idea of sending and receiving information makes sense when we're talking about human beings talking with one another or exchanging letters or emails. Similarly, the idea of "coded information" is at home in the realms of human communication and, by extension, some forms of electronic engineering that have the function of helping people communicate. Is there any literal sense in which genes encode information? Certainly, some philosophers have dismissed this whole way of speaking as a metaphor that's gotten out of hand (Sarkar [1996] suggests this position; Griffiths [2001] endorses this view). This way of thinking is sometimes called *eliminativism*. For example, one is an *eliminativist* about "biological information" if one thinks that biology would be better off if scientists just stopped talking about things that way, because such talk can only obscure the true causes of things.

My own inclination is to think that the idea of genetic information is *not* a metaphor, but possesses some literal meaning. The reason is that talk of biological information is rampant not only in genetics, but also in neuroscience, ethology (animal studies), and developmental biology. Many neuroscientists, for example, seem to think that the primary function of the nervous system as a whole is to *receive*, *transmit*, and *process* information (Garson 2003, forthcoming). The same goes for animal studies. One branch of ethology (behavioral biology) is the study of animal communication, that is, the way that animals signal various pieces of

information to one another, though this way of speaking is controversial – see Section 7.4. And developmental biologists are comfortable describing how organisms extract information from various environmental cues about fitness-relevant challenges or opportunities. So perhaps if we figure out what neuroscientists, or behavioral biologists, or developmental biologists, mean when they talk about information, we could decipher what geneticists mean when they say that genes encode information. Moreover, just because one believes in genetic information, this doesn't make one some kind of crude genetic determinist. We can say that genes "carry information" about developmental outcomes without saying that genes *alone* carry such information (Sterelny et al. 1996).[8]

Even if we accept this approach to innateness, it's not clear how this would allow us to make any progress on figuring out *which* traits are, in fact, innate. This is for two reasons. First, the concept of biological information is, as it currently stands, highly indeterminate. Even if genes carry information about *something*, that doesn't tell us *what* they carry information about. This is called the problem of content indeterminacy. Short of a solution to the content indeterminacy problem, the idea of genetic information won't allow us to say with any precision what's innate and what's not. Peter Godfrey-Smith (1999) makes this point in terms of coding: even if a sequence of DNA "codes for" *something*, this doesn't allow us to say it "codes for" some observable trait such as eye color. Indeed, geneticists typically describe DNA as "coding for" proteins, and little else. In Chapter 7 I'll develop this kind of "content indeterminacy" problem.

Secondly, even if we solve the indeterminacy problem, presumably the only way to find out whether or not a gene *carries information* about a trait is to see whether or not the gene usually *causes* that trait. But this throws us right back to the problem of determining genetic causation, which, as I've noted, is very difficult to measure. If we don't have a context-independent way to figure out how a gene contributes to a trait, then we have no way to figure out what information the gene actually encodes. That should be cold comfort to the champions of innateness. Even one of the contemporary proponents of this idea of genetic information, the philosopher Nicholas Shea, doesn't think that we should appeal to genetic information to explain what *causes* actual development. This genetic information, he thinks, isn't part of the "causal mechanism" that takes us from embryo to adult (Shea 2007, 319).

A final idea about innateness that deserves mention here is the idea that innateness is a relic of a prescientific way of thinking about the world. Some have argued that the "innate/acquired" distinction reflects a way of thinking that children typically pass through as they try to get a grip on what makes things alive ("folkbiology") (Griffiths 2002; Griffiths and Machery 2008; Linquist et al. 2011). From this perspective, despite the temptation to embrace a simple distinction between the innate and the learned, we might come to see *that very temptation as a psychological phenomenon to be explained* rather than an indicator of a deep fact about the nature of reality, kind of like the temptation to think that people who look different than us are probably dangerous.

On the basis of these kinds of criticisms, some recent philosophers have argued that we should get rid of the concept of innateness altogether, or substantially reform it. In the next section, I'll develop the idea – argued most notably by the biologist Patrick Bateson – that we replace the innate/acquired distinction with the distinction between robustness and plasticity. This is not to define "innateness" in terms of "robustness." Rather, it's to acknowledge that the innate/acquired distinction represented a fumbling attempt to get at something very real.

4.5 ROBUSTNESS AND PLASTICITY

If we scrap the innate/acquired distinction, is it true, as Lorenz forewarned, that we'd have to give up the project of analyzing human behavior or its development? Not at all. In the last section, I noted, following Wimsatt, that the distinction represented an attempt, albeit a confused and fumbling one, to get at real biological phenomena. Getting rid of innateness opens the door to the more constructive project of describing the phenomena in question and coming up with a better way of thinking about them. (A second question is why this new way of thinking isn't just another definition of "innateness.") In this section I'll develop the idea that we replace the innate/acquired distinction with the distinction between robustness and plasticity (Bateson and Gluckman 2011; also see Keller 2010, 75).[9]

To clarify, there are two important positions here, which differ in how extreme they are.[10] The first position is that the innate/acquired distinction is deeply problematic, but can be salvaged or reformed. The second is that the innate/acquired distinction should be abolished. The latter view goes on to claim that, to the extent that there is a legitimate biological phenomenon at issue here, it can be adequately captured by some other distinction, such as that between robustness and plasticity. William Wimsatt and André Ariew argue for the former position. Paul Griffiths and Matteo Mameli (see Mameli and Bateson 2006) argue for the latter, as does the biologist Patrick Bateson. I place myself in the latter camp as well.

Here is a striking feature of biology: certain characteristics are reliably produced, or repeated, generation after generation. Human beings have one head, rather than two or none. We also give birth to other humans, rather than to ostriches or mice. We all have much the same inner cluster of organs, and, at a very general level, the building blocks of our minds are very similar. People have thoughts and emotions, and they make inferences about cause and effect. Of course, sometimes things go wrong, and a child is born without a limb, or an organ. But these are the exceptions that prove the rule. These traits can be termed "robust."

Because biologists and philosophers often use terms differently, I'll take a moment to clarify what I mean. To say that a trait is "robust" is to say that it appears reliably, in spite of environmental and genetic variation. Robustness is not mere repetition. Biologists are very good about ensuring repeatability in their model organisms through careful genetic selection and by maintaining highly artificial, uniform environments. But this doesn't mean that the traits they've selected in this way are robust. "Robustness" has the additional sense of repeatability across a wide variety of environmental and genetic changes. "Robustness" is sometimes used interchangeably with "canalization," an idea that the English biologist Conrad Waddington (1957) promoted in the mid-twentieth century. Nijhout (2002), for example, uses the terms interchangeably. People use the term "canalization" in different ways, however, so I'll stick with robustness, because it's a newer term and there is probably less historical baggage.

Three things to notice about robustness are that it is a degree property, it is a relative property, and it describes a phenomenon rather than a mechanism.[11] First, robustness comes in degrees. One trait can be more robust than another with respect to a set of environmental and genetic features. It's not all-or-nothing. The property of having eyes is robust. The property of having brown eyes is also robust, but less so. Second, strictly speaking, we shouldn't say

that a trait is "robust," but robust-relative-to-certain-developmental-factors. The property of having four limbs is robust with respect-factors to many different genetic mutations, and it is robust with respect to many different changes to the fetal environment, such as changes to maternal nutrition. But it is not robust relative to the presence or absence of thalidomide, a morning sickness drug widely sold to pregnant women in the 1950s and 1960s that caused a large number of limb deformities. Finally, when I talk about robustness I'm just referring to a statistical phenomenon, rather than any particular mechanism that causes it. The question "what causes robustness?" is an interesting and lively research question.

The alternative to robust is "plastic." This term, like "robust," has multiple meanings. One sense of the term used in the last section, typically associated with "phenotypic plasticity," describes the way that one and the same gene can give rise to different phenotypes in different environments, such as the example of antisocial personality disorder (see Section 4.3). Plasticity, in the sense I'll focus on here, is merely the opposite end of the spectrum from robustness. It just means, "not very robust." It varies quite readily in the face of genetic or environmental changes.

A major difference between robustness and plasticity, on the one hand, and innate and acquired, on the other, is that the innate/acquired distinction is framed in terms of inner and outer: what's "inside" and what's "outside." I suspect that one reason people tend to slide from the gene-environment distinction to the innate/acquired distinction is because they both attempt to distinguish the contributions of inner and outer causes in making us the way we are. The robustness/plasticity distinction doesn't fit into the inner-outer distinction, but cuts across it. The property of having a normal body mass index is robust for human beings, but involves both genes and environment, inner and outer, without assuming that we can segregate strictly the specific contribution of each. So does the property of having one head rather than two or none. To put it in sophisticated terms, it does not involve "apportioning causal responsibility" to genes and environment.

Another good example of a robust property, one that's intuitively *not* "innate," is the tendency of baby geese to follow around their mothers. One of the crucial mechanisms here is imprinting, where the gosling forms a strong preference for an object it encountered at a certain period early in life (see Section 8.4). In artificial conditions, geese can imprint on people, on other animals, or even on toy trains. As a consequence, if anything is "acquired," then the tendency of geese to follow around their mothers is. Yet it is reliably produced in many different environments even under many different genetic changes, so it is relatively robust.

The biologist Patrick Bateson is the main proponent of scrapping the innate/acquired distinction and using the idea of robustness and plasticity as defined here. However, two other approaches, due to the philosophers of biology William Wimsatt and André Ariew, respectively, are very similar in spirit, though they differ in fine details. Wimsatt urges that we reform the idea of innateness via the concept of "generative entrenchment."[12] "Generative entrenchment" points to the way that certain features of the organism, or certain stages of development, rely on others. To say that a feature or a developmental stage is "deeply entrenched" is to say that many other features or stages depend on it.

This has the implication that major changes to, or modifications of, these "deeply entrenched" features may have massive ramifications for everything that depends on them. The process of neurulation, for example, is essential for the development of the brain and spinal cord, and

hence for all of the anatomical, psychological, and behavioral characteristics that depend on it. (Neurulation takes place about four weeks after conception. A sheet of cells folds into a tube, the neural tube, which develops into the brain and spinal cord.) Disruptions to the process of neurulation are typically devastating and fatal.

The idea of generative entrenchment is closely related to robustness. That's because generative entrenchment can *explain* robustness. If a trait, or developmental stage, is deeply entrenched, that means lots of other things depend on it. As a consequence, we should expect deeply entrenched properties to be protected from disruption. That is, we should expect that evolution would have managed to come up with adaptations that protect them from being disrupted. As a consequence, we should expect them to be robust – to arise reliably in the face of a lot of environmental or genetic variation.[13] Interestingly, we should also expect robust traits – traits that develop reliably in the face of variation – to become deeply entrenched over time. Because they are stable, we should expect evolutionarily novel traits to be built "on top of" the robust ones, that is, evolutionarily novel traits should come to depend on robust traits over time.

Ariew adopts a slightly different approach. His idea is that we should define "innateness" in terms of what he calls "canalization." The first thing to notice, then, is that Ariew doesn't think we should eliminate "innateness." Canalization, however, is closely related to what I've called "robustness." As a consequence, there is a kind of natural affinity amongst these approaches. A second difference between Ariew's approach and Bateson's is that Ariew defines canalization in terms of a trait's being insensitive to *environmental* changes, rather than insensitive to both environmental and genetic changes. Traditionally, the idea of developmental canalization (owing mainly to the biologist Conrad Waddington in the 1940s and 1950s) was meant to describe traits that are reliably produced in the face of *both* environmental and genetic changes (Waddington 1957, 42). For example, gene duplication is a mechanism for ensuring the constancy of a phenotypic trait in the face of novel genetic mutations, and hence it contributes to robustness (or canalization in the traditional, broad sense).

The ideas of robustness and plasticity, thought of as statistical properties of groups rather than as specific mechanisms, open up a vast research field because they invite us to explore the various biological mechanisms that cause them (Bateson and Gluckman 2011, 20–29). In the simplest case scenario, a trait is robust with respect to some variation because the developmental process that produces the trait is shielded from that variation. The mammalian placenta functions quite literally as a barrier that protects the fetus from a lot of physical changes taking place on the other side. Hence it is a mechanism of robustness. Another mechanism is redundancy, one example of which is duplication. Our kidneys are paired for the same reason that our chromosomes are paired, to provide a crucial backup system in case one member of that pair breaks down. Repair and regulation refer to other ways that the body ensures constancy in the face of change. The same point goes for plasticity – there are several mechanisms that explain it. I'll come back to this in Chapter 8, where I'll suggest that certain mental disorders may represent the outcome of phenotypic plasticity, and that this may have profound implications for how we think about psychiatric research and classification.

CHAPTER SUMMARY AND SUGGESTED READINGS

The Greek philosopher Plato, in his dialogue, *Meno*, wondered whether virtue is innate or whether it can be taught. By asking this, Plato was opening the "nature/nurture" dispute: which parts of who we are come from nature? Which from nurture? Another way of putting it is in terms of what's "innate" and what's "acquired." For nineteenth-century scientists like Francis Galton, solving the nature/nurture riddle not only was a matter of intellectual satisfaction, but had urgent social weight. The purpose of this chapter is not to *answer* the question, but to "deconstruct" it, that is, to suggest that the formulation is not entirely coherent.

First things first: what does it even *mean* to say that something is "innate"? In Section 4.1, I went back to the seventeenth-century philosopher John Locke to show why the question is more perplexing than it might seem on the surface. In the next three sections, I examined three different attempts to answer the question. One defines what's "innate" as the opposite of what's "learned" (Section 4.2). Another says that a trait is "innate" if it is under "genetic," rather than "environmental," control (Section 4.3). I pointed out that there are actually two versions of this thought, depending on how we think about genes. In Section 4.4, I examined the idea that a trait is "innate" if the *information* about the trait is encoded in the genes. All of these attempted characterizations of "innate" came up short.

Finally, in Section 4.5, I suggested that we scrap the "innate/acquired" distinction and replace it with the distinction between "robust" and "plastic." The robustness/plasticity distinction recognizes that certain traits are developmentally "robust" – that is, they appear reliably under substantial environmental and genetic variation. Yet, unlike the concept of innateness, the concept of robustness makes no attempt to specify *which* of genes or environment plays a more important role in the development of a trait. It cuts across the innate/acquired distinction, yet manages to capture an important element of it. I contrasted this view with two other ideas, that of generative entrenchment and that of canalization. I think the distinction between robustness and plasticity, unlike the distinction between innate and acquired, heralds an exciting research paradigm.

The best recent starting point for exploring the controversial concept of innateness is the third volume of the three-volume set, *The Innate Mind*, edited by Carruthers et al. (2007). The first part of that volume has articles by philosophers of biology and gives a good overview of the state of play. Fiona Cowie's (1999) *What's Within?* gives an overview of the troubles with innateness in psychology. Paul Griffiths' (2002) short article, "What Is Innateness?" is a provocative call for abolishing innateness to which many of the papers in *The Innate Mind* are responding.

The classic text on "apportioning causal responsibility" between genes and environment is Lewontin (1974). He claims, effectively, that we have no way of answering the question of "how much" of a trait is caused by the genes, and "how much" by the environment. For other criticisms of the notion of heritability, particularly when it is used to attempt to quantify the contribution that the genes make to a trait, see Block (1995), Sarkar (1998, ch. 4), and Bateson and Gluckman (2011).

A good entry point for thinking about the concept of *information* in biology is a collection of articles published in 2000, in the journal *Philosophy of Science* (volume 67). A good, recent entry point for thinking about the complexities involved in defining "gene" is Griffiths and Stotz (2013).

The best entry on the concepts of robustness and plasticity is a recent, short book by the biologists Patrick Bateson and Peter Gluckman (2011), *Plasticity, Robustness, Development, and Evolution*.

NOTES

1 Evelyn Fox Keller (2010, 24–6) argues that Galton was able to put the "nature/nurture" distinction so sharply because he was relying on Darwin's "particulate" theory of inheritance, which is the idea that the hereditary factors that make children like their parents were largely due to atom-like substances thrown off by the cells. This idea made it easier, she argues, for Galton to think of them as strictly separate from the child's environment.

2 The continuity versus discontinuity debate set the biometricians against the so-called "Mendelians" such as Hugo de Vries, William Bateson, and Wilhelm Johannsen. The debate was ultimately resolved through the statistical acumen of Ronald Fisher (1918), who showed that the kind of continuous variation that the biometricians discussed could be modeled as the outcome of a large number of small, discontinuous changes (Provine 1971; Sarkar 2004).

3 Sometimes, proponents of "innateness" are called "nativists," but the distinction between "nativism" and "empiricism" is a debate within psychology, and I want to examine the concept of innateness more generally – regardless of whether it applies to ideas or abilities – so I will avoid that way of framing the question.

4 E.g., Hebb (1953, 47) describes learning as, "unidirectional change of neural function, resulting from sensory stimulation." See Kandel et al. (2013, ch. 65), for an overview of contemporary theories of learning. Also see Garson (2012) on activity-dependent and activity-independent synapse formation.

5 Sober points out that one could reformulate the manner of delegating causal responsibility so that the analogy to Newtonian forces is not relevant; we could ask, instead, "how tall would I be if my genes were different?", and this is a meaningful question. Yet this response, as he points out, makes the answer heavily dependent on which alternative "worlds" we choose to entertain.

6 It's true that the standard approach to assessing heritability, the analysis of variance, includes a "gene-environment interaction" factor, often designated "G × E." But, as Elliott Sober (1988, 308) puts it, G × E is merely a "fudge factor" to make the calculations turn out right; it is not intended, nor is it able, to characterize these systematic interactive effects. "G × E" does not even appear in some models (e.g., Layzer 1974).

7 Thomas Bouchard, a scientist who has been promoting heritability research on twins for decades, provides a recent defense (Bouchard 2007) but neglects to take long-standing conceptual problems with heritability into account.

8 No doubt, part of the hostility amongst philosophers against talk of "genetic information" is connected with the idea that what makes genes a "special" kind of cause is that genes *alone* carry such information (Oyama 1985; Griffiths 2001). (Maynard Smith 2000 is one of the main advocates of this "exclusivist" view of genetic information.)

9 Wimsatt (1986, 1999) and Ariew (1999) develop similar kinds of approaches. I'll characterize their differences later in this section.

10 I thank Sahotra Sarkar for encouraging me to clarify this point.

11 See Nijhout (2002) for a clear and careful exposition. My sense is that Waddington (1957) accepts the same three points. With regard to the last, he states that, "the epigenetic landscape … expresses properties which are essentially formal in nature" (47). In other words, he seems to want to abstract the idea of canalization from the biological mechanisms that give rise to it.

12 Sometimes Wimsatt seems to suggest that he adopts eliminativism about innateness (e.g., Wimsatt 1986, 186). At other times he suggests that generative entrenchment is part of his proposed definition of "innate" (see, e.g., Wimsatt 1999, 153).

13 Two other interesting consequences are these. First, deeply entrenched features should be *taxonomically general*, that is, common to many different species. The early stages of embryo formation, for example, look almost identical in many different species, presumably because they are deeply entrenched – that is, because so many other developmental processes and features depend on them. Second, deeply entrenched properties should be slow to evolve, since they are "buffered" from typical causes of variation.

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