1. INFORMATION AND CAUSAL DEMOCRACY

Significant progress has been made in philosophy of biology concerning the nature of biological information since an exchange between Philip Kitcher and myself a decade ago. At that time I argued that the idea of biological information was a barrier to understanding gene-environment interaction. Here, however, I describe two new accounts of biological information that provide powerful tools for characterizing gene-environment interaction and implementing Kitcher's principle of "causal democracy."

All phenotypes depend on both genes and environment for their development. This truism constitutes the "interactionist consensus" in biology and philosophy of biology. The interesting question is how genes and environment interact in the development of particular phenotypes. Kitcher (2003a, 290) has argued that this can be settled only by patient empirical research that obeys his principle of causal democracy: "Interactionists ought to support a principle of causal democracy: if the effect E is the product of factors in set S, then, for any $C \in S$, it is legitimate to investigate the dependence of E on C when the other factors in S are allowed to vary."

1. This paper appeared in Singh et al. (2001) before being reprinted in Kitcher's (2003a) collected papers. Kitcher himself cites the paper as 2000 (Kitcher 2003a, 13), which was the physical publication, as opposed to the imprint, of the original volume.
Kitcher’s principle has been widely misrepresented. As the quotation makes clear, causal democracy requires equality of opportunity, not equality of outcomes. Nevertheless, like conservative political commentators, conservative philosophical commentators have represented causal democracy as the demand that all causes be dragged down to the same level and the refusal to acknowledge that some causes are more significant than others.\(^2\) But like any good liberal, Kitcher is merely asking that all causes be given a chance to reveal whether they play a significant role in development. He believes that current empirical evidence suggests that genes and environment interact in many different ways, depending on the phenotype being studied, so that the relative significance of genes and environment must be assessed on a case-by-case basis. He recognizes that the existence of powerful, standardized techniques for investigating genetic factors provides a practical justification for focusing on genes. But he thinks, or at least thought at that time, that the focus on genetic factors to the exclusion of environmental factors is greater than can be justified by these practical considerations.

Kitcher’s (2003a) key message in “Battling the Undead” is that patient reiteration of interactionism and causal democracy are all that is needed for a balanced and accurate assessment of the role of the genes in development. He denies that there are any deeper conceptual reasons for the persistent neglect of the role of the environment in development, contra Richard Lewontin (1983), Susan Oyama (2000b), and Russell Gray and myself (Griffiths and Gray 1994). Kitcher agrees that simplistic genetic determinism can seem like a vampire, rising from the grave each time it seems to have been dispatched, but he counsels patience rather than searching for a conceptual “stake in the heart” (Kitcher 2003a, 283).

The decade since Kitcher proposed the causal democracy principle has seen greatly increased interest in the role of environmental factors in development (Griffiths and Stotz 2013, ch. 5). A major reason for this has been the rise of the “developmental origins of health and disease” paradigm in medicine, which has redirected some of the vast resources available for biomedical research. Research into obesity, for example, now targets not

\(^2\) See, for example, Franklin-Hall 2015; French 2012, 197; Okasha 2009, 724; Rosenberg and McShea 2008, 174; Thornhill 2007, 206; Weber 2006, 607; Woodward 2011, 249. Most of these authors attribute the idea of causal democracy to Susan Oyama (2000a, S333), citing her response to Kitcher where she says that she will not adopt his phrase “causal democracy” because it “introduces into already-complicated discussions rather more additional baggage than is likely to be helpful.” Perhaps these authors have been misled by the fact that the phrase occurs in the title of Oyama’s paper. Kitcher’s democracy principle is not dissimilar to Oyama’s demand for “parity of reasoning” when comparing genetic and nongenetic factors in development, a demand that is usually parodied in the same way as Kitcher’s democracy principle.
just "obesity genes" like LEPR but the epigenetic effects of maternal nutritional state on offspring physiology and broader exogenetic pathways from parent to offspring, such prenatal and neonatal influences on food preferences. In evolutionary biology there has been a parallel rise of interest in adaptive phenotypic plasticity, both within and between generations. In many species a significant component of fitness differences results from transgenerational environmental influences, or "parental effects." Even behavioral geneticists, the target of some of Kitcher's strongest criticisms, have recognized the need to broaden their research to embrace an interactive picture of behavioral development (Hamer 2002). Some of the powerful techniques for studying genetic factors that seemed to provide practical reasons to focus on genes have simply been repurposed for studying non-genetic factors. To take just one example, second-generation sequencing can be used for high-throughput screening of epigenetic marks. The spirit of democracy seems to have been handsomely vindicated.

2. CAUSAL DEMOCRACY AND GENETIC INFORMATION

Kitcher acknowledges that there is something puzzling about how readily people default to monocausal genetic explanations. If we are all interactionists, "why, then, do we always end up discussing whether genotypes are all-powerful in development?" (Kitcher 2003a, 290–300). Susan Oyama has argued that apparently commonsensical recommendations like Kitcher's causal democracy principle are stymied by "the notion that some influences are more equal than others, that form, or its modern agent, information, exists before the interactions in which it appears and must be transmitted to the organism either through the genes or by the environment" (Oyama 2000b, 13). Oyama has carefully dissected the role of this idea in distorting, as she sees it, the interpretation of research and the kind of research that takes place. Ideas like causal democracy must fight against a deep-rooted conviction that while phenotypes have many causes, only some of those causes contain the information that specifies the phenotype, the others playing a merely supportive role for the expression of that information. Her work builds on that of behavioral development researchers such as Daniel Lehrman, Robert Hinde, and Gilbert Gottlieb (see Griffiths and Tabery 2013).

Kitcher is skeptical that there is any problem deeper than the usual preference for overly simple, often monocausal explanations. A decade ago I defended Oyama's diagnosis (Griffiths 2006a), arguing that genetic information is frequently read as "intentional" information that is "about" something in the same way that sentences and thoughts are "about" something.
As a result the relationship between genes and the outcomes about which they contain information is assumed to be more deterministic than is supported by the actual data. For example,

if we describe the same gene as a "genetically encoded instruction" to be a homosexual, then, intuitively, the presence of different genes at other loci, or prenatal environments that do not support the cascade of gene expression, or postnatal environments that lead the brain to mature differently, all merely cause the organism to misinterpret or disobey the instruction contained in the gene. Furthermore, the gene retains its identity as a gay gene even in an individual... who is, phenotypically, a heterosexual. (Griffiths 2006a, 187)

This picture of how the "gay gene" causes same-sex preference does not reflect the actual scientific content of behavioral genetic research (Hamer et al. 1993), but it was clearly the picture operating in public discussion of that work. The idea that genes are units of information meant the gay gene was understood as an intentional cause—the brain is constructed using a homosexual blueprint, or it is instructed to be homosexual. But intentional causation is utterly different from the interactive, context-sensitive, "difference-making" role of genes envisaged by the interactionist consensus (Sterelny and Kitcher 1988).

In more recent work I and my collaborators have tried to bolster such anecdotal arguments with a program of experimental research on the "folkbiology" of behavioral development—how development is understood by people without formal education in biology (Griffiths et al. 2009; Linquist et al. 2011). This work provides some empirical support for Oyama's contentions that people hold a "dichotomous" view of development in which some phenotypic traits express the organism's inner "essence" while others are imposed on it by the environment; that traits that come from the "inside" are thought to be resistant to modification by the environment; and that this inner essence is nowadays thought to be "in the genes."

In this essay, however, I want to consider how the concept of information can play the opposite role, helping to vindicate the principle of causal democracy.

3. PROXIMATE AND ULTIMATE INFORMATION

In 2006 I worked on the assumption that biological information must be either "causal" information or "intentional" information (Sterelny and Griffiths 1999). Causal information is the systematic dependence of one
variable on another, the kind of dependence that is measured in information theory. One variable carries information about another whenever the values of the two variables are systematically related. Intentional information is the context-insensitive “aboutness” described earlier. Intentionality was introduced into philosophy over a century ago as the distinctive feature of human thought and language. In recent decades, however, philosophers and biologists have argued that intentionality can be created by natural selection. On this “teleosemantic” view a variable carries intentional information if it evolved for the purpose of representing another variable (Maynard Smith 2000; Millikan 1984). Hence intentionality can be ascribed to bacteria and to genes, not just to humans.

Causal and intentional biological information can be thought of as “proximate” and “ultimate” information, respectively (Griffiths 2013; Mayr 1961). Causal biological information is a way to describe the causal structure of a living system: How do the parts of the system depend on one another? It can be characterized mathematically using information theory. Hence the study of causal information is part of proximal biology. However, if intentional biological information is teleosemantic information, then it is a way to describe the purpose for which some aspects of a living system evolved: What is the evolutionary function of these parts of the system? This is an ultimate or evolutionary biological question. Knowing how a system works will not tell us anything about the teleosemantic information it contains, unless we also know the selection pressures that created and maintain the system.

In sections 4 and 5 I describe a new theory of causal/proximate biological information. This is in the spirit of Sterelny and Kitcher’s (1988) difference-making analysis of the sense in which a gene may be a gene “for” a phenotype despite the interactionist view that every phenotype depends on many variables. The new theory uses the recent “interventionist” view of causal explanation (Woodward 2003). Some of my earlier claims about causal/proximate biological information need to be revised in light of this recent work. I previously argued that causal/proximate information could not distinguish genetic from nongenetic causes because it is ubiquitous. Any variable that has an effect on the development of a phenotype will contain information about that phenotype in the sense of information theory; knowing the state of the causal variable reduces our uncertainty about the state of the phenotype (Griffiths and Gray 1994; Maynard Smith 2000). In this more recent work, however, my collaborators and I have used the concept of “causal specificity” to discriminate between causes that provide information for their effects and those that do not (Griffiths et al. 2015; Griffiths and Stotz 2013). This reintroduces the possibility that genetic
causes are the sole or main locus of developmental information. However, I will show that our new approach provides a powerful, quantitative way to state the principle of causal democracy and that at least some environmental causes contain enough information to deserve citizenship. In section 6 I describe and build on a debate between Nicholas Shea and myself over whether “ultimate” information can play a role in developmental explanations (Griffiths 2013; Shea 2013). Shea (2007) has developed a sophisticated teleosemantic approach to information and claims that the best way to defend the significance of nongenetic causes is to show that it is not only genes that carry what he calls “inherited information” (Shea 2011). I accept that inherited information is a useful concept in evolutionary theory. However, I have argued that inherited information cannot cause development. I revisit this criticism in the light of significant developments in philosophical accounts of biological teleology in the past decade. These open the way to construct an ahistorical teleosemantics. This defines teleosemantic intentional information in terms of the current causal structure of organisms and makes the presence or absence of this information a potential causal difference-maker in development.

4. CAUSAL SPECIFICITY

It has long been argued that because the effect of an allele substitution depends on many other factors, both other genes and the environment, it is misleading to identify a single allele as the “gene for” a phenotype. However, the fact that alleles produce phenotypes interactively does not prevent their being salient causes of those phenotypes in the interactionist picture favored by Kitcher. Alleles cause phenotypes by making a difference to those phenotypes against a background of other factors. This idea was spelled out in detail by Kitcher in an article with Kim Sterelny:

An allele A at a locus L in a species S is for the trait P* (assumed to be a determinate form of the determinable characteristic P) relative to a local allele B [at the same locus] and an environment E just in case (a) L affects the form of P in S, (b) E is a standard environment, and (c) in E organisms that are AB [genotype] have phenotype P*. (Sterelny and Kitcher 1988, 350)

Gray and I offered two criticisms of this definition (Griffiths and Gray 1994). The first concerned the definition of “standard environment,” to which Kitcher (2003a, 291–92) responded with an amended definition. The second criticism was that the definition could equally license “epigenetic
marks for" or "incubation temperatures for" phenotypes. Kitcher does not regard this as a criticism and believes that we can and should treat genes and environment symmetrically in this respect: "Far from being a reductio of the interactionist view, this point simply testifies to the democracy principle introduced above" (293).

The idea that there are genes (and other factors) for phenotypes is closely related to the idea that genes contain information about phenotypes. Sterelny and Kitcher's difference-making approach can be readily translated into information-theoretic terms. Their analysis identifies a covariance between gene and phenotype when other factors are held constant. We can regard the gene as a signal source, the phenotype as a signal receiver, and the other factors as channel conditions. When the channel conditions are stable, we can reduce our uncertainty about the state of the phenotype by observing the state of the gene, so the gene carries information about the trait (Griffiths and Gray 1994). I observed earlier that "information" in this sense is ubiquitous. All developmental factors carry such information. For many authors, the fact that causal information is found in all factors affecting development is a reason to look for another kind of information that is found only in genes, or in genes and some special selection of environmental factors. This has typically been teleosemantic intentional information (Maynard Smith 2000; Shea 2007; Sterelny et al. 1996). However, an alternative strategy is to develop a more discriminating causal account of information using resources from the philosophy of causation and information theory. It is this strategy that my collaborators and I have pursued in our recent work.

The influential interventionist theory of causal explanation provides new resources for the study of causal difference-making (Woodward 2003). It provides formal criteria that distinguish causal from noncausal relationships, based on the insight that "causal relationships are relationships that are potentially exploitable for purposes of manipulation and control" (Woodward 2010, 314). The theory treats causation as a relationship between variables in a scientific model, using causal graph theory as a canonical format in which to express these models. There is a causal relationship between variables X and Y if it is possible to manipulate the value of Y by intervening to change the value of X. "Intervention" here is a technical notion with various restrictions. For example, changing a third variable Z that simultaneously changes X and Y does not count as "intervening" on X. Causal relationships between variables differ in how "invariant" they are. Invariance is a measure of the range of values of X and Y across which the relationship between X and Y holds. But even relationships with very small ranges of invariance are causal relationships.
The basic interventionist criterion of causation is deliberately weak, admitting even causal relationships that hold only under very narrow conditions. In more recent work Woodward and others have examined why some causes are singled out as more salient or significant than others. They have focused on how such “causal selection” occurs in biology (Stotz 2006; Waters 2007; Weber 2006; Woodward 2010). One of the most prominent proposals is that causes differ in the degree to which they are “specific” to their effects.

Causal specificity can be illustrated by contrasting the tuning dial and the on-off switch of a radio. Hearing the news is equally dependent on the dial taking the value “576” and on the switch taking the value “on.” But the dial seems to have a different kind of causal relationship with the news broadcast than the switch does. The switch is a nonspecific cause, whereas the dial (or digital tuner) is a specific cause. Interventions on a specific causal variable can be used to produce a large number of different values of an effect variable, providing what Woodward (2010, 302) terms “fine-grained influence” over the effect variable.

The existing literature on causal specificity is mostly qualitative, relying on examples and intuition, and the authors recognize that greater precision is needed (Weber 2006, 606). Woodward has suggested that the limit of fine-grained influence is a bijective mapping between the values of the cause and effect variables: every value of E is produced by one and only one value of C and vice versa. The idea of a bijective mapping does not admit of degrees, but we have developed an information-theoretic framework with which to measure the specificity of causal relationships within the interventionist account, with a bijective mapping as a limiting case (Griffiths et al. 2015). Our measure formalizes the idea that, other things being equal, the more a cause specifies a given effect, the more knowing how we have intervened on the cause variable will inform us about the value of the effect variable. This led us to propose a simple measure of specificity:

Spec: The specificity of a causal variable is obtained by measuring how much mutual information interventions on that variable carry about the effect variable.

The mutual information of two variables is simply the redundant information present in both variables. Where $H(X)$ is the Shannon entropy of X, the mutual information of X with another variable Y, or $I(X; Y)$, is given by:

$$I(X; Y) = H(X) - H(X | Y)$$
Mutual information is symmetrical: $I(X; Y) = I(Y; X)$. So variables can have mutual information without being related in the manner required by the interventionist criterion of causation. However, our measure of specificity does not simply measure the mutual information between $C$ and $E$. Instead it measures the mutual information between interventions on $C$ and the variable $E$. This is not a symmetrical measure because the fact that interventions on $C$ change $E$ does not imply that interventions on $E$ will change $C$: in general, $I(\hat{C}; E) \neq I(\hat{E}; C)$, where $\hat{C}$ is read “do $C$” and means that the value of $C$ results from an intervention on $C$ (Pearl 2009).

Any two variables that satisfy the interventionist criterion of causation will manifest some degree of mutual information between interventions and effects. If the relationship $C \to E$ is minimally invariant, that is, invariant under at least one intervention on $C$, then $C$ has some specificity for $E$, that is, $I(\hat{C}; E) > 0$. Conversely, if $C \to E$ has some degree of specificity, then the relationship is invariant under at least one intervention on $C$.

Elsewhere we have argued that a causal relationship in biology should be regarded as an informational relationship when it is highly specific (Griffiths and Stotz 2013). We are not the first to draw a link between information and specificity. Woodward (2010, 312n21) has written, “The ideas of causal specificity and information are obviously closely linked. . . . Biologists tend to think of structures as carrying information when they are involved in causally specific relationships. I regret that I lack the space to explore this connection in more detail.” Sahotra Sarkar (2004) and Ulrich Stegmann (2014) have also argued that the salient causes in development are the most biologically specific causes. Sarkar’s set-theoretic analysis of biological specificity is very similar to Woodward’s idea of a bijective mapping, and I suggest that biological specificity is simply causal specificity in a biological system.

Using our measure, a causal relationship will be highly specific whenever $C$ and $E$ can take many values and there is a high degree of mutual information between them. In informal terms the cause can make the difference between many different states of the effect and can be used to exercise fine-grained control over that effect. This is actually what Francis Crick (1958, 153) meant by information when he introduced the “sequence hypothesis” and the “central dogma of molecular biology”: “Information means here the precise determination of sequence.” The distinction between “instructive” and merely “permissive” causal interaction in developmental biology is also a distinction between more and less specific causes in our sense.

The proposal to identify biological information with causal specificity in biological systems is thus a classic explication (Carnap 1950). We construct
a more precise substitute for an intuitive idea: Informational causes are causes for which $I(\hat{C}; E)$ is substantial. We then demonstrate that there is significant intellectual continuity with the original, intuitive notion. In the next section I show that our analysis of biological information meets another requirement for a good explication: it provides a useful tool for the relevant sciences.

5. SPECIFICITY AND CAUSAL DEMOCRACY

Some authors have suggested that the very idea of causal specificity refutes the principle of causal democracy (see note 2). This relies on the misrepresentation of causal democracy as the view that all causes are equally significant, so it can be refuted merely by showing that it is possible to discriminate. But Kitcher’s principle is that all causes should be given a chance to show their significance. This is evidently compatible with the idea of causal specificity and with other theories of causal selection.

When Crick advanced the sequence hypothesis and central dogma he assumed that the sequence of the gene not only precisely determined the sequence of the product but also completely determined it. The discovery of alternative splicing in the 1970s showed that the sequence of the gene can underdetermine the sequence of the product. Since then, alternative splicing has turned out to be ubiquitous in eukaryotes and has been joined by other mechanisms of pre- and post-transcriptional processing: mRNA editing, co-transcription, programmed frame shift, trans-splicing, translational recoding, and protein trans-splicing. The transcriptome—the total population of RNAs found in the cells of an organism—is at least an order of magnitude greater than the number of genes.

These mechanisms are employed because there are many different types of cell, each of which uses the same genetic resources to make a different set of products; even a single cell uses those resources differently at different stages in its life cycle. This requires additional specificity of a kind not captured by the original “sequence hypothesis.” Crick’s biographer Robert Olby (2009, 251, italics added) notes:

Clearly, in concentrating on this aspect of informational transfer he [Crick] was setting aside two questions about the control of gene expression—when in the life of a cell the gene is expressed and where in the organism. But these are also questions of an informational nature, although not falling within Crick’s definition.
This point was immediately obvious to Crick’s contemporaries and led the ciliate biologist David L. Nanney (1958, 712) to introduce the idea of “epigenetic control systems”:

This view of the nature of the genetic material . . . permits, moreover, a clearer conceptual distinction than has previously been possible between two types of cellular control systems. On the one hand, the maintenance of a “library of specificities,” both expressed and unexpressed, is accomplished by a template replicating mechanism. On the other hand, auxiliary mechanisms with different principles of operation are involved in determining which specificities are to be expressed in any particular cell . . . To simplify the discussion of these two types of systems, they will be referred to as “genetic systems” and “epigenetic systems.” The term “epigenetic” is chosen to emphasize the reliance of these systems on the genetic systems and to underscore their significance in developmental processes.

The philosophical literature on causal specificity has not been blind to this aspect of specificity, and Woodward (2010, 304–5, italics added) has noted that specificity includes “systematic dependencies between a range of different possible states of the cause and different possible states of the effect, as well as dependencies of the time and place of occurrence of E on the time and place of C.”

It is evident that the additional research questions to which Olby refers concern additional sources of specificity. Our account of biological information as causal specificity chimes neatly with the way many biologists use information in these contexts. Regulatory mechanisms that affect how coding sequences are used in a particular cell at a particular time have been described as “amplifying” the information in those coding sequences. Biologists in this field search for the “target sequence specificity” of forms of editing (Davidson 2002) or search for the “missing information” needed to supplement the information in the coding sequence (Wang and Burge 2008).

Nanney hypothesized that the utility of the epigenetic control systems “lies precisely in their ability to respond specifically to altered environmental conditions” (1958, 713, italics added). He suggested that the influence of these systems should be understood in terms of their “specificity of induction” of developmental effects (715). We see the same language of specificity employed in developmental biology when biologists distinguish between the more specific instructive and the less specific permissive inductive interactions.
Identifying biological information with causal specificity provides a rigorous analysis of what is meant by information in these contexts and explains why these very disparate physical processes influencing the phenotype share a common informational currency. Causal specificity can provide that currency because it obeys Kitcher’s causal democracy principle. It does not build into the definition of biological information any features that prejudge the issue of which causal factors contain information.

My concern in this and the previous two sections has been with proximate information, information that does causal work in living systems. In the following section I turn to ultimate information, to definitions of information based on the process of evolution. I raise the prospect that these two kinds of information, currently serving very different kinds of biological explanation, can be brought closer together.

6. ULTIMATE INFORMATION

The most developed account of ultimate information in the current literature is Shea’s (2007, 2011) version of teleosemantic intentional information, which he calls “inherited information.” Shea accepts the causal democracy principle. It is an empirical question whether environmental factors contain inherited information:

To make the case against gene centrism, DST [developmental systems theory] should be pointing to theundoubted specialness of genes and saying, “You know that property, the one that makes genes so special? Well that property is found not just in genes but in several other factors in development.” That special role is to transmit information, generated through a process of natural selection, down the generations to inform development. (Shea 2011, 61)

Shea differs from earlier teleosemantic theorists by requiring that representations actually correlate with what they represent, so he describes his account as an “infotel” theory, combining information theory and teleosemantics.

A cause contains “inherited information” if

(a) there is a consumer system which is caused by a range of tokens, including tokens of type R, to produce a range of outputs, with a specific evolutionary function for each type of output, and where every token satisfies (b) to (d) with respect to some content;
(b) Rs carry the correlational information that condition C obtains;
(c) an evolutionary explanation of the current existence of the representing system adverts to Rs having carried the correlational information that condition C obtains; and
(d) C is the evolutionary success condition, specific to Rs, of the output of the consumer system prompted by Rs. (Shea 2013, 5)

Shea defines an “evolutionary success condition” thus: “The proximal evolutionary explanation of the survival and reproduction of the representing system adverts to C’s obtaining when Rs were tokened” (5).

I agree with Shea that both genetic and environmental causes in development can carry inherited information and that this fact can be used to show that they sometimes play the same role in evolution. My concern is that the presence of inherited information, whether in genes or in environment, cannot contribute to proximate explanations of biological development. Conditions (c) and (d) imply that which information a gene carries depends on the selection pressures that acted on ancestral copies of the gene. It follows that physically identical genes—an inherited beneficial allele and a de novo mutation that produces the same allele, for example—need not share any “information” in Shea’s sense. They will both share what he calls “correlational information,” but the new mutation will not meet conditions (c) and (d) and so will contain no inherited information. But this difference in “information” can make no difference to how these genes affect the developmental process, since the two are physically identical. The presence or absence of “information” in Shea’s sense is a difference that makes no difference in development (Griffiths 2013).

Shea (2013) argues that, despite this point, inherited information can explain how organisms develop. He describes models that treat both genes and environmental factors as sources of information about future environments and describes how these models are used to study the conditions under which a phenotype will develop with or without an environmental cue. Similarly biologists can study whether an organism should respond to its own developmental environment or to its parents’ environment, signaled by a cytoplasmic cue, by asking which is the more reliable source of information about the environment in which the organism evolved. Shea’s examples are compelling, and his teleosemantic definition of information is appropriate for such explanations. But these are evolutionary, ultimate explanations and not proximate explanations. By modeling the adaptive advantage of that design in past environments they explain why development is designed in a particular way. They don’t answer the proximate “How?” question any more than an evolutionary model predicting the optimal design of a mitochondrion could have been used to solve the mystery of how oxidative
phosphorylation is possible in biochemistry (see the excellent account in Weber 2005). If a plant adds something to its seeds that causes those seeds to flower early, it must add something more than inherited information.

In the remainder of this section I will explain how a concept of information that can figure in proximate developmental explanations can be constructed by taking Shea’s definition and censoring the claims it makes about history. To do this I will need an ahistorical account of biological teleology. Fortunately there has been an upsurge of interest in such accounts in recent years (Christensen 1996; Griffiths 2009; McLaughlin 2001; Schlosser 1998; Weber 2005; Wouters 2007). The common theme in these accounts is that the function of a biological trait is the contribution it currently makes to survival. But to make such an account work, survival must be understood as survival and reproduction (Griffiths 2009). Focusing on survival without reproduction makes it impossible to understand many functions of physiology and behavior, since many life-history strategies sacrifice health and physical integrity for increased reproductive fitness. In fact the whole apparatus of evolutionary theory is needed to identify “what an organism is doing” because some physiological mechanisms have functions that cannot be characterized except by referring to the evolutionary process, such as controlling the variance of the distribution of numbers of offspring.

The functions of a trait in this ahistorical sense are those features in virtue of which the trait has “survival value” (Tinbergen 1963). Questions of survival value are questions about the causes of current fitness. This aspect of Niko Tinbergen’s “four questions” framework is often misunderstood, but in fact, these questions ask “whether any effect of the observed process contributes to survival, if so how survival is promoted and whether it is promoted better by the observed process than by slightly different processes” (418). The answer to these questions provides vital data with which to ask further, evolutionary questions about the selection pressures that produced the trait. Survival value itself, however, is not a historical matter: “Even if the present-day animals were created the way they are now, the fact that they manage to survive would pose the problem of how they do this” (423).

This ahistorical yet entirely evolutionary approach identifies the functions of a trait with the features that are adaptive, whereas the historical approach identifies functions with the features for which the trait is an adaptation. Although adaptiveness and adaptation are both essential to the theory of natural selection, many philosophers are skeptical about whether an ahistorical definition of function is possible. This skepticism may stem from the deficiencies of one well-known ahistorical theory (Bigelow and Pargetter 1987). But the view that any ahistorical evolutionary view of
function will be incoherent seems to me to be a non sequitur. Here are four versions of the argument: 3

1. What a trait is adapted for is fully determined by facts about the past, although admittedly these may be hard to discover. But there is no determinate fact about what a trait is currently adaptive for. It may do something for one organism but not another, or in one locality but not another. Reply: What a trait is “adapted for” is defined as what it was “adaptive for” in the past. So the first cannot be determinate if the second is not. To identify the evolutionary forces now acting on a population we have to look for general patterns that may not hold for every organism, but we have to do that to identify the evolutionary forces that acted in the past too.

2. Fitness depends on the environment. When studying adaptation we know exactly which environments are relevant: those in the actual past. But when studying why a trait is currently adaptive we have no objective basis for rejecting abnormal environments. Some animals live in zoos—should we include them? Reply: This is a more concrete version of the first argument. There were animals in zoos in the past too. We need to make decisions about what constitutes a single selective process whether we are looking at the past or the present.

3. The evolutionary trajectory leading to the current population is determinate, a matter of past facts. But its future trajectory is indeterminate because future events may interfere with our best prediction. Reply: The ahistorical account does not need to predict the future. It only needs to establish the causes of current fitness. Just as a moving object has an instantaneous trajectory in space, a population has an instantaneous evolutionary trajectory. In experimental studies of evolution, it is this instantaneous trajectory that we actually study. Even what might seem to be essentially “predictive” traits, such as variance of offspring number, can be defined at an instant—that is why we can design games of chance like lotteries rather than having to construct them by trial and error!

4. If functions are defined ahistorically, they will not explain why the traits that have these functions exist. Reply: Ahistorical function is not meant to replace historical function, any more than “adaptive” can replace “adaptation.” The two are complementary. To explain why a trait exists

3. It has also been argued that we cannot describe organisms ahistorically because their parts are defined by their adaptive function (Neander 2002; Rosenberg and Neander 2009). For a refutation, see Griffiths (2006b).
you need the concepts of adaptation and historical function. To explain what a trait currently does for the organism (and to define adaptation) you need the concepts of adaptiveness and ahistorical function.

With this ahistorical account of biological teleology, I can construct a definition of teleosemantic intentional information free of history. References to past evolution in Shea’s conditions are simply replaced by references to present evolution. Rather than inherited information, we get adaptive information:

A cause contains adaptive information if

(a) there is a consumer system which is caused by a range of tokens, including tokens of type \( R \), to produce a range of outputs, with a specific survival function for each type of output, and where every token satisfies (b) to (d) with respect to some content;

(b) \( R_s \) carry the correlational information that condition \( C \) obtains;

(c) an explanation of the current fitness of the representing system adverts to \( R_s \) carrying correlational information that condition \( C \) obtains; and

(d) \( C \) is the success condition, specific to \( R_s \), of the output of the consumer system prompted by \( R_s \) (that is, \( R \) increases fitness because \( C \) obtains).

So if we remove the historical content from Shea’s theory of inherited information, we are left with the claim that some state \( R \) (a) has an effect on the organism, (b) carries information about the environment, (c) the resulting pattern of \( R_s \) has survival value, and (d) each output increases fitness because it fits a specific \( C \). For example, the North American seed beetle \textit{Stator limbatus} follows alternative developmental pathways on different hosts. To survive on Blue Palo Verde seeds, offspring must grow faster and attain a larger final size than those developing on seeds of Catclaw Acacia. Mothers bring this about by laying fewer, larger eggs on the Palo Verde seeds than they do on the Acacia seeds (Fox et al. 1997). Having detected which kind of seed it is depositing eggs upon, the mother signals to the offspring to adopt one growth strategy rather than another. The egg mass contains adaptive information because growth rate (output of the consumer system) is caused by seed mass (\( R \)), which is correlated with the species of tree on which the egg is laid (\( C \)), and fitness is enhanced because different growth rates suit different tree species (in this case the egg mass also contains Shea’s inherited information, since this system is an adaptation, but this will not always be so).
This proposal can be made more comparable to the causal/proximate notion of information described in sections 4 and 5 by stating it is as a relationship among three variables. $C$ continues to denote Shea's environmental condition; $R$ continues to stand for the state that signals this condition; and I introduce $E$ (effect) to denote the outputs of Shea's consumer system.

A causal variable contains adaptive information if

(a) there is a variable $E$ whose value depends on a variable $R$ of which conditions (b) through (d) hold:
(b) $R$ correlates with some third variable $C$;
(c) $E$ contributes to fitness by responding to $R$ because $R$ is correlated with $C$; and
(d) for each $r$, there is a "success condition" $c_k$ such that if the $e_i$ caused by $r_i$ contributes to survival then it does so because $c_k$ is the expected value of $C$ given $r_i$.

A connection to the idea of causal specificity from section 4 can now be seen. $R$ is specifically caused by the environmental variable and specifically causes a state of the organism. It is straightforward to express conditions (a) through (d) formally using the specificity measure introduced in section 4 and adding a fourth variable to denote fitness. Many readers will also have noticed the resemblance between the system described by (a) through (d) and a signaling network (Skyrms 2010). However, a formal treatment would reveal that conditions (a) through (d) are inadequate in ways that are hidden by their merely verbal formulation. A general version of the conditions for either inherited or adaptive information requires a measure of causal influence that does not break down when other variables interact with $C$ to determine $R$ or interact with $R$ to determine $E$. A full treatment is in preparation.

It is important to recognize that adaptive information is entirely compatible with Shea's inherited information. The two relate just as adaptiveness relates to adaptation. For example, just as something needs to be adaptive in the past to be an adaptation in the future, a representation needs to have contained adaptive information in the past if it is to contain inherited information in the future. Another important parallel is that when an adaptation is useful in the current environment, this implies it is still adaptive. Just so, if a system that contains inherited information produces an adaptive fit between organism and environment, this must be because it contains the corresponding adaptive information. Possessing inherited information without adaptive information does not, by definition, produce an adaptive match.
The added value of the idea of adaptive information is that it can feature in proximate explanations of the operation of living systems. This is easiest to see when the variable $C$ is in the external environment, for example, the presence of predators. The statistical and causal relationships among $R$, $C$, and $E$ explain how the organism detects the predators. But adaptive information can also feature in proximate developmental explanations, when $C$ is inside the organism. The statistical and causal relationships among $R$, $C$, and $E$ might, for example, explain how a transcription factor succeeds in relating a stimulus received by a cell to a developmental response (Calcott 2014).

7. CONCLUSION

Since my exchange with Kitcher a decade ago significant progress has been made in philosophy of biology on the nature of biological information. Then I argued that the idea of biological information was a barrier to understanding gene-environment interaction. Here I have argued that biological information can be a powerful tool with which to characterize gene-environment interaction and to implement Kitcher's principle of causal democracy.

In sections 4 and 5 I described a new theory of causal/proximate biological information. This is in the spirit of Sterelny and Kitcher's (1988) difference-making analysis of the sense in which there are genes “for” phenotypes. The new theory combines information theory with the interventionist view of causal explanation to develop a quantitative measure of difference-making.

In section 6 I described a new theory of ultimate, evolutionary information based on an ahistorical teleosemantics. The new theory defines teleosemantic intentional information in terms of the current functioning of organisms and makes the presence or absence of this information a potential causal difference-maker in development.

Both these accounts of information are in principle equally applicable to genetic and nongenetic causes in development. They are powerful resources for the patient, empirical exploration of the relative importance of different causes in the development of phenotypes that Kitcher recommended a decade ago.4

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Reply to Griffiths

PHILIP KITCHER

The genomic research of the past two decades has yielded increasingly complicated pictures of both development and evolution. Along with Karola Stotz and John Dupré, Paul Griffiths has been at the forefront of attempts to bring new discoveries about the dynamic genome to the attention of philosophers. As Paul's illuminating essay reveals, even those who campaigned in the 1980s and 1990s against the oversimplified scenarios often spun in discussions of the "biological basis of human behavior," tales woven by biologists as well as by philosophers, underrepresented the wide variety of ways in which epigenetic and environmental factors enter into proximate and ultimate causation. Even the most ardent interactionists were unaware of the intricacies of the interactions.

Griffiths sees how biologically informed philosophy can make far more precise the principle of causal democracy I advocated in my critique of three prominent ventures in articulating general models of development and evolution that hoped to rival (and displace) the dominant gene-centric approaches. Susan Oyama, Richard Lewontin, and Griffiths (along with Russell Gray) aimed at a novel picture of ontogeny and of evolution, one that would prevent the hasty and unwarranted forms of genetic determinism, seemingly arising with monotonous and potentially harmful regularity. Although I shared these thinkers' concerns, I believed that no such general account was needed. Recognizing causal democracy would suffice.

The principle of causal democracy was intended, as Griffiths sees, to offer equal opportunity to a wide variety of potential factors. It allows different investigators to focus on a particular type of cause and to explore the effects as other factors vary. It also encourages them to consider a variety of potential causes. Perhaps at the end of the day, when different processes are analyzed in terms of different types of causes, it will turn out that DNA sequences play the most important role in a plurality of cases. Or perhaps not—some other factor might dethrone the gene. Alternatively we might discover that no single type of cause is "the most fundamental" across any significant range of developmental and evolutionary phenomena. The whole idea that some particular biological factor is more important than all others might turn out to be a mistake.

Griffiths's views have evolved since our debate of a decade ago—and so have mine. Although I continue to believe that the principle of causal democracy offers valuable methodological counsel in the design of biological research and the appraisal of biological claims, I now think my earlier
objections to the ventures in general modeling were too harsh. Although there are good reasons to doubt whether any fully general model can be precise enough to be helpful, the models Oyama, Lewontin, and Griffiths tried to elaborate are useful tools for explaining families of biological phenomena, and, by demonstrating that, it's possible to destabilize the monocular fixation on genes. I should have recognized the potential worth of some vivid examples of causal democracy in action.

In the rest of this reply I want to consider four debates in which the principle of causal democracy proves useful. The first is the controversy at the heart of Griffiths's essay: Are some causes privileged in virtue of their character as bearers of information? The second is the disagreement about strategies in biomedical research. The third is the ancestral home of my own worries about genetic determinism (and consequent enthusiasm for causal democracy): the continued emphasis on "genetic bases" for complex human behavioral traits. The fourth is an oddity of contemporary analytic philosophy: the uninformed enthusiasm for attempts to show that evolution debunks this or that philosophical position.

A gene-centered view of the biological world often takes flight from the thought that the genes provide "a set of instructions" for the developing organism. As gene-centrists willingly concede, gene action is possible only when other "ancillary" factors are in place. The genes, however, are the "teachers," and nobody reflecting on education should deny that teachers play a more fundamental role than their aides or the kitchen staff or the janitors or the classrooms or the blackboards. Educational democrats might protest that evaluation, and both Griffiths and Nicholas Shea offer an analogous rebuttal in the biological domain. They are both concerned to show that other causal factors can be information-bearing. Hence there is no basis for singling out the genes as privileged.

There's no doubt that appeal to "information" has allowed useful formulations of research questions at some points in the history of molecular inquiry; it did, after all, inspire the search for the genetic code. Griffiths and Shea want to sanction the usage and—democratically—extend the class of potential information-bearers. Their different programs depend on the success of teleosemantics. As a naturalistically inclined philosopher, I'd be delighted were some version of teleosemantics to succeed, but the formulations available so far face well-known difficulties. I'm inclined to suspect that any fully adequate semantic theory will outrun the conceptual resources present-day theorists deploy. I appreciate the possibility that some future semantics will endorse many types of biological cause as information-bearing. But I don't want to leave attacks on the thesis that "genetic causes..."
are privileged because the genes are the sole information-bearing biological entities” hostage to debates about the future of semantics.

So I prefer a different strategy. Sterelny and Kitcher (1988) and Kitcher (2003a) eschew talk of information in terms of a more austere causal idiom; as Griffiths rightly points out, those articles look at the differences causes make, an approach elaborated and much improved in the recent work of James Woodward and Kenneth Waters. I regard the talk of information as a metaphor, useful when it helps to make some biological issue vivid, but potentially misleading, especially when users forget that the coinage is not literal. The ultimate test of a use of the metaphor is to translate back into the primitive austere idiom and to frame questions in terms of difference-making causation. Causal democracy recognizes that there are all kinds of difference-making causes: sometimes allelic differences are important across a wide range of environments; on other occasions an environmental factor (the absence of an important source of some nutrient, say) makes a large and uniform difference across a spectrum of genotypes. The basic causal idiom provides no basis for singling out some causes as always more crucial than others. Treating informational language as metaphor thus diagnoses gene-centrists as people who have allowed a figure of speech to run riot in their thinking.

Griffiths touches on my second debate in his optimistic assessment of the ways biomedical research is broadening its menu of options for exploring the causation of disease. I am less hopeful. In my judgment the narrow focus on genes continues to dominate. A symptom of that dominance is the current fascination with “personalized medicine.” Many universities with prominent schools of medicine—my own among them—are giving very high priority to the establishment of centers in which researchers will use information about DNA sequences in attempts to identify drugs and other forms of treatment that can be beneficial for people who carry a particular sequence (or some member of a family of sequences). This form of gene-centered inquiry is often heralded as “the next phase” in the progress of medicine.

Who could complain? Surely it would be better if physicians knew in advance which of a collection of medical regimes would be optimal for an individual patient. Yet it’s worth asking who the beneficiaries are likely to be. The universities who rush to achieve eminence in personalized medicine are not driven purely by a laudable wish to ameliorate the human condition. Personalized medicine and translational research are terms that often appear in close proximity—and the juxtapositions should remind us of a basic fact: there’s money in it. Visions of profitable patents already dance before administrative eyes. When the universities receive the returns on
their wise investments, it's easy to predict the patients who will benefit most from the new advances. They will be those whose medical insurance will cover the costs of expensive drugs and procedures. The rich will continue to get healthier.

The more resources devoted to personalized medicine, the less money and talent will flow to other biomedical research initiatives. Perhaps there are some genetic determinists who believe that the health gap between rich and poor is to be explained in terms of the prevalence of different genotypes in the two groups, but determinists of this stripe are (fortunately) rare. Higher rates of asthma, diabetes, hypertension, and heart disease among poor people are likely explicable as effects of environmental factors, some of them already known—but not easy to eradicate—and others that remain to be identified. Well-ordered science, used as a diagnostic tool to pick out promising ways of improving overall human health, would probably recommend a turn away from gene-centrism toward programs aimed at discovering more of the harmful environmental factors or developing better strategies for eliminating those already known.

At this point the principle of causal democracy enters discussions of biomedical research policy. So long as gene-centrists can insist on “the fundamental role of genes” in biological processes, the search for molecular causes will seem the most efficacious strategy. The principle of causal democracy challenges this response. Griffiths and Shea are not embroiled in a purely academic debate when they seek different ways of vindicating causal democracy. I prefer a different line of objection, but we agree on the goals: first, the dethronement of genes as privileged causes and, in consequence, the broadening of medical research so that it is less skewed toward further attending to those who are already relatively well off.

Turn now to my third debate. Griffiths correctly points out how contemporary behavioral genetics is beginning to absorb the lesson of causal democracy. Yet a form of genetic determinism continues to infect some of the enterprises that march under the banner of “evolutionary psychology.” Here too there are encouraging exceptions: Griffiths alludes to studies of the effects of maternal nutrition, and there have been some subtle and rigorous investigations of how the nutritional deficiencies of pregnant females affect aspects of the behavior of their daughters (see the work of Patrick Bateson and Daniel Nettle). Some of the investigators (Bateson, for example) were long-standing causal democrats avant la lettre. Their approach contrasts with that of the most prominent style of evolutionary psychology, beloved of journalists needing a “sexy” science story: the “Santa Barbara paradigm.”
The principle of causal democracy descended from my efforts to expose the flaws in the ambitious style of human sociobiology practiced in the 1970s and early 1980s (Kitcher 1985). The mainstream evolutionary psychology of today rarely commits all the errors of its sociobiological ancestor, but there's often a simplified way of thinking about evolution and development, akin to those I criticized, that is essential to the attention-grabbing conclusions. We're told that a particular behavioral propensity—the tendency of young females to find attractive those males who can supply resources, say—would have been advantageous on the savannah. This serves as the basis for a hypothesis about a modular psychological capacity, which can then be seen as operative across a range of experimental or survey data. Contemporary women's behavior is thus interpreted as expressing their savannah-selected propensity to be attracted to well-provided (typically older) men. Add the thesis that selection can operate only where there is genetic variation, and the way is open for the conclusion that this propensity is "hard-wired." At this point, of course, the journalists pick up the story.

Even from the far more limited perspective of the 1980s, there were many evolutionary and developmental alternatives. Today, with our richer awareness of genomic complexities, the route to genetic determinist conclusions is even more crowded with rival scenarios, not to be dismissed out of hand. If it is to fulfill its ambitions, evolutionary psychology must go beyond the simple narratives that dominate many—but not all—ventures under the Santa Barbara paradigm. It should emulate the approach of Bateson and Nettle, with its thorough incorporation of causal democracy.

The simplifications of parts of evolutionary psychology are, however, as nothing compared with the recent fad for "evolutionary debunking arguments" in philosophy. Since the publication of Street (2006), meta-ethics in particular has succumbed to a deluge of articles debating whether, if moral realism is correct, a human capacity for morality could have evolved. (Interestingly Street's original article is more sophisticated about evolution than all those I know that have come after it.) Underlying the stream of papers lies a common trio of ideas: "evolution" means "evolution by natural selection"; natural selection favors or frowns upon very specific traits, things like a capacity for detecting moral truths; and underlying those traits are genes "for" them.

To hold any of these ideas you have to be very innocent with respect to contemporary evolutionary theory. First, when the animal in whose evolution you are interested is Homo sapiens, cultural transmission and cultural selection can play a not inconsiderable part. Thanks to Robert Boyd and Peter Richerson (1985), it's been known for thirty years that cultural
selection can lead to outcomes different from those to which natural selection alone would tend and that the pertinent regime of selection can be maintained under natural selection. More recently Boyd and Richerson (2005) have offered a new “folk theorem”: under cultural selection, just about anything can evolve. That seems close to a debunking of debunking arguments.

Second, the idea that any psychological trait that happens to strike a philosopher’s fancy would be a target of selection is unwarranted speculation. Gould and Lewontin (1979) reminded biologists, as well as some interested philosophers, that the relations between genes and traits are many-to-many, not one-to-one. Assuming we have a single capacity for detecting moral truth, it’s overwhelmingly likely that its genetic basis consists of several (possibly many) loci, and that the alleles at these loci influence a whole spectrum of other characteristics. Natural selection would favor the best combination of such characteristics, and the capacity for moral knowledge might be part of the package quite independently of its direct effects on survival and reproduction. Matters are even worse for the debunkers if there’s no single capacity but rather the environmentally (culturally) contingent interaction among several distinct psychological abilities.

Finally, the general thesis that evolution is all about the selection of underlying genes is belied by the upsurge of recent work in “evo-devo,” by niche construction theory—and by the earlier ventures pioneered by Oyama, Lewontin, Griffiths, and Gray. Like the earliest sociobiologists, the debunkers know nothing of cultural selection, nothing of developmental constraints and the perils of adaptationism, and, unfortunately, nothing of causal democracy either.


References


References


References


