Conceived This Way: Innateness Defended[1]

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Abstract

We propose a novel account of the distinction between innate and acquired biological traits: biological traits are innate to the degree that they are caused by factors intrinsic to the organism at the time of its origin; they are acquired to the degree that they are caused by factors extrinsic to the organism. This account borrows from recent work on causation in order to make rigorous the notion of quantitative contributions to traits by different factors in development. We avoid the pitfalls of previous accounts and argue that the distinction between innate and acquired traits is scientifically useful. We therefore address not only previous accounts of innateness but also skeptics about any account. The two are linked, in that a better account of innateness also enables us better to address the skeptics.

1. Introduction

Scientists find it useful to divide biological traits into innate and acquired ones.[2] But it is now a commonplace that biological traits result from the complex interplay of genetic and environmental factors. Therefore, they cannot be labeled innate or acquired simpliciter; a more sophisticated analysis is required.

We will argue that biological traits are innate to the degree that they are caused by factors intrinsic to the organism at the time of its origin, while they are acquired to the degree that they are caused by factors extrinsic to the organism. We will ground this account in a rigorous notion of degree of causation. We will then compare it with previous accounts. After that, we will address skepticism about innateness and argue that the concept remains valuable.

Our account explicates 'innate' and 'acquired' to fit clear uses of these terms within the biological sciences. These uses are informed by the sciences of evolution and development. We appeal to judgments about what's innate and acquired that should be uncontroversial, upon reflection, among subjects educated in current biology. To the degree that our account departs from *untutored* judgments (Griffiths et al. 2009), such judgments ought to be revised—as even non-scientists are capable of doing (Knobe and Samuels 2013).

2. Intrinsic Factors at the Time of Origin

To motivate our account, let's begin with two popular yet inadequate explications of innateness. First, innateness cannot be what's genetic or caused by genes because being caused by genes is

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[2] Increasingly so: searching PubMed for "innateness" on 5/29/2017 resulted in 97,954 hits, of which 68,186 are from the last 10 years and 40,205 from the last 5 years.

neither necessary nor sufficient for being innate. Some innate factors are not genetic, such as mitochondria, the cytoplasm, and inherited epigenetic factors. And some genetic factors are not innate, such as genes inserted into an organism via genetic engineering or bacterial conjugation. Second, innateness cannot be the same as what's inherited. Some innate factors are not inherited, such as mutations, while some inherited factors are not innate (Mameli 2004).

There is something in the vicinity of the genome and inherited factors that will help us develop a better account: it is the factors that are intrinsic to an organism at the time it originates. Organisms originate either through synthesis of non-living materials or through reproduction, either sexual or asexual. In sexually reproducing organisms, new individuals originate at the time of conception. In asexually reproducing organisms, new individuals originate at the time of fission or cloning.[3]

We will appeal to the factors that are intrinsic to an organism at the time it originates as a distinct individual. By intrinsic factors, we mean properties that organisms have in virtue of what's within their boundaries. For ease of reference, we will refer to these factors as Intrinsic Factors at Origin (IFOs).[4] IFOs include the cell membrane and any factor that is included within the cell membrane, such as the genome, epigenetic factors, mitochondria, or the cytoplasm. IFOs exclude any factors that are in the environment of, and thus external to, the new biological individual. The same IFOs may give rise to different phenotypes depending on differences in environment. By the same token, a given environment may give rise to different phenotypes depending on which specific IFOs interact with it.

The first step in our account is that IFOs are innate. This stipulation is consistent with common usage. Any causal contributions to the phenotype that are not from IFOs, and hence are extrinsic to the organism, are environmental. Traits that were not present at origin are the effect of the interplay between IFOs and environmental causes. These latter traits are innate to the degree that they are caused by IFOs as opposed to environmental factors; acquired to the degree that they are caused by environmental factors as opposed to IFOs.

3. Degree of Causal Contribution

At the heart of the common interest in innateness is the efficacy of interventions. Intuitively, a trait is acquired to the degree that intervening on its developmental environment changes it; it is innate to the complementary degree that intervening on its developmental environment leaves it unchanged. This is what our analysis is after. Already, this coheres with deeming IFOs themselves to be innate: interventions on IFOs will (trivially) have an effect on IFOs, but interventions on environment will have zero effect on IFOs because any environmental intervention can by definition only occur after an organism's origin.

[3] We assume that biological individuals are sufficiently well defined. This may not always be the case (Pradeu 2016). On our account, innateness is only a useful concept when the positing of biological individuals is also useful.
[4] We will use the singular, 'IFO,' for a specific subset of an organism's intrinsic factors at origin (e.g., a portion of its genome); context will make clear whether the plural, 'IFOs,' refers to analogous factors from multiple organisms (e.g., the same portion of their genome) or to *all* factors that are intrinsic to one or more organisms at their origin.

The notion of an intervention is in turn intimately tied to that of causation. It is not a coincidence, therefore, that causal notions such as contribution and sensitivity will prove central to innateness. Indeed, we will define innateness in explicitly causal terms. This approach has several advantages: it can be anchored in the rich causation literature, it will always be readily applicable, and it enforces conceptual clarity. Notably, it will make clear just how much, and why, ascriptions of innateness and related terms are relative to explanatory context.

We have demarcated the factors that determine a trait's development into two subsets, namely, IFOs and environment. These are the relevant causes; the trait itself is the relevant effect. So armed, we may proceed.

The heart of our analysis is *degree of causal contribution*. Overwhelmingly, this notion has been analyzed in philosophy of science and metaphysics as degree of difference making. Roughly speaking, a cause's contribution is defined as how much difference it makes to an effect. More formally: Let X be a cause variable and Y an effect variable. Y is a function of the state of the world—i.e., of X and W—where W is background conditions (i.e., formally a set of variables representing the state of the world just excluding X). In causal graph terms, there are arrows into Y from both X and W. Let X_A denote the actual value of X, let X_C denote the salient counterfactual value of X, and let Y_A and Y_C denote the values that Y takes given X_A and X_C respectively. Then define the degree of causal contribution of a cause variable X with respect to an effect variable Y, to be: $Y_A - Y_C$.[5] We gloss over many further technical details here (Northcott 2013, 2012b).

Degree of causal contribution is thus a fundamentally comparative notion: what is the value of an effect variable with a cause compared to its value without that cause? A cause's degree of contribution is therefore multiply *relativized*.

First, degree of contribution will depend on the salient contrast level or levels of the cause variable. For example, a particular allele might be an important (probabilistic) cause of schizophrenia relative to some contrast allele – but only a minor one relative to a different contrast allele. The contribution of a particular cause, such as a particular IFO, is not well defined until we have further specified a salient contrast. Thus, the same actual IFO may be associated with many different degrees of causal contribution, depending on choice of contrast IFOs. The same applies to the contribution of a particular environment.

Second, even given a specified contrast, causal contribution will also vary with background conditions, i.e., with the value of non-focal IFOs or environmental variables. Take the prevailing cultural environment, for instance: perhaps in the stress of a modern city a particular allele

[5] Not surprisingly, something like this understanding of causal contribution, often labeled 'causal effect', has a long history in several different literatures: measures in psychology, psychiatry, statistics, epidemiology, law and computer science are similar. Still other measures are closely related, being again essentially comparative of an effect with and without a cause. Within philosophy, this understanding reflects the common emphasis on causation's difference-making aspect – a cause is something that makes a difference to its effect. Thus, naturally, degree of contribution is how *much* difference it makes. This understanding can be incorporated into the contemporary Bayes net and causal modeling literatures, and arguably is endorsed by experimental practice, at least in the case of quantitative variables (Woodward 2003, Pearl 2000, Spirtes et al. 2000). More generally, it is also consistent with the mainstream literature on probabilistic causation (Hitchcock 1996).

greatly raises the chance of developing schizophrenia, yet in hunter-gatherer conditions it has almost no effect.

Third, causal contribution is also relative to choice of effect variable – in two ways. First, obviously, something may make a big difference to one thing but none at all to another. Second, less obviously, choice of contrast value of the *effect* variable is in general significant for assessing a cause's *explanatory* strength (Northcott 2013).

4. Innateness

Several notions of innateness arise. The simplest concerns a single trait of an individual, as when we declare your eye color to be innate. A second notion concerns a trait type within a population, as when we declare human eye color in general to be innate. A third concerns the more abstract notion of sensitivity, as when a trait's development is declared to be insensitive to environmental influence. A fourth concerns the range of values a trait may take, as when we say that a trait's development is 'under a tight leash'. At first glance, these various senses may seem heterogeneous. Upon closer examination, they are different manifestations of degree of causal contribution. We will now clarify and unify them under a common framework.

4.1 Contribution – token case

Consider the trait of height. For simplicity, let's pretend that the only relevant environmental factor is diet. Consider an animal population, its range of heights, and its range of diets. The different phenotypes (heights) are not just an unanalyzable mix of IFOs and environmental contributions. On the contrary, for any given IFOs there is a range of possible heights and the same for any given diet. The specific height of any individual is the outcome generated when that individual's IFOs are exposed to that individual's diet. Our suggestion is to call the effects on height coming from IFOs the *innate contribution* to height, and the effects on height coming from diet the *acquired contribution* to height.

In making this suggestion more precise, it will help to tackle the token and type cases separately. Begin with the token case. Formally, denote the relevant effect variable by T. This may be some quantitative variable such as height or qualitative one such as eye color. As we will see, what exactly T denotes may be interpreted with considerable flexibility. T's value is a function of its causes. In particular, for our purposes we partition these causes into two groups, namely IFOs and environment E. Then, following the causal analysis from the previous section, the quantity of contribution of IFOs to T is defined as follows:

$$\mathbf{T(IFO, E) - T(IFO^*, E)} \qquad \mathbf{(1)}$$

The asterisk denotes the salient contrast IFOs. We may define the contribution of environment similarly, with some contrast E* instead of IFO* in the right-hand side of the formula.

In the height example, in the token case (of you, say) the effect variable T is your height. The two causes of interest are a specific IFO—the subset of your IFOs that is relevant to height—and diet. What would your height have been if you'd had your friend's IFO instead of your own – and, thus, how much difference did having your IFO make? The answer to that is, per formula

(1), your IFO's causal contribution. And similarly for the causal contribution of diet. The counterfactuals are evaluated like all causal counterfactuals, i.e., by keeping background conditions constant except for changes caused by the change of IFOs itself (Lewis 1973, Woodward 2003).

Two important corollaries follow. First, degrees of contribution by either IFO or diet are relative both to choice of contrast and to background conditions.[6] Thus, my IFO might make a large difference to my height compared to one friend's IFO but not compared to another's, and a large difference to my height in one environment but not in another. Therefore, innateness itself is also a relative rather than absolute matter.

Second, the contributions of IFO and environment need not 'add up' to a trait's whole value. Suppose, for instance, that my height is 2 meters. If the contrast IFO would have seen my height be 1.8 m, then the causal contribution of my IFO is 0.2 m. Similarly, if the contrast diet is identical to my actual one save that it included less protein, then relative to that my actual diet perhaps only contributed 0.1 m. And $0.1 + 0.2 < 2$. For some choices of contrasts the two contributions might add up to exactly 2, but in general they will not. Once we accept the first corollary that causal contribution is a relative matter, this second corollary is inevitable.[7]

Another important point is that the ubiquitous causal entanglement of IFOs and environmental factors does not present any particular analytical difficulty. In the same way that counterfactuals generally are definable, so is the causal contribution of either IFOs or environment (Northcott 2012b) – against the skepticism of, for instance, Ariew (1996) and Garson (2015, 83).

4.2 Quantity of contribution – type case

In the type case the issue becomes, what is the contribution of IFOs to the height of a population's members as opposed to the height of a particular organism? Formally, in (1) the interpretation of T must be adjusted accordingly. It will be some measure of the height of a population – we must choose between mean, variance, or something else. There is now a richer range of possible contrasts. Are we comparing human heights or those of other species? If only human heights, which particular human populations? Suppose we are interested in height in a contemporary Western population; that still leaves open whether we are comparing this to non-Western populations, to counterfactual populations (perhaps some hypothetical enhanced human population of the future), to past populations, or to other contemporary Western populations. Formally, each such choice implies a commitment to some particular set and contrast set of IFOs, and any value for IFOs' causal contribution to height will vary accordingly. Analogous remarks apply to the type-level causal contribution of environmental factors.

All this duly noted, the type-level contribution of IFOs to height is defined, per (1), as how much difference the actual range of IFOs makes relative to some contrast range of IFOs.

But yet more disambiguation is required. First, the interpretation of 'contrast range of IFOs' is underdetermined. Suppose, for instance, it is taken to be the population of some currently poorer

[6] 'Background conditions' is represented in (1) by all non-focal components of IFOs and E.

[7] We do not address here what determines choice of contrast – for discussion, see Schaffer 2005, Northcott 2012a. But for any plausible answer, the second corollary follows.

country. What then would the contrast background conditions, such as diet, correspond to? We would want in the usual way the contrast population's diet to be the same as the actual population's diet. Would the diet and background conditions experienced by 'Person 1' in the actual population be exactly that experienced by 'Person 1' in the contrast population, and likewise for all other persons? The designation of who in the contrast population would be 'Person 1' is arbitrary and yet will impact the value assigned to the causal contribution, given the ubiquity of interactive effects, i.e., that different individuals' heights in general respond differently to the same change in diet.

What of causal contribution within a single population? Many different token-level contributions are definable, corresponding to different pairs, both actual and counterfactual, of members of the population, and for each pair there is a range of possible background environments. Any overall score for the population as a whole must presumably then be some aggregate of these token scores. Which particular aggregate is most appropriate is again interest-relative.[8]

We list these intricacies in order to emphasize again that any type-level score for causal contribution, and thus any ascription of innateness, is relativized in many ways. But we are *not* implying that type-level innateness is therefore so underspecified as to be useless. Quite the opposite. Consider degree of causation in general: even though multiply relativized in the same way, it is very useful. In practice, the explanatory context usually makes sufficiently clear what the relevant relativizations are, and they can always be noted explicitly if necessary. Accordingly, it can be meaningful to claim, and useful to know, that, for instance, smoking is a stronger cause of lung cancer than is air pollution. There is no reason why we cannot establish similarly useful claims about causal contributions to the development of a biological trait – indeed lung cancer itself is one example.

4.3 Sensitivity

Turn next to what initially seems a rather different notion of innateness. As a first pass—and assuming a discrete trait and a linear interaction between IFOs and environments—on this alternative view, the degree of innateness is the proportion of environments (relative to the entire environmental range of interest) in which a trait manifests itself given certain IFOs rather than others. Similarly, the degree of acquiredness is the proportion of IFOs (from the range of interest) that give rise to a trait within a certain kind of environment rather than others.

As it stands, this approach is incomplete because it does not specify that the range of environments of interest must include non-actual ones. For example, the belief that we cannot breathe under water arises in almost all actual environments for almost all human IFOs, and accordingly would be awarded a high degree of innateness. But, of course, this belief is a paradigm case of something *acquired*. The reason is that typically we consider those counterfactual scenarios where a human never experiences enough water to worry about breathing in it nor is she ever taught about it. Focusing only on actual cases obscures this

[8] One of the problems with traditional measures of causal contribution within a single population, such as statistical heritability (or, more generally, analysis of variance), is an implicit lack of such sensitivity to investigator interest (Northcott 2006, 2008a). There are several other problems too: statistical heritability is a notoriously bad instrument for assessing causal contribution (Lewontin 1974, Northcott 2006, 2008a, Shipley 2000, Spirtes et al. 2000).

dependence on environment. The remedy is to include some non-actual cases too by analyzing innateness in terms of the causal notion of sensitivity.

So, in a more refined version, the more a trait is *sensitive* to variation in IFOs and *insensitive* to environmental variations (within the relevant ranges), the more innate it is. Conversely, the more a trait is *insensitive* to variation in IFOs and *sensitive* to environmental variations, the more acquired it is. If a trait is completely insensitive to environmental variation (within the relevant range), it is innate simpliciter. If a trait is completely insensitive to variation in IFOs (within the relevant range), it is acquired simpliciter.

An example: Down syndrome occurs when people have a particular extra chromosome, pretty much regardless of environment (within any range that will normally be relevant). And similarly for other so-called genetic conditions. These traits are therefore innate, even though there may well be special environments in which they do not develop or they develop in milder forms, and of course medical researchers ought to investigate and design such special environments. (The *absence* of Down syndrome symptoms in a hypothetical environment that is specifically designed to prevent its development in people with the extra chromosome, then, would be an acquired trait.) Similarly, calluses and scars occur when certain environmental interventions affect people's skin, pretty much regardless of what someone's IFOs are (within a normal range). These traits are therefore acquired, even though there may well be rare IFOs that prevent their occurrence, and it may well be important to investigate those rare IFOs. (The absence of calluses and scars under the relevant environmental conditions, then, would be an innate trait.)

The sensitivity of a trait is connected to causal contribution, but how exactly? Recall that token-level degree of contribution, per (1), is the impact of a single intervention on a trait of a single organism. Next, type-level degree of contribution is defined in the same way, i.e. per (1), except it tracks the impact of an intervention on a trait in general. This is a function of many token-level degrees of contribution. The sensitivity understanding of innateness now expands still further the degrees of contribution taken into account. In particular, not only do we consider a range of organisms as in the type-level contribution, but we also consider a range of *interventions* too. Sensitivity amounts to some function of different interventions' own type-level contributions, each of these type-level contributions defined per (1). A trait's sensitivity to variation in IFOs is understood as the extent to which changes in IFOs produce change in the trait.[9] Similarly, a trait's *in*sensitivity to environment is understood as the extent to which changes in environment do *not* produce change in a trait. There is also no reason why sensitivity cannot be defined at the token level too, i.e. for an individual organism's trait.

This sensitivity notion of innateness is thus a relativized affair in the same way as causal contribution is. There exists no univocal degree of sensitivity for any given trait; rather, all depends on choices of contrasts and background conditions.[10]

[9] For many purposes, a standard reaction function will represent this same information more usefully. But the point here is to make clear the relation to the underlying notion of causal contribution.

[10] Birch (2009) and O'Neill (2015) define innateness in a similarly relativized way, namely as a trait's lack of environmental inducement (Birch) or as a trait's insensitivity to some explicitly specified environmental input (O'Neill). We discuss O'Neill's view below.

4.4 Innate range

The range of values that a trait can have for any given IFO in a certain range of environments is that trait's *innate range for that IFO*. The range of values that a trait can have in a certain environment given a certain range of IFOs is that trait's *innate range for that environment*. The idea here is that both environments and IFOs constrain the value that a trait may take, and it's often useful to know by how much. Contributions of IFOs and environments to a trait, as well as innate ranges, can be plotted on a chart with either IFOs or environments as the independent variable (Figures 1&2). The implicit notion of innateness is that the more the range of values is constrained by a given IFO, the more innate that trait is.[11]

[Insert Figures 1&2 here]

How does this version of innateness relate to the base notion of degree of causal contribution, as defined by (1)? Suppose we wish to know how strongly my IFOs constrain my height. This corresponds to T being the trait of height, IFOs being fixed at their actual value, and an interest in how T varies as environment varies across some salient range. In other words, like the sensitivity understanding of innateness, the range understanding corresponds to a function of a particular group of causal contributions. Instead of perhaps the average value of these contributions (as in the case of sensitivity), in the case of range we might be interested rather in the difference between the greatest and smallest contributions.[12]

Because of this underlying similarity to the other understandings of innateness, the range understanding shares the now familiar properties of being relativized to choice of contrasts and to background conditions. It also shares the appeal to counterfactuals, since many cited IFO-environment pairs may not actually occur.

5. Summary: an account of innateness

We have defined innateness informally as follows:

> Base clause: intrinsic factors at origin (IFOs) are innate.
> Recursive clause: other traits are innate to the degree that they are caused by IFOs and not by environmental factors.

We have defined acquiredness informally as follows:

> Traits are acquired to the degree that they are caused by environmental factors and not by IFOs.

These definitions, we saw, can be developed in several ways. First, degree of causation may be explicated as what we called *quantity of contribution*. On this understanding, a trait is innate to the degree that IFOs make a large contribution to it and environmental factors make a small

[11] Formally, innate range may be a property either of an individual IFO or of a population of them, and so, like sensitivity, may be defined at the token as well as type level.

[12] Of course, the simple gap between upper and lower bounds is just one property of a set of trait values and we might be interested in others.

contribution to it. On a second understanding, a trait is innate to the degree that it is *sensitive* to variations in IFOs and *insensitive* to environmental variations (within salient ranges). On a third understanding, a trait is innate to the degree that it has a *small range* of variation across salient environments (when holding IFOs fixed).[13] All three of these come in type and token versions. (Mutatis mutandis for defining 'acquired'.)

When analyzed causally, the close relation between these different understandings becomes apparent. They are all variations on degree of causal contribution, as defined by formula (1). In particular, in all cases innateness is identified with a large causal contribution from IFOs and a small one from environment. This causal contribution may be at the *token* level, e.g. to an individual organism's height, or at a *type* level, e.g. to a population of individuals' heights. Further, it may be that we are interested in a range of causal contributions, i.e. in a range of contrast levels of IFOs or environments, not just one. A trait's innate *sensitivity* is then the extent to which variation across this range of IFOs (or environments) produces large (or small) changes in the trait, i.e. it is some function of a range of causal contributions. A trait's innate *range*, meanwhile, is a specific such function, namely the difference between the greatest and least trait values produced by the relevant range of IFOs or environments.

Because they are all versions of degree of causal contribution, all of these understandings of innateness are multiply relativized and are a matter of degree. Moreover, they share two implications: first, the inefficacy of salient environmental interventions on highly innate traits; and second, a predictive guide as to what to expect to observe, and to be robust, across a range of environments. All also allow innateness to be readily measured, although how best to do so may be highly sensitive to local details (Griffiths et al. 2015).

Our account easily explains why judgments of innateness often correlate with traits that are insensitive to environmental factors during development, are typical of a population, or are adaptations, and why environments that disrupt the development of innate traits are often judged to be abnormal (Griffiths et al. 2009). Insensitivity to environmental factors is a component of innateness that is built into our account. Being typical of a species is not a component of innateness; but traits that are typical of a species are often highly innate because members of a species share important aspects of their IFOs, and causation by shared IFOs is the simplest way to produce species-typical traits. Finally, being an adaptation is not a component of innateness; but adaptations are usually highly innate because selecting IFOs is the most common way that natural selection produces adaptations. Of course, adaptations are usually also typical of a species. In light of the above, many highly innate trait types are species-typical adaptations. And since adaptations contribute to fitness, environments that disrupt them disrupt fitness, which may lead to judgments that such environments are abnormal.

So, on our account, innate traits do not *necessarily* have many of the properties often associated with innateness. They need not be species-typical, essential to a species, or adaptations. By the same token, acquired traits need not be learned. This independence from problematic associations will come in handy when we rebut skepticism about innateness.

[13] To repeat – the salient range of environments in this latter definition will typically include counterfactual ones, so the definition is *not* simple actual prevalence.

A special caveat pertains to whether innate traits are present at birth. Etymologically, that is what 'innate' means. But the term 'innate' is much older than our scientific understanding of how organisms originate. In past days, it might have made sense to focus on the traits that organisms possess at birth. For one thing, it was difficult if not impossible to study the development of traits *before* birth. For another thing, it was not always known that many organisms, such as bacteria, do not give birth in the sense in which animals do.

The notion of innateness should be general enough to cover all organisms. That's why we must focus on the traits that organisms possess at the time of origin and what those traits contribute to the development of other traits. Insofar as that's not what innateness used to mean, it's what it *should* mean. That being said, anyone who finds it useful to focus on the special case of traits that are present at birth, or at any other time in development, can easily adapt our account. They can define specialized notions of innateness as follows: a trait is innate$_t$ either if it is intrinsic to an organism at time t (where t = birth or any other salient time) or to the degree it is caused by traits that are intrinsic to an organism at time t. We will leave such specialized notions of innateness aside and focus on the basic case.

Next, we will compare our approach to previous ones. We contend that it does better both at tracking scientifically informed judgments about innateness and at doing the jobs we need the innate/acquired distinction to do. After that, we will address arguments that advocate abandoning the notion of innateness altogether.

6. Compare and Contrast

We can now assess other recent proposals that are either *accounts of* or *replacements for* innateness. Our account is the first to articulate explicitly the separate notions of innate contribution, sensitivity to IFO variation, insensitivity to environmental variation, and innate range, as well as how they all stem from degree of causal contribution.

The most influential previous account, by contrast, is that innateness is simply insensitivity to variations in the developmental environment (Ariew 1996, 1999; O'Neill 2015; see also Stich 1975, Sober 1998).[14] This core idea is indeed a condition that contributes to innateness—accordingly, it is built into our account as a component of it. But this condition alone is not enough, for several reasons. For one thing, as well as insensitivity to environment, an account of innateness should also include the mirror notion of sensitivity to IFO, as when, say, eye color

[14] Ariew motivates this insensitivity criterion by appeal to the developmental notion of canalization, due originally to Waddington, itself motivated by the explanatory concerns of some evolutionary theorists. O'Neill objects that canalization is a stronger condition than mere insensitivity to environmental variation – it is insensitivity to environmental variation *due to developmental buffering*. She points out that some innate traits are insensitive to a certain environmental variation simply because that variation does not affect its development, not because of buffering (O'Neill 2015, 214-15). However, Ariew's definition of innateness (1999, 128) does not make such buffering a requirement: "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." O'Neill's criticism applies better to Mallon and Weinberg's proposal, according to which a trait is innate just in case it is both insensitive to environmental variation and caused by a developmental process that produces "relatively few phenotypic outcomes" (Mallon and Weinberg 2006, 340). Yet polymorphic traits (e.g., human eye color), whose developmental process produces *many* phenotypic outcomes, may still be highly innate.

being innate implies that (in context) your and my different eye colors are due to our different IFOs.

Another lacuna is the lack of a satisfactory account of degree of causal contribution. One symptom of this is inadequate appreciation of the crucial role of contrasts. Recent accounts have made innateness relative to a contrast class of outcomes (Birch 2009) or to a contrast class of environments (Ariew, O'Neill). But innateness is relative to contrasts in both cause and effect slots—relative both to contrast IFOs or environments and also to contrast trait values. This double-contrastivity proves useful in many examples in the literature, such as the birdsong cases discussed below. We also get clearer on a number of other issues: what other relativizations are necessary; how these relativizations are often not – and need not be – made explicit (see Section 8 below and contrary to O'Neill's account); the relevance of counterfactuals; the distinction between type and token cases; and that innateness must be a matter of degree (again contrary to O'Neill's account, although not to Ariew's).

Another insensitivity account is Woodward's suggestion that innateness is causation by genes that is insensitive to salient environmental variation (2006, 41-2). Although it is rather brief and suffers from some of the same lacunae as other insensitivity accounts, Woodward's suggestion is closer to ours in that it is rooted in a contextual difference-making theory of causation.

A different recent proposal is that the distinction between innate and acquired traits should be *replaced* by the distinction between robust and plastic traits (Bateson and Gluckman 2011; also Keller 2010, 75; Garson 2015, Section 4.5). A trait is robust to the extent that it appears reliably in the face of both environmental and genetic variation. Thus, robustness entails insensitivity to variations in the developmental environment but not vice versa. A trait is plastic to the extent that it's not robust. Robustness and plasticity are also important notions; they point at the valuable project of investigating the mechanisms through which some traits can develop reliably through generations in spite of wide environmental and genetic variation. But this is clearly not the same as innateness, as traits can be innate without being robust. For example, a point mutation may lead to a trait that is innate yet not at all robust with respect to IFO variation. More generally, often innateness signals precisely that a trait *is* sensitive to genetic variation – it's the IFOs, not the environment, that make the difference. So, the robust-plastic distinction is neither equivalent to nor a good replacement for the innate-acquired distinction.

Wimsatt (1986, 1999) makes a sophisticated replacement proposal along related lines, namely that innateness should be replaced by generative entrenchment. A trait is generatively entrenched to the extent that the development of other traits depends on it. Because of this dependence, the more a trait is generatively entrenched, the more disrupting it would have widespread and potentially catastrophic consequences for other traits, and so the more it tends to be robust (so as to avoid these catastrophic consequences). An immediate counterexample is, as with the robustness account, a trait caused by a point mutation that may be innate yet not generatively entrenched. But Wimsatt's proposal is part of a larger project concerning the evolution of phenotypic structure, because of which he is explicitly concerned only with traits that are species-typical. His account is not meant to be applicable to innate traits that vary within species, such as human eye color. Generative entrenchment is therefore not a perfect conceptual substitute for innateness, as Wimsatt is well aware. The real issue is whether those aspects of

innateness not captured by generative entrenchment are of value. Wimsatt argues that they are not. His position on innateness is thus one of partial *skepticism*. In the next two sections we will explain why we disagree.

7. Against Innateness Skepticism I

A large strand of the literature has treated the very notion of innateness with suspicion—the old labels of 'innate' and 'acquired' are said to have many liabilities. Innate traits were often assumed to be adaptations, species-typical, or present at birth, and acquired traits were often assumed to have the opposite characteristics. But innate traits need not be adaptations, species-typical, or present at birth. More fundamentally, the innate/acquired dichotomy is accused of resting on an essential/inessential dichotomy, and evolutionary theory is taken to have fatally undermined essentialism about biological types. Accordingly, it is charged, any focus on innateness is scientifically unhelpful. It risks invoking misleading essentialist connotations, which leads to confusion by conflating various biological properties that are better kept separate (Griffiths 2002, Mameli & Bateson 2006, 2011). Moreover, these connotations may be harmful socially as well as scientifically, perhaps by stigmatizing members of atypical sub-groups or by entrenching culturally specific notions of humanness (Hull 1986, Sahlins 2008, Lloyd 2012).

We will reply in two stages. First, in this section we argue that many of the most widespread criticisms do not apply to our account. Then, in the next section, we consider remaining criticisms that do apply and so require a longer response, including elaboration of the innateness concept's positive value.

The key is that, from the practical point of view of advice and interventions, often what matters is whether – not how – a trait's development is sensitive to a particular variable of interest, be that variable an IFO or an environmental factor. Our account abstracts away from underlying biological mechanisms or evolutionary history. It therefore makes no appeal to the notion of an evolutionary adaptation, so it is left unscathed by the many cases of innate traits that are not adaptations. It is compatible with evolutionary histories of niche construction and more generally of continuous reciprocal interaction between genes and environment (Laland & Brown 2007). And it is compatible with evolution making development reliable by stabilizing environmental parameters at the right value or by exploiting pre-existing environmental regularities.

It may be that universal (or almost universal) traits derive their relative stability across environments and cultures not solely from inherited genes but equally from extra-genetic influences. But again our account is compatible with this observation. For instance, if the explanatory context is the dependence of a trait's universality on a particular environmental cue, then the salient contrast will be the absence of that cue, which in turn will generate a large causal contribution for environment, yielding a verdict of 'acquired'. Thus, universality will *not* imply a verdict of 'innate' when the explanatory focus is the trait's dependence on a particular environmental input. (When the focus is instead the *in*efficacy of some other environmental input, then the verdict of 'innate' usefully returns.)

A related skeptical motivation draws on work by developmental psychobiologists suggesting that any trait is sensitive to various environmental cues. For example, one species of cowbird

acquires a species-typical song without exposure to parental singing, but with a regional dialect depending on its interaction with local females (see Griffiths 2009 for references). Our account, asserting as it does that traits are innate or acquired only relative to the range of environments that we consider, easily accommodates such complexities. Different explanatory contexts yield different innateness verdicts. If we are interested in why the bird sings its species-typical song rather than a song typical of another species, then IFOs make the difference but environment does not, thus yielding a verdict of 'innate'. If, by contrast, we are interested in why the bird has this particular song dialect rather than another, then it is environment not IFOs that make the difference, thus yielding a verdict of 'acquired'. That is, the song type is innate, the dialect acquired. (Formally, the two cases may be represented in (1) by different choices of contrast in both the effect and cause slots.)

Making no appeal to the mechanism of learning, our account accommodates examples where innateness and (lack of) learning come apart. Mameli and Bateson (2011, 438) give a hypothetical such case: a bird has a species-typical song but developing the ability to sing it requires a learning process, so the trait seems innate even though it requires learning. But the correct account is that the anatomical structures required to sing the particular song type are highly innate, i.e., caused primarily by IFOs in a way that is insensitive to the learning input, while the ability to actually sing it is acquired in response to the learning input. It is to our account's credit that it captures this distinction.[15]

Another source of skepticism about innateness is the non-additive nature of gene-environment interactions during development, which allegedly makes it impossible to parcel out responsibility between the two factors in a meaningful way (Griffiths 2009). We agree that responsibility cannot be 'parceled out' in the sense that the two factors' causal contributions may not add up to the total value of a trait (Section 4). Nevertheless, as we have seen, those causal contributions remain perfectly well defined, and that is what matters.

More generally, because innateness is a relativized affair, the very same trait may come out as highly innate in some explanatory contexts and highly acquired in others. The deeper picture is that traits are the result of both innate and environmental influences, which may interact in complex ways, yet our account provides objective measures of both. Sometimes we focus on innate contributions to a trait and ignore environmental contributions, other times the reverse. Innateness is a useful guide to outcomes of salient interventions, and such a guide must inevitably be contextualized as per our account – the connection to causation makes this inevitable. The specter of a single trait being 'both innate and acquired' is perhaps a hangover of a misplaced essentialism – in this case that a trait can have a fixed and absolute property of being one or the other.[16]

[15] Mameli and Bateson take lack of learning to be essential to innateness and therefore the hypothetical bird example to undercut any account of it. But lack of learning is not essential to innateness for at least two reasons. First, lack of learning is insufficient for innateness, as any non-mental acquired trait attests (e.g., scars). Second, learning itself is a range of complex processes that result from the interaction between innate and environmental causes. For a more detailed critique of defining 'innate' as not learned, see Garson 2015, Section 4.2.

[16] In principle, one could formulate counterfactuals that vary IFOs and environment simultaneously in such a way as to render no clear innateness verdict. In practice though, such 'combined' counterfactuals do not seem to be salient in actual disputes. Indeed, this anti-combined point seems to be true generally (van Fraassen 1980, 126). Even if

Perhaps biological influences on human behavior have been greatly exaggerated compared to the influences of society and culture, and the one thing that is strongly characteristic of all humans is their developmental plasticity (Sterelny 2012)? If so, many human traits are innate to a lesser degree than often supposed. But precisely this conclusion would be automatically tracked by our account. (Meanwhile, the plasticity itself would be innate because it is insensitive to salient environmental changes, even while other traits – namely the plastic ones – are acquired.)

8. Against Innateness Skepticism II

We now turn to two lines of criticism that we consider more challenging. The first, mentioned already, is that the term innateness comes with too much baggage. In particular, it taps into essentialist intuitions that arguably are part of a deep-rooted 'folk biology'. Any proposed account inevitably invites more confusion than it is worth, in particular by conflating several distinct biological phenomena (Griffiths 2002, Mameli & Bateson 2006).

The second line of criticism begins with the thought that an account of innateness should elucidate scientific practice. For example, Ariew's account of innateness as environmental canalization was originally (1996) motivated by the wish to make sense of the nativist assumption common in cognitive psychology. By fleshing out what that assumption could amount to biologically, it elucidates the nativist framing of scientific arguments such as Chomsky's poverty of the stimulus. On the other side, Griffiths and others' skepticism is motivated in part by a different area of science, namely the findings in developmental biology that environmental cues are ubiquitous in the development even of traits often labeled innate (Griffiths 2009). In contrast to these two examples, our own account of innateness is 'thin', having no richer connection to scientific practice than the partition of a trait's causes into IFOs and environment. When declaring a trait innate, it appeals to the values of contextually salient counterfactuals and associated interventions, but it does not explain those values.

In reply to the first line of criticism: formally, our account does not conflate anything. Rather, in this respect its thinness is a virtue, enabling it to judge innateness without commitment to any underlying biological mechanisms. Nevertheless, that does leave the risk of inheriting mistaken essentialist attitudes. Our reply is twofold: that any mistaken essentialist attitudes should be filtered out, as even non-scientists can do (Knobe and Samuels 2013); and that retaining innateness also brings with it compensating benefits. We think the latter is the appropriate reply to the second line of criticism too—it is *beneficial* to define innateness in a way that abstracts from any particular scientific practice.

The heart of our case is that innateness is a useful *umbrella term*. This line of defense is standard for any macro-property that is multiply realizable, such as 'money' or 'erosion'. According to our account, the common theme to innateness ascriptions is an assertion of sensitivity to a contextually salient change of IFO and insensitivity to a contextually salient change of environment, but what change is salient may vary greatly and the mechanisms explaining the sensitivity and insensitivity may similarly vary greatly. For many purposes, it is useful to abstract

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such a counterfactual ever were salient, our deeper defense of the innateness concept is merely that it is useful frequently and that claim would survive occasional odd cases where it isn't.

away from the causal details. Analogous remarks apply to money, erosion and the rest (cf. Maley and Piccinini 2014, Boone and Piccinini 2016).[17]

Precisely because the value of the innateness concept lies in abstracting away from specific biological mechanisms, it would be a mistake to replace it by referring instead each time to one of those specific mechanisms, any more than always referring specifically to coins, bank transfers, conch shells and so on would usefully replace the concept of money. Nor, therefore, is it a problem if innateness conflates its different particular instantiations, any more than doing so is a problem for money or erosion.[18]

Our account abandons any absolute, context-free division of traits into innate and acquired. Judgments of innateness and acquiredness are relativized to the salient range of IFOs and environments for a population or individual. As we saw, this tracks our judgments of innateness well, and certainly better than do other proposed accounts. The same contextualism is true of umbrella terms generally – thus conch shells, for instance, will count as money in some contexts but not in others. The value of an umbrella term is in part precisely this flexibility. Indeed, arguably such flexibility is implied by *any* causal account, given the sensitivity of all causal claims to contextual considerations (Northcott 2008b, Schaffer 2005).

Such causal claims are perfectly objective, notwithstanding their contextual relativization. They also allow for elision – and so does innateness. That is, we may without cost often speak *as if* causal (and therefore innateness) claims were not contextually relativized because the salient contrasts, background conditions and so on are obvious. It is only when they are not obvious that explicit relativization is required (Northcott 2012a, 2008b, Schaffer 2005).

Much of the innateness debate within philosophy has focused on psychology and biology. Yet research in the behavioral sciences often presupposes the possibility of generalizations over all or almost all humans – this is the case in many areas of anthropology, psychology, economics, sociology, and history (Kronfeldner et al 2014). There are also examples from animal training, biological anthropology, education theory, transhumanism, and other fields.[19,20] Such generalizations over humans are often assumed to be relatively immune to environmental

[17] We are *not* committed to innateness being a natural kind term – any more than money or erosion are. This defuses Mameli and Bateson's objection (e.g. 2011) that innateness is not a 'cluster' term, therefore (following Boyd) cannot be a natural kind term, and therefore should be eliminated from scientific use. Whether or not innateness is a natural kind, it is a useful one. It also defuses Mameli and Bateson's counterexample of 'jade'. As they point out, jade was erroneously supposed to be a single chemical kind before being discovered to include two different such kinds, namely jadeite and nephrite. But the issue should be whether 'jade' is a useful umbrella term, not whether it is a natural kind. (Although we have no particular commitment regarding 'jade', we note that jewelers at least still find it a useful term.)

[18] Sometimes our interest might be in the underlying mechanisms themselves, in which case indeed one will want to delve deeper than an invocation of innateness. But even then our account's thinness is an advantage, since it enables it to stay agnostic about these separate investigations. Indeed, in many cases the underlying mechanisms are unknown – but the relevant environmental insensitivity is still valuable knowledge.

[19] Moreover, several recent strands in ethics also appeal to the notion of an innate human nature (Hursthouse 1999, Foot 2001, also Buchanan 2011 regarding human enhancement).

[20] Perhaps the division between IFOs and environment is also sometimes useful indirectly (Pinker 2004, 14-17). Waters (2007, although see Northcott 2009) and Stegmann (2014) argue that a focus on genetic causation is justified by several of its formal properties.

intervention, and thus the traits they identify are naturally labeled 'innate' in our sense. Often, we are not interested in the specific underlying mechanisms, thus favoring our thin, abstract account. Moreover, the generalizations frequently play an explanatory role, thus linking them (at least in many cases) closely to causation.

Mameli and Bateson themselves mention the example of *immunology*. This science routinely distinguishes between the innate and acquired components of the immune system. The innate component provides immediate defense against infection, utilizing mechanisms that work in a non-specific way; the acquired (or 'adaptive') component develops and changes in response to specific past infections, as when one acquires immunity to a virus after having caught it once.[21] The 'innate' label here has a clear meaning readily understood by all. It highlights that the relevant distinction is similar to that recurring in many other areas (thereby also aiding comprehension), and it both accurately describes an organism's medical history and accurately guides future interventions. All is in accordance with our account. As is often the case, the invocation of innateness is not only unobjectionable, it is positively helpful.[22]

The innate/acquired distinction is ubiquitous in medicine more generally. It is central to, for instance, the analysis of different vulnerabilities of different groups – thus the tailoring of treatments for particular age cohorts, sexes, ethnicities, and bearers of particular genes. It is useful to know which vulnerabilities can reasonably be inferred (even if only statistically) for a particular patient based on their innate dispositions, and which cannot. What unites these inferences is not the specific mechanisms involved, which indeed sometimes are unknown, but rather the more abstract similarity captured by the concept of innateness.

Several other umbrella terms in medicine perform similar roles and are useful for similar reasons. One familiar example is 'fatal': this is easy to define and is usefully applied to a wide range of situations. It would be perverse to eliminate the term or to insist on disaggregating it. Yet it shares the same alleged weaknesses as innateness: it abstracts over a large range of mechanisms; the very same injury or condition may or may not be fatal depending on context; it is arguably associated with misleading folk notions such as fate or destiny that ignore sensitivity to environmental inputs (e.g. an open wound need not itself be fatal until infected and left untreated); and it may not be a natural kind. Moreover, it carries a new imprecision of its own, namely the exact probability of death required for a condition to qualify. Still, despite all of this it remains frequently useful to know whether a condition is fatal. As with 'fatal', so with 'innate'.

9. Conclusion

Innateness remains a ubiquitous notion within many sciences because it usefully tracks and predicts which interventions affect trait development. Accordingly, we propose to explicate it as follows: factors that are intrinsic to organisms when they originate are innate; other traits are innate to the degree they are caused by factors intrinsic at origin. And factors that are extrinsic to

[21] Mameli and Bateson are concerned that even the adaptive component of the immune system features no learning in the psychological sense (2011, 437). We agree that it doesn't – but this is no barrier to declaring it non-innate. The example again tells only against defining innateness (in our view mistakenly) in terms of absence of learning.
[22] O'Neill (2015, 219) discusses several other examples where innateness is useful to scientific practice.

organisms are environmental; traits are acquired to the degree they are caused by environmental factors.

Skeptics argue that usages of 'innate' in the sciences are mistaken and the concept should be eliminated. They point to some debates within evolutionary and developmental biology where some scientists find it unhelpful. But we should not be parochial or imperialistic: debates among some scientists should not automatically trump the practices of many others. It behooves us to give an adequate account of innateness if at all possible – one that licenses the relevant claims about interventions without any troublesome commitments to particular mechanisms or histories. A contextual-causal account does the job.

References

Ariew, A. (1996). 'Innateness and canalization', *Philosophy of Science* 63, S19-S27.

Ariew, A. (1999). 'Innateness is canalization: a defense of a developmental account of innateness', in V. Hardcastle (ed), *Where Biology Meets Psychology*. (Cambridge: MIT Press.)

Bateson, P., and Gluckman, P. (2011). *Plasticity, Robustness, Development and Evolution*. Cambridge: Cambridge University Press.

Birch, J. (2009). 'Irretrievably confused? Innateness in explanatory context', *Studies in History and Philosophy of Biological and Biomedical Sciences* 40.4: 296-301.

Boone, W., and Piccinini, G. (2016). 'Mechanistic Abstraction,' *Philosophy of Science*, 83.5: 686–697.

Buchanan, A. (2011). *Beyond Humanity? The Ethics of Biomedical Enhancement*. (Oxford.)

Foot, P. (2001). *Natural Goodness*. (Oxford: Clarendon Press.)

Garson, J. 2015. *The Biological Mind: A Philosophical Introduction*. London: Routledge.

Godfrey-Smith, P. (2007). 'Innateness and genetic information', in Carruthers, Laurence, and Stich (eds) *The Innate Mind: Foundations and the Future*. (Oxford University Press.)

Griffiths, P., E. Machery, and S. Linquist (2009). 'The vernacular concept of innateness', *Mind and Language* 24, 605-630.

Griffiths, P., A. Pocheville, B. Calcott, K. Stotz, H. Kim and R. Knight (2015). 'Measuring causal specificity', *Philosophy of Science* 82, 529-555.

Griffiths, P. (2002). 'What is Innateness?', *The Monist* 85.1, 70-85.

Griffiths, P. (2009). 'The Distinction Between Innate and Acquired Characteristics', *The Stanford Encyclopedia of Philosophy* (Fall 2009 Edition), Edward N. Zalta (ed.), URL = <http://plato.stanford.edu/archives/fall2009/entries/innate-acquired/>.

Hitchcock, C. (1996). 'The Role of Contrast in Causal and Explanatory Claims', *Synthese* 107, 395-419.

Hull, D. (1986). 'On Human Nature', *PSA: Proceedings of the Philosophy of Science Association* 2, 3-13.

Hursthouse, R. (1999). *On Virtue Ethics*. (Oxford: Oxford University Press.)

Keller, E. F. (2010). *The Mirage of a Space between Nature and Nurture*. Durham, NC: Duke University Press.

Knobe, J. and R. Samuels (2013). 'Thinking like a scientist: innateness as a case study', *Cognition* 126, 72-86.

Kronfeldner, M., N. Roughley, G. Toepfer (2014). 'Recent work on human nature: beyond traditional essences', *Philosophy Compass* 9, 642-652.

Laland, K. and K. Brown (2007). 'The Niche Construction Perspective', *Journal of Evolutionary Psychology*, 51–66.

Lewis, D. (1973). 'Causation', *Journal of Philosophy*, 70, 556-67.

Lewontin, R. (1974). 'Analysis of variance and analysis of causes', *American Journal of Human Genetics*, 400-411.

Lloyd, G. (2012). *Being, Humanity, and Understanding: Studies in Ancient and Modern Societies*. (Oxford.)

Maley, C. J., and Piccinini, G. (2014). 'The Metaphysics of Mind and the Multiple Sources of Multiple Realizability,' in M. Sprevak and J. Kallestrup (eds) *New Waves in the Philosophy of Mind*, Palgrave Macmillan, pp. 125-152.

Mallon, R., and J. Weinberg (2006). 'Innateness as closed-process invariance', *Philosophy of Science* 73, 323–344.

Mameli, M. (2004). 'Nongenetic selection and nongenetic inheritance', *British Journal for the Philosophy of Science* 55, 35-71.

Mameli, M. and P. Bateson (2006), 'Innateness and the sciences', *Biology and Philosophy* 21, 155-88.

Mameli, M. and P. Bateson (2011). 'An evaluation of the concept of innateness', *Philosophical Transactions of the Royal Society B* 366, 436-443.

Northcott, R. (2006). 'Causal efficacy and the analysis of variance', *Biology and Philosophy* 21, 253-276.

Northcott, R. (2008a). 'Can ANOVA measure causal strength?', *Quarterly Review of Biology* 83, 47-55.

Northcott, R. (2008b). 'Causation and contrast classes', *Philosophical Studies* 39, 111-123.

Northcott, R. (2009). 'Is actual difference making actually different?', *Journal of Philosophy* 106.11, 629-634.

Northcott, R. (2012a). 'Genetic traits and causal explanation', chapter 4 in Kathryn Plaisance and Thomas Reydon (eds) *Philosophy of Behavioral Biology* (Springer: Boston Studies in Philosophy of Science 282), 65-82.

Northcott, R. (2012b). 'Partial explanations in social science', chapter 7 in Harold Kincaid (ed) *Oxford Handbook of Philosophy of Social Science* (Oxford), 130-153.

Northcott, R. (2013). 'Degree of explanation', *Synthese* 190.15, 3087-3105.

O'Neill, E. (2015). 'Relativizing innateness: innateness as the insensitivity of the appearance of a trait with respect to specified environmental variation', *Biology and Philosophy* 30, 211–225.

Pearl, J. (2000). *Causality*. (New York: Cambridge University Press.)

Pinker, S. (2004). 'Why nature and nurture won't go away', *Daedalus* 133.4, 5-17.

Pradeu, T. (2016). 'The many faces of biological individuality', *Biology and Philosophy* 31.6, 761-773.

Sahlins, M. (2008). *The Western Illusion of Human Nature*. (Chicago: Prickly Paradigm Press.)

Schaffer, J. (2005). 'Contrastive Causation', *Philosophical Review* 114.3, 297-328.

Shea, N. (2012). 'Genetic representation explains the cluster of innateness-related properties', *Mind & Language* 27, 466-493.

Shipley, B. (2000). *Cause and Correlation in Biology*. (Cambridge: Cambridge University Press.)

Sober, E. (1998). 'Innate knowledge'. In: Craig, E. and L. Floridi (eds) *Routledge encyclopedia of philosophy*. Routledge, Cambridge, pp. 794–797.

Spirtes, P., C. Glymour, and R. Scheines (2000), *Causation, Prediction, and Search* (2nd edn). (Cambridge, MA: MIT Press.)

Stegmann, U. (2014). 'Causal control and genetic causation', *Nous* 48, 450-465.

Stich, S. (1975). 'Introduction: the idea of innateness'. In: Stich, S. (ed) *Innate ideas*. University of California Press, Berkeley, pp. 1–22.

Sterelny, K. (2012). *The Evolved Apprentice: How Evolution Made Humans Unique*. (MIT Press.)

van Fraassen, B. (1980). *The Scientific Image*. (Oxford: Oxford University Press.)

Waters, C. K. (2007). 'Causes that make a difference', *Journal of Philosophy* 104, 551-579.

Wimsatt, W. C. (1986). 'Developmental constraints, generative entrenchment, and the innate-acquired distinction'. In: Bechtel, W. (ed) *Integrating scientific disciplines*. Martinus Nijhoff, Dordrecht, pp 185–208.

Wimsatt, W. C. (1999). 'Generativity, entrenchment, evolution, and innateness', in V. Hardcastle (ed), *Where Biology Meets Psychology*. (Cambridge: MIT Press.)

Woodward, J. (2003). *Making Things Happen*. (Oxford: Oxford University Press.)

Woodward, J. (2006). 'Sensitive and insensitive causation', *Philosophical Review* 115.1, 1-50.