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Lehrman's Dictum: Information and Explanation in Developmental Biology

ABSTRACT: *The integration of concepts from evolutionary developmental biology, such as the homology concept, into developmental psychobiology has great potential. However, evolutionary developmental biology is an attempt to integrate evolutionary and developmental explanation and developmental psychobiology has traditionally been concerned to avoid conflating these two kinds of explanation. This article examines a recent attempt to explain development in terms of "inherited information." The resulting explanation is an evolutionary explanation of development of a kind typical of evolutionary developmental biology. But its proponent mistakes it for an actual developmental explanation. Any integration of evolutionary developmental biology and developmental psychobiology should pay close attention to longstanding concerns about conflating evolutionary and developmental explanations.* © *2012 Wiley Periodicals, Inc.* Dev Psychobiol 55: 22–32, 2013.

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INTRODUCTION

The special issue in which this article appears is an attempt to integrate ideas from the successful new field of evolutionary developmental biology (Hall, 1999) into developmental psychobiology. Evolutionary developmental biology, commonly known as "evo-devo," sets out to integrate evolutionary and developmental explanation. The concept of homology plays a central role in this endeavor (Hall, 1994; Wagner, 2001). Most people are familiar with the idea that homologous features of organisms reflect their descent from a common ancestor, and provide evidence for evolutionary relationships. Evolutionary developmental biologists go beyond treating homologies as products of evolution and regard them as units of evolutionary change. Through understanding the developmental basis of homology they seek "to explain why certain parts of the body are passed on from generation to generation for millions of years as coherent units of evolutionary change . . ." (Wagner, 1994, p. 279). Similar issues about "character identity" confront developmental psychobiologists. When are behaviors in related species, or behaviors in different development stages of an individual, really "the same" behavior? One way to pose this question is to ask whether those behaviors are homologous.

However, in contrast to evo-devo, developmental psychobiology has traditionally been concerned to keep evolutionary and developmental explanations at arm's length, a concern embodied in Robert Lickliter's concept of the "phylogeny fallacy" (Lickliter & Berry, 1990). Developmental psychobiology has been particularly suspicious of explanations of development in terms of a genetic program, or coded information in the genome, favoring instead mechanistic explanations of development in which DNA is one physical resource amongst others. This suspicion of the genetic program concept is not shared by evo-devo. The growth of evo-devo has been closely tied to discoveries in developmental genetics. Many practitioners regard understanding gene regulatory networks as more or less the same thing as understanding development, and their work

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reflects this assumption. A more radical strand of thought in evo-devo known as ''developmental evolution'' is an exception to this generalization (e.g., Müller & Newman, 2003). But the idea that factors outside the genome are part of the developmental system, as opposed to permissive conditions allowing the system to operate, is regarded as misguided by most mainstream evolutionary developmental biologists (Robert, Hall, & Olson, 2001).

Developmental psychobiology has documented the extensive dependence of development on highly specific aspects of what Meredith West and Andrew King have termed the developmental niche (West & King, 1987). A classic strategy to downplay the importance of such discoveries is to argue that no matter what role the environment plays in development, the ''program'' or ''information'' that controls the process is located solely in the genome (for an extended critique of this strategy, including its use in evo-devo, see Robert, 2004). This strategy can readily be applied to the idea of homology. One example is the definition of homology as continuity of genetic information (van Valen, 1982): if the same feature develops in different organisms, or repeatedly in the same organism (serial homology) this shows that the same genetic information has been utilized. It is irrelevant that the environment may have made the same contribution to the development of the trait on each occasion. Van Valen's definition has not been widely adopted, but more recently the leading evolutionary developmental biologist Günther Wagner has suggested that the homologous features may reflect the operation of a conserved circuit of regulatory genes distinctive of each homologue (Wagner, 2007). There is nothing to be said against this as an attempt to answer the question Wagner posed above, but it does embody the assumption that the role of the environment in explaining why organisms develop the features they do is strictly secondary. It would be unfortunate if such overtones were to be imported into studies of behavioral development along with the homology concept.

Whilst the integration of evolutionary developmental biology with developmental psychobiology has great potential, the characteristic concern of developmental psychobiology to distinguish developmental from evolutionary explanations remain important for clear thinking about development. If the concept of homology is to be useful in developmental psychobiology, it must be a version of that concept which does not conflate evolutionary and developmental questions.

LEHRMAN'S DICTUM

One of developmental psychobiology's founding figures, Daniel S. Lehrman, wrote 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, p. 35). Lehrman was writing in response to Konrad Lorenz's later theory of innateness, which defined innate traits as those whose adaptive fit to the environment is explained by the inheritance of ''phylogenetic information'' transmitted in the genes (Browne, 2005; Lorenz, 1965). 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 that genetic information. The idea of genetic information, like the idea of innateness, is a Trojan horse that helps to disguise an evolutionary explanation as a developmental explanation, and obscures the fact that no actual explanation of development has been produced.

The questions about behavioral development that Lehrman has in mind request an explanation of how organisms are able to change from one stage of their development to the next. These are what philosophers of science have termed *mechanistic* explanations (Bechtel & Abrahamsen, 2005). The phenomenon to be explained, in this case the change from an earlier developmental stage of the organism to a later one, is shown to be a consequence of the arrangement of some set of components and the way in which those components behave under accepted physical principles. Explanations of this sort are familiar from fields such as cell biology, where molecular mechanisms produce cellular behavior (Bechtel, 2006), and neuroscience, where molecular and cellular mechanisms explain the activity of the brain (Craver, 2009). Developmental psychobiologists are increasingly also working at the molecular and cellular level, but traditionally they have worked at a slightly higher level of analysis, showing how demonstrated capacities of the organism at an earlier stage, together with aspects of the developmental niche, produce the capacities of the organism at a later stage (for examples, see Michel & Moore, 1995).

George Michel (personal communication, December 15, 2011) has pointed out that Lehrman used the phrase ''development from'' to refer to a perspective in which the investigator asks how the current state of the organism, interacting with its social and physical environment, produces the changes in the organism which give rise to the next stage of development (Lehrman, 1970). Work of this kind leads to detailed fleshing-out of developmental mechanisms. Lehrman contrasted it to a ''development to'' perspective, which takes the later stage of development as a starting point and tries to

identify difference makers with respect to whether organisms reach that stage. This kind of work can be useful as an initial step in discovering developmental mechanisms, but in itself it can only identify salient causal factors and does not address how those factors, in interaction with others, produce the effect. It is useful to know that the light switch turns on the light, but this is no substitute for a wiring diagram. A common example of the ''development to'' perspective is an explanation which shows that the development of a trait depends on the presence of a particular genetic variation. Lehrman's dictum suggests that discovering that there is a ''gene for'' a trait or that the trait is ''genetically encoded'' will never be more than a starting point for the elucidation of an actual developmental mechanism.

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 this information explains how development proceeds (Shea, 2011b, in press). Although Shea's work is a significant advance on previous attempts to use the idea of information to analyze the relationship between evolutionary and developmental explanations, I will argue here that Lehrman is still right: information in Shea's sense of the term does not explain development in the sense that Lehrman intended. Lehrman's developmental explanations capture the mechanisms by which a developmental system constructs a trait. The developmental explanations Shea offers are evolutionary explanations of why these developmental mechanisms have evolved their present form. This is an explanation characteristic of evo-devo. Shea's attempt to substitute one of these explanations for the other is an object lesson in what can go wrong when the lessons of sixty years of developmental psychobiology are disregarded.

Before outlining Shea's proposal I will sketch the earlier debate between biologists and philosophers of biology about the concept of genetic information on which he builds.

TWO KINDS OF INFORMATION

Accounts of genetic and developmental information have been divided into those that treat them as *causal* or statistical information and those that treat them as *semantic* information. The causal family of information concepts are all in the spirit of what philosophers have called ''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 correlated. Sophisticated notions in this family include the quantitative measure of information due to Shannon and Weaver (1949) and its relatives. Philosophers have tried to devise a purely causal notion of information content to complement this account of information quantity using modified versions of Shannon's apparatus (e.g., Dretske, 1981).

The second, semantic family of information concepts ascribe to information-bearing states the property of *intentionality*. If I see a kangaroo at twilight and think that I have seen a kangaroo, the *intentional object* of my thought is simply the object which caused me to have the thought—the kangaroo. But if I see the kangaroo and think I have seen Ned Kelly's ghost, the *intentional object* of my belief is the ghost. The fact that my belief was caused by a kangaroo does not affect the semantic content of the belief, which is that I saw Kelly and not that I saw a kangaroo. It is because the intentional objects of mental states can differ from their causal objects that the content of mental states can be false. Moreover, intentional states can be about things that do not exist at all, like ghosts. Whatever dictates that a state contains certain semantic information, it is not that the state was caused by or correlated with that information. Intentionality has traditionally been taken to be what marks out mental states, and linguistic expressions of those states, as distinctive from the rest of the natural world. Causal information is ubiquitous in the natural world, but semantic information is not. 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*, and so forth. This means that for each intentional state there is a corresponding 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. The indicative statement ''I saw a ghost'' is doomed to fail to match its truth condition because there are no ghosts. It is weighed in the semantic balance and found wanting.

Scientifically inclined philosophers are concerned to ''naturalize'' semantic information by showing that it can be reductively explained in terms of less problematic features of the natural world. This has proved extremely difficult. The 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). In broad terms, this ''teleosemantic'' approach suggests that an indicative representation is true when it is produced in accordance with the evolutionary design of the system that produces it. The truth condition of the statement is the state of affairs in the context of which

the system would produce such a representation if it were functioning as it was designed to function by natural selection. My belief has the content "I saw a kangaroo" because the mental mechanisms which produced that belief fulfill their evolutionary purpose by producing such a belief when I am confronted by a kangaroo. Conversely, my belief has the content "I saw Ned Kelly's ghost" because unless I really did see the ghost, the mental mechanisms which produced that belief have not fulfilled their evolutionary purpose by producing it. False beliefs are possible because adaptations do not always do what they are designed to do.

The overall success or failure of the teleosemantic program for reducing semantic content to facts about evolutionary design need not concern us here. I introduce it only because both biologists and philosophers have used this approach to explain what they mean by "genetic information." These definitions of "genetic information" stand or fall on their own merits and not on whether the same definition can be used to explain the semantic content of thought and language.

INFORMATION IN DEVELOPMENT

Building on the work of the developmental theorist Susan Oyama (1985), Russell Gray and I proposed that all concepts of information obey a "parity thesis" (Griffiths & Gray, 1994): if a conception of information can be applied to genes because they play a particular role in development, then it can also be applied to non-genetic causes which play the same role 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 developmental causes. It has now been widely accepted that causal 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. The eminent evolutionary biologist John Maynard Smith wrote that:

> 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, pp. 189–190).

Maynard Smith hoped a teleosemantic approach to information would vindicate the idea that only genes carry information relevant to the development of evolved traits. As I and others have pointed out, teleosemantic information actually obeys a form of the parity thesis (Griffiths, 2001; Shea, 2011a; Sterelny, Dickison, & Smith, 1996). 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., methylation patterns, parental care). These non-genetic developmental inputs are designed to influence the development of evolved traits and so by definition they carry teleosemantic information about developmental outcomes.

However, even if some more sophisticated version of teleosemantics defined a concept of information that defeated the parity thesis, there is an obvious reason why teleosemantic information cannot play a role in a mechanistic explanation of how development unfolds. The fact that a developmental cause, genetic or otherwise, has teleosemantic content is a fact about its evolutionary history. For example, an allele that has been subject to selection carries teleosemantic information. A physically identical allele that arises by mutation in a new individual does not carry that teleosemantic information, because it does not have that history. But in developmental science it is a given that two organisms with the same physical developmental inputs will develop in the same way (or at least have the same chances of developing in any given way). It follows that the teleosemantic content of a developmental cause makes no difference to what happens as a result of that cause. This point was made by Shea in an earlier publication:

> It has often been argued that any information about phenotypes carried by genes cannot form part of an explanation of the course of individual development . . . The reason is that the semantic properties of genes are a species of selectional property . . . So if we seek to explain the course of individual development—the chain of processes by which an embryo becomes an adult—we

should not advert to the semantic information in the genome (Shea, 2007b, pp. 318–319).

The fact that the phylogenetic origins of a trait cannot directly affect ontogeny is part of the original inspiration for Lehrman's dictum. It also underlies the idea of a "phylogeny fallacy" (Lickliter & Berry, 1990). The phylogeny fallacy occurs when a step in a mechanistic explanation of development is filled by a reference to the evolutionary advantages of developing in that way. This is to answer a "proximal biology" question with an "ultimate biology" answer (Mayr, 1961).

TELEOSEMANTIC TRANSMISSION INFORMATION

In the past decade a new and powerful defense of the scientific value of treating genetic and other causes in development as signals carrying teleosemantic information has emerged. In an early presentation of the idea Eva Jablonka argued that a developmental factor carries information if there is a "receiver" for that factor: a developmental mechanism whose activity depends on which of a range of inputs it receives, and whose differential activity is "functional" (Jablonka, 2002). If "functional" is taken to imply that the receiver has been designed by natural selection to respond to the input in this way, then this is a form of teleosemantics. The developmental factors Jablonka had in mind were all those via which the state of a parent affects the development of its offspring. She argued that the value of treating heredity as a 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 emphasize that the signal itself, as well as the receiver, has been designed by natural selection (Bergstrom & Rosvall, 2011). But they do not develop an account of the teleosemantic content of these signals. In their work they emphasize Shannon's quantitative notion of information. The task of applying teleosemantics to transmission information has been taken on by Nicholas Shea with his "infotel" theory of developmental information (Shea, 2007a, b, in press). The infotel theory combines teleosemantics with a requirement that developmental causes carry correlational information about the developmental environment. It is very much in the spirit of Lorenz's (1965) concept of "phylogenetic information," as Shea acknowledges.

Shea defines the teleosemantic content of signals in the context of a "representing system" like that depicted in Figure 1. It consists of a "producer" which can produce a range of "intermediates" R_i and a "consumer" which can produce a range of behaviors. The behavior of the consumer depends on which intermediate state is produced. According to Shea, if there is a system with this structure, then the conditions for an intermediate state *R* to have semantic content are as follows:

R has the indicative content C if and only if:

- 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.
- 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 that output 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, some water fleas of

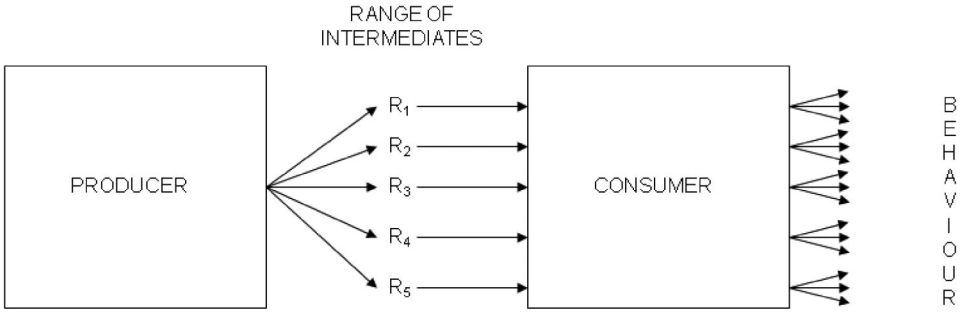

FIGURE 1 The infotel theory of biological information. See text for explanation (from Shea, in press).

the genus *Daphnia* can develop down two alternative developmental pathways. One of these involves the production of a defensive morphology that makes the flea more resistant to predation. This pathway is triggered by the presence of chemical traces of predators (kairomones) during development (Gilbert & Epel, 2009). This mechanism of developmental plasticity can be described as one of Shea's representing systems. The mechanisms that detect kairomones are the producer. The mechanisms that allow the embryo to develop down either developmental pathway are the consumer. The molecular signal that passes between them meets Shea's three conditions:

- It carries the *correlational information* that predators are present.
- The representing system evolved because kairomones are correlated with predation.
- The presence of predators is the relevant *evolutionary success condition*. The consumer system is designed to put the embryo down this developmental pathway only when predators are present.

So according to the infotel theory, that molecular signal carries the indicative semantic information "predators present" and the imperative semantic information "grow defenses." The defensive morphology can also be produced as a parental effect, in which offspring of fleas with the defensive morphology develop that morphology without themselves being exposed to kairomones. In this case, the molecular signal by which the mother induces this developmental response in offspring will carry the semantic information "predators present, grow defenses."

The application of Shea's model to genetic heredity is more complicated (Fig. 2). The first thing to grasp is that the representation system is partly at the population level and partly at the individual level. The producer system is the selective history of a *lineage* of organisms, so it exists at the level of the whole population. But the consumer system is an individual developmental process—there is a separate consumer system for each individual organism. The intermediate states (representations) that the producer sends to the consumers are individual DNA sequences. In Figure 2 a particular genetic variant has gone to fixation, but this does not seem to be an essential feature of the model.

In figure two, natural selection leads to one kind of DNA sequence (G') being eliminated from the population

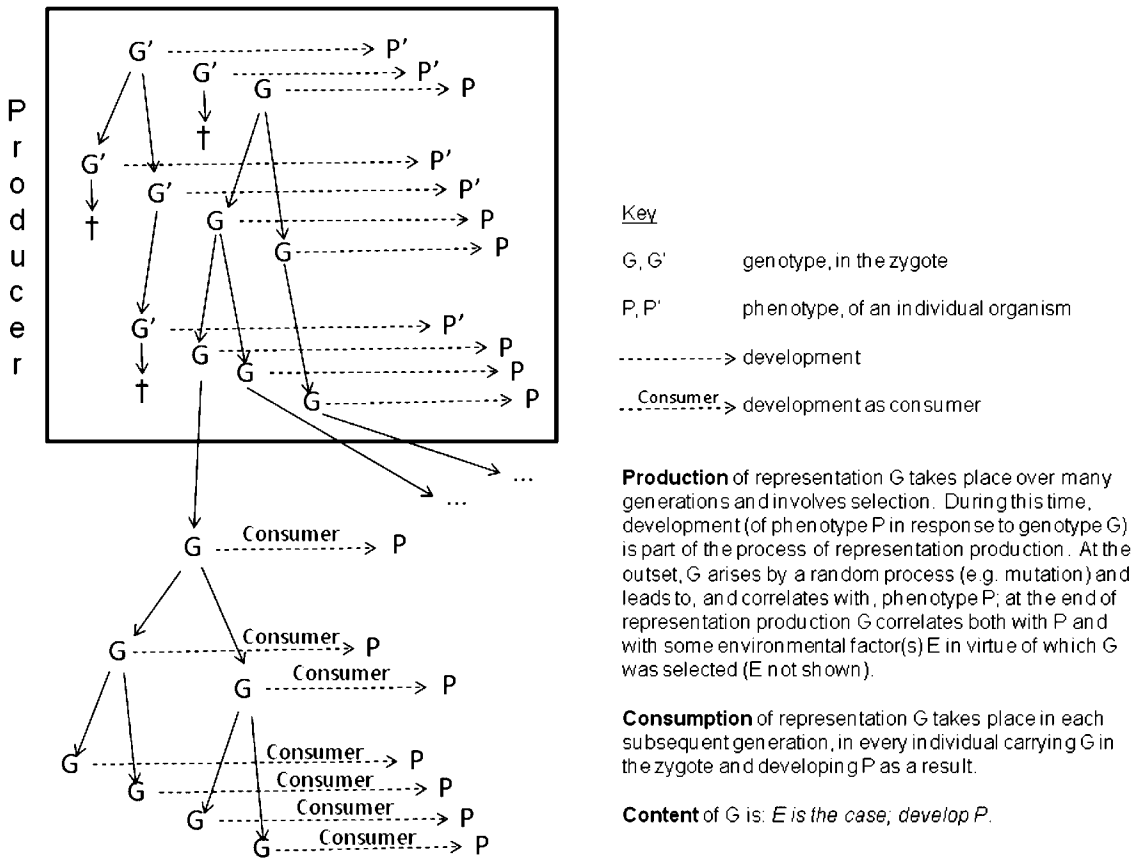

FIGURE 2 The infotel theory applied to genetic inheritance (from Shea, in press).

while another (G) remains. Shea interprets this as the producer system producing one representation rather than another. The development of the individual is affected by receiving G instead of G'. Shea interprets this as the consumer producing a specific behavior in response to a specific representation. According to Shea, G has semantic content because it meets his three conditions. First, it carries correlational information about the selection pressures in past environments: which DNA sequences make it through the process of natural selection depends on what the environment is like. Second, the entire representing system evolved because DNA sequences are correlated with selection pressures in past environments. This is a broad claim about the evolution of nucleic acid-based heredity systems, which Shea defends at length. Third, he claims that the selection pressures in past environments, whatever they were, are the evolutionary success condition for G. He argues that the evolutionary explanation for why developmental systems respond differentially to different DNA sequences is because this allows them to match phenotypes to the likely selection pressures in their environment.

I accept that the infotel theory may be a useful tool in modeling the evolution of heredity systems. Its leading advocates all 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 merely want to argue that TTI cannot be part of a mechanistic explanation of how development unfolds. This is what is claimed by Lehrman's dictum.

INHERITED REPRESENTATIONS ARE *NOT* READ IN DEVELOPMENT

There is something apparently paradoxical about the position I seek to defend—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.

In a thoroughly documented case of a ''maternal effect'' on development, the North American seed beetle *Stator limbatus* follows alternative developmental pathways in response to the challenges posed by the seeds of different species. Eggs laid on seeds of the Catclaw Acacia (*Acacia greggii*) have very high rates of survival. Seeds of the Blue Palo Verde (*Cercidium floridum*) pose more of a challenge. In order to have a reasonable probability of survival when laid on Palo Verde seeds, offspring must grow faster and attain a larger final size than those developing on the Acacia seeds. Mothers bring this about by laying fewer, larger eggs on the Palo Verde seeds than they do on the Acacia seeds (Fox, Thakar, & Mousseau, 1997; Fox, Waddell, & Mousseau, 1995; I owe this example to Tobias Uller. For a review of parental effects and their evolutionary significance, see Uller, 2008).

This case, like the intergenerational variant of the *Daphnia* case described above, can be modeled as a signaling system. Having detected which kind of seed it is depositing eggs upon, the mother signals to the offspring to adopt one growth strategy rather than another. Using the infotel theory, we can assign the larger egg mass the indicative content ''you are on *Cercidium floridum*'' and the imperative content ''grow fast and get large.''

However, this teleosemantic transmission information does not translate into a mechanistic explanation of development. If we ask the developmental question ''how does the egg mass produce faster growth and larger size'' and answer ''by transmitting to the mechanisms of development the instruction to grow fast and get large'' or ''by transmitting to the mechanisms of development the information that the egg has been laid on *Cercidium floridum*'' it is evident how vacuous this is as an explanation.

It is striking that the vacuity of an explanation of development in terms of teleosemantic transmission information is less obvious when the intergenerational signal is a DNA sequence, or even a methylation pattern on a DNA sequence. In such cases it is relatively easy to think of the cause that acts in development as coded information. I suggest that this is a simple confusion deriving from the fact that the molecular signal that carries TTI also carries information in another sense. For example, whatever TTI we may assign to a DNA coding sequence, it will still carry its usual payload of information in Francis Crick's original sense—the specification of sequence in the corresponding protein (Crick, 1958). But this is not teleosemantic information, it is straightforward causal information: a sequence of DNA nucleotides specifies the same sequence of amino acids whether or not it has a particular evolutionary history. Unlike teleosemantic information, information in the sense of the textbook genetic code, or newer ideas like a ''methylation code,'' *can* function as a component of mechanistic explanations of development (Griffiths and Stotz, in press; Stotz, 2006a, 2006b, 2008).

WHAT INHERITED INFORMATION EXPLAINS AND WHAT IT DOES NOT

To see how it is possible for inherited information to explain why offspring resemble their parents but still

not form part of a mechanistic explanation of how development unfolds we can use the ''four questions'' framework (Tinbergen, 1963). A full biological explanation of a trait involves answering these questions:

(1) *Causation*: what is the mechanism by which the trait produces its effects?
(2) *Survival value*: how does the trait contribute to the organism's fitness?—''how survival is promoted and whether it is promoted better by the observed process than by slightly different processes.'' (Tinbergen, 1963, p. 118)
(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 unraveling of its dynamics.'' (Tinbergen, 1963, p. 428)

The questions of causation and ontogeny both call for mechanistic explanations, but one asks how a trait works once it has developed, and the other asks how it develops. The distinction between the two questions depends on the ability to distinguish the development of a trait from its operation, something that is not always clear. In Tinbergen's example of the eye, the distinction is clear enough. How the eye focuses light on the retina and transduces that light into neural firing is one thing. How the eye forms in the embryo is quite another. But other cases are not so clear. Phototropism in plants might be thought of as either the operation of an existing trait (causation) or the development of a new trait (ontogeny).

The question of survival value is quite distinct from the question of how the trait evolved: ''survival value has to be studied in its own right'' (Tinbergen, 1963, p. 423; see also Griffiths, 2009). The idea is to analyze how the trait currently contributes to fitness, not what contributions it made during the evolution of the trait. The distinction is particularly clear in invasion biology, where the selection pressures that explain the success of the invader may be very different from those in the environment in which the invader evolved. The results of analyzing survival value will inform hypotheses about the evolution of the trait, but those results are valid whether or not the same effects on fitness were important in the evolution of the trait. Moreover, the question of evolution is not merely about the selection pressures that operated in the past, but includes the study of other factors that influence population dynamics, and also the basic question of which evolutionary events happened in which order.

The ''questions concerning behavioral development'' that Lehrman asserts cannot be answered by alluding to information coded in the genes correspond to Tinbergen's third question of ontogeny. The ontogeny question asks for a mechanistic explanation of the series of changes that result in the organism having the trait.

The explanations which Jablonka and Bergstrom and Rosvall provide using transmission information primarily address Tinbergen's second question of survival value. Transmission information is used to address the design of heredity mechanisms as an evolutionary optimization problem: what information-theoretic properties should a heredity system have in order to maximize fitness? An answer to this question of survival value is of obvious relevance to Tinbergen's fourth question of evolution. However, the two questions should not be conflated: how a trait currently contributes to fitness and what pressures caused its evolution are distinct questions. The recent work of population geneticist Michael Lynch on the origin of fundamental features of genomic architecture such as introns and *cis*-regulatory elements is a reminder of the importance of this distinction (Lynch, 2007). Lynch argues that, while these features may currently be of adaptive value in facilitating genome regulation, their origin in eukaryotic cells is best explained by drift. Whether Lynch is correct or not, the fact that this can be the topic of an important scientific controversy shows the importance of Tinbergen's distinction.

The examples Shea gives of the value of TTI in ''explaining development'' address both the question of survival value and the question of evolution. These explanations point to the adaptive advantages of certain developmental mechanisms (survival value question) and propose that this was an important factor in the historical evolution of such mechanisms (evolution question). But an evolutionary explanation of a development mechanism is not the same thing as a mechanistic explanation of development (ontogeny question).

Shea has been led astray because he quite correctly wishes to distinguish two different explanations. Early in his article ''Inherited representations are read in development'' he states that,

> We can distinguish two broad questions that can be asked about an individual episode of development: why did it arrive at a particular outcome; and how did the process unfold? This section focuses on the former, arguing that genetic representation explains some of the cases in which the outcome matches a feature of the organism's environment. We return in section 6 to questions about how the developmental process operates (Shea, in press, ms8).

The first of the explanations that Shea promises is an explanation of why development produces a certain, adaptive outcome. It is transparently an evolutionary

explanation. But Shea suggests that in section six of his article we will find explanations which use inherited information to answer Tinbergen's question of ontogeny—to provide mechanistic explanations of how development unfolds. But section six actually argues that

> the informational perspective can help explain why the internal mechanisms of development—developmental programs, somatic cell inheritance, etc.—take the form that they do (Shea, in press, ms24).

Instead of offering mechanistic explanations of how traits develop from the fertilized egg, Shea takes explanations of that kind that already exist, or have already been hypothesized, and uses his informational framework to look at the adaptive advantages of those features. Whereas in the earlier part of the article Shea offered evolutionary explanations of why development produces a particular outcome, in the later part he offers evolutionary explanations of why development uses a particular mechanism to produce that outcome. But these are still evolutionary explanations, not mechanistic ontogenetic explanations. They are adaptive explanations of the mechanisms of development and, in fact, representative of one kind of work found in evolutionary developmental biology.

The distinction between mechanistic explanations of development and evolutionary explanations of development is evident when we apply the four questions framework to a paradigmatically developmental trait. The four questions were originally applied to behavioral phenotypes, although it was always evident that they applied to morphological phenotypes as well. But they also apply to developmental traits, although when dealing with development there may be no clear distinction between questions one and three. Take, for example, the process of gastrulation, in which an undifferentiated ball of cells invaginates and the inside and outside differentiate to form germ layers. The four questions about gastrulation look 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 does gastrulation contribute to fitness: "how survival is promoted and whether it is promoted better by the observed process than by slightly different processes." (Tinbergen, 1963, p. 118)
(3) *Ontogeny*: the development of the blastula, and especially of the specific factors that will cause it to gastrulate. In this case the difference between the causation and ontogeny question is basically one of timescale. Over a longer timescale the cells involved change so as to acquire the competence to perform the operations involved in gastrulation. If we idealize the cells at a late stage in this process, we can describe relatively fixed mechanisms that explain the process of gastrulation.
(4) *Evolution*: the phylogeny of gastrulation and an explanation, perhaps adaptive and perhaps drawing on the answer to 2, of why this stage arose and was preserved.

Although the trait to be explained, the fact that an organism gastrulates, is transparently developmental, only the first and third questions are answered by providing a mechanistic account of how the process unfolds. Question two is answered by looking for adaptive advantages of this process over potential alternative processes. Evo-devo researchers often address this question, asking how a developmental mechanism enhances fitness (Lynch (2007) argues that evo-devo researchers are too quick to infer the adaptive origins of developmental traits from these studies of current utility). Question four asks for the phylogeny of the trait and the dynamics that drove transitions in that phylogeny. Although this question is asked about a developmental trait, it is an evolutionary question.

Shea's two specific examples of TTI "explaining development" are evolutionary explanations of development. He explains why many developmental processes are canalized by arguing that canalization optimizes the transmission of TTI (Shea, in press, ms22–23). He also hypothesizes certain advantages for multicellular development from the existence of a nuclear membrane in eukaryotes (Shea, in press, ms23–24). But he does not use TTI to address either Tinbergen's first or third question for either of these developmental traits. He does not use it as part of a mechanistic account of how a developmental process unfolds.

When Shea says that TTI can answer "questions about how the developmental process operates" (Shea, in press, ms8), he does not mean this in the mechanistic sense that Lehrman had in mind, namely answering Tinbergen's third question. Instead, he applies the whole Tinbergen framework again to a different trait, which is a feature of how the first trait develops, and answers the evolutionary question for that new, developmental trait. Shea has correctly seen that there are two different questions, and that one of them is a question about development. But he has mistaken how these two questions fit into the Tinbergen framework. They are not an evolutionary and a developmental question about the same trait. They are an evolutionary question about the original trait, and a second evolutionary question about a different trait, a feature of how the first trait develops.

CONCLUSION

Lehrman's dictum stands. When integrating evolutionary explanations into developmental psychobiology there is always a risk of committing the ''phylogeny fallacy'' or one of its relatives. The fact that the evolutionary explanation is itself focused on development is no insurance against this. An evolutionary explanation of development is not the same thing as an explanation of development in the sense that Lerhman understood it—a mechanistic account of how the developmental system consisting of the fertilized egg and its developmental niche undergoes a series of transformations that give rise to a trait at some later stage in the life cycle of the organism.

I have argued that Shea makes mistakes because he is not sensitive enough to the risk of conflating developmental and evolutionary explanation, an issue of which developmental psychobiologists have long been acutely aware. Because evolutionary developmental biologists seek connections between development and evolution they are more worried that strictures like Lehrman's dictum, Tinbergen's four questions framework, or Ernst Mayr's distinction between proximate and ultimate biology can blind researchers to the genuine connections between evolution and development (Laland, Sterelny, Odling-Smee, Hoppitt, & Uller, 2011). But the Tinbergian framework here should not raise this concern. Tinbergen stressed that the four questions were mutually illuminating (for an extended discussion of the complementarity of evolutionary and developmental explanations of behavior, see Hochman, in press). Ontogeny is a valuable resource in addressing evolutionary questions. But questions of ontogeny are not themselves evolutionary questions, and for students of behavioral development those ontogenetic questions are at least as interesting as evolutionary ones.

NOTES

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