The Fearless Vampire Conservator: Phillip Kitcher and Genetic Determinism*

1. Introduction

Genetic determinism is the idea that many significant human characteristics are rendered inevitable by the presence of certain genes; that it is futile to attempt to modify criminal behavior or obesity or alcoholism by any means other than genetic manipulation. Recent discussion of human cloning has revealed how real a possibility genetic determinism seems to many people. Surveying this discussion, developmental biologist Lewis Wolpert was amused to see so many 'moralists who denied that genes have an important effect on behavior now saying that a cloned individual's behavior will be entirely determined by their genetic make-up' (Wolpert, 1998). His observation is accurate, and the vehemence of many attacks on behavioral genetics probably reflects an underlying belief that if genes affect behavior at all, then they must determine it. In fact, genes are very unlikely to be deterministic causes of behavior, for reasons I will come to in a moment. But if genetic determinism is unlikely to be true, why are we as a community so afraid of it? Wolpert seems to think that moral and political commentators on biology are simply ignorant, but the facts of which they are supposedly ignorant have been widely available for a very long time. Perhaps there is more to the strange persistence of genetic determinism.

The psychologist Susan Oyama has famously compared arguing against genetic determinism to battling the undead:

> "But wait," the exasperated reader cries, "everyone nowadays knows that development is a matter of interaction. You're beating a dead horse.
>
> I reply, "I would like nothing better than to stop beating him, but every time I think I am free of him he kicks me and does rude things to the intellectual and political environment. He seems to be a phantom horse with a thousand incarnations, and he gets more subtle each time around. ... What we need here, to switch metaphors in midstream, is the stake-in-the-heart move, and the heart is 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, 1985: 26-7)

Oyama suggests that genetic determinism is inherent in the way we currently represent genes and what genes do[1]. As long as genes are represented as containing information

---

* To appear in *Genes in development: Rereading the Molecular Paradigm* Neumann-Held, E.M and Rehmann-Sutter, C (Eds), Duke University Press (In Press).

[1] Oyama's influential book *The Ontogeny of Information* (Oyama, 1985), from which the above quotation is drawn, has recently been reprinted with a new introduction by Richard Lewontin (Oyama, 2000b), as have

about how the organism will develop, they will continue to be regarded as determining causes no matter how much evidence exists to the contrary. The denial that developmental information is localized in the genes is the 'stake in the heart' that will lay the vampire of genetic determinism to rest.

Philip Kitcher has strongly disputed Oyama's diagnosis, arguing that the conventional 'interactionist' perspective on development is the correct framework for understanding the role of the genes in development. The persistence of genetic determinism is explained, not by any conceptual problem in current representations of genetic causation, but by two much simpler facts: the universal human preference for simple explanations over complex ones and the sheer difficulty of communicating complex science to a wider audience. He argues further that calls for radical new approaches to understanding the role of the genes in development will only alienate working scientists from efforts to take account of non-genetic factors in development:

> 'critics of conclusions about the important effects of genotype on phenotype will be seen as taking refuge in nebulous appeals for a new general view of causation of behavior and as driven to this predicament solely by their sense of outrage at the determinist claims' (Kitcher, 2001: 408)

While acknowledging the legitimacy of many of Kitcher's observations, I believe that Oyama's view is substantially correct. In this paper I try to support the Oyama diagnosis in three main ways. I break down genetic determinism into a number of component fallacies and argue that each is made more plausible by arguments that rest essentially on attributing semantic properties to the gene. I use data from an empirical study of biologists to document an apparent association between endorsing informational representations of the gene and being relatively uninterested in contextual effects on gene expression. I do not want to place too much weight on this one, preliminary result, but it does suggests that efforts to determine whether Oyama is correct need not be confined to philosophical argument: the claim that genetic determinism is *caused* by a certain representation of the gene can be bolstered by documenting a *correlation* between determinist thinking and that representation. Finally, I suggest that Kitcher is mistaken in thinking that 'neither Lewontin's "dialectical biology" nor Oyama's "developmental systems theory" offer anything that aspiring researchers can put to work' (Kitcher, 2001: 408). There is a substantial research tradition in developmental psychobiology that fits the prescriptions of developmental systems theory (DST) for the simple reason that DST is an attempt to abstract a theoretical framework from research in that tradition. Philosophers of science and other commentators on the biological sciences need to become more aware of this tradition and its achievements. Popular presentations of those achievements may also offer a practical route to improving public understanding of the role of the genes in development.

---

many of her papers (Oyama, 2000a). Another recent collection contains new and classic papers by Oyama and other authors on the developmental systems approach (Oyama, Griffiths, & Gray, 2001).

2. What *is* genetic determinism?

The concept of 'innateness' was inherited by the Darwinian tradition from natural theology (Richards, 1987). In its theological incarnation, innate behavior is behavior that cannot be explained by an animal's use of reason. Innate behavior shows the hand of divine providence, equipping the organism in advance for the challenges that it will face when it is born. Complex instincts like those of the social insects provided the natural theologians of the early nineteenth century with numerous 'evidences of Christianity'. In the Darwinian incarnation of innateness, natural selection takes the place of God in explaining how organisms can manifest behavior that is adaptive when they have had no opportunity to learn that behavior. The existence of complex, instinctive behaviors provides 'evidences of evolution'. The founders of ethology, particularly Konrad Lorenz, continued this tradition with their extensive use of the 'deprivation experiment'. Behaviors that develop when the organism is experimentally deprived of the opportunity to learn are innate whilst those that fail to develop are acquired. But it has been agreed for a several decades that a dichotomy between innate behavior and acquired behavior makes little sense. All behaviors have both genetic and non-genetic causes. For any behavior there will be some genetic modifications that stop in occurring and some non-genetic modifications that stop it occurring. On the one hand, social deprivation of young rhesus monkeys will prevent them from displaying their 'innate' sexual behaviors as adults (Harlow, Dodsworth, & Harlow, 1965). On the other hand, a rat and a bird will emerge from an identical program of conditioning having learnt very different behaviors: their genetic endowment affects what is 'acquired' (Garcia, McGowan, & Green, 1972). Similar examples formed the empirical core of Daniel Lehrmann's influential critique of the use of the innateness concept by early ethologists (Lehrman, 1953), a critique that was widely accepted. Ethologists came to realize that questions about the development of a behavior and questions about its evolution should not be conflated (Tinbergen, 1963). Many evolutionary adaptations require complex and highly specific inputs from the environment, and not all traits that are robust in the face of variations in the developmental environment are evolutionary adaptations. Even Konrad Lorenz grudgingly admitted that he had offered an overly simplistic 'understanding of the relations between phylogenetic adaptation and adaptive modifications of behaviour. It was Lehrman's (1953) critique which, by a somewhat devious route, brought the full realisation of these relations to me.' (Lorenz, 1965: 80)

Once it is accepted that all traits develop as a result of the interaction of genetic and non-genetic factors, genetic determinism becomes a view about *how* these factors interact. This view can be conveniently represented using 'norms of reaction' - graphical representations of a phenotypic variable as a function of genotypic and environmental variables. The strongest form of genetic determinism claims that a norm of reaction show no response to the environmental variable. An organism needs *an* environment for the trait to develop, but it doesn't matter *which* environment (Figure 1). Kitcher suggests that some modern genetic determinists think norms of reaction have this form, but only in some limited, but perhaps contextually important, range of environments. Someone might

claim, for example, that ‘genetic diseases’ develop in any environment except those specifically structured as clinical interventions to cure the disease.

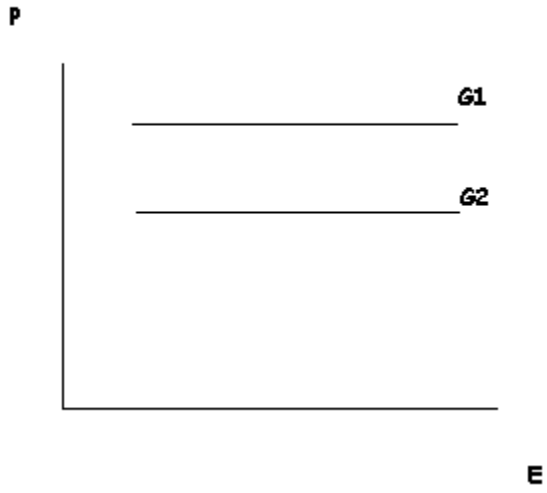

Figure 1. A norm of reaction vindicating genetic determinism

A more moderate form of genetic determinism claims that genetic and environmental factors interact additively (Figure 2). Genotype makes a *constant difference* across some range of environment. A determinist picture of the relationship between genetic factors and education in the determination of IQ might take this form.

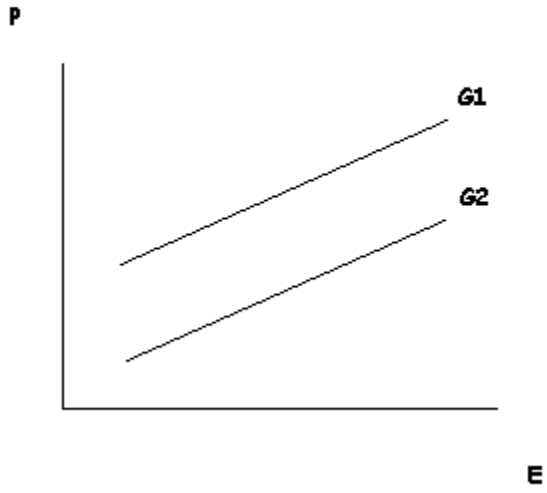

Figure 2. Pure additive interaction between genotype and environment

Perhaps the single most influential contribution to the literature on the interpretation of behavioral genetics is Richard Lewontin paper 'The analysis of variance and the analysis of causes' (Lewontin, 1974). Lewontin pointed out that the empirical evidence suggests that actual norms of reaction are likely to be non-additive (Figure 3). In that case, it makes no sense to talk of a particular genotype 'determining' a particular phenotypic difference. Genotype and environment *jointly* determine the outcome in the straightforward sense that the effect of each factor on the outcome is a function of the particular value taken by the other factor. Nor, as Lewontin further points out, does it make any sense in the context of Figure 3 to perform an analysis of variance on trait differences in a population and interpret the resulting statistic as indicating the percentage contribution of genes and environment to the trait. The very same causal system can produce a pattern of trait differences that correlate 100% with the environmental factor (if everyone lives in the environment where the lines cross) or correlate strongly with genotype (if everyone lives at one extreme of the graph). According to Lewontin and many others, because gene-environment interactions are typically non-additive, heritability studies do not yield information about the relative importance of genetic and environmental developmental factors in the actual causal process that gives rise to a phenotypic trait.

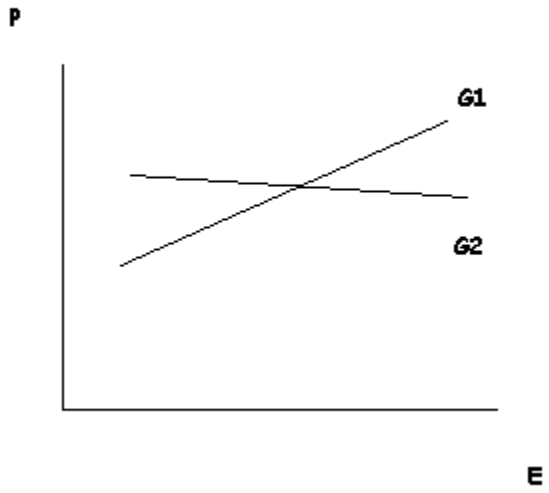

Figure 3. Non-additive interaction between genotype and environment

Figures 1 and 2 represent two senses in which a genotype can be said to 'determine' a trait or a trait difference. For Kitcher, genetic determinism as a general intellectual position is simply the claim that *many* norms of variation have 'determinist' shapes. If

this claim is true, then for many scientific purposes the role of the environment in producing traits or trait differences can be neglected. Hence, according to Kitcher genetic determinism arises from the widespread and understandable human desire for simple, mono-causal explanations. His antidote to genetic determinism is the careful, case-by-case investigation of how genetic and environmental factors interact to determine phenotypes. Kitcher is confident that in many cases, genetic determinism will prove to be false. He sees behavioral genetic research as the most scientifically tractable approach to such investigations. Specific genetic loci that are shown to correlate with behavioral traits in certain environments provide valuable entry points to the complex molecular pathways that construct the behavior. Once these pathways are understood, Kitcher is confident that the importance of non-genetic factors will become evident. I discuss in section six some other scientific approaches to developmental interactions that I take to be at least equally tractable and that have been unduly neglected by Kitcher and other commentators.

It is hard to disagree with Kitcher that the careful elucidation of specific developmental pathways will provide evidence bearing on the issue of genetic determinism. I also share his confidence that in many cases the norm of reaction will turn out not to have a 'determinist' shape. But I am less confident that simply publicizing more examples of non-additive gene-environment interaction will lay the specter of genetic determinism. Unlike Kitcher, Oyama sees the persistence of genetic determinism as a puzzling phenomenon that requires special explanation. Consider, for example, a footnote to Kitcher's paper containing an anecdote about a leading population geneticist's irritated response to the assertion by a behavioral geneticist that heritability figures reveal something about the role of genes in the development of behavioral traits. Heritability measures, Kitcher comments are 'irrelevant' and the fact that behavior geneticists persist in using them is 'an unfortunate tic from which they cannot free themselves' (Kitcher, 2001: 413). It is this sort of anomaly that sends Oyama in search of a cause. Why do so many intelligent scientists appear to ignore facts that are well known to them, such as the likely non-additive interaction of genotypes and environment? For Oyama, 'genetic determinism' refers to something deeper than a pattern of interaction between genotype and environment that may or may not hold in any particular case. It is an underlying attitude to genes and their role in development that makes it hard to assimilate certain facts and easy to assimilate - or to assume - others. Genetic determinism is whatever it is that leads the unreflective to infer from evidence that genes have a causal role in the development of a trait that:

- the prevalence of the trait in the population can never be reduced below the proportion of variance in the trait found to be correlated with genetic factors

- development of the trait will be insensitive to environmental factors in development in rough proportion to 'how genetic' the trait is (the proportion of variance in the trait in some study population which is due to genetic factors)

- a given genetic change will make a constant difference irrespective of the values of other developmental variables

- as a consequence of the last point, the variance accounted for by genetic factors in one population can be safely extrapolated to other populations

It is these and other, similar inferences that Oyama takes to result from the idea that genes contain *information* about the phenotypic outcomes of development.

3. Information in biology

Although biologists think of genes as key parts of the molecular machinery that assembles a protein product, they also think of them as instructions or programs for the production of particular phenotypic traits[2]. In popular science writing this second representation of the gene predominates, leading to assertions like the following:

'An organism's physiology and behaviour are dictated largely by its genes. And those genes are merely repositories of information written in a surprisingly similar manner to the one that computer scientists have devised for the storage and transmission of other information...[biology] is itself an information technology.' (Economist, 1999: 97)

This way of thinking about genes has its roots in Mendelian or transmission genetics - the discipline that first postulated genes. In the absence of any molecular understanding of the gene a tractable theoretical and experimental framework was constructed in which genes were identified by the phenotypic characters with which they correlated in breeding experiments. Developmental biology - the investigation of how characters seen in the parent are reconstructed in the offspring was put to one side in favor of a black box strategy in which genes, identified in the manner described, were treated as if the transmission of a chunk of chromosome explained in and of itself the 'transmission' of the phenotypic character. Making use of metaphors from the new sciences of information theory, cybernetics and computing, biologists came to describe genes as containing 'blueprints', 'programs' and 'instructions' concerning the traits with which they correlate in breeding experiments (Kay, 2000; Keller, 1995). The results of the molecular revolution in biology have been explained to the general public almost entirely in these terms.

The popular understanding of the nature of the molecular revolution, and the common metaphors used by scientists themselves when explaining their work, are in stark contrast to the views of many contemporary philosophers of biology. The biologist and philosopher Sahotra Sarkar has noted that, 'there is no clear, technical notion of "information" in molecular biology. It is little more than a metaphor that masquerades as a theoretical concept and ...leads to a misleading picture of possible explanations in

[2] The contrasting scientific roles of these two ways of thinking about genes are explored at length in (Moss, 2001, 2002). Some exciting potential implications of the first, molecular way of thinking about genes are explored by (Neumann-Held, 1998).

molecular biology.' (Sarkar, 1996:187). The leading philosopher of biology Peter Godfrey-Smith concludes that, 'All the genes can code for, if they code for anything, is the primary structure (amino acid sequence) of a protein' (Godfrey-Smith, 1999: 328). The point is not that there is no useful way to apply formalisms from the information sciences to the study of molecular developmental systems - there are many such ways. The point is that the facts of molecular developmental biology do not correspond to the popular idea that the genetic code is a language in which the genome contains instructions about phenotypes. Kenneth Schaffner has made this point by saying that there are no tiny 'traitunculi' living in the genome (Schaffner, 1998). The slippage from a code for protein structure to a language for specifying phenotypes embodies a systematic confusion about the meaning of the term 'information'.

Concepts of information can be divided into two very broad classes, which Kim Sterelny and I have called 'causal'[3] and 'intentional' (Sterelny & Griffiths, 1999: 101). Causal notions include the measure of quantity of information which is at the heart of the mathematical theory of communication as well as the measures found in algorithmic complexity theory and various notion of information *content* inspired by these mathematical measures of information quantity (e.g. (Dretske, 1981). The simplest causal accounts of the information content of a signal - what the signal is about - define the content as whatever the signal is reliably correlated with. Smoke contains information about fire because, as the saying goes, 'where there's smoke there's fire'. The weakness of this causal account of information content - and of many of its more complex relatives - is that it makes information ubiquitous. As John Maynard Smith has noted:

> With this definition, there is no difficulty in saying that a gene carries information about adult form; an individual with the gene for achondroplasia will have short arms and legs. But we can equally well say that a baby's environment carries information about growth; if it is malnourished, it will be underweight. (Maynard Smith, 2000: 189)

Maynard Smith concludes that a definition of information that can be used to capture the traditional idea that genes carry information while other developmental causes merely support the expression of that information will have to be a definition that includes an element of what philosophers refer to as 'intentionality'.

Intentional information is information in the sense derived from human thought and language. The distinctive feature of intentional information is that it can be false (Godfrey-Smith, 1989). The utterance 'There are fairies at the bottom of my garden', and the thought that accompanies it, have never occurred in response to the presence of fairies in someone's garden, because fairies do not exist. But this has no effect on what the

[3] It has been suggested that we should have used the term 'correlational' in recognition of the fact that classical information theory makes no reference to causation. Instead, we chose to introduce acausal information channels later as a special case. In the biological contexts we were concerned with it is assumed on all sides that correlations between developmental factors and phenotypes are of interest precisely because they provide evidence of an underlying causal network.

utterance means or on the content of the thought. The idea that genes have meaning in something like the way that human thought and language have meaning is lurking in the background in many discussions of genetic information. For example, it has been suggested that under starvation conditions, human mothers methylate growth-enhancing genes in their children, and thus block transcription of those genes. Children with identical genomes and identical nutrition will reach one adult height and weight if they have well-nourished mothers and another if they have starving mothers. When, as in this case, such a response is thought to be an adaptation, it is referred to as a 'disjunctive genetic program' (Figure 4). The genes contain the information ‘grow fast if your mother was adequately nourished, slowly if she was starved’.

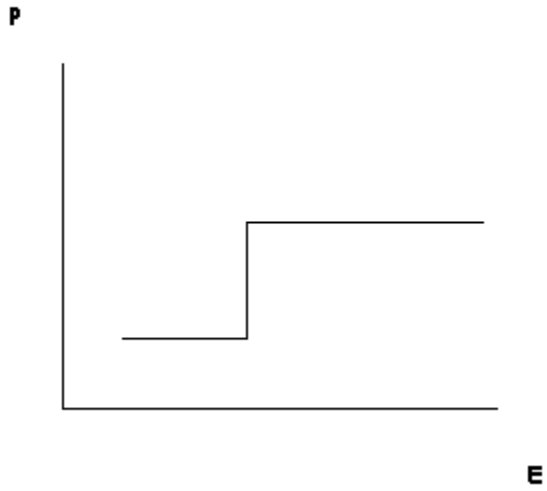

Figure 4. Norm of reaction corresponding to a ‘disjunctive genetic program’

However, there are innumerable cases where the norm of reaction for a trait resembles that in Figure 4, but where we do not talk of disjunctive programs or regard the norm of reaction as an expression of conditional information in the genome. These cases are the pathological or merely quirky effects that revealed by experiments, either those of developmental biologists or those of nature. The claim that the Drosophila genome contains the instruction ‘develop a second thorax when given an ether shock’ sounds like metaphor mingled with hyperbole, as does the claim that the macaque genome encodes the conditional instruction that a mother should kill her babies if she is raised in social isolation. If the concept of information in play were a causal concept, then this would be puzzling, since the causal information in the genome is more or less the same thing as the genome’s norm of reaction. If, however, the concept in play is that of intentional information, then it is clear why some outcomes are regarded as part of the informational content of the genome and others are not. The *intentional* content of an instruction is the behavior it is intended to produce, not the behavior it actually produces. No matter how many students ignore the instruction to write a term paper based closely on the set texts,

the meaning of that instruction remains the same. That is why it is legitimate to deduct grades for not following the instruction even when it is predictable that most students will ignore it!. Likewise, if the human genome contains the intentional information specifying a normal human phenotype, then the information content of the genome is unaffected by the many cases of what Lorenz used to call 'bad rearing'. In such cases, the phenotype misrepresents the information in the genes.

In various contexts, I and others have argued that any analysis of intentional information that makes it part of the natural world will reveal that if genes contain intentional information, so do many other non-genetic developmental inputs. The molecular biologist Robin Knight and I have called this the 'parity thesis'[4]. In this paper, however, the question is not whether the intentional concept of information content is *legitimately* restricted to the genes but only whether it is *as a matter of fact* used when discussing genes and not used when discussing other developmental causes. This latter claim is relatively uncontroversial. The fact that the intentional concept of information is used in this asymmetric way explains why the proposal that all developmental resources contain developmental information has been so controversial. As mentioned above, Maynard Smith has argued that only an intentional concept of information can capture the intent of the many biologists who have used the idea of information to distinguish what genes do in development from what other causes do (Maynard Smith, 2000). I will rest my case, therefore, with just one more example of the asymmetric treatment of genetic and non-genetic causes. A critical temperature range in the nest plays a role in sex determination in crocodiles strikingly similar to that played by the SRY gene on the Y chromosome in mammals. Both initiate a biochemical cascade that masculinizes the fetus. Both causal factors are brought into existence by a complex system that has evolved to ensure that the masculinizing factor generates the correct sex ratio. Despite this, it is not intuitively correct to describe the temperature using locutions that suggest intentional information. Like the SRY gene, the nest temperature can 'cause', 'determine' and even 'signal' the fetus to masculinize, but it sounds odd to say that the molecular kinetic energy in the nest provides the fetus with information about masculinity. It seems natural to say that the SRY gene contains the 'instruction' to masculinize the fetus, but this would seem forced in the case of nest temperature. One might try to justify this asymmetry by arguing that the effect of the temperature is strongly context dependent. It is only in the very precise

[4] The term 'parity' derives from Oyams's call for 'parity of reasoning' in dealing with genetic and environmental causes (Oyama, 1985). '*Parity* is the idea that genes and other material causes are on a par. The 'strawman' parody of developmentalism says that all developmental causes are of equal importance. The real developmentalist position is that the empirical differences between the role of DNA and that of cytoplasmic gradients or host-imprinting events do not justify the metaphysical distinctions currently built upon them. Nucleic acid sequences and phospholipid membranes both have distinctive and essential roles in the chemistry of life and in both cases there seems no realistic substitute for them. But the facts of development do not justify assigning DNA the role of information and control while inherited membrane templates get the role of 'material support' for reading DNA.' (Griffiths & Knight, 1998: xxx). See also (Griffiths, 2001; Griffiths & Gray, 1997, 2001; Sterelny, Dickison, & Smith, 1996). The obvious candidate for a naturalistic account of intentional information is a 'telosemantic' account, according to which a representation contains information about the state of affairs that it is an adaptation to represent (Millikan, 1984; Papineau, 1987).

context of a crocodile fetus that this temperature has this effect. The same, however, is true of the SRY gene, whose effects can be blocked by mutations affecting receptors for its products or by the environmental conditions in the womb whose tragic results create work for gender reassignment units around the world (Money, 1993). Both the temperature and the gene act as switches, causing certain other genes to be transcribed. Neither has any connection to a phenotype outside of a specific class of developmental systems.

4. Intentional information and genetic determinism

I have suggested that the asymmetric use of information talk can help to explain the persistence of genetic determinism. The proposed explanation is, in essence, very simple. The predominant vernacular meaning of 'information' is intentional information: the truism that the internet contains a lot of information means, not that it has a high degree of entropy, but that it contains a large number of intentional representations. It is a central feature of intentional information that it retains its identity in the face of misrepresentation or, in the case of imperative representations, non-compliance. This is what makes it possible for intentional representations to be false and for intentional imperatives to be disobeyed. The relationship between an intentional imperative and its effect is thus quite different from that between a material cause and its effect. If we describe a gene as a switch that initiates a cascade of gene transcription leading to, for example, an initial state of the brain which, under some range of environmental conditions, produces a behavioral preference for homosexual relationships, it is evident that the link between the switch and its final effect is a function of the complex causal system in which the switch is embedded. If the context is changed, the gene is no longer a switch that controls homosexuality, any more than a light switch remains a light switch when it is wired to an exhaust fan. If, however, 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 misinterpret the instruction. Furthermore, the gene retains its identity as a 'gay gene' even in an individual to which it has made some other biochemical contribution, and who is, phenotypically, a heterosexual. In other words, intentional information is intrinsically context insensitive and thus intrinsically unsuited to express the causal link between genes and complex phenotypes, because that link is intrinsically context sensitive[5].

[5] Sarkar has proposed that we regard phenotype as 'genetic' only when the phenotype itself can be characterized in terms of a specific molecular product, or, more usually, its absence (Sarkar, 1998). Thus, for example, muscular dystrophy can be defined as the inability to synthesize a key protein. The link between the phenotype and the loss of gene that templates for this protein is context-insensitive because the phenotype more or less *is* the loss of that template capacity.

Representing genes as intentional imperatives that contain a representation of a phenotype supports genetic determinism because it allows genes to retain a link to a specific phenotype when they are moved from one context to another. A causal intervention that removes the causal pathway between a gene and the phenotype with which it was previously associated does not change the 'meaning' of the gene, it merely prevents that gene from being expressed. It does not put the old phenotype on a par with all the other phenotypes that form part of that gene's norm of reaction. The new phenotype with which the gene is associated as a result of the intervention is not the new meaning of the gene, it is merely a misrepresentation of the information embodied in the gene. Allowing genes to retain their imperative link to a particular phenotype across changes in causal context creates a background assumption that if the gene expressed, it will produce the phenotype about which it contains information. The intentional representation of the gene also makes it natural to think that environments in which the gene does not 'express' its meaning are qualitatively different from those in which it does; such environments are somehow abnormal, or pathological because they create a *mismatch* between gene and phenotype. In all these related ways, the intentional representation of the gene supports the idea that genes have a constant effect across context, and hence the idea that genetic and environmental factors interact additively. If genes contain intentional information, then changing the environment either facilitates the expression of this information or hinders expression of the same information. This view is naturally represented by something like Figure 2 above, rather than Figure 3. For example, if genotype G1 in that figure contains the instruction 'be intelligent', for example, changing the environment merely determines the extent to which this instruction is obeyed. It cannot turn G1 into an instruction to be stupid, as would seem to happen in Figure 3. Thus, intentional representations of genes lead almost immediately to one of the central fallacies identified in Lewontin's critique of behavioral genetics - the default assumption that associations between a gene and a phenotypic difference observed in one environment can be extrapolated to any other 'normal' or 'healthy' environment.

The intentional representation of the gene is connected to the other, more vulgar fallacies described in section two by various simple misunderstandings of the sort that Kitcher and others have observed to bedevil public understanding of genetics. If the claim that a behavior is 30% genetic is understood to mean that in 30% of cases studied the behavior can be traced back to the presence of a particular gene, then the intentional representation of the gene suggests that these form a 'hard core' of cases which will be insensitive to environmental variation. It is all too easy to imagine the claim that schizophrenia or same-sex preference is 30% genetic being understood in this way. Alternatively, if a continuous trait such as height or obesity is described as being 30% genetic, and of this is understood as partitioning the actual trait into a portion that can be ascribed to the genes and a portion than can be ascribed to the environment, then the intentional representation of the gene will suggest that, while that part of the trait due to the environment can be modified, the part due to the genes can only be hidden or suppressed my massive environmental manipulation that will be hard to sustain.

5. Can the influence of 'information talk' be tested empirically?

Oyama claims that representing genes and gene action in terms of information leads to certain errors in reasoning. In the last section, I tried to spell out some plausible ways in which this could occur. But Oyama's claim is an empirical one and ought to be capable of empirical testing. If Oyama is correct, then there should be an association between using the informational concept of the gene, in which genes are type-identified by the developmental information they contain, and neglect of the role of contextual factors in gene expression. A questionnaire study conducted on eighty-one post-PhD Australia biologists by Karola C. Stotz and myself in 1999 produced a result that suggests that such an association is worth testing for more carefully (Stotz & Griffiths, In preparation). In that study, biologists with training and experience in developmental biology were much less likely to endorse the idea that the gene can be adequately defined as a unit of information than those with backgrounds in biochemistry and pure molecular genetics (Figure 4).

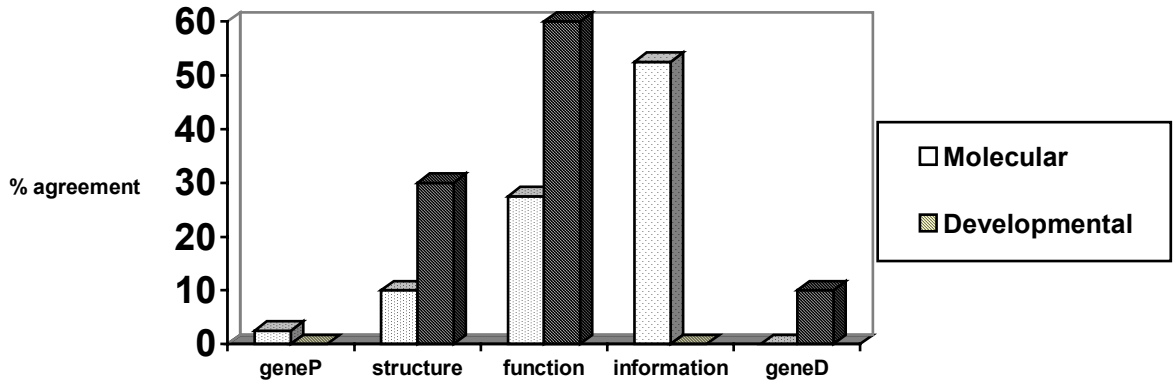

Figure 4. Molecular and developmental biologists were offered a list of 'short definitions' of the gene and asked which they would endorse if forced to choose only one:
1. That which makes the difference between two phenotypes [gene P]
2. A nucleic acid sequence with a certain characteristic structure [structure]
3. A nucleic acid sequence with a certain characteristic function [function]
4. A carrier of heritable information [information]
5. A resource for development [geneD]
[Comments in square brackets are keys to the graph and did not appear on the questionnaire.]
Association 0.553; Significance 0.008.

This result is consistent with the informal comments of many biologists. For example, the leading developmental biologist, Scott Gilbert has remarked that:

> Evolutionary biologists and population geneticists treat genes as markers or patterns analogous to morphological characters - effectively, as modules of structure. In contrast, developmental geneticists and molecular biologists see genes as causal agents, the basis of specific functions, or elements in networks of functionally interconnected units: modules of process or of function. (Gilbert, 2001: xxx)

Other results from the same study are consistent with the equally widely held view that developmental biologists view DNA sequences in the light of contextual factors that affect the expression of gene products. The responses of developmental biologists to questions about whether two DNA sequences are 'the same gene' were significantly influenced by information about such contextual factors. Molecular biologists without experience in developmental biology tended to neglect these contextual factors, in the sense that their survey responses were not affected by information about them. Putting these two results together suggests that those scientists who are least concerned with contextual effects on gene expression are the happiest to endorse the idea that genes are, fundamentally, carriers of information. Hence, I believe that there are good prospects for operationalizing and empirically testing the claim that the informational gene concept has a distorting effect on either research itself or the interpretation of research results by the wider community.

6. The research agenda of developmental systems theory

An important part of Kitcher's critique of Oyama, and of others who argue that the persistence of genetic determinism has a deeper explanation, rests on the observation that, 'neither Lewontin's "dialectical biology" nor Oyama's "developmental systems theory" offer anything that aspiring researchers can put to work' (Kitcher, 2001: 408). Kitcher argues that Oyama and Lewontin call for a radical, new approach to genetic causation when no such approach is available. The result, he suggests, will be to convince practical scientists that whatever the shortcoming of mono-causal genetic explanations, there is no practical alternative. In this respect, I suspect, Kitcher has been misled by the heavy emphasis amongst philosophers of science on evolutionary biology and evolutionary explanations of human behavior. There is a vast philosophical literature on this topic, some of the best of it by Kitcher himself (Kitcher, 1985), and in the evolutionary context, Kitcher's complaint has real substance. Russell Gray and I have described the sort of evolutionary research that might be facilitated by a developmental systems perspective (Gray, 2001; Griffiths & Gray, 2001) and recent work on the evolutionary significance of epigenetic inheritance and niche-construction can be regarded as a partial vindication of these claims (Avital & Jablonka, 2001; Jablonka & Lamb, 1995; Laland, Odling-Smee, & Feldman, 2001; Odling-Smee, Laland, & Feldman, 1996). However, the case for the

practical relevance of developmental systems theory is much easier to make in the developmental context. Until recently, philosophers of science have paid very little attention to developmental biology, and still less to the developmental biology of behavioral traits. But there is a rich experimental tradition in developmental psychobiology dating back several decades and developmental systems theory is to a large extent an attempt to make explicit and reflect on the core concepts of this research tradition. Developmental psychobiology might perhaps be defined as the experimental elucidation of the effects of genetic and environmental factors and their interactions in the ontogeny of gross behavioral traits. This sort of research was pioneered work in the inter-war years by American comparative psychologists and continued after WWII in the work of their students and of developmentally-oriented workers in the new science of ethology, especially those influenced by Daniel Lehrman (Gottlieb, 2001; Johnston, 2001). Developmental psychobiology differs from behavioral genetics in its methodological emphasis on experimental intervention in the laboratory, as opposed to the descriptive-statistical study of natural populations, a trait that places it closer to developmental biology. A textbook presentation of this kind of work can be found in George Michel and Celia Moore's excellent *Developmental Psychobiology : An interdisciplinary science* (Michel & Moore, 1995). When I talk above of the importance of interactions between genes and environment, I am referring not so much to the statistical interactions revealed by behavioral genetics at the population level, but to the causal interactions as revealed by experiments in developmental psychobiology. The call to pay more attention to developmental interactions is not merely an appeal to complexity. Instead, it is an appeal to move beyond using genes as statistical markers for phenotypes and to understand them as biochemical causes of development. As an imperative to researchers, this can mean something very practical and not at all unappealing, like 'going molecular' and investigating the causes of, for example, mental illness at the level of functional genomics, proteomics and developmental neurobiology (Schaffner, 2001).

7. Developmental systems and public understanding of science

I suggest that it will be difficult to improve popular understanding of genetics while continuing to rely on what has so far been the main conceptual tool of popularization - the idea that genes are blueprints or programs. As I have argued, this formulation makes a deterministic reading of claims about the role of genes in development almost inevitable. This poses a considerable problem. 'Information talk' in molecular biology is not going to disappear in the foreseeable future. There really is a genetic code, numerous legitimate applications of technical notions of information play a role in research in molecular biology and the informal, quasi-cybernetic notions of 'signaling', 'switching' and 'feedback' are the *patois* of molecular developmental biology. Unfortunately, the mathematical meaning of 'information' is too unintuitive and too far from the usual meaning of the word to become part of popular consciousness and even terms like 'signaling' irresistibly suggest that what is being signaled is an intentional message. Hence, neither eschewing information talk not explaining it properly to a wide audience seems to be practicable.

The only practical solution, I suggest, is the popularization of another kind of biological research that can act as a counterweight to popular misinterpretations of information talk. That counterweight ought to be developmental biology and developmental psychobiology. Both disciplines have the advantage that they can discuss gross phenotypic characters that are easily and intuitively grasped by a popular science audience - the carapace of the turtle, or the mutual recognition of parent and offspring in ducks. They explain these characters using causal, rather than informational, locutions and by recounting experimental interventions that often involve macro-level physical processes that are also relatively easy to grasp. Genes often figure in these developmental narratives as things activated by other factors, and those other factors are often environmental. Explanations of development thus automatically correct the impression that genes are God-like 'prime movers themselves unmoved'. Developmental psychobiological explanations have a strong tendency to focus on gene-environment interactions, so they also automatically stress context dependence. Fortunately, popular science writing about this tradition has started to appear in recent years (Bateson, 1999; Gottlieb, 1997; Moore, 2001). It can only be hoped that some stock examples from this tradition become firmly entrenched in the popular imagination as a counterweight to the dim awareness that 'scientists have discovered' genes for this, genes for that and the genes for other.

Paul E. Griffiths,
Department of History and Philosophy of Science,
University of Pittsburgh,
Pittsburgh,
PA 15232,
USA

pauleg@pitt.edu

References

Avital, E., & Jablonka, E. (2001). *Animal Traditions : Behavioural Inheritance in Evolution*. Cambridge: Cambridge University Press.
Bateson, P. P. G. (1999). Design for a Life: How behavior and personality develop. London: Jonathan Cape.
Dretske, F. (1981). *Knowledge and the Flow of Information*. Oxford: Blackwells.
Editorial. (1999, June 26th). Drowning in data. *Economist,* pp. 97-98.
Garcia, J., McGowan, B. K., & Green, K. F. (1972). Biological constraints on learning, In Selgiman, MEP & Hager, J.L (Eds) *Biological Boundaries of Learning*. Appleton, Century Crofts, New York, 1972. (pp. 21-43).

Gilbert, S. F. (2001). Homologies of Process: Modular elements of embryonic construction. In G. P. Wagner (Ed.), *The Character Concept in Evolutionary Biology* (pp. xxx-xxx). New Haven: Yale University Press.
Godfrey-Smith, P. (1989). Misinformation. *Canadian Journal of Philosophy, 19 (5)*, 533-550.
Godfrey-Smith, P. (1999). Genes and Codes: Lessons from the Philosophy of Mind? In V. G. Hardcastle (Ed.), *Biology Meets Psychology: Constraints, Conjectures, Connections* (pp. 305-331). Cambridge, M.A: MIT Press.
Gottlieb, G. (1997). Synthesizing Nature-Nurture : Prenatal Roots of Instinctive Behavior. Hillsdale, NJ: Lawrence Erlbaum Assoc.
Gottlieb, G. (2001). A Developmental Psychobiological Systems View: Early Formulation and Current Status. In S. Oyama & P. E. Griffiths & R. D. Gray (Eds.), *Cycles of Contengency: Developmental Systems and Evolution* (pp. 41-54). Cambridge, Mass.: MIT Press.
Gray, R. D. (2001). Selfish genes or developmental systems? In R. S. Singh & C. B. Krimbas & D. B. Paul & J. Beatty (Eds.), *Thinking about Evolution: Historical, Philosophical and Political Perspectives* (pp. 184-207). Cambridge: Cambridge University Press.
Griffiths, P. E. (2001). Genetic Information: A Metaphor in Search of a Theory. *Philosophy of Science, 68*(3), 394-412.
Griffiths, P. E., & Gray, R. D. (1997). Replicator II: Judgment Day. *Biology and Philosophy, 12*(4), 471-492.
Griffiths, P. E., & Gray, R. D. (2001). Darwinism and Developmental Systems. In S. Oyama & P. E. Griffiths & R. D. Gray (Eds.), *Cycles of Contingency: Developmental Systems and Evolution* (pp. 195-218). Cambridge, Mass.: MIT Press.
Griffiths, P. E., & Knight, R. D. (1998). What is the Developmentalist Challenge? *Philosophy of Science, 65*(2), 253-258.
Harlow, H. F., Dodsworth, R. O., & Harlow, M. K. (1965). Total isolation in monkeys. *Proceedings of the National Academy of Sciences, 54*, 90-97.
Jablonka, E., & Lamb, M. J. (1995). *Epigenetic Inheritance and Evolution: The Lamarkian Dimension*. Oxford, New York, Tokyo: Oxford University Press.
Johnston, T. D. (2001). Towards a systems view of development: An appraisal of Lehrman's critique of Lorenz. In S. Oyama & P. E. Griffiths & R. D. Gray (Eds.), *Cycles of Contingency: Developmental Systems and Evolution* (pp. 15-23). Cambridge, Mass.: MIT Press.
Kay, L. E. (2000). *Who Wrote the Book of Life : A History of the Genetic Code*. Palo Alto: Stanford University Press.
Keller, E. F. (1995). *Refiguring Life: Metaphors of Twentieth Century Biology*. New York: Columbia University Press.
Kitcher, P. (1985). *Vaulting Ambition*. Cambridge, MA: M.I.T. Press.
Kitcher, P. (2001). Battling the undead: How (and how not) to resist genetic determinism. In R. Singh & K. Krimbas & D. Paul & J. Beatty (Eds.), *Thinking about Evolution: Historical, Philosophical and Political Perspectives (Festchrifft for Richard Lewontin)* (pp. 396-414). Cambridge: Cambridge University Press.

Laland, K. N., Odling-Smee, F. J., & Feldman, M. W. (2001). Niche Construction, Ecological Inheritance, and Cycles of Contingency in Evolution. In S. Oyama & P. E. Griffiths & R. D. Gray (Eds.), *Cycles of Contingency: Developmental Systems and Evolution* (pp. 117-126). Cambridge, Mass.: MIT Press.
Lehrman, D. S. (1953). Critique of Konrad Lorenz's theory of instinctive behavior. *Quarterly Review of Biology, 28 (4)*, 337-363.
Lewontin, R. (1974). The analysis of variance & the analysis of causes. *American Journal of Human Genetics, 26*, 400-411.
Lorenz, K. (1965). *Evolution & the Modification of Behaviour* (US ed.). Chicago: University of Chicago Press.
Maynard Smith, J. (2000). The concept of information in biology. *Philosophy of Science, 67*(2), 177-194.
Michel, G. F., & Moore, C. L. (1995). *Developmental Psychobiology : An interdisciplinary science*. Cambridge, MA: MIT Press.
Millikan, R. G. (1984). *Language, Thought & Other Biological Categories*. Cambridge, MA: M.I.T. Press.
Money, J. (1993). *The Adam principle : genes, genitals, hormones & gender : selected readings in sexology*. Buffalo, NY: Prometheus Books.
Moore, D. S. (2001). *The Dependent Gene: The Fallacy of "Nature versus Nurture*". New York: W.H Freeman/Times Books.
Moss, L. (2001). Deconstructing the gene and reconstructing molecular develomental systems. In S. Oyama, Griffiths, P.E, Gray, R.D (Ed.), *Cycles of Contingency: Developmental Systems and Evolution* (pp. 85-97). Cambridge, Mass.: MIT Press.
Moss, L. (2002). *What Genes Can't Do*. Cambridge, Mass.: MIT Press.
Neumann-Held, E. M. (1998). The Gene is Dead - Long Live the Gene: Conceptualising the gene the Constructionist Way. In P. Koslowski (Ed.), *Sociobiology and Bioeconomics. The Theory of Evolution in Biological and Economic Theory* (pp. 105-137). Berlin: Springer-Verlag.
Odling-Smee, F. J., Laland, K. N., & Feldman, F. W. (1996). Niche Construction. *American Naturalist, 147*(4), 641-648.
Oyama, S. (1985). *The Ontogeny of Information: Developmental systems and evolution*. Cambridge: Cambridge University Press.
Oyama, S. (2000a). *Evolution's eye: A systems view of the biology-culture divide.* Durham, North Carolina: Duke University Press.
Oyama, S. (2000b). *The Ontogeny of Information: Developmental systems and evolution* (Second edition, revised and expanded. ed.). Durham, North Carolina: Duke University Press.
Oyama, S., Griffiths, P. E., & Gray, R. D. (Eds.). (2001). *Cycles of Contingency: Developmental Systems and Evolution*. Cambridge, M.A: MIT Press.
Papineau, D. (1987). *Reality and Representation*. NY: Blackwells.
Richards, R. J. (1987). Darwin and the Emergence of Evolutionary Theories of Mind and behavior. Chicago: Univ. of Chicago Press.
Sarkar, S. (1996). Biological information: A sceptical look at some central dogmas of molecular biology. In S. Sarkar (Ed.), *The Philosophy and History of Molecular Biology: New Perspectives* (Vol. 183, pp. 187-232). Dordrecht: Kluwer Academic Publishers.

Sarkar, S. (1998). *Genetics and Reductionism*. Cambridge: Cambridge University Press.
Schaffner, K. (1998). Genes, Behavior and Developmental Emergentism: One Process, Indivisible? *Philosophy of Science, 65*(2), 209-252.
Schaffner, K. F. (2001). Nature and Nurture. *Current Opinion in Psychiatry*, xxx-xxx.
Sterelny, K., Dickison, M., & Smith, K. (1996). The extended replicator. *Biology and Philosophy, 11*(3), 377-403.
Sterelny, K., & Griffiths, P. E. (1999). *Sex and Death: An Introduction to the Philosophy of Biology*. Chicago: University of Chicago Press.
Stotz, K., & Griffiths, P. E. (In preparation). *How scientists conceptualise genes: An empirical study*.
Tinbergen, N. (1963). On the aims and methods of ethology. *Zietschrift für Tierpsychologie, 20*, 410-433.
Wolpert, L. (1998, 7-13 January). Dolly the Sheep. *Independent International,* pp. 18.