Information, Homology, and Lehrman's dictum

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Introduction

This talk is motivated by some worries about integrating concepts from evolutionary developmental biology (EDB) into developmental psychobiology (DP). DP has traditionally been concerned to keep evolutionary and developmental explanations at arm's length, an approach exemplified by Robert Lickliter's concept of the 'phylogeny fallacy' (Lickliter and Berry 1990). In contrast, EDB is an attempt to integrate evolutionary and developmental explanation. DP has been suspicious of the concept of genetic information, favouring causal-mechanistic explanations of development. EDB researchers, in contrast, see the idea of genetic information as an important theoretical construct. Both these differences are relevant to the concept of homology. Homology sometimes been defined as continuity of genetic information (Roth 1984; but see Roth 1999), and EDB approaches to homology have integrated developmental and evolutionary elements. Whilst the integration of EDB concepts, including the homology concept, into DP has great potential, ideas from DP about the independence of developmental from evolutionary explanations, and about the idea of genetic information, remain important for clear thinking about development.

Lehrman's dictum

Developmental psychobiologists are well-acquainted with Daniel S. Lehrman's saying that "although the idea that behavior patterns are 'blueprinted' or 'encoded' in the genome is a perfectly appropriate and instructive way of talking about certain problems of genetics and evolution, it does not in any way deal with the kinds of questions about behavioral development to which it is so often applied." (Lehrman, 1970: 35). According to Lehrman, it is legitimate to regard the genome as a means for the transmission of information from parent to offspring, but not to explain the development of phenotypic traits as the expression of genetic information.

Applying Lehrman's dictum to homology defined as continuity of genetic information would suggest that even if this is the right approach to understanding homology as an evolutionary relationship, it is not be the right approach to understanding homology as part of an explanation of development.

Lehrman's dictum has recently been challenged by Nicholas Shea, who argues explicitly that the information transmitted from parent to offspring in the genes is also 'read in development' and that the inheritance of this information explains developmental outcomes (Shea In Press). Although Shea's work is a significant advance on previous attempts to use the idea of information to analyse the relationship between evolutionary and developmental explanations, I will reject this particular use of it below. Lehrman is still right on this matter.

But first I outline some of the older debates about genetic information that are the background to my argument.

Causal v semantic information

Accounts of genetic and developmental information have been divided into those which treat these things as 'causal' or statistical information and those that treat them as semantic information.

The first family of information concepts are all in the spirit of 'natural meaning' (Grice 1957). Dark clouds mean rain and tracks in cloud chambers mean particles have passed that way - an event carries information about things with which it is causally correlated. Sophisticated notion in this family include the quantitative measure of information due to Claude Shannon (Shannon 1949) and its relatives. Philosophers have tried to devise a qualitative notion of information (meaning) using Shannon's apparatus (Dretske 1981).

Semantic notions of information ascribe to information-bearing states the very special property that philosophers call 'intentionality'. An intentional state carries the same information irrespective of whether it stands in a correlative or causal relationship to its content, or 'intentional object'. That is to say, an intentional state can be false – it can contain information about states of affairs that do not obtain. Intentional states can not only contain false information about things which exist, they can contain (semantic) information, true or false, about things that do not exist at all – unicorns, or fictional characters. Intentionality has traditionally been taken as one of the things which marks out mental states as distinctive from the rest of the natural world. Another way to look at the distinctive nature of semantic information is that it is *normative*: indicative statements have truth-conditions, imperatives have compliance conditions, etc. Each statement has a state of affairs which may or may not exist, but which is nonetheless the normative condition against which the success of the statement is judged.

Scientifically-inclined philosophers are concerned to 'naturalise' semantic information by showing that it can be reductively explained in terms of less problematic features of the world. One obvious route would be to reduce it to statistical information, but it is widely agreed that attempts to do this have been unsuccessful (Godfrey-Smith 1989). Another popular route seeks to derive the normative features of semantic information from the 'design' process of natural selection (Millikan 1984).

Russell Gray and I proposed (1994) that information obeys the 'parity thesis': if a conception of information can be applied to the role played by genes in development, it can also be applied to the role played by non-genetic causes in development. We made this claim to counter the common use of 'information' as the mark that separates the role of genes in development from the role of other resources. It has now been widely accepted that statistical notions of information obey the parity thesis. One response has been to shift to arguing that genetic information is semantic information, so as to save the idea that genes and only genes contain such information:

> "With this *[statistical]* 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.. ... Informational language has been used to characterize genetic as opposed to environmental causes. I want now to try to justify this usage. I will argue that the distinction can be justified only if the concept of

> information is used in biology only for causes that have the property of intentionality.... A DNA molecule has a particular sequence because it specifies a particular protein, but a cloud is not black because it predicts rain. This element of intentionality comes from natural selection." (Maynard Smith 2000, 189-190)

The 'teleosemantic' approach to genetic information defines the semantic content of a representation as the state of affairs which that representation was designed by natural selection to be caused by. As I and others have pointed out, at least this simple version of teleosemantics also obeys a form of the parity thesis (Sterelny, Dickison et al. 1996; Griffiths 2001). Many of the non-genetic causal inputs to development are present at the relevant stage of the life-cycle as a result of previous episodes of natural selection (e.g. parental care).

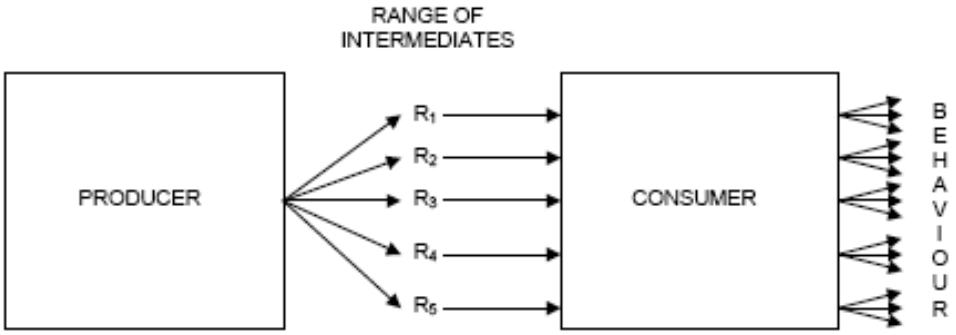
Moreover, Lehrman's dictum would suggest that the very idea that teleosemantic information could play a role in developmental explanation is wrong headed. The teleosemantic content of a representation depends on its evolutionary history. A physically identical state with a different history would not have the same (teleosemantic) content. It would seem to follow that the teleosemantic content cannot be a property in virtue of which the representational states causes something to happen.

Lehrman's dictum is closely related to the 'phylogeny fallacy' criticised by developmental psychobiologist Robert Lickliter (Lickliter and Berry 1990). The phylogeny fallacy occurs when a step in a mechanistic explanation of phenotypic development is filled by a reference to the past evolutionary advantages of a mechanism which could accomplish this step. This is to answer a 'proximal biology' question with an 'ultimate biology' answer (Mayr 1961), although, as we will see, that it not the most adequate framework in which to assess what has gone wrong with such an explanation.

The transmission sense of information

A convincing account of the scientific value of treating genetic and other causes in development as signals carrying teleosemantic information has been developed in recent years. This is sometimes referred to as the 'transmission sense of information' (Bergstrom and Rosvall 2009). The idea is clearly presented by Eva Jablonka (Jablonka 2002). Jablonka argued that a state carries information if there is a 'receiver' which systematically alters its own state in a way that is functional on receipt of states of this kind. If 'functional' is taken to imply that the receiver has been designed by natural selection to respond to the signalling state in this way, we get a form of teleosemantics. Jablonka argued that the value of treating such processes as the flow of information is that we can compare the properties of different heredity systems and assess the selective advantages of one form of heredity compared to another.

Biologists Carl Bergstrom and Martin Rosvall have developed this idea in some detail and given examples of the value of this approach for understanding the evolution of heredity systems. They emphasise the idea that the signal itself, as well as the receiver, has been designed by natural selection (Bergstrom and Rosvall 2009). Perhaps the most thoroughly developed version of this class of approaches is Nicholas Shea's 'infotel' theory of biological information (Shea 2007a; Shea 2007b; Shea In Press). The conditions for a state R to have semantic content are as follows:



R has the **indicative** content C iff:

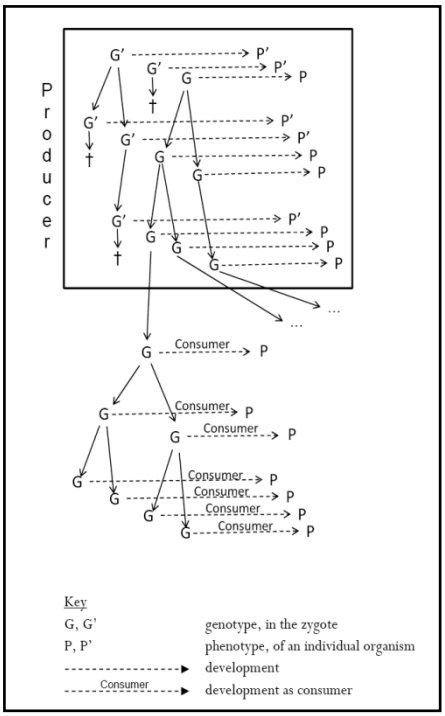
- R's carry the *correlational information* that condition C obtains;
- An evolutionary explanation of the current existence of the representing system adverts to R's having carried the correlational information that condition C obtains; and
- C is *the evolutionary success condition*, specific to R's, of the output of the consumer system prompted by R's. That is, C is the background environmental condition that made producing X adaptive for a consumer in the past.

It is easy to see how these conditions are satisfied by an environmental input to an evolved mechanism of phenotypic plasticity. For example, the kairomones whose detection leads to the development of the defensive morph in Daphnia carry semantic information about predators:

- Kairomones are correlated with predators
- The system evolved because kairomones are correlated with predation
- The consumer system is designed to divert resources to defense when there is increased *risk of predation*

Where the defensive morph is a parental effect, the signal by which the mother induces this developmental pathway in offspring will also carry semantic information about predators.

It is a little harder to see how Shea's model applies to genetic heredity. He gives the following diagram:



In this picture, the producer system is the selective history of a *lineage* of organisms. The representation is a DNA sequence, and the consumer system is the developmental process including interaction with other genes and the environment. The odd feature here is that the 'representation system' flips between the population and individual level. But this is very close to Maynard Smith's vision in which genes carry teleosemantic information because the process of natural selection is analogous to the design of computer code by a 'genetic algorithm' (Maynard Smith 2000). The information is 'programmed' at the population level but 'read' at the individual level. Other authors have criticised Shea because, they argue, his 'development' is not an actual mechanism but a theoretical abstraction.

However, these criticisms are not my concern. I am very happy to embrace the advantages of the 'transmission sense of information' for evolutionary theory. This is even more the case, since its leading advocates accept that it is not *only* genes that carry teleosemantic transmission information (hereafter TTI), but also other adapted developmental causes such as those involved in intergenerational adaptive plasticity, thus accepting a form of the parity thesis. I am also happy to accept Shea's careful development of this approach to give a precise account of the truth-conditions of states which carry TTI. I merely want to argue that TTI is not something which causes developmental or causally explains development.

Why inherited representations are *not* read in development

My thesis seems paradoxical – if the reason offspring resemble their parents is the transmission of information, then surely that information explains the development of the trait in the offspring? To see why not it is useful to think about an example of non-genetic TTI. A recent review of the evolution of parental effects describes a typical effect in the plant *Campanulastrum americanum* (Uller 2008). There is a complex relationship between maternal environment and germination characters in offspring, but one effect is that a herb is more likely to germinate in the autumn and

grow as an annual if the seed comes from a plant growing in high lighting conditions. From an evolutionary point of view, pollen disperses over a larger distance than seeds, so it makes sense for the plant's life-history strategy to reflect the maternal environment and ignore the paternal environment. Phenotypic plasticity and a maternal effect provide an elegant means to achieve this. It is almost impossible to resist describing phenomenon like this in terms of information transmission. The offspring acquires TTI about the environment in which it will develop from its parent.

However, this kind of informational analysis does not translate easily into a causal explanation of development. If the intergenerational signal is a methylation pattern it is relatively easy to think of it as coded information, although we will see below, that would be an equivocation on 'information' as it would not be TTI that does the work in development. But the signal might equally well be seed mass or the thickness of the seed coat. If we ask the question 'how does the seed mass cause late germination' and answer 'by transmitting to the mechanisms of development the information that the plant is likely to grow in high lighting conditions' it is evident how vacuous an explanation this is.

We can see what has gone wrong by applying the 'four questions' framework (Tinbergen 1963). A full biological understanding of a trait involves answering four questions:

1. Causation: what is the mechanism by which the trait produces its effect?

2. Survival value: how does the trait contribute to the organism's fitness? [1]

3. Ontogeny: how is the trait constructed in development?

4. Evolution: "the elucidation of the course evolution must be assumed to have taken, and the unravelling of its dynamics." (1963, 428)

If we look back at the explanatory value which Jablonka and Bergstrom and Rosvall claim for TTI we can see that it is primarily intended to answer the question of Survival Value. It is used to address the design of heredity mechanisms as an evolutionary optimisation problem. What information-theoretic properties should a heredity system have to maximise fitness? This answer to the question of survival value is of immediate relevance to going on to answer the question of evolution, as Tinbergen intended, although it should not be conflated with that question, as Tinbergen stressed in his original presentation.

If we look at the examples Shea gives of the value of TTI in 'explaining development' it turns out that they, too, are all explanations of why development is structured in a particular way. That is to say, they are evolutionary explanations of developmental mechanisms. But an evolutionary explanation of a development mechanism is not the same thing as an explanation of development in the sense of Tinbergen's Ontogeny question. To see the distinction clearly we need to apply the four questions framework to a developmental mechanism. The four questions were originally presented as explanations of a behavioural phenotype, although it was evident that they applied to morphological

[1] In most presentations of the four questions, Survival Value disappears and the two halves of Evolution are separated to make up the numbers. What appears here is what Tinbergen actually says, and it is also a better analysis of biological explanation than the subsequent modifications: see Griffiths, P. E. (2009). "In what sense does 'nothing in biology make sense except in the light of evolution'?" Acta Biotheoretica **57**(1-2): 11-32.

phenotypes as well. A moment's thought will show that they apply to developmental phenotypes. Take, for example, the process of gastrulation, which is explained like this:

> 1. Causation: the molecular and cellular mechanisms by which cells in the blastula migrate and differentiate to form the gastrula
>
> 2. Survival value: 'how survival is promoted and whether it is promoted better by the observed process than by slightly different processes." (1963, 118)
>
> 3. Ontogeny: the development of the blastula, and especially of the specific factors that will cause it to gastrulate
>
> 4. Evolution: the phylogeny of gastrulation and an explanation, perhaps adaptive, of why this stage arose and was preserved

Where will TTI figure in these explanations? Certainly not in Causation. That answer much be given in terms of the physical structure of the blastula and its environment immediately before gastrulation. Nor do I think TTI can play a role in answering Ontogeny. If anywhere, TTI will find a role in answering Survival Value and Evolution.[2]

To summarise, the idea that 'inherited information explains development', where information means TTI, can only mean that TTI figures in an evolutionary explanation of the design of heredity mechanisms or developmental mechanisms. It cannot mean that TTI causally explains the course of development.

Equivocating on information

It was evident that TTI could not causally explain the course of development, since the TTI content of a representation is an historical property, and any causal powers of a representational state must be shared by physically identical state with a different history. The confusion might arise because the very same state that carries TTI also carries information in another sense. For example, if a gene carries TTI, it will also still carry information in Crick's sense – the precise determination of nucleotide sequence. But this is a form of causal/statistical information: a particular sequence of DNA nucleotides specifies the same sequence of amino acids whether or not it has a particular evolutionary history. Insofar as it causally explains development, the 'information' or 'signals' that flows through gene-control networks is also causal information, defined cybernetically in terms of the network structure, not by its phylogenetic origins. Teleosemantic properties can often be ascribed to the same events, but they do not cause developmental in virtue of having those properties.

Once ultimate, evolutionary explanations are distinguished from proximate, developmental explanations the only grounds for denying that environmental factors in development 'carry information' are claims about the structure of the causal mechanism

For example, C. Kenneth Waters has argued that the gene is the sole or main source of molecular specificity, with other causal factors in development at the cellular level having merely 'permissive'

[2] I am not clear if there is, in fact, any role for TTI in this case, since gastrulation is not a heredity mechanism. Perhaps there is a mitotic analogue of TTI that can be optimised in developmental processes?

roles (Waters 2007). Karola Stotz has refuted Water's position using arguments for 'molecular epigenesis' whose general structure will be familiar to any developmental psychobiologist. She documents the instructive role of factors outside the DNA base-sequence which co-determine the precise sequence of the mRNAs and polypeptides that will be derived from the base-sequence (Stotz 2006).

Conclusion

Lehrman's dictum stands. When integrating evolutionary concepts such as homology, in some of its senses (Griffiths 2006; Griffiths 2007; Griffiths 2007), into developmental psychobiology there is a risk of committing the 'phylogeny fallacy' or its relatives.

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