7 Innateness Is Canalization: In Defense of a Developmental Account of Innateness

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Lorenz proposed in his (1935) articulation of a theory of behavioral instincts that the objective of ethology is to distinguish behaviors that are "innate" from those that are "learned" (or "acquired"). Lorenz's motive was to open the investigation of certain "adaptive" behaviors to evolutionary theorizing. Accordingly, since innate behaviors are "genetic," they are open to such investigation. By Lorenz's lights, an innate/acquired or innate/learned dichotomy rested on a familiar Darwinian distinction between genes and environments. Ever since Lorenz, ascriptions of innateness have become widespread in the cognitive, behavioral, and biological sciences. The trend continues despite decades of strong arguments[1] that show, in particular, the dichotomy that Lorenz invoked in his theory of behavioral instincts is literally false: No biological trait is the product of genes alone. Some critics[2] suggest that the failure of Lorenz's account shows that innateness is not well defined in biology and that consequently the practice of ascribing innateness to various biological traits should be dropped from respectable science.

Elsewhere (Ariew 1996) I have argued that despite the arguments of critics, there really is a biological phenomenon underlying the concept of innateness. On my view, innateness is best understood in terms of C. H. Waddington's concept of "canalization," that is, the degree to which a trait is innate is the degree to which its developmental outcome is canalized. The degree to which a developmental outcome is canalized is the degree to which the developmental process is bound to produce a particular endstate despite environmental fluctuations both in the development's initial state and during the course of development. The canalization account differs in many ways from the traditional ways that ethologists such as Konrad Lorenz originally understood the concept of innateness. Most importantly, on the canalization account the innate/acquired distinction is not a dichotomy, as Konrad Lorenz had it, but rather a matter of degree—a difference that lies along a spectrum with highly canalized development outcomes on the one end and highly environmentally sensitive development outcomes on the other end. Nevertheless, I justified the canalization account on the basis of a set of desiderata or criteria that I suggested falls out of what seemed uncontroversial about Lorenz's account of innateness. Briefly: Innateness is a property of a developing individual; innateness denotes environmental stability; and innate-ascriptions are useful in certain natural-selection explanations (more below). From that same set of desiderata I argued (1996) that neither the concept of heritability nor of norms of reactions—two concepts from population genetics—suffices to ground innateness.

In this chapter, I wish to provide further support of the canalization account in two ways. First, I wish to better motivate the desiderata by revisiting a debate between Konrad Lorenz and Daniel Lehrman over the meaning and explanatory usefulness of innate ascriptions in ethology. Second, I wish to compare my canalization account of innateness with accounts proposed by contemporary philosophers, one by Stephen Stich (1975), another by Elliott Sober (1998), and a third by William Wimsatt (1986).

1 Lorenz's Theory of Instincts

Within Lorenz's theory of instincts, the theoretical significance of identifying innate traits is threefold: it is useful in taxonomy, in individual explanation, and in evolutionary explanation (Richards 1974). Roughly:

(1) Taxonomy involves characterizing species-specific traits as opposed to traits that pick out differences between individuals in a species or a population.

(2) Individual explanation: Citing innate behavior may explain why individual organisms act as they do in certain circumstances. For example, why does a stickleback attack a wax model of a fish that lacks all structural resemblance to a rival stickleback? The answer is that the model has a red underbelly, which serves in sticklebacks to release an innate attacking behavior (Richards 1974).

(3) Innate traits are genetically determined and hence are subject to evolutionary investigation and explanation. Lorenz sought to provide natural-selection explanations for certain "adaptive" species-specific behaviors.

According to Lorenz, field observations and "isolation experiments" constitute the empirical investigation of behaviors. In an isolation experiment, the organism under study is deprived of the opportunity to "learn" or acquire the candidate behavior from environmental cues. If the organism undergoing isolation develops the trait "normally" then the trait or behavior is said to be innate. For example, Lorenz observed that female mallards raised to reproductive age in exclusive company of pintail ducks show no sexual affinity for the pintail drakes. But upon seeing a male mallard for the first time, the females immediately engage in the sexual courtship behavior particular to their species. Remarkably, a mallard expresses courtship behavior even if it has had no opportunity to learn it. That is, mallards that are naturally or experimentally deprived of the opportunity to observe courtship behavior tend to develop it nonetheless. Determining that the mallard's courtship behavior is

innate allows the investigator to (1) include the behavior as part of the taxonomy of mallards, (2) explain why mallards exhibit courtship behavior, and (3) open the study of courtship behavior to evolutionary investigation.

As Lorenz's critics have demonstrated, however, Lorenz's account is unsatisfactory. To attribute behavior to the genes is to misunderstand how biological traits are manifested. No biological trait is the instantaneous product of genes alone or of the environment alone. Biological traits are the product of a developmental process that depends on the actions and interactions of *both* genes and environment. Lorenz thought that the results of deprivation experiments showed that some traits appear "out of a vacuum" (Lehrman 1953). Yet, even behaviors manifested in deprivation depend on *some* environmental interaction, namely the environment of the deprivation device (assuming that the creature is kept alive). It follows from this truism that no phenotype is the product of genes alone.

2 The Lorenz-Lehrman Debate

Lorenz devoted a small book to respond to his critics (Lorenz 1965). In his response, Lorenz makes some concessions though he maintains that the category of innateness is useful to ethology. Lorenz's main concession is to acknowledge that attributing behavior to the genes is misleading in the context of a developmental explanation of behavior. Nevertheless, Lorenz asserts that the categories of innate and acquired (or learned) are explanatorily useful (and hence ought not be dropped) as they serve to articulate the "adaptiveness" of certain behaviors. I think that Lorenz's response is significant. It reveals that the exchange between Lorenz and his critics is in part a clash of divergent explanatory strategies or approaches to explain the nature of organic life.

What is clear is that Lorenz's interest in the category of innateness is to explain an evolutionary phenomenon, adaptiveness. What does it mean to be interested in explaining adaptiveness? Adaptiveness is a relation between an organic form and its habitat. Within a particular habitat, an adaptive trait is one that confers relative survivability and/or reproductive success to those individuals who possess it. Darwinists explain adaptiveness by appeal to the theory of natural selection. More precisely, since evolution is a population-level phenomenon, what Darwinists explain is the origins and prevalence of adaptive traits in a population of organisms.

In contrast, Lorenz's critics are in large part developmental biologists, interested not in the relation between organic form and its habitat but in the organic form itself. Further, developmentalists are not necessarily interested in prevalence of organic form, but rather in its origins in an individual embryo.

Given this distinction between what evolutionary theorists and developmentalists seek to explain, I think we can clarify Lorenz's response to his critics. He admits that attributing behavior to the genes is an error if the point is to explain how a trait comes to develop in an individual (the developmentalist project). However, if the point is to explain the characteristics of adaptiveness (the evolutionist project), then attributing behavior to the genes is part of a standard Darwinian evolutionary explanation. Let me briefly explain why Lorenz might have thought this.

There are many points of intersection between evolution and development, but one difference is that evolutionary explanations often "black-box" development. That is, evolutionary theorists often talk about the genetic transmission of phenotypes without regard to what might be a complex correspondence between genotypes and phenotypes. From the evolutionary theorist's point of view, what is important is not knowledge of the causal factors that lead individuals with a given genotype to *develop* particular phenotypes, but that genotype and phenotype are correlated in a way that allows investigators to examine an evolutionary phenomenon (Sober 1984; Amundson 1994). In other words, the developmental story that links genes to phenotypes is not the central issue for evolutionary theorists, though it is important that that there is some developmental story to tell.

Although both explanatory strategies, the developmentalist's and the evolutionary theorist's, appeal to the concept of "gene" and "environment," in the case of genes what counts as the significant causal role diverges for each strategy, and in their uses of the term "environment," their meanings (and purported references) differ. For evolutionary theorists (especially adaptationists), genes serve necessarily as the unit of inheritance and sometimes serve as the unit of selection. The laws that dictate how genes play that role (e.g., Mendelian and non-Mendelian laws of segregation and assortment) are insensitive to the molecular composition of genes (i.e., the fact that genes are bits of chromosomes composed of DNA in the form of a double-helix, etc.). But for the developmentalist, genes play a crucial biochemical role in the determination of organic form. Genes code for amino acids that determine the 3-D folding structure of protein molecules, and so on. Further, in regard to what counts as the "environment," what distinguishes modern-day epigeneticism from pre-nineteenth-century preformationism is the tenet (truism) that genes alone do not produce biological traits. Development involves complex interactions between a few or many genes, between genes and other components of a cell, etc. Everything outside of a particular gene is "the environment" for developmentalists.

The evolutionary theorist (again, especially the adaptationist) regards the environment as part of the "selective regime" including all conditions external to the organism itself (or outside the unit of selection): resources (food, shelter), existence of predators, climatic conditions, topography, location of mates, etc. Typically, they gloss over the role of the environment in the development of individuals. Are they justified in doing so?

Consider how natural selection explains the prevalence and maintenance of biological traits. Selection operates over *variants* in a population. Certain organisms, because of particular traits they possess, are better suited to survive and reproduce than their conspecifics in the environment they all inhabit. The key is that an adaptationist is interested in trait differences between organisms exhibited in an environment where selection is going on. In a particular selective regime, the factors of the environment that instigate selection (climate, resources, etc.) are *fixed* as far as the evolutionary theorist is concerned. The phenotypic differences are all due to genetic differences (much of those genetic differences are likely responsive to selection). This is not to say that interactions between gene and environment do not occur. In other words, it is not to say that the development of the organism is due solely to the genes. That would violate the central tenet of epigenetics and make evolutionary theory incompatible with the best theory of development. Rather, it is *assumed* that these various ways in which environment affects development does not account for the phenotypic variation. It is this sense in which, vis-à-vis the evolutionary project, the environmental role in development is said not to matter and is often taken for granted.

If the evolutionary project were the extent of Lorenz's methodology, I think that Lorenz's response to his critics is relatively uncontroversial. For example, it might interest an evolutionary theorist to consider the selection pressures that might have been present to favor individuals that possessed innate behavior capacities rather than individuals who had to learn their behaviors. In the spirit of adaptationism a model may be constructed to speculate on possible answers (see Sober 1994a).

But Lorenz fails to distinguish the question of explanatory significance from the question of what makes a trait innate. Recall that Lorenz originally identified innateness with the ability of an individual organism to *develop* a trait in isolation of environmental cues. In response to his critics, Lorenz asserted the value of innateness from an evolutionary theorist's point of view. But his response fails to answer the original question: What sort of *developmental* phenomenon is innateness?

Lorenz recognized this problem and provided an answer consistent with his evolutionary approach. His answer went roughly as follows: For adaptive behavior to develop in an individual, the organism requires *information* about its environment. There are two possible sources of this information. Either it is provided by the individual's interaction with its environment, or it is provided by the evolutionary process of natural selection, in which case it is encoded in the organism's genes. When

the source of the information is provided by natural selection, Lorenz argued, the trait is said to be "innate." So, the category of innateness is important not only to the study of evolutionary biology, but also to the study of *development*. It allows one to distinguish traits whose developmental information is due to natural selection from those traits whose developmental information is in the individual organism's environment.

Setting aside Lorenz's associations between innateness and "information," let us focus on his views of the explanatory role of natural selection. I take it the key point is that knowing that a trait is a product of natural selection helps explain its development in an individual. But as psychologist Daniel Lehrman (1970) pointed out, Lorenz's theory about the role of natural selection in development is mistaken. According to Lehrman, knowing that a trait is a product of natural selection does not settle any questions about the developmental process by which the phenotypic characteristic is produced in an individual organism.

Lehrman writes, "The clearest possible genetic evidence that a characteristic of an animal is genetically determined in the sense that it has been arrived at through the operation of natural selection does not settle any questions at all about the developmental processes by which the phenotypic characteristic is achieved during ontogeny" (1970, pp. 25–28). Lehrman's argument rests on an example of a trait, species-specific mating preferences, that is widely under selective control. Compare the development of this trait in doves and cowbirds. For both the trait is a product of natural selection, yet individual doves develop their mating preferences through "learning" whereas the cowbird does not.

I think the most perspicuous way of illustrating Lehrman's point is to consider an example used by Elliott Sober (1984), which I alter to fit our purposes. Imagine that gaining entrance to a school requires that individuals read at the third-grade level. Suppose that Sam, Aaron, Marisa, and Alexander pass the test and so are admitted, whereas other students do not pass, and so are excluded. The selection process explains why the room contains only individuals that read at the third-grade level. But the selection process does *not* explain why *those individuals* in the room (Sam, Aaron, and so on) read at the third-grade level. As Lehrman put it, the selection process favors certain *outcomes*, not the processes by which those outcomes are achieved. Notice that it is quite possible that the story about *how* each individual came to read at the third-grade level varies. For example, suppose Sam and Aaron have parents who taught them how to read at a young age, Marisa took a pill which enhances her reading ability, and Alexander reads well despite living with parents who did nothing to aid his progress. These individual differences are not part of the selection explanation for the prevalence of children in the room who read at the third-grade level. But they are part of the developmental explanation for how each child came to read at the third-grade level.

Lorenz's mistake was to misapply natural-selection explanation. Selection explanations are tailor-made for the investigation of evolutionary phenomenon. Yet they are inappropriate for the investigation of what is essentially a developmental phenomenon, the "innateness" of biological traits. Further, by defining innateness as a product of natural selection (via genetic transmission), we are left without an adequate definition of innateness as a *developmental phenomenon.*

Let me put the point in a different way. Attributing biological traits to genes as Lorenz would have it does not answer the central question that innateness ascriptions were meant (by Lorenz) to address.[3] The issue is, broadly, why does an individual have the traits that he or she does? (Or, how does an individual come to manifest the traits that he or she has?) To say that a trait is innate, that is, to say that it is genetically determined, is only to deny that it is, in some unclear sense, not environmentally acquired. Identifying traits as innate as opposed to acquired says nothing of the developmental *process* involved in the production of the trait (Lehrman, p. 344). If we want to know why an individual has the traits he or she has we need to ask *how* environment and genes interact to produce the trait in question. Attributing a trait "mostly" to the genes says nothing about the developmental process that gets us from genes to the trait in question.

3 Apportioning Casual Responsibility between Genes and Environments

Is there another way to preserve Lorenz's genetic account and still have it that innateness is a feature of development? Some authors, most notably Robert Richards (1974) and Michael Levin (1994), have suggested that the distinction between innate and acquired traits is in fact useful to the developmental project of *decomposing* phenotypes into genetic and environmental components. The thought is that the fact of development does not preclude the possibility that the causal role of each may be distinguished. As both Richards and Levin point out, the decomposition of phenotypes is consistent with standard practice in physics and chemistry. Levin (1992) writes that "the causal role of genes can be isolated, just as the causal role of any single chemical in a reaction can be" (p. 50). Richards (1974) concurs, "The discernment of the sources of behaviour, a little like the discernment of spirits, is rarely easy.... The situation is no different, of course, in any of the other sciences.... [T]he problem of holding constant or eliminating factors other than those of interest is one which ... all experiments in natural science share" (p. 127).

Unfortunately, Levin's and Richards's approach won't work. The method of decomposition to which they refer is not applicable to the investigation of phenotypes. Let us see why.[4]

In chemistry and physics, one can meaningfully ask, "How much did two causal factors contribute to an event?" We can ask this about chemical or physical systems because events of physics and chemistry both obey J. S. Mill's principle of the composition of causes. Consider a Newtonian example where the event is a particle's acceleration, one causal factor is an effect of gravity, and the other is an effect of electricity. By Mill's principle, the result of the two forces is just the sum of what each would have *achieved had each acted alone* (Sober 1994b, p. 184). To answer the question, "How much did gravity and electricity contribute to the particle's acceleration?" we ask two further questions: (1) How much acceleration would there have been, had the gravitational force been present, but the electrical force been absent? and (2) How much acceleration would there have been had the electrical force been present, but the gravitational force been absent?

Such questions have no analogue in developmental biology. The nature and behavior of phenotypes do not obey Mill's principle. It follows from the fact of development that one cannot ask what effect genes or the environment would have had on the production of a biological trait if genes or environment had acted *in isolation*. For example, one cannot ask of Sally how tall would she have been if genes acted in isolation or if the environment acted in isolation.

Interestingly, Sober (1994b) argues that Mill's principle is inessential to the issue of whether it is possible to ask how much genes and environment contribute to a biological trait. If this is right, then the truism of the phenotype does not automatically yield the negative conclusion. What is essential to the conclusion is that the contributions of genes and environments to the production of a biological trait are incommensurable; as Sober puts it, "their contributions are not made in a common currency" (Sober 1994b, p. 193).

Consider the following example to illustrate the notion of commensurability between two causal factors and to demonstrate why the truism of the interaction of genes and environments is inessential to the conclusion.[5] Suppose two brick-layers are responsible for building a wall. Each begins work on one end of the wall and works her way to the middle, both involved in the task of laying down the mortar and the bricks. Since both perform the same task, it is possible to ask which brick-layer contributed more to the job. To answer, we simply count the number of bricks each worker contributed to the job. There is a way to determine the relative causal contribution of each worker for each makes her contribution in a common currency.

Notice that to ask how much each brick-layer contributed to the wall, it is not necessary to ask how much would each have contributed *had she acted alone*. That is, to answer the question about causal contribution we do not need to appeal to Mill's principle of contribution of causes. We just need to compare the number of bricks each brick-layer laid.

One might object that counting each brick-layer's contribution just amounts to asking how much each would have contributed had she acted alone.[6] A slightly different example throws some doubt on that intuition. Suppose the masons coordinate their workload such that each takes turns laying down one brick before the other lays down hers. So, one mason sets down brick number 1, 3, 5, etc., while the other sets down brick number 2, 4, 5, etc. After the wall is built, we cannot answer the question how much did each contribute had each acted alone, because each worker's contribution by itself does not constitute a wall at all. A wall with successive bricks missing is not a wall, but a heap of bricks. Again, if we want to know the relative contribution of each worker we simply count the bricks each mason laid down.

Now suppose that the division of labor is such that each brick-layer has a specific task: one lays down the mortar and the other sets the bricks. Is it meaningful to ask which brick-layer contributed more to the task of building the wall? Since, in this scenario, each brick-layer does not make her contribution in a common currency, this question has no answer. The interaction between genes and environments is akin to this second brick-laying example, in which the contributions the factors make are incommensurable.

What would it be like for biological traits to be decomposable and for their relative contributions to have a common currency? Suppose height were the result of an accumulation of height particles whereby both genes and environments contributed a certain number of particles to make up a person's height. If so, then we could compare the number of particles genes and environments contributed to, say, Jane's six-foot stature (Sober 1994b, p. 193). To return to innateness, if the term is meant to denote a trait that owes a great deal of its structure to genes, and if the contributions genes and environments made were commensurable in this way (e.g., if there were height particles), then innateness ascriptions would be helpful in apportioning causal responsibility among genes and environments. We could determine the relative number of particles genes and environments contributed to the trait by elaborating various deprivation experiments. But alas, there are no height particles; the contributions that genes and environments make to biological traits are not commensurable; we cannot answer the question, *how much* each factor, genes and environments, contribute to a trait. Hence innateness cannot be ascribed in answering such questions.

The nature/nurture case is not unique. The distance a cannonball travels depends on both the angle of the cannon's barrel and the muzzle velocity. But the two factors do not make their contributions in a common currency; there is no telling how much muzzle velocity and barrel angle contribute to the distance the cannonball travels (Sober 1994b, p. 197).

I conclude that Levin's and Richards's suggestion intended to save Lorenz's genetic account of innateness fails. Does it follow, as Lehrman and his modern-day counterparts (Oyama, Johnston, Griffiths, Gray, and others) would have us believe, that the gene/environment dichotomy upon which the innate/acquired dichotomy rests is unexplanatory? Is there no refinement of the notion of innateness that is useful for any field of inquiry?

No. Lorenz's naive account is not the only one available. There are other biological definitions of innateness, the best of which are not falsified by the fact that genes alone do not produce biological traits. I will offer one and criticize three others in this essay. But first I need some criteria of evaluation. I take it that the best source of these criteria comes from what remains fairly uncontroversial about Lorenz's theory.

(a) **Development.** An account of innateness should make it a feature of development. In the birdsong literature, for example, you often find a contrast between birds that develop their songs "innately" and those that don't. Recall that part of Lorenz's project was to pick out certain adaptive behaviors that particular animals exhibit even if they had no opportunity to "learn" or "experience" the behavior in the wild. Ethologists are still interested in such behaviors. For example, ethologists of birdsong have discovered, via the use of deprivation experiments, various ways in which species-specific birdsong develops in different species of birds. Certain birds develop their species-specific song regardless of whether or not they hear another bird sing that song. Other birds seem to require experiencing the song before they themselves can sing it. Here we have a contrast in developmental requirements: some birds require a specific environmental cue for the development of their own song, whereas others do not require that specific environmental cue.

(b) **Environmental Stability.** Innateness should denote an environmentally stable trait. In the ethology and biology literature, innateness seems to have something to do with what environment does not do to influence development in an individual. Evidence from deprivation experiments and observations in the wild suggests that some traits develop normally in a range of environments, including impoverished and abnormal ones. In such cases, the environment does not prevent the trait from being manifested. Ethologists since Lorenz sometimes associate innateness with environmentally stable, as opposed to labile, traits (Hinde 1982, p. 86).

(c) **Natural Selection Explanandum.** An account of innateness should make clear how natural selection can effect the prevalence of some adaptive traits. Recall that Lorenz was interested in applying natural selection to explain the prevalence of certain highly adaptive species-specific traits. For example, he was interested in how selection might explain the prevalence of ducklings that display courtship behavior without any need of experiencing other ducklings displaying that behavior. I object only to defining innateness solely as a product of natural selection. I have no in-principle objection to natural-selection explanations of the prevalence of innate features. The natural-selection project seems promising although it is an open question whether all such traits are explainable via natural selection.

First, I'll propose my own account of innateness, which stems from C. H. Waddington's concept of "canalization." After demonstrating how the canalization account of innateness captures the desiderata I'll compare it with three other accounts, one put forward by Stephen Stich, another by Elliott Sober, and a third by William Wimsatt.

4 The Canalization Account

In 1936, developmental biologist C. H. Waddington along with his associates (Joseph Needham and Jean Branchet) made a remarkable discovery (see Gilbert 1991). They had set out to determine the specific substance that induces competent ectoderm tissue to develop neural plates. Waddington had thought that the substance was a particular steroid and that only that steroid could induce neural-plate development from competent ectodermal tissue. Waddington's crew discovered, however, that a large variety of natural and artificial compounds rather than a specific steroid induce neural-plate development. Waddington proposed that since lots of different substances serve as an inducer and not all cells can form neural plates, there must be something special about the competent tissues of the ectoderm that allows it to respond to the range of the inducing chemicals. What is special, Waddington conjectured, is that competency to respond to a range of inducing agents is a genetically controlled feature of certain tissues. The wide-ranging competency of the ectoderm allows neural plates to develop in a wide range of environments.

If competency is genetically inherited, it might be susceptible to forces of natural selection. For instance, neural plates develop out of ectoderm in a wide range of developing environments, even in environments lacking steroids. For certain selective regimes the capacity to develop in a range of environmental conditions might confer reproductive success to individuals possessing them, say, in unstable environments.

In such a case, Waddington said that the pathway became *canalized* by natural selection. Canalization denotes a process whereby the endstate (the product of development) is manifested despite environmental perturbations (Waddington 1940).

According to Waddington, canalization explains why developing organisms tend to produce a number of distinct and well-defined body types despite environmental variation between the individuals. Waddington envisioned development as "an epigenetic landscape," a branching out of various developmental pathways, each leading to the production of a distinct endstate. Once development starts (e.g., in the egg) any number of a range of inducing agents force the developing organism down one or another canalized pathway (Waddington 1957).

Further, the canalization model could account for why, for some quantitative characters, there appears to be a "normal" range such that deviant morphologies are difficult to produce. Waddington writes, "For most animals there seems to be a 'normal' size, to which the adult individual often closely approximates even though it may have suffered various enhancements or retardations of growth during its lifetime as a consequence of factors such as the number of its litter-mates, the level of its feeding and so on" (Waddington 1975, p. 99).

For our project of providing an adequate biological account of innateness, canalization fits the bill. Recall the three desiderata for an adequate account of innateness. An adequate account ought to (a) make innateness a feature of development, (b) contrast traits that are environmentally stable rather than labile, and (c) be part of natural-selection explanations for the prevalence of certain traits. Waddington provided an account of the development of environmentally stable traits that occur in individuals and whose prevalence can be effected by natural selection, satisfying the three desiderata. I propose the following as an adequate account of innateness:

The degree to which a biological trait is innate for individuals possessing an instance of a genotype (or set of genotypes) is the degree to which the developmental pathway for individuals possessing an instance of that genotype (or set of genotypes) is environmentally canalized.

The degree to which a developmental pathway is canalized is the degree to which development of a particular endstate (phenotype) is insensitive to a range of environmental conditions under which the endstate emerges.

The concept of canalization is useful because there often exists a high degree of constancy ("robustness") of phenotypes over a fairly well-defined "normal" range of environmental conditions.

Several features of the canalization account of innate traits are worth noting. First, the canalization account preserves the idea that traits are innate with respect to certain genotypes. It may turn out that, for example, some of the genotypes that typically express blue eyes are canalized whereas others are more sensitive to environmental fluctuations.

Further, although Waddington's account of canalization was intended to provide an illustration of the genetic control of phenotypic characters, it is important to note that Waddington recognized that development is an interactive process involving both genes and environmental conditions. Certain environmental conditions are essential for the development of certain traits. No developmental pathway is resistant to all possible environmental perturbations. As Waddington states, "Even the most well-canalized character is, of course, never entirely invariant" (Waddington 1975, p. 100). That there is a limit in the environmental ranges to which a developmental pathway produces a single phenotypic endstate should come as no surprise, given the fact of development.

To reflect the significance of development, on the canalization account innateness is treated as a matter of degree: The greater the environmental range against which a developmental pathway is buffered, the more canalized is the developmental pathway. The steepness of an entrenched pathway (or "chreode") in Waddington's representation of development represents the degree to which a pathway is canalized. The steeper the sloping sides of the pathway, the more resistant the pathway is to disturbances.

On the canalization account, development too is a matter of degree. Loosely speaking, limb development in many organisms is highly canalized; limbs develop in all but only the harshest environments. However, a trait requiring several very specific environmental cues to develop in an individual, for example, the ability to speak French (as opposed to linguistic ability in general), is not highly canalized.

Finally, consider that Waddington's comparisons are made within a specific environmental range. This leads to the question of what counts as an appropriate environmental range to determine innateness. I doubt that there is a uniquely correct answer to this. We probably cannot avoid having to determine the appropriate range on pragmatic considerations, say, depending on the interests of the biologist. As Waddington himself recognized, as a consequence of the fact that no biological trait develops independently of biological factors, no trait is canalized simpliciter. Mutatus mutandis for innateness. Recall that Lorenz's mistake was to think innateness denoted traits that appear in vacuo. An adequate account of innateness should not make the same error.

However, biologists and ethologists tend to ascribe innateness to traits whose development are to some surprising degree insensitive to certain environmental conditions (we made this environmental stability condition as one of our desiderata of an adequate account of innateness). Let us look at three examples illustrating that what constitutes the appropriate range depends on the trait in question.

First, Lorenz was interested in understanding the development of goslings who exhibit a "follow-mother" behavior even in the absence of mother geese. For Lorenz's purposes, the exhibition of a gosling's follow-mother behavior is insensitive to whether mothers are present or not. Lorenz was not interested in how fluctuating embryonic temperatures affect the development of the behavior although it may be an open question as to whether temperature does affect the outcome. Lorenz was impressed that a behavior that is specifically designed to allow an individual to follow mother (that is, a well-adapted activity) is exhibited without the need of the actual mother or any other gosling present. According to Lorenz, there is something interesting about the behavioral development of goslings that would allow such a trait to emerge. In the terms of the canalization account, what's interesting is that the endstate is canalized within the range of environments whereby mothers are present or not. (Note that it may be that the development of the follow-mother behavior is canalized or innate within environments where mothers are either present or not, but not canalized within environments in which, say, embryonic temperature is fluctuating.)

Second, birdsong ethologists are interested in the contrasting developmental pathways of individuals of different sparrow species. Some sparrows produce their characteristic song even if they are reared in silence, whereas in other species, a sparrow produces its song only if it first hears that song performed (Gould and Marler 1991). Birdsong ethologists are interested in contrasting the developmental pathways of individuals representing different songbird species, that is, ones that develop when reared in silence with ones that require their specific song as a recondition for development.

Third, Waddington expressed interest in traits whose development appeared to be insensitive to "normal" environmental conditions. Waddington sought to provide selection explanations for why certain traits persist in "normal" environments and express variants only in "stressful" ones. What counts as a "normal" and "stressful" environmental range is vague, but it probably denotes the range of environments a population of individuals typically shared throughout their selective history.

To reflect the varied interests of biologists, what counts as an appropriate range might change depending on the trait in question. But at least one caveat is in order: At minimum biologists should restrict themselves to environments in which the organism can *develop*. This is an important condition vis-à-vis an account of innateness. For example, one does not prove that hair color is not innate (for individuals possessing a genotoken) by showing that there are environments in which the individual does not develop hair at all (Sober, personal communication).

5 Innateness and Natural Selection

In accepting that Waddington's notion of canalization applies well to the idea of innateness, we open the door to a range of parallels between the work of Waddington and the work of ethologists like Lorenz. A striking example will further strengthen the case for thinking that innateness is canalization. In this instance, we find Waddington solving a problem that Lorenz failed to solve: How can we invoke natural selection to explain the prevalence of highly adaptive traits, such as sexual affinity in mallards, that are seemingly environmentally acquired yet turn out upon further investigation (e.g., by performing a rearing in isolation experiment) to manifest in isolation? For Waddington, the key is the concept of canalization.

Recall Waddington's notion of competence. Competent tissues are inducible by a range of compounds. Most importantly, the range can extend to compounds found either to be internal or external to the developing organism. Waddington hypothesized that once a developmental pathway is canalized, the competent tissue may transfer its responsiveness from one inducing agent to another (Gilbert 1994, p. 851). The possibility of transfer of competency is significant for evolutionary biology. For example, callouses are typically environmentally induced by friction between skin cells and some external surface. Here, the genes play a role in proliferating cells to form the callous. Suppose both that the competence of the skin cells to form a callous structure when induced by friction is a product of natural selection and that the pathway initiated by the friction that leads to the callous structure is canalized. The canalization of callous formation depends on an external agent in this instance. But if the skin cells are competent for a range of agents, it is possible for an internal agent to substitute for the external agent. For instance, suppose a mutation appears that enables the skin cells to respond to a stimulus within the developing embryo. Then the ability to form a callous due to friction may become part of the "genetic heritage" of the organism. That is, what starts as an externally induced trait undergoes a mutation that in effect transfers its competence to internal inducers. The end result is an organism that develops a callous in the absence of friction. In some selective regimes the transfer of competence from external to internal inducer may confer fitness to organisms possessing the capacity. Waddington called the transfer of competence "genetic assimilation." He hypothesized that selection aiding in the

genetic assimilation of the capacity of ostriches to develop callouses explains why ostrich *embryos* possess callouses (Waddington 1975). If the transfer of competence is due to a mutation, genetic assimilation is said to be an instance of what is known as the "Baldwin effect."

Unfortunately, Waddington did not support his hypothesis concerning ostrich callouses with experimental evidence. However, he did find evidence of genetic assimilation in experiments on thorax development in *Drosophila*. To illustrate the phenomenon, Waddington (1975) managed to induce an extreme environmental reaction in the developing embryos of *Drosophila*. In response to ether vapor, a proportion of embryos developed a radical phenotypic change, a second thorax. At this point in the experiment we should say that bithorax isn't innate; it is a kind of chimera induced by an unusual environment. But then Waddington continually selected for *Drosophila* with the developmental capacity to respond to the environmental stress. After about twenty generations of selection, some *Drosophila* were obtained that developed *bithorax without being exposed to ether treatment*. What happened, according to Waddington, is that selection favored a particular pathway that led to the production of the optimal (in this case desired) effect. Eventually the pathway became canalized, and hence the endstate, bithorax, appeared regardless of environmental conditions.

6 Critique of Stich's, Sober's, and Wimsatt's Theories of Innateness

Now I wish to compare the canalization account of innateness with three other developmental accounts, one by Stich (1975), one by Sober (1998), and a third by Wimsatt (1986). Using certain counterexamples, I hope to demonstrate the superiority of the canalization account over these. Further, in demonstrating why the canalization account is superior, I will highlight its key features. For example, I have emphasized that canalized development is (relatively) insensitive to developmental environments. Resiliency, not interaction with environmental conditions, accounts for the developmental invariance of the phenotype in question. This feature is not clearly a part of either Stich's or Sober's developmental account of innateness.

Stich's aim is to provide an account of innate *ideas*. Following Descartes, his strategy is to provide a more general account of innateness (actually, one of innate diseases) and then apply it to the case of ideas: "In calling ideas innate, Descartes tells us, he is using the same sense of the word we use when we say certain diseases are innate. So let us launch our analysis of innateness by pursuing Descartes's hint and asking what it is to be afflicted with an innate disease" (p. 3).

Stich's account resembles Descartes's own; both are "disposition accounts." On Descartes's view, individuals that suffer an innate disease "are born with a certain disposition or liability to acquire them" (Stich 1975, p. 6). Stich's account unpacks the disposition to provide the following account:

> A person has a disease innately at time *t*, if and only if, from the beginning of his life to *t* it has been true of him that if he is or were of the appropriate age (or at the appropriate stage of life) then he has or in the normal course of events would have the disease's symptoms. (1975, p. 6)

The Cartesian approach (of which Stich's is a piece) is sensitive to the intuition that "inborn" or "present at birth" is not a necessary condition for "innateness." Muscular dystrophy is, intuitively, an innate disease, yet the symptoms do not appear until later in a child's life. Stich's Cartesian account preserves this intuition: A child born with muscular dystrophy has the disposition to experience the associated symptoms, and hence muscular dystrophy is an innate disease. Or, more precisely, it is true of the diseased child that at the appropriate age she will begin to experience the symptoms (Wendler 1996, p. 91).

Stich's account does not fare well with diseases associated with parasites that are acquired in the normal course of one's development, as Wendler (1996) demonstrates. Humans typically possess an abundant supply of a particular species of bacteria *clostrium difficile* (*c. diff.*) in our intestines. Humans are not born with *c. diff.*; we typically acquire it by ingesting food and water. *C. diff.* is not harmful to healthy humans, but it can make sick people on antibiotic treatment sicker by colonizing in areas which the antibiotic treatment (to which *c. diff.* may be resistant) is killing off harmful bacteria. Unabated colonization of *c. diff.* produces toxins that lead to diarrhea and associated symptoms. Is the possession of *c. diff.* in one's intestines innate?

Innateness is generally and intuitively contrasted with environmentally acquired (albeit as a matter of degree). *C. diff.* is acquired from ingesting food and water, so, intuitively, the possession of *c. diff.* is not innate. However, in the normal course of events humans eat and drink water and hence acquire *c. diff.* As Descartes would say, humans have a disposition to suffer the symptoms of *c. diff.* in the intestines. It follows, on Stich's Cartesian account, that *c. diff.* is an innate disease of the intestines. This is an unfortunate consequence of Stich's account.

Stich recognizes the loophole in his account: "There are commonly a host of necessary environmental conditions for the appearance of the symptoms of a disease. If these conditions all occur naturally or in the normal course of events, the symptoms will be counted as those of an innate disease" (p. 7). Stich's response amounts to hoping for vagueness in one's intuitive judgments per case: "it is often unclear whether the occurrence of a certain necessary [environmental] condition is in the

normal course of events. So it will often be unclear whether a person is afflicted with an innate disease or is, rather, susceptible to a (noninnate) disease" (p. 7). But Wendler's counterexample is a paradigm. Clearly *c. diff.* is, in the normal course of events, at least part of the normal task of eating and drinking.

It must be admitted that the problem that Stich alludes to is difficult for any account of innateness. Stich's problem can be generalized as follows: For the development of any biological trait, disease, or otherwise, there will be a host of necessary environmental conditions for the appearance of that trait. This just follows from the fact of development, that genes do not operate in vacuo.

The canalization account handles this fact in two ways, first by making innateness a matter of degree, and second by noticing that for the development of some (special) traits, once a certain environmental (or genetic) condition is met, development becomes resilient to further environmental perturbations—in other words, development becomes canalized. For instance, suppose a developmental system is found to correspond to the developmental "rule of thumb": "Develop *X* no matter what the state of the world happens to be." This exemplifies an extreme form of canalization. Less extreme are systems that conform to the conditional rule: "Develop *X* if experience is *C*" or "Develop *X* if experience is *C*, develop *Y* if experience is *D*." The more sensitive to the environmental condition the developmental system is, the less inclined we are to say that the developmental endstate is canalized. Let's see why this is an improvement over Stich's account.

Stich's account fails to take into account the *causal dependency* of development on the environmental conditions that constitute the normal course of events. Sober's account suffers from a similar problem. For Sober, "a phenotypic trait is innate for a given genotype if and only if that phenotype will emerge in all of a range of developmental environments" (1998). In other words, innateness amounts to phenotypic invariance across a range of environmental conditions. Both Stich and Sober fail to recognize that there are two ways in which a trait emerges (invariantly) in a (e.g., normal) course of development, namely (both conditions come from Johnston 1982, p. 420)[7]:

(1) By means of strict genetic control over development so that the outcome of development is *insensitive* to the conditions under which it occurs. Such outcomes are said to be strongly canalized against environmental perturbation.

(2) By means of a developmental sensitivity *only* to environmental factors that are themselves invariant within the organism's (e.g., normal) developmental environment.

The second outcome is not canalized in Waddington's sense but is invariant under the normal conditions of development. The *c. diff.* case is a paradigm example of the second case. Contracting the symptoms associated with *c. diff.* (or even contracting *c. diff.* itself) is part of one's normal course of environment. Further, acquiring the disposition to suffer the symptoms associated with the possession of *c. diff.* emerges across quite a range of (normal) human environments. So we would say the outcome is invariant and hence innate on Sober and Stich's account. But acquiring *c. diff.* and having the disposition to suffer the consequences are both normal and developmentally invariant *because* humans are susceptible to an environmental condition that is itself invariantly part of human development. *C. diff.* is in the food we eat and the water we drink, and we obviously need to eat and drink to develop at all. Although the outcome is invariant it is acquired, not innate and not canalized.

Next, I turn to Wimsatt's "generative entrenchment" account of innateness. I reject it because generic entrenchment is not at all an essential condition for innateness.

The "crucial feature" of Wimsatt's account is the idea that "features that arise early in development have a higher probability of being required for features that appear later ... and tend to have a larger number of downstream traits depending on them" (1986, p. 198). Accordingly, Wimsatt defines "generative entrenchment" of traits "to the degree that they have a number of later developing traits depending on them" (p. 198). There appear to be two central claims here. One is that innate traits tend to appear early in development. The second is that innate traits have a number of later developing traits depending on them. I'm not altogether sure how Wimsatt conceives of the relationship between these two concepts, though it does seem that the concept of generative entrenchment is most central to Wimsatt's notion of innateness judging from his quip, "on this analysis, if it is generatively entrenched, it is 'innate'" (p. 200). Nevertheless I think neither are *necessary* features of innate traits. To see what's wrong with Wimsatt's theory, consider the innate development of pubic hair in adolescents. Because (i) pubic hair appears late in development and (ii) pubic hair has no further trait (at least prima facie) depending upon it, Wimsatt must say that pubic hair is not innate. Whether or not a trait is innate has nothing to do with how late in development it appears or what further trait depends on it.

Wimsatt might retort that pubic hair is in fact not innate. But that would contradict his motivation to unify many philosophical and ethological accounts of innateness under the concept of generative entrenchment. The idea that pubic hair is innate is supported by several of these accounts, including this one: "Innate behavior for a given species is universal among normal members of that species in their normal

environment either: (a) because the behavior has a genetic base [or] (b) because the behavior is 'canalized' or homeostatically regulated in development" (p. 187).[8]

According to Wimsatt, an interesting consequence of his developmental model is that some environmental experiences count as being innate. For example, Wimsatt claims that "not only is the imprinting mechanism of the greylag goose at birth 'innate' ... but the object of imprinting is also 'innate'" (p. 200). Take one of the minimal requirements for an environmental condition to be called innate: "the acquisition of that kind of information at that stage of development is deeply generatively entrenched with respect to subsequent behavior" (p. 200). Accordingly, imprinting is innate because a lot of later developing traits depend on it: "there is a very high probability that the young goose will properly imprint on its mother and will, in short order, learn to distinguish her cries and her appearance from that or other female greylag geese nearby" (p. 201).

To see what's wrong with this account, try applying it to the case of the fetal deformities resulting from the mother's taking thalidomide during pregnancy. For those who wish to preserve the contrast between innate and acquired, the effects of thalidomide are not at all innate when we consider that the presence of thalidomide, an environmental condition, makes all the difference between a child being born with a limb or without. Whether or not a lot of later traits depend on the taking of thalidomide does not sway our intuition that the child's deformities are not innate. I conclude that whether or not a trait is innate has little to do with whether it is generatively entrenched.

7 Conclusion

Taking my cue from critics of Lorenz, I have presented an account of innateness that avoids the fallacy of claiming that traits can develop by genetic causes alone. Innateness, on the canalization account, is a property of a developing individual. At the same time, the proposal captures what is thought to be distinguishing features of innateness: satisfying the ontogeny condition, referring to the capacity to produce environmentally stable traits, and making sense of the idea that natural selection can install innate traits in a natural population.

Acknowledgments

Parts of this essay were read at ISSHP in Seattle, the University of Arizona, the University of Rhode Island, Arkansas State University, and Virginia Tech. I wish to thank the audiences for helpful comments. In addition I wish to thank Denis Walsh, Elliott Sober, Chris Stephens, Robert Cummins, Tom Bontly, Joel Pust, Paul Bloom, and Henry Byerly for extensive comments on earlier drafts.

Notes

1. E.g., Kuo (1921), Lehrman (1953), Oyama (1985), Johnston (1988), Griffiths and Gray (1994).

2. Especially Oyama (1985), Johnston (1988), and Griffiths and Gray (1994).

3. Johnston writes on behalf of Lehrman: "The main point of Lehrman's argument was not to take a stand on the issue of whether, or how much, behavior is due to the environment as opposed to the genes, but rather that any such stand simply reflects a misunderstanding about the nature of development" (1987, 153).

4. My demonstration is borrowed heavily from Sober (1994b).

5. The example is taken from Lewontin (1974).

6. I thank Joel Pust and Tom Bontly for pointing this out to me and helping me with a solution.

7. Note that the distinction between the two means of invariant outcomes is not a dichotomy but a matter of degree.

8. I should point out that both intuitively and on my canalization account, the universality condition is neither central nor necessary to identify innateness. Consider those unfortunate persons who possess a gene that increase one's chances of developing Tay-Sach's syndrome. It appears that Tay-Sachs syndrome is canalized to a large degree for those members possessing the gene. Neither possessing the gene nor developing the syndrome is universal among humans.

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