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Genes, Causation and Intentionality

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Abstract

I want to exhibit the deeper metaphysical reasons why some common ways of describing the causal role of genes in development and evolution are problematic. Specifically, I show why using the concept of information in an intentional sense in genetics is inappropriate, even given a naturalistic account of intentionality. Furthermore, I argue that descriptions that use notions such as programming, directing or orchestrating are problematic not for empirical reasons, but because they are not strictly causal. They are intentional. By contrast, other notions that are part of the received view in genetics and evolutionary theory are defensible if understood correctly, in particular the idea that genes are the main replicators in evolution. The paper concludes that dropping all intentional or intentionally laden concepts does not force us to accept the so-called causal parity thesis, at least not in its stronger form.

Genes have been occupying center stage in biological research for almost a century now, and there is no end in sight yet. The rationale for spending so much time studying genes is often seen in their role as bearers of genetic information. In addition, the genome is often described as containing a 'genetic program' or a 'genetic blueprint' for making an organism. However, historians and philosophers of biology have launched a sustained wave of criticism at these popular notions. Sahotra Sarkar has argued that 'genetic information' is a metaphor (Sarkar 1996). This conclusion forms the starting point for the historical work of Lily Kay (2000). She has published a monumental study trying to show that information language in genetics is a product of the military technology of the Cold War period, in particular cybernetics and cryptography. This is an interesting historical claim, but it turns critically on the assumption that genetic information is not a part of the real world that was simply *discovered* by molecular biologists. But whether or not information belongs to the world's ontological furniture is a metaphysical, not a historical question. This metaphysical question has not really been settled to date.

A somewhat different critique of the orthodox theory of molecular genetics came from the proponents of the 'developmental systems theory' or 'DST' for short (Griffiths and Gray 1994, Oyama 1985). DST tries to displace genes and DNA as the master molecules of life. According to DST, a developing organism should not be viewed as a little sack of disorganized, passive matter that takes its instructions from the clever genes. A more democratic view of development is called for according to DST. In this view, control is distributed in the entire developmental system. Genes and DNA are not distinguished as the sole carriers of genetic information, nor do they organize or instruct the developmental process in any sense. Some DS theorists even claim that there is no sense at all in which genes play any kind of privileged role in development. This claim is also known as the 'causal parity

thesis' or sometimes as the thesis of 'causal democracy' (Kitcher 2001; see also Rosenberg, this volume).

Due to these various criticisms, I think it is fair to say that, in the history and philosophy of biology, there is a total lack of consensus on the causal role of genes as well as on the reasons why genes are conceptualized by biologists the way they are. Of course, biologists have no such disputes; there is a widespread consensus about the role of genes in the scientific community. In the philosophical debate there is at best a very minimal consensus: *that* genes are causally involved in development and evolution. Any claim stronger than this is likely to incur criticism from one side or another. I don't think that this is all a matter of conceptual confusion; there are salient philosophical issues involved about causation, intentionality and the concept of information. However, some conceptual confusion there is, as I will show later.

A systematic appraisal of the different critiques of the orthodox theory of the gene is beyond the scope of this paper. I will focus on the following questions: (1) is there a genetic program encoded in DNA? (2) Do genes contain information in an intentional sense? (3) Are genes the only (or the main) replicators in evolution? (4) Is the causal parity thesis or the thesis of 'causal democracy' true? I shall address these questions in turn.

1. Is there a genetic program?

Rosenberg (this volume) defends the concept of genetic program and argues that it singles out a unique role for genes. To this idea, it must be objected that to be a program for some process or mechanism means more than to stand in a certain causal relation to that mechanism. A part of a physical system can only be a program in relation to an agent who uses this system for a particular purpose (see also Hunziker, this volume). Without an

intentional user, a program is just one causal component among many. The program of a machine is that part that allows the user to control the operations of the system in a manner that does not require her to intervene once the system is running. Programming presupposes intentionality.

To this line of reasoning, it can be objected that an intelligent designer is not necessary for generating computer programs. There are artificial selection algorithms that can perform this task. The great evolutionary theorist John Maynard Smith has argued that a computer program generated by a selection algorithm can be indistinguishable from a program created by an intelligent designer (Maynard Smith 2000). But if they are indistinguishable and one is a program, then so is the other. The same reasoning can now be applied to genetic programs: DNA sequences that were shaped by many rounds of natural selection could be indistinguishable from sequences that were written by an intelligent designer. Therefore, if the designed sequences are programs, then so is the natural product.

The main problem with this argument is that in the case of the artificial selection algorithms, there is still an intelligent programmer, namely the author of the selection algorithm. Whatever machine code that algorithm generates, its status as a program or code will be parasitic on the status of the selection algorithm as a program. By contrast, nobody has laid down the rules of natural selection. Natural selection is not an algorithm (cf. Dennett 1995). Its rules have no author, and it serves no purpose. It isn't even clear if there are general rules of natural selection (Weber 1998, Chapter 7). Therefore, it has not been shown that there is such a thing as a natural program in biological organisms. As is stands, programs are still necessarily artifacts of rational beings.

To conclude, my claim is that the property of being a program is not a strictly causal property, nor is it reducible to causal properties. To the extent that a developing organism is a causal mechanism, it does not literally execute a program.[1]

2. Do Genes Contain Information in an Intentional Sense?

That there is *some* sense in which genes carry information is not controversial. For example, the Shannon-Weaver information measure is easily applied to DNA. But such an information measure can be applied to any system whose states are reliably correlated; it does not distinguish DNA. Molecular biologists use the term in a sense in which only DNA and sometimes RNA contains genetic information. Several other considerations suggest that biologists use the term 'information' in the context of genes in a very strong sense. A single nucleotide substitution can destroy the genetic information of a gene, for example, if it leads to the insertion of a stop codon. But the mutant DNA sequence still contains the same amount of Shannon-Weaver information. What this suggests is that 'information' is actually used in an *intentional* or *semantic* sense, that is, in a similar or even the same sense in which we say about English sentences that they contain information. It is the existence of genetic information in this strong sense that is controversial.

It might seem absurd to say that DNA has semantic properties. It could be argued that the cell would have to be a conscious interpreter of DNA in order for DNA to have such properties, which is indeed absurd. However, this argument is based on a strong assumption with regards to the nature of intentionality. I think it is this assumption that has made people jump to the immediate conclusion that information in an intentional sense makes no sense in genetics. For it is not obvious that in order to have semantic properties or intentionality, a system needs to be conscious. There are many philosophers of mind who have denied exactly that. There are

naturalistic accounts of intentionality that attribute semantic content on the basis of strictly causal relations, and those don't require consciousness. In fact, some of these accounts see a very intimate relation between information and intentionality.

One example is Fred Dretske's account of natural information, which is at the same time an account of intentional content. Dretske defines natural information with the help of conditional probabilities (Dretske 1981, 65):[2]

A signal r carries the information that s is F iff $p(Fs|r) = 1$ and $p(Fs|\ \overline{r}) < 1$

Now let us see if this account can be applied to genetics. I shall apply Dretske's definition to the idea that genes contain information about the amino acid sequence of proteins:

Gene g carriers the information that the amino sequence of P is Φ iff
$p(\Phi P|g) = 1$ and $p(\Phi P|\ \overline{g}) < 1$

At first sight, this seems plausible. Note that the conditional probability is relative to an appropriate cellular context that includes, in particular, the presence of specific tRNAs and aminoacyl-tRNA synthases (i.e., the set of molecules that determine the relevant genetic code). This is how it should be. For there is nothing wrong with saying that some signal carries the information that s is F only in a specific context. After all, the content of linguistic devices is also relative to a group of speakers who share a common language.

The problem with natural information *sensu* Dretske is rather that it is ubiquitous. Consider:

tRNA sequence τ carriers the information that the amino sequence of P is Φ iff
$p(\Phi P|\tau) = 1$ and $p(\Phi P|\ \overline{t}) < 1$

Note that the antecedent says *t*RNA, not mRNA. tRNA (transfer-RNA) sequences are causally relevant to the production of a specific protein molecule (they determine the assignment of triplets to amino acid), however, tRNA does obviously not contain information about proteins in the same sense as DNA or mRNA (messenger-RNA) does. Unlike the latter, tRNA does not function as a template for protein synthesis. Dretske's concept of natural information seems to be blind to this difference, and, therefore, fails as an analysis of genetic information. I believe that it fails for the same reason as an analysis of intentional content in general, however, this is not the place to argue this.

Probably the best-known naturalistic account of intentionality has been developed by Ruth Millikan and others (Millikan 1984, Price 2001). It is known as 'teleosemantics'. Teleosemanticists want to capture in particular the *normative* aspect of intentionality, that is, the fact that intentional content can be right or wrong. Other naturalistic accounts had difficulties to capture this aspect.

The core of the teleosemantic account is the idea of reducing intentionality to biological functions. Biological functions reproduce a crucial aspect of normativity, namely that a function can fail to deliver, much like an intentional state can fail to represent a state of affairs. These properties seem to make the teleosemantic approach look like the ideal candidate in order to ascribe intentional content to genes. Surely, genes can have biological functions, why not intentionality? I will show now that, in general, this doesn't work. Obviously, this requires that we take a closer look at the specifics of the teleosemantic approach.

Most versions come roughly in three steps: First, biological functions are defined in accordance with the etiological account of functions. Second, it is suggested that the etiological function of certain biological mechanisms generates intentionality. Finally, in a third step, additional constraints are introduced that serve the purpose of stopping us from ascribing intentional content to anything with an etiological function. Here we go:

(1) Etiological theory of biological functions:

> X's function in system S is to ϕ iff X's presence in S is a causal consequence of the ϕ-ing of some tokens of X in S.[3]

(E.g., the heart's function in the circulatory system is to pump blood iff the presence of the heart in the circulatory system is a causal consequence of the blood pumping of some heart-tokens in the circulatory system)

(2) Ascribing intentional content:

> State Φ of the frog's visual system VS means that there is a fly nearby iff it is a biological function of VS to produce Φ exactly when there is a fly nearby.

So far, the account is too permissive. This can be shown with the following counterexample:

> State β of the chameleon's skin means that the environment is brown (or has set of surface properties B) iff it is a biological function of the chameleon's skin to produce β exactly when the environment is brown (or has set of surface properties B)

This is undesirable, because the intentional content threatens to explode. The chameleon's skin color, if it represents anything, would have to represent not just the color of the surroundings, but also the fact that the environment contains predators, that these predators use color vision to locate prey, and so on. Therefore, additional constraints must be introduced (Price 2001, 75ff.):

(3) Additional constraint on naturalized intentionality:

> Intentionality is restricted to mechanisms whose biological function is to control the operation of a second mechanism such as to coordinate the operation of that second mechanism with some condition in the environment.

E.g., it is a function of the frog's visual system to control the mechanism that makes the frog snap at flies.

It is not my goal here to defend this naturalistic account of intentionality.[4] What I want to show instead is that even if it were possible to fly a teleosemantic theory of content in the style of Millikan and others, this would not license the ascription of intentional content to genes in general.

To begin, it must be noted that certain genetic systems satisfy some of the constraints that teleosemanticists impose. Take the following example:

> State *R*: 'repressor bound to *lac* operator' of the bacterium's *lac* operon means that there is no lactose in the medium iff it is a biological function of the *lac* operon to produce *R* exactly when there is no lactose in the medium

This seems fine. The case of the *lac* repressor even satisfies the additional constraint:

> The *lac* operator functions in controlling the operation of the gene expression mechanism such as to coordinate the operation of that mechanism with the presence of lactose in the medium

This example pulls into several interesting directions. First, it ascribes semantic content to an extremely simple life form. Teleosemanticists always choose much more complex organisms, namely metazoans, to illustrate their theory. What this suggests is that they implicitly use some additional constraints on intentional content. It seems to me that they really want to ascribe semantic content only to organisms with a central nervous system. But what is it about brains or ganglions that generates intentionality? An answer to the effect that the central nervous system is a system for information processing will not do, as the concept of information is what we are trying to explicate here. A vicious circle looms here, especially if the information processed by the CNS is of the intentional variety. In other words, the implicit constraint that makes teleosemanticists chose brainy metazoans instead of brainless bacteria as examples threatens to collapse the whole account on being made explicit.

The other interesting point about this example is that, while we succeeded in ascribing intentional content to a simple genetic system, this is a special case. Most genes have no such function as to control the operation of a second mechanism in order to coordinate it with a condition in the environment. Some genes are making proteins all the time, come rain or come shine (some of them are called 'household genes'). Some serve a purely structural, and no regulatory role. But when biologists say that genes are carriers of genetic information they are talking about all genes, at least the ones that code for a protein or RNA molecule.

Therefore, a teleosemantic account fails as an analysis of the concept of genetic information. First appearances notwithstanding, teleosemanticists from the philosophy of mind and language are really false friends for those who like to grant genes intentional status.

To this, it could be objected that the teleosemantic friends from the philosophy of mind and language are really too restrictive on intentionality. Why couldn't we just drop the additional constraints and be happy?

On such a view, any gene that has a history of natural selection would count as a bearer of intentional content and/or of information in an intentional sense. This was the view of John Maynard Smith (2000). However, on pains of trivializing the concept of intentionality, such a course can hardly be recommended. For this again – not unlike Dretske's account – would mean that intentionality is ubiquitous. To identify intentionality with etiological function leads to a strange kind of naturalistic panpsychism. For example, think of all the states of affairs an increased heart rate could be said to represent. Again, we have an explosion of content if we admit functions like these to the category of intentionality.

Can't naturalists define intentionality the way they please in order to find some sense in which genes are carriers of intentional information? I don't think this is acceptable. The notion of intentionality was introduced into philosophy in order to capture a certain phenomenological aspect of minds, namely the directedness or 'aboutness' of certain mental states. This includes the idea that the objects of intentional states are represented as having certain properties (whether or not these objects exist). Anyone who uses this term must show that her theory of intentionality somehow accounts for this phenomenology. Teleosemanticists try to give such accounts, however, they do so precisely in those parts of their theory where additional constraints on intentionality are introduced (e.g., that intentional mechanisms must coordinate

the operation of another mechanism with an environmental condition) and that will keep most genes out of the category.

Again, I am not here to defend or criticize a naturalistic account of intentionality. My point is rather that even *if* such an account were accepted, it wouldn't follow that genes contain information in an intentional sense.

Two final points on this issue. First, if I reject genetic information in the intentional sense, I don't mean to question that this notion has played a very important role historically. I think that viewing the hereditary material in terms of a message that needs to be read by the cell has been highly fruitful as a heuristic. The reason probably is that our minds are strongly accustomed to thinking in terms of meaning and interpretation. The analogy between DNA and a message or code has probably enabled scientists to tap into these cognitive resources as a tool of scientific discovery (Weber 2005b). I think it is not the first time in the history of science that a metaphysically inadequate idea has nevertheless proven to be heuristically important.

Second, my conclusion is merely negative. I don't want to exclude that there is some specifically biological definition of 'information' that is adequate.[5] My present thesis extends only to information in the intentional sense.

3. Is DNA the Only or the Main Replicator?

A replicator is a structure that has a large number of alternative states that can be transmitted to subsequent generations. Genes are replicators; their alternative states are the different possible combinations of the DNA-building blocks A, G, T, and C. The extent to which there

are non-DNA based replicators is controversial. It is sometimes claimed that cellular structures like membranes, centrioles, cytoplasmic gradients, endosymbionts and epigenetic modifications of the DNA molecule (such as DNA methylation) also replicate. However, care must be taken not to confuse the mere copying and subsequent material transmission of a biological structure from parent to offspring with replication. For example, cells receive their membranes from a parent cell. In fact, membranes can only be made from pre-existing membranes. But it does not follow from this that membranes are replicators. For it is not the case that a change in a membrane can be transmitted to future generations. At least I know of no evidence for this. To put it differently, membranes, even though they are materially transmitted, are not capable of transmitting a *causal mark* to future generations. The same holds for centrioles, the proteinaceous structures that are required for cell division and for cytoplasmic gradients that are involved in pattern formation in embryonic development (see Weber 2005a, Chapter 8). DS theorists tend to grant replicator status to all of these structures, even where there is absolutely no empirical evidence or theoretical plausibility.[6]

All this does not mean that DNA is the only replicator known. Mounting evidence suggests that certain chemical modifications of DNA itself or of certain scaffolding proteins in the chromatin are heritable, and that these modifications are functionally and perhaps even evolutionarily relevant (Jablonka and Lamb 2005). These structures are replicators in the strict sense; they can transmit marks to future generations. However, it looks like these epigenetic systems only affect some very specific phenotypic properties. By contrast, changes to a DNA molecule can affect any aspect of the phenotype, even an organism's fundamental *Bauplan*. Thus, DNA is not the only replicator, but there is a sense in which it is the main replicator.

4. Is the Causal Parity Thesis True?

What could it mean when DS theorists say that DNA and genes are 'causally on a par' with other parts of the developmental system (Sterelny and Griffiths 1999, 95)? I suggest that this could mean two things ('CPT:' causal parity thesis):

> Strong CPT: Genes have the same causal relations as other developmental resources.
>
> Weak CPT: Genes belong to the same category of causes as other developmental resources.

Following a philosophical convention, the terms 'strong' and 'weak' refer to the relative logical strength of these statements. The strong CPT is stronger because it implies the weak CPT, but not vice versa.

On the basis of this distinction, it could be argued that, depending on the exact interpretation of the CPT, it is either false or it is not an empirical claim.

The strong CPT is clearly false. DNA has a very unique place in the causal nexus of life. I have already discussed one aspect of DNA's distinctness, namely its status as the main replicator. But in addition, there is a relation of *causal asymmetry* between genes and proteins to the effect that genes determine the linear sequence of proteins, but not vice versa. In some contexts, there is also a causal asymmetry between DNA and RNA. This asymmetry is basically the content of Francis Crick's well-known 'Central Dogma of Molecular Biology.' Crick couched it in terms of information flow, which, for obvious reasons, I can't use here. But I think there is a strictly causal corollary to Crick's Central Dogma, namely the causal

asymmetry inherent in the mechanism of gene expression. For these reasons, the strong CPT fails. What is also important to note is that the strong CPT fails due to *biological facts*.

What about the weak CPT? I am quite happy to accept it. But then I must insist that it is not a biological fact. It is a consequence of the metaphysics of causation. If we are serious about physicalism, we can't introduce special kinds of causes in biology. At least I know of no theory of causation that respects physicalism but at the same time leaves room for 'master causes' or something like that. For a physicalist, all causes are on a par by being physical causes or being realized by physical causes, and no empirical biological fact could possible change that.

Note also that verbs like 'organize', 'instruct', or 'program' (see Section 1), which are sometimes used in the context of genes, do not refer to strictly causal properties. All of these properties are intentional, and I have shown why intentionality has no proper place in genetics.

The final question that I want to address is whether different metaphysical accounts of causation treat the case of the gene any differently. Is there a theory of causation that privileges genes as causes of developmental processes?

If we start from a regularity theory of causation such as Mackie (1980), we recognize that – like any other cause – genes are neither necessary nor sufficient for their phenotypic effects. They are insufficient but non-redundant parts of sufficient but non-necessary conditions ('inus-conditions'). As such, they do not differ categorically from other parts a developing organism. Now, regularity theories of causation allow that some causes be privileged by virtue of some special interests. Such a pragmatic privileging is often expressed by speaking

of '*the* cause' as opposed to merely '*a* cause.' In the case of genetics, the special interests that could mandate a special status of genes could be their technological utility (Gannett 1999). However, such an account is wanting, because we can always ask: But *why* is biotechnology interested in genes so much? The answer 'because they make money' is not really an answer, because what we want to know is what makes genes so useful for producing pharmaceuticals and so on. Note that pharmaceutical companies usually don't sell genes; they sell products that were made with the help of knowledge about genes, and sometimes direct gene products (e.g., proteins). It must be the causal properties of genes that make them so interesting industrially. I suggest that it is their special place in the causal nexus of life – that is, their stability and their role as replicators that allows them to be multiplied easily in the laboratory or industrially. If a pure regularity theory of causation misses this, then so much the worse for a pure regularity theory of causation.

In a similar vein, genes can be privileged because focusing on genes in science is an investigative choice that has turned out to be enormously fruitful. Waters (forthcoming) argues that, while there is always a multiplicity of possible ways of construing biological processes, gene-centered biology is a successful practice that has served the investigative interests of scientists extremely well. From the days of the Morgan school until the Human Genome Project, focusing on genes has allowed scientists to study a great variety of phenomena, from microbiology to cell biology, embryology, immunology, neurobiology, and so forth. Genes have continuously opened new opportunities for research, and this is what drives scientific research (not some fundamental theory or the need to fill in the blanks in some abstract scheme of nature, as traditional philosophers of science have assumed).

While I am sympathetic to such an approach, it is not the full story either, because we can still raise the question of what it is about genes that served scientists' investigative interests so

well. I claim that it is, again, the place of genes in the causal nexus of life that explains why gene-centered biology has been so successful. It is the fact that genes replicate, that they can transmit a causal mark across many generations of living organisms. It is this property that explains the success of gene-centered biology. Genes may be metaphysically ordinary, but they clearly are biologically special.

Notes

[1] To be complete, my argument would have to show that the concept of program couldn't be naturalized. I lack the space to do this here. But note that such a naturalization will probably have to rely on a naturalized account of intentionality. I show in the following section why such accounts fail if applied in genetics.

[2] Dretske uses an equality sign instead of a biconditional to present this definition. Since this makes no sense logically, I have inserted a biconditional – assuming that this is what Dretske actually meant to say.

[3] Note that X's ϕ-ing explains why S has X, which is not the case in some alternative accounts of function

[4] I am inclined to think that intentional content is a *holistic* property of the mind-brain, in the sense that Esfeld (2001) has explicated.

[5] The question is if this concept will ascribe information just to DNA and RNA, or to other biological structures as well (see Rosenberg, this volume).

[6] For example, Sterelny and Griffiths's (1999) textbook is guilty of this.

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