

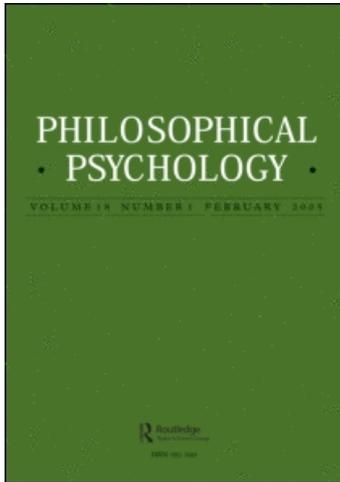
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The Nature of Nurturant Niches in Ontogeny

Jeffrey R. Alberts

The concept of ontogenetic niche is used here to interpret how species-typical behaviors develop through active, context-dependent processes. Ontogenetic niches typically include social stimuli, such as those arising from parents, siblings, and others that provide 'nurturing' in the form of resources, stimulation, and affordances for development. This approach is a useful alternative to wrestling with artificial dichotomies such as nature-nurture.

Keywords: Cowbird; Huddling; Instinct; Ontogenetic Adaptation; Ontogenetic Niche; Rat Pup; Suckling

1. Ontogenetic Niches

The analysis and comprehension of complex problems usually finds only brief and limited value in simple dichotomies. Thus, the nature–nurture dichotomy is remarkably odd in its persistence. Rather than add to numerous expert and incisive critiques, including those in the present collection, I shall discuss the *ontogenetic niche* as an alternative construct in the analysis and comprehension of behavioral development, particularly the ontogeny of species-typical behavior. Ontogenetic niche is an especially satisfying framework in which to view organismal, especially behavioral, development. By necessity, this will be a brief presentation but the interested reader can find broader and more thorough discussions elsewhere (e.g., Alberts, 1987, 1994; Alberts & Cramer, 1988; Alberts & Ronca, 2005; Galef, 1981; West, King & Arberg, 1988; West, King & White, 2003).

Ontogenetic niche is a concept related, in part, to the traditional idea of an ecological niche, a definable locale that affords its inhabitants a peculiar set of

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environmental parameters within which they live. Some would refer to the physical environment itself as a *habitat*. With the organism living and adapting therein, a habitat becomes a *niche* (cf. Alberts & Cramer, 1988). By adding *ontogenetic* to the niche concept, we further specify a dynamic view, one that recognizes that niches change in ways that are sequential and orderly. This basic step is powerful because, for example, it can immediately alert us to the myriad ways in which a developing organism, even an immature and incomplete one, is finely specialized for adaptation to its environment. In addition, this same conceptual step also alerts us to ways in which the niche can change, shift, or be replaced during an organism's development. The view that is created, then, is one that frames development as a sequence of adaptations to a sequence of niches.

We have quickly arrived at a view of individual development as a journey through a sequence of niches. An example, Figure 1 illustrates a sequence of four ontogenetic niches in the life of the Norway rat (*Rattus norvegicus*). The fetal niche (A) is first. Depicted in Figure 1a are a few fetuses, each 'at work' in its amniotic sac within the mother's uterus. To earn a living in this uterine habitat, a fetus must express a slew of adaptive specializations. For instance, the working fetus obtains oxygen and discards CO₂ via the umbilical cord, the same conduit used for nutritive uptake. Indeed, on the levels of its cells, systems, and its overall behavior, fetal life is a story of adaptation to the uterine niche.

The next niche, located just on other side of the birth canal, is an entirely different world. Whereas the uterine habitat is aqueous, the postnatal environment is gaseous. It is also generally colder, replete with stronger light, a spectrum of acoustic vibrations, and more extreme gravity-related forces. In the postnatal niche(s), oxygen must be absorbed from the atmosphere. The newborn rat, like all vertebrates, accomplishes this via the surfaces of the lungs. It interacts with its new, gaseous environment with an uninterrupted rhythm of breathing cycles. Nutrition continues to come from the mother but the newborn must obtain it orally and digest and absorb it in new ways. Where the fetus' habitat was a chamber inside the mother, its

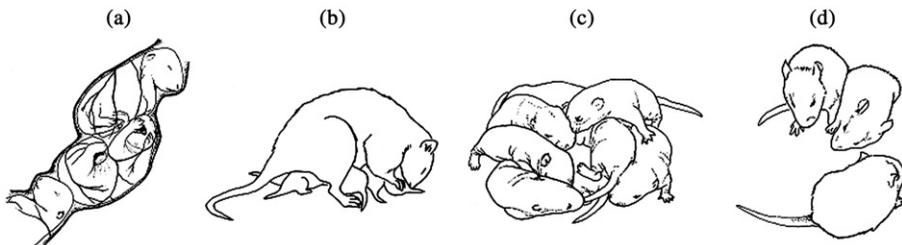


Figure 1. Four ontogenetic niches of rat ontogenesis. (a) The uterine niche, showing two adjacent fetuses and the anterior portion of a third fetus late in gestation; (b) The mother's body as niche, with a dam in nursing repose above some pups; (c) The huddle niche, as illustrated by some week-old pups; (d) The coterie as niche, depicted by pups converging on a piece of food.

early postnatal habitat is largely the exterior of the mother's body (Figure 1b). To survive and thrive in this niche, the newborn must behave in dramatic new ways, especially in relation to the mother and the littermates. The mother's body behaves, and each pup's behavior must adjust adaptively to her presence, her postures, her physiology and that of the littermate siblings (Alberts, 2007; Alberts & Cramer, 1988).

Continuing with this sequential view of development, the next niche in the pup's life is the pile of littermates in the natal nest (Figure 1c). After the first postnatal week, the rat dam's behavior includes longer and more frequent departures from the nest (Grotta & Ader, 1969; Leon, Croskerry, & Smith, 1978; Thiels, Alberts, & Cramer, 1990). With her increasing excursions, the mother's body diminishes as the offspring's main habitat. In her absence, the remaining clump of pups, called the huddle, becomes a niche in which the pups function both individually, in coordination with others, and as a group (Alberts, 2007).

Figure 1(d) represents the social cohort as the next niche. The drawing depicts weanling pups interacting over a piece of solid food. In this developmental niche, the pups are exquisitely sensitive to cues of other rats, both age mates and adults. Together, they form habits of feeding, exploring, playing, and living a social life.

The niche concept embodies the purely physical habitat. The niche requires that individuals be seen in context, thus bringing into focus those aspects of the organism that are linked (fit) to the proximate environmental features—and there are always many such aspects. In this way, the niche concept also directs us to embrace the biological idea of *adaptation*, summoning its immediate, proximate meanings as well as the historical processes of natural selection (cf. Pittendrigh, 1958). Thus, ontogenetic niche and adaptation are closely-related concepts, for they *require* us to view the organism in relation to its environment and then to understand how the relations may (or may not) serve functional roles and contribute to biological fitness.

Ontogenetic adaptation, as a paradigm for analyzing and comprehending development, extends the themes of the niche concept, calling for recognition that selection operates *at each point* in development (Oppenheim, 1981), not simply on some idealized adult form or through the funnel of reproduction. Moore (2001) refers to the stream of 'event-moments' that constitute a life cycle, with each moment subject to testing by natural selection.

So, how does the framework of ontogenetic niche contribute to understanding the development of species-typical behavior? And, more specifically, how do we use the same concept to reframe the kinds of issues that have sustained the nature–nurture dichotomy?

The answers lie, I think, in 'the nature of niche.' Recall that we applied the term 'habitat' to refer to the physical parameters of the niche environment (the organism's 'address'). Each habitat in an ontogenetic sequence is a distinct context in which the developing organism must 'fit' or adapt (*in sensu*, adaptation = *ad* [toward] + *aptus* [fit]).

If we return to ontogenetic niches as illustrated in Figure 1 and consider the life of the developing rat in each one, we can recognize some important commonalities,

despite the dramatic contrasts across them. Common to each niche are channels of sustenance for the developing organism. For instance, in the uterine niche, the mother's body pumps nutrients into each fetus. The natal niche (b in Figure 1) provides mother's milk, some of her body heat, and various forms of behavioral stimuli such as licking and transport. The huddle, depicted in Figure 1c, provides insulation and conductive heat exchange at ages when body temperature and energy conservation are physiological priorities. Social stimulation and augmentation of feeding are among the numerous forms of sustenance present in the niche of the coterie (Albers, 2007; Albers & Cramer, 1988; Albers & Ronca, 2005; Galef, 1981).

Research in the field of developmental psychobiology offers insight into the ontogeny of species-typical behavior of the Norway rat, particularly as displayed by a common domesticated strain (Sprague Dawley) in a laboratory setting. The developing rat provides a host of invariant, stereotyped, and adaptive species-typical behaviors. Many of these behaviors are controlled or directed by olfactory cues, and I will focus on a couple of these to pursue the theme of the present essay.

1.1. Suckling

Consider the suckling behavior of the newborn rat, keeping in mind that this is a behavior both universal among and unique to mammalian infants. Within minutes of abandoning the uterine niche in the mother's body and taking up residence in the natal niche under the mother's body, the newborn must locate a nipple and begin oral ingestion. At this early point in development, the rat pup is blind, deaf, spatially naïve, and has limited strength and motoric competence. Nasal chemoreception in air is also completely new. Nevertheless, the newborn actively locates one of the 12 nipples on the dam's ventrum, attaches to the teat and expresses milk from it. How does the newborn accomplish this remarkable and vital behavior in a new and alien world?

Olfactory stimuli are the key to the pup's suckling behavior. A pup's suckling capabilities can be seen by placing it against the ventrum of a lactating dam rendered motionless by anesthesia. The infant rat will autonomously orient to the dam's body, actively scan and probe along the surface, orally grasp a nipple and suck vigorously on it. Anosmic pups do not attach to nipples or suckle; they lie passively next to an anesthetized dam.

Olfactory cues on the dam's body stimulate the activities that culminate in the pup; findings and attaching to a nipple. These cues can be washed off and then replaced. The pup's suckling behavior vanishes when the cues are removed from the dam's body and is reinstated when a distillate of the wash is painted on the dam's body (Teicher & Blass, 1976, 1977).

Some would assert with confidence that the rat pups' suckling sequence is 'innate'. After all, suckling behavior is expressed stereotypically by naïve newborns and is controlled by a seemingly simple stimulus.

But, as we discover the developmental antecedents of suckling, it is no longer tenable to think of the newborn innately endowed with odor recognition for the

nipple or with a behavioral module for suckling. Instead, the following picture has emerged: While in the uterine niche, fetal respiratory and swallowing movements bring amniotic fluid in contact with developing olfactory sensoria. The fetus can detect chemical cues in the amniotic fluid and the odors are learned. During the birth process, amniotic fluid is left on the dam's body; she grooms herself frequently during and after parturition and further spreads some of these fluids on her fur. The amniotic odors are a bridge of familiarity spanning the pre- and postnatal worlds. Newborns respond to the odors by becoming active and the configuration and texture of the mother's body shape their behavior into an organized form of scanning and probing.

That the amniotic odor is learned and used for suckling activation was shown by Pedersen and Blass (1982) when they added a nonbiological odor (citral) to the amniotic fluid of rat fetuses on gestational Day 17. About 5 days later, newly-delivered pups respond to citral odors on a washed dam and attach to her nipples. Saline-treated (control) fetuses do not respond to citral, and remarkably, the original citral-exposed pups do not respond to normal amniotic odors.

Mechanistically, there is reason to expect that large surges of the catecholamines norepinephrine and dopamine in the fetal brain during parturition may mediate the fetus' odor-learning. Compressions such as those experienced by the fetus during maternal labor and parturition induce these catecholamine surges (Ronca, Abel, Ronan, Renner & Alberts, 2006). There are indications that catecholamines mediate neural and behavioral activation, and contribute to learned associations (e.g., Rangel & Leon, 1995; Wise, 2004).

1.2. Social Huddling

Contact behavior, or huddling, is a species-typical behavior of *R. norvegicus*. Onset of the rat's social, or filial huddling, is developmentally 'fixed', with an onset on Postnatal Day (PD) 15. Prior to about PD 15, rat pups show no species preference: they huddle equally with a conspecific (member of the same species) *versus* a gerbil. Filial preference is demonstrated when a rat prefers to huddle with another rat, rather than a gerbil or a warm tube (Alberts & Brunjes, 1978). The basis of the filial huddling preference is the olfactory cues of rats. Deprived of the sense of smell, rats continue to huddle indiscriminantly with members of other species or with inanimate, warm objects.

The olfactory-guided species preference of rats is also learned. Anointing a mother rat with a novel odor induces a filial preference for that odor in her offspring (Brunjes & Alberts, 1979). Onset of the altered filial preference was on PD 15, the same as the 'normal' preference in rats. Alberts and May (1984) 'labeled' different experiences with different odors and then measured the preferences for the odors. Experiences labeled with odors that became preferred in tests of filial huddling were seen as 'inductors' of filial huddling. Surprisingly, suckling and receipt of milk proved uninvolved in the induction of filial preference, but thermotactile stimulation (conductive heat transfer from a warm body) was a potent inductor. Thus, scented

nonlactating dams were as potent as scented dams that nursed and gave milk. Even more dramatically, a warm scented tube induced a strong filial preference, again implicating conductive heat transfer from a contact surface.

Again we find a stereotyped, species-typical, developmentally-fixed behavior is learned, with all of the key components—odors and thermotactile ‘reinforcement’ in the present case—existing as natural features of the ontogenetic niche. While in contact with mother (Figure 1b) and with littermates (Figure 1c), pups experience an inherent association of species-typical odors, tactile stimulation, and conductive heat transfer. These associations induce a filial preference.

Mechanistically, we now hypothesize that the neuropeptide oxytocin is released during contact behavior in the nest. Oxytocin is now recognized as a transmitter located in neural regions mediating a variety of social behaviors (Ferguson, Aldag, Insel & Young, 2001; Insel, 1992) as well as primary processes such as social memory (Ferguson et al., 2000; van Wimersma & Wintzen, 1980). And there is evidence that oxytocin plays a role in some forms of learning (Keverne & Curley, 2004; Lim & Young, 2006; Nelson & Panksepp, 1996). In support of the general hypothesis of oxytocin mediation of filial odor learning are the recent data from my lab that blockade of oxytocin binding in the brain interferes with the formation of filial preference during a controlled exposure to a scented foster dam (Kojima & Alberts, 2007).

1.3. Nurturant Niches

The association of mother and offspring plays a central role in both of our empirically-based examples of how species-typical behaviors develop. At different points in development, the mother-offspring association takes different forms (e.g., Figure 1a–c). Indeed, each form *is* an ontogenetic niche. Specific features of these niches elicit specific reactions and responses in the developing offspring. These reactions and responses constitute conditions sufficient for the formation of a learned association and, as a result, the differentiation of behavior.

The uterine niche nurtures with total physical support. During the transition from uterine niche to niche in the natal nest, the fetus is necessarily exposed to a mélange of stimuli. Many of these stimuli are effects of other primary processes. For instance, the primary function of parturition is to move the fetus from inside the mother to the outside. During the transition catecholamine levels soar. This is understood to be a primary adaptation serving the transition to pulmonary respiration. The catecholamine surge augments removal of liquid from the newborn lungs and protects the fetus from neural damage during possible episodes of hypoxia, as pulmonary respiration is established (Lagercrantz & Slotkin, 1986).

We think the catecholamine surge may be uniquely capable of creating a learned association between activated behavior and an odor cue. Set in the context of the natal niche, the consequence is a chain of behavioral reactions we call ‘nipple search’ and suckling. Milk transfer as a result of suckling may well reinforce and shape

the behavior. Again, it is the sustaining, guiding, and facultative aspects of the niche that does the job. For this reason, it is appropriate to view niche as ‘nurturant,’ recognizing that the developing offspring may be actively involved in eliciting and extracting the nurturing. The utter reliability of the ontogenetic niches and the affordances that exist in each are inherited as surely as are genes. An offspring’s behavioral interactions with the dam or with its siblings in the nest can be framed as active ‘niche construction,’ adding another dimension to ontogenetic adaptation (e.g., Alberts & Schank, 2006).

The process of inducing a huddling preference similarly involves an association co-opted from other, primary processes. Contact behavior results from the mother’s presence in the nest, her nursing behavior, and the pups huddling for energetic benefits (Alberts, 1978; Alberts & Gubernick, 1983). These are primary adaptations to the natal niche and huddling niche. Within the mélange of stimuli involved in these nurturing activities, there exist natural associations and the developing pups’ behavior is shaped by these. In both instances, we see that species-typical behaviors arise from development occurring in species-typical niches.

2. A Local Synthesis

The story of vocal learning in brown-headed cowbirds (*Molothrus ater*) as related by West and King (this volume) provides a view concordant with that offered herein. Historically, the cowbird’s nest parasitism set the stage for generations of observers to conclude that *M. ater* was ‘hard-wired’ for its species-typical song. After all, the young are reared entirely by members of other species so that the entirety of the cowbird’s early development is in the presence of other species.

The beauty and power of West and King’s investigations, derives from their continuous focus on the ontogenetic niches, rather than on the temporal dimensions of development (or genetic dimensions, for that matter). Their adherence to the process of niche inheritance and especially the breadth of their perspective on the contents of the cowbirds’ ontogenetic niches was a key to their marvelous discoveries.

Whereas the presentation of ontogenetic niches that I offered emphasized physical and chemical components, West and King emphasized *social* components in their analysis. In addition, West, King and their students exploited comparative dimensions and geographic variation in their analyses.

The similarities between West and King’s (this volume) presentation and the present one include manipulations of experience during development. They suggest the term “experiential knock-out” (EKO) to refer to manipulations of flock composition so that the social structure lacked the presence of adult males. In contrast, most of the manipulations described in the studies of rodents, left intact the social and physical structures of the environment, but inserted novel olfactory stimuli as markers or ‘labels.’

West and King’s invocation of the term EKO is purposively ironic, with its clear reference to the ‘knock-out’ technologies of genomic studies, which are so often used

with cavalier flair to assert the 'genetic control' of features with complex developmental origins. West and King's insightful 'deconstruction' of presumed innateness, coupled with a comprehensive view and treatment of ontogenetic niche, give support and hope to a future in which observers and analysts conceptualize the richness of development in ways that recognize the experience of context and environment at each point in the life cycle.

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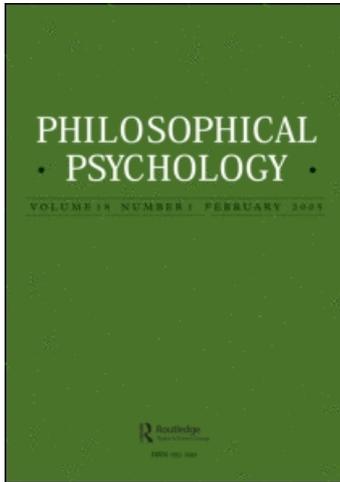
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The Nurture of Nature: Hereditary Plasticity in Evolution

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The Nurture of Nature: Hereditary Plasticity in Evolution

Ehud Lamm and Eva Jablonka

The dichotomy between Nature and Nurture, which has been dismantled within the framework of development, remains embodied in the notions of plasticity and evolvability. We argue that plasticity and evolvability, like development and heredity, are neither dichotomous nor distinct: the very same mechanisms may be involved in both, and the research perspective chosen depends to a large extent on the type of problem being explored and the kinds of questions being asked. Epigenetic inheritance leads to transgenerationally extended plasticity, and developmentally-induced heritable epigenetic variations provide additional foci for selection that can lead to evolutionary change. Moreover, hereditary innovations may result from developmentally induced large-scale genomic repatterning events, which are akin to Goldschmidtian “systemic mutations”. The epigenetic mechanisms involved in repatterning can be activated by both environmental and genomic stress, and lead to phylogenetic as well as ontogenetic changes. Hence, the effects and the mechanisms of plasticity directly contribute to evolvability.

Keywords: Epigenetic Inheritance; Genome Organisation; Macroevolution; Natural Genetic Engineering; Richard Goldschmidt

‘Hereditary plasticity’ sounds like a contradiction in terms, a category mistake. Taking the view of heredity that dominated the last century, what is plastic, is, by definition, not hereditary, because plasticity refers to individual ontogeny, to the ability of a single genotype to develop several different phenotypes depending on environmental/developmental conditions. During the long and stormy history of the nature/nurture debate, the relationship between hereditary/genetic inputs and

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environmental/developmental inputs has been under scrutiny, and the supposed dichotomy between them, as well as their distinctness, was questioned. Many biologists, psychologists and sociologists (e.g., Gottlieb, 1976, 1992; Oyama, 1985), argued convincingly that within an individual's ontogeny, genetic inputs and environmental inputs interact, and it is this developmental interaction that constructs the phenotype. The critics used the interactive developmental perspective to show the problematic nature of terms such as 'instinct' (Gottlieb, 1992; Lehrman, 1953), 'innateness' (Griffiths, 2002; Mamei & Bateson, 2006), and 'genetic program' (Fox-Keller, 2000; Lewontin, 2000). They showed that individual development provides a unifying framework, in which a separation between nature and nurture makes no sense.

We want to approach this issue from the complementary perspective—that of heredity and evolution. We argue that the traditional distinctions between evolution–heredity on the one hand and development–plasticity on the other hand, are breaking down, at least for some sets of processes and problems. Heredity may be developmentally constructed, and this has far reaching effects on our concepts of heredity and evolution (Jablonka, 2007; Jablonka & Lamb, 2007a, b). In this article, we shall focus (i) on the developmental origins of heritable epigenetic variations, which introduce a temporal, trans-generational dimension to developmental plasticity; (ii) on the role of epigenetic control mechanisms in the generation of macroevolutionary innovations. Not only heredity, but evolvability as well, may have epigenetic, developmental aspects.

Before we develop our arguments, we would like to position our approach within the modern framework of developmental evolutionary biology.

1. The Evo-Devo synthesis

The relationship between development and evolution has recently become the focus of evolutionary studies. The emerging synthesis between development and evolution, known as 'Evolutionary Developmental Biology' or Evo-Devo, focuses on the processes of evolutionary innovation (Müller & Newman, 2005), on the constraints and generic properties of developmental systems (Hermisson & Wagner, 2004), on comparative studies of developmental genes with major effects (Carroll, 2005), on the architecture of genetic-developmental networks (Davidson, 2006), and on the evolution of the ability to develop and learn (Gilbert, 2003). From the Evo-Devo perspective, in order to explain the evolution of a morphological, physiological or behavioral trait, it is necessary to explain the developmental processes that contribute to its construction during ontogeny. Whatever the origin of a new variation, it must become integrated into a network of developmental interactions: its expression, its modifiability, and the scope of its effects depend not only on its own intrinsic nature but also—and often much more—on the regulatory structure of the network in which it is integrated. Processes leading to developmental flexibility and sensitivity to environmental variations on the one hand (plasticity), and to the buffering of

environmental and genetic ‘noise’ on the other hand (canalization), are therefore important subjects of research in Evo-Devo.

The focus on plasticity and canalization and the processes underlying them was central to the views of Waddington (e.g., Waddington, 1957) and Schmalhausen (1949), but was peripheral to the interests of most other evolutionary biologists until recently. This developmental perspective on evolution has now been developed and extended by West-Eberhard (2003), who suggested it as an integrated framework for evolutionary theory. The focus of enquiry and the starting point of this approach are the integrated phenotype, its development, and its responsiveness to the environment. An adaptive novelty begins as a new developmental response to a new input—a new mutation, or, more commonly, a novel, recurrent environmental-inductive change. The phenotypic response to the novel input leads to the restructuring of developmental units: the deletion, amplification, temporal, and spatial re-organization of body parts, stages in the life-cycle, etc. Following this phenotypic adjustment (or *phenotypic accommodation*) to the new input, genetic changes that simulate, facilitate, complement, or ameliorate the effects of the phenotypic accommodation may be selected, a process that West-Eberhard calls *genetic accommodation*. The genetic variation selected for (or against) can affect the focal trait itself or its side-effects.

The processes that underlie plasticity and evolvability seem to lead to conflicting consequences, and involve selection at different levels. Plasticity is defined in terms of the potential of a single genotype to generate several *different* phenotypes. According to West-Eberhard (2003), plasticity is:

The ability of an organism to react to internal or external input with a change in form, state, movement, or rate of activity. It may or may not be adaptive (a consequence of previous selection). Plasticity is sometimes defined as the ability of a phenotype associated with a single genotype to produce more than one continuously variable alternative form of morphology, physiology and/or behaviour in different environmental circumstances (Stearns 1989). It refers to all sorts of environmentally induced phenotypic variation (Stearns 1989). Plasticity includes responses that are reversible and irreversible, adaptive and nonadaptive, active and passive, and continuously and discontinuously variable. (p. 33)

Plasticity is therefore, defined within the framework of the life cycle of a single individual organism. It can be said to be a property exhibited by an individual’s development.

Evolvability, in contrast, is a property of lineages or clades, not of individuals, because it is defined in terms of the potential for evolutionary change. Evolvability was defined by Kirschner and Gerhart (1998) as “an organism’s capacity to generate heritable phenotypic variation” (p. 8420). Wagner distinguishes between two related but nonidentical meanings of evolvability: (i) a system is evolvable if its properties show heritable genetic variation and if natural selection can thus change these properties; (ii) a system is evolvable if it can acquire novel and adaptive functions through genetic change (Wagner, 2005). The difference between the two notions of evolvability is that the second focuses not just on any heritable and

selectable variation, but on heritable and selectable variations that also affect the developmental system in a way that leads to real innovation.

The structure of the developmental system as well as that of the hereditary system is of central importance within this framework. The basic organization of biological systems is hierarchical and modular, that is, they are made up of more or less autonomous developmental units, which are delimited by developmental switches. There are degrees of modularity and of integration between modules, but in general, it seems that a high degree of developmental modularity is associated with increased evolvability, since the combinatorial possibilities are greater than those of a system with limited modularity (Wagner, 2005; West-Eberhard, 2003). Statistical analysis of the modular architecture of genetic networks suggests that modules are mostly conserved during evolution, with intermodule connections serving as a source of evolutionary innovation (reviewed in Koonin & Wolf, 2006). It should be noted, however, that the evolutionary origin of modularity remains an open question and does not seem to be the result of direct selection for evolvability (Wagner, Mezey, & Calabretta, 2005).

Plasticity and evolvability clearly differ with respect to the level at which natural selection operates. Moreover, it may be argued that plasticity reduces evolvability: by allowing an organism to survive in an environment for which it is not adapted plasticity may reduce the selection pressure leading to evolutionary change. Furthermore, plasticity seems to conceal the differences between genotypes from the direct scrutiny of selection, thus preventing the selection of better-adapted genotypes. However, as we show below, the same molecular mechanisms may underlie both plasticity and evolvability; the two processes are, we argue, complementary and continuous.

The role of epigenetic inheritance and epigenetic control mechanisms in evolution is rarely discussed within the framework of Evo-Devo. However, epigenetic developmental control mechanisms are directly related to the generation of heritable variations in two important ways: (i) epigenetic variations generate some of the variations that are the raw material for natural selection and introduce new foci of selection, and (ii) systemic epigenomic (genetic and epigenetic) variations lead to some types of genome repatterning and macroevolutionary changes. We argue that the incorporation of these mechanisms within an evolutionary framework extends the notions of plasticity and evolvability, and affords new interpretation of evolutionary phenomena.

2. Epigenetic Inheritance: Transgenerationally Extended Plasticity

The types of plastic responses on which we focus in this article are persistent phenotypic responses—responses, which once induced, can last for a long time: for a significant portion of the life-time of an individual or throughout life, or for generations. Examples of persistent ontogenetic variations are the kind of plastic responses associated with the processes of cellular differentiation in multicellular

organisms, where different cell types are generated (e.g., skin cells, kidney cell, liver cells) from the same genotype, and once generated, are very stable and unlikely to change. Another well-known example is that of the inactivation of the X chromosome in female mammals: in different somatic cells of (eutherian) mammalian females, one of the X chromosomes, either the paternally derived or the maternally derived, becomes stably inactivated (Lyon, 1961). There are many other types of stable, life-long changes in the attributes of the phenotype, including stable changes in behavior that arise as a result of various inputs into development. For example, susceptibility to diseases and obesity is affected by early maternal inputs to the embryo's development and by postnatal inputs, in both humans and rats (Gluckman & Hanson, 2005; Gluckman, Hanson, & Beedle, 2007). In plants, vernalization is a good example for a long-term change that is induced by a developmental input: exposure to the prolonged cold of winter results in the acquisition of the competence to flower in the spring (Henderson & Dean, 2004). In all cases of plasticity, an organism with the same genotype can develop along different trajectories, depending on the inputs it receives.

Environmentally-induced maternal effects are examples of plasticity, which is extended beyond one generation. The term refers to environmentally-induced changes in the mother that influence gene expression in the offspring. For example, in many insects, the photoperiod, temperature, or host availability experienced by an ovipositing female will determine the probability of diapause in her offspring (Mousseau & Fox, 1998). Environmentally-induced maternal effects are also known in plants, where they can affect seed, seedling, and adult characteristics of offspring (Roach & Wulff, 1987). Maternal behavior has obvious effects on progeny characteristics, for example, the level of resistance to stress (Avital & Jablonka, 2000). There are also paternal effects, both physiological and behavioral, for example, when the father contributes to the offspring postnatal development as in song birds. Parental effects—both maternal and paternal—can be seen as responses to inputs into both parent and offspring development, with the effect on offspring mediated by the effect on the parent. Parental effects are manifestation of developmental plasticity, and are often, though not invariably, adaptive (Mousseau & Fox, 1998).

What are the developmental mechanisms that can underlie such persistence of plastic alternative responses? Can plasticity be extended beyond the offspring generation, for two, three, or even more generations? The concept of *epigenetic inheritance* refers to plastic responses that persist over several, possibly many generations. Environmentally-induced parental effects are therefore a special and limited case of epigenetic inheritance. Epigenetic inheritance occurs when environmentally-induced and developmentally-regulated variations, or variations that are the result of developmental noise, are transmitted to subsequent generations of cells or organisms (Jablonka & Lamb, 2005, 2007a). The term epigenetic inheritance is used in a broad sense and in a narrow sense. The broad usage includes cellular epigenetic inheritance through mitotic or meiotic cells, as well as information-transfer that by-passes the germline, for example, through early developmental (prenatal and early postnatal) inputs that depend on physiological feedback loops that regenerate previous

developmental conditions (e.g., hormonal and neural conditions). An example is the persistence of a male-biased sex-ratio in some Mongolian gerbil lineages, which is the result of interactions between testosterone concentrations in utero, maternal behavior, and offspring development, which re-creates in pregnant female offspring the hormonal conditions that they themselves experienced in the uterus (Clark & Galef, 1995). Another example is induction of a stress-sensitive phenotype in rats by altered maternal care (reduced licking and grooming) during a sensitive period. This phenotype is perpetuated in the lineage because when the daughters become mothers, they reproduce the early maternal care style of their mothers (Meaney, 2001; Weaver et al., 2004, 2005). Gluckman and his colleagues (Gluckman et al., 2007) have argued that the new obesity epidemic in human populations, which is driven by a change in diet, is affected by events that happened in the parental generation (and possibly in earlier generations as well). In all these cases, alternative phenotypes can persist for several, possibly many, generations. Inputs from the external environment that depend on the activities of the organism and are to a large extent the result of such activities (transgenerational ecological niche-construction, see Odling-Smee, Laland, & Feldman, 2003) often contribute to such trans-generational transmission of alternative phenotypes. Socially learnt activities are important inputs for the reconstruction of niches in social animals; socially learnt behaviors can persist in a group of animals for many generations and form traditions (Avital & Jablonka, 2000), and in our species, symbolically encoded communication and learning construct the persistent aspects of our society and culture (Boyd & Richerson, 2005; Jablonka & Lamb, 2005). However, as the earlier examples illustrate, recreating the developmental inputs causing the plastic change, or other plasticity-inducing developmental inputs, is not the only mechanism leading to persistent plastic responses.

A more restricted usage for epigenetic inheritance refers to the *cell-to-cell* transmission of variations that are not the result of differences in DNA sequence. This occurs during cell division in prokaryotes and mitotic cell division in eukaryotes, and during the meiotic divisions in the germline that give rise to sperm or eggs. Somatic cell heredity is manifest in the stability of different phenotypes of determined and differentiated cells within the same multicellular body. However, cellular inheritance also includes transmission through the germline, following meiotic divisions.

Jablonka and Lamb (2005) characterized several types of cellular mechanisms that underlie cellular epigenetic inheritance, mechanisms referred to as *epigenetic control mechanisms*. They include: (i) *Self-sustaining metabolic loops*, which are dynamic regulatory circuits that maintain cellular patterns of activity of genes and their products. The transmission of the components of the circuit (proteins, RNAs and metabolites) to daughter cells leads to the same patterns of gene activity being reconstructed in them (Ferrell, 2002; Malagnac & Silar, 2003). (ii) *Structural inheritance*, in which pre-existing cellular structures act as templates for the production of similar structures, which become components of daughter cells, for example, prion-based inheritance in fungi (Shorter & Lindquist, 2005), the inheritance of cortical structures in ciliates (Grimes and Aufderheide, 1991), and

the reconstruction of what Cavalier-Smith (2004) calls ‘genetic membranes’.

(iii) *Chromatin marking*, in which chromatin configurations (that consist of histone and nonhistone proteins that are noncovalently bound to DNA, and small chemical groups, such as methyls, that are covalently bound directly to DNA) are reconstructed in daughter cells (Henikoff, Furuyama, & Ahmad, 2004).

(iv) *Heritable RNA-mediated variation in gene expression*, in which transcriptionally silent states are maintained through repressive interactions between small RNA molecules and the mRNAs or DNA to which they are partially complementary (Meister & Tuschl, 2004). The state of gene expression is maintained in daughter cells that inherit the small RNA molecules. Current studies suggest that the chromatin marking and the RNA-mediated epigenetic inheritance systems play a particularly large role in intergenerational inheritance in sexual multicellular organisms. As we argue in the next section, the epigenetic control mechanisms underlying chromatin and RNA-mediated epigenetic cellular inheritance also play a central role in the generation of systemic mutations that may lead to macroevolution.

Is epigenetic inheritance an aspect of developmental plasticity or an aspect of heredity? It is clear that one needs to treat it as both, and the perspective one chooses is dictated by the biological question one asks. Epigenetic inheritance can be a direct agent of evolutionary change, for the frequency of heritable epigenetic variation can change in a population and such variations may accumulate. Moreover, epigenetic inheritance may accelerate the process of genetic accommodation, and often an adaptation or an accidental divergence process may start with an epigenetic heritable variation, which later becomes accommodated genetically. When the direct effects of epigenetic inheritance on evolutionary change are the focus of attention, the heredity aspect of the phenomena is most important. On the other hand, if we are interested in the evolution of epigenetic inheritance as an adaptive strategy, the preferred focus is on how it leads to extended plasticity (Jablonka & Lamb, 2005). A single perspective is both too limiting and potentially misleading.

The hereditary aspect of plasticity means that when heredity is developmentally constructed, as it is in the case of epigenetic inheritance, ‘nurture’ (inputs from the environment that affect ontogeny) participates in the construction of ‘nature’ (heredity). This is a Lamarckian mode of inheritance, which for a long time was considered a biological impossibility, and was explicitly excluded from the modern synthesis. We suspect that the historical stigma associated with it is the main reason for its reluctant incorporation within the developing Evo-Devo Synthesis.

3. Epigenetic Control Mechanisms in Evolution: A Source of Systemic Mutations

The relationship between plasticity and evolvability is illuminated from an additional angle if we consider developmentally-controlled DNA rearrangements. The differentiation of some cell-types, for example immune system cells that undergo targeted rearrangements during development, the polytenization

(repeated replication of chromosomes without separation of daughter chromatids resulting in multi-stranded giant chromosomes) of the trophectoderm in female mammals, the many cases of polyploidization (replication of chromosomes not followed by cell division resulting in multiple number of chromosomes in a cell) of somatic tissues in plants and insects, and developmentally-regulated gene amplification and deletions, are all well-known examples in which the developmental processes of determination and differentiation occur via genomic rearrangements (Jablonka & Lamb, 1995). A survey of different taxa suggests that developmentally-regulated genome rearrangements are an ancient feature of eukaryotes, and are brought about by epigenetic control mechanisms (Zufall, Robinson, & Katz, 2005). For example, chromatin marking systems, involving histone modifications and RNA silencing, guide the DNA rearrangements in the immune system of mammals (Busslinger & Tarakhovsky, 2007). In the normal life cycle of ciliates, the (somatic) macronucleus is extensively rearranged (noncoding segments are deleted, coding segments are amplified, etc.), a process directed by RNA-mediated chromatin marking mechanisms (Mochizuki & Gorovsky, 2004). It is interesting to note that sequence-specific information is transferred to the new zygotic macronucleus from the parent macronucleus via RNA-mediated epigenetic processes, an adaptive process which is part of the normal life cycle of ciliates, and which provides an extreme form of transgenerational adaptive plasticity (Mochizuki & Gorovsky, 2004).

As Shapiro (1999) has noted, the existence of a cellular genetic engineering kit in all existing prokaryotic and eukaryotic cells that enables cutting, sewing, and reorganizing the genome during development, suggests that these same developmental mechanisms may also lead to extensive and rapid evolutionary alterations under some conditions. The plasticity mechanisms of ontogeny may therefore operate in phylogeny, and can be considered as evolvability mechanisms. We suggest that these repatterning mechanisms, that were discovered in the context of development and are mechanisms of epigenetic control, underlie the systemic mutations suggested by Richard Goldschmidt, and that they lead to these systemic epigenomic (epigenetic and genetic) alterations in conditions of stress.

Goldschmidt (1940) applied the Evo-Devo-like notion of *norm of reactivity*, according to which “the genotype is... the inherited norm of reactivity to the ensemble of conditions which may influence phenotypic expressions” (p. 250), to the discussion of macroevolutionary change. Goldschmidt observed that the range of modifiability of one species under conditions of developmental stress is on a similar scale as the range of phenotypic differences between related species under natural conditions (p. 253); differences which he argued are the result of *systemic mutations*—a term by which he meant chromosomal repatterning. He observed that new species are usually chromosomally different from their parental species, and suggested that evolution above the species level usually involves such repatterning. McClintock suggested that genomic repatterning occurs under conditions of stress and that transposable elements play a major role in this process (McClintock, 1984).

In order to re-evaluate Goldschmidt's and McClintock's ideas about systemic mutations, we need to establish that processes of chromosomal and genomic repatterning are indeed common during evolution, uncover the conditions under which such repatterning takes place, and identify the cellular mechanisms that bring it about. Contemporary experimental results show that stress can lead to large-scale genomic restructuring events (Jablonka & Lamb, in press). For example, nutritional stress causes epigenetic and genetic changes in r-RNA genes and repetitive sequences in flax (Cullis, 2005), heat shock causes similar epigenomic stress in *Brassica* (Waters & Schaal, 1996), and hydrostatic pressure causes genome wide changes in methylation patterns in rice (Long et al., 2006). Radiation seems to induce both genetic and epigenetic genome-wide instabilities that last for several generations in both animals and plants (Dubrova, 2003; Moliner, Ries, Zipfel, & Hohn, 2006). Transposable elements—usually relatively silent—are activated as a result of various stresses, such as wounds and pathogen attacks, just as McClintock suggested, and the activity is in many cases restricted to germ cells, and hence transgenerational (reviewed in Kidwell & Lisch, 2001). Following McClintock, Kidwell and Lisch argue that while the evolution of this behavior of transposable elements may be attributed to the benefits it provides to the transposable elements themselves (which increase their chance of survival into the next generation of offspring of the stressed individual), the increased genomic variation may be evolutionarily advantageous to the stressed host plant. In microorganisms, stress leads to specific responses, such as the SOS response (a postreplication DNA repair system that allows bypassing of lesions or errors in the DNA) that increases variations, and Radman (1999) argued that this system evolved not only to patch up a damaged genome but also because it increases evolvability in an adaptive manner—mutations happen just when an evolutionary response to catastrophe is required (Caporale, 2003).

Genomic stresses due to hybridization and polyploidization are frequent in plant evolution. In most flowering plants (70–90%) speciation through hybridization has occurred, and in some clades this is a recurrent process. Studies of the effects of genomic stress caused by auto- and allo-polyploidization indicate that the genomic reorganizations exhibit repeatable, wide-ranging, yet specific, genomic, and chromosomal changes that involve massive epigenetic changes involving DNA methylation and histones modifications, transcriptional and posttranscriptional gene silencing through the RNAi system, as well as targeted genetic changes (see, e.g., Comai et al., 2000; Levy & Feldman, 2004; Mittelsten Scheid, Afsar, & Paszkowski, 2003; Pikaard, 2000, 2001; Rapp & Wendel, 2005). Polyploidy and hybridization are not restricted to flowering plants and were also important in the phylogeny of bryophytes (Natcheva & Cronberg, 2004), parthenogenetic fish, and some groups of rodents and frogs (Arnold, 2006). These events and the wide-ranging epigenomic changes that accompany them, therefore underlie speciation in many taxa. They cannot be considered as having a minor importance in evolutionary history, and are relevant to the recent discussions about the universal applicability of the Tree of Life metaphor for describing the relationship between species and the significance of

hybridization and horizontal gene transfer in speciation events (Doolittle & Bapteste, 2007; Goldenfeld & Woese, 2007).

In animals, behavioral stress can lead to rapid heritable morphological changes. This was observed by Belyaev and co-workers in their work on the domestication of silver foxes, and later the domestication of other mammalian species (Belyaev & Borodin, 1982; Belyaev, Ruvinsky, & Trut, 1981a; Belyaev, Ruvinsky, & Borodin, 1981b; Jablonka & Lamb, in press; Popova, 2006; Ruvinsky, Lobkov, & Belyaev, 1983a, 1983b, 1986; Trut, Plyusnina, & Oskina, 2004). The neuro-endocrine system is destabilized under domestication, and this seems to lead to heritable changes in gene expression, as well as an increase in the frequency of microchromosomes (Belyaev, Volobuev, Radjabli, & Tryt, 1974). A heritable effect of hormonal stress on extensive epigenetic heritable variations was also observed in rats. Anyway, Cupp, Uzumcu, and Skinner (2005), Anyway, Memon, Uzumcu, and Skinner (2006a), Anyway, Leathers, and Skinner (2006b), and Crews et al. (2007) injected pregnant females with vinclozolin, and showed that the abnormalities induced in male offspring were inherited through the male line for at least four generations. They found several different DNA sequences that had altered methylation patterns in the F1 males, and these were transmitted from the F1 to the F3 generation.

Our understanding of the mechanisms involved in hereditary changes, especially under stress, is challenging the accepted dichotomy between plasticity and evolvability and many of the related terms and assumptions that this dichotomy suggested. For example, the assumption that evolution is driven solely or mainly by 'random' mutations is misleading, and the term itself is used in different ways that should be distinguished and questioned (Jablonka & Lamb, 2007a). The assumptions about random mutations are that: (i) '*Random mutations are not highly targeted*', i.e., identical (or very similar) changes in DNA do not occur in many different individuals within a population. However, as we have seen, programmed response to genomic stress, as seen in some cases of polyploidization (Levy & Feldman, 2004), are recurrent and directed to the same genomic and chromosomal regions. The recurrence in this case seems dictated by the nature of these sequences (e.g., their repetitive sequences and transposable elements), which are targets of the epigenetic control mechanisms, and targeting is therefore dependent on the genetic architecture of the parent species, the level of divergence between them, and the direction of the cross. (ii) '*Random mutations are not developmentally or environmentally induced*', i.e., identical changes in conditions do not result in identical mutations. However, as the cases we discussed suggest (e.g., the induced mutational changes in flax) this is often not the case under stress. (iii) '*Random mutations are not adaptive*', i.e., they do not increase the chances that the individuals carrying them will survive and reproduce. However, there is evidence that the mutational mechanisms that generate certain classes of mutations may be adaptive (Caporale, 2003). Of course, the nature of the mechanisms involved, and the unpredictability of the environmental triggers, cannot guarantee that the variation produced is adaptive, but the processes that give rise to these mutations increase the probability of survival and reproduction as compared to chance mutations alone.

It is possible to see these genomic reorganization processes as part of the processes of phenotypic accommodation, which is in this case, also a process of genetic accommodation. (iv) *'Random' mutations are local: they do not involve coordinated changes to the genotype.* However, as we saw, large scale genome reorganization, of the type emphasized by Goldschmidt and seen in hybridization, seems to be triggered in stressful conditions. Thus, all the four notions associated with the assumption that all evolutionarily important mutations are 'random' are challenged by recent molecular studies. There is a developmental aspect to evolutionary change, which the assumption about the 'randomness' of mutations fails to acknowledge.

'Randomness' is just one concept that needs to be reconsidered and qualified in the light of an extended perspective on heredity. The ubiquity of epigenetic inheritance and the new information about the origin and nature of genetic variations, problematize all the traditional concepts of evolutionary biology.

4. Conclusions

The nature/nurture dichotomy, which has been shown to be problematic and incoherent in the context of an individual's ontogeny, remains embodied in the notions of plasticity and evolvability, as commonly understood. We argued that plasticity and evolvability, like development and heredity, are mechanistically and conceptually continuous. The very same mechanisms may be involved in both, and the choice of perspective depends to a large extent on the type of problem being explored, and the kinds of questions being asked.

Attention to the mechanisms that underlie both epigenetic changes and genome repatterning highlights the complex relationships between plasticity and evolvability: a continuum from counter-acting processes to complementary ones, based on the very same molecular phenomena. Instead of an unchanging relationship, the epigenetic mechanisms may serve both ontogenetic and evolutionary purposes to varying degrees, and the relationship between the two aspects may change over time. It is possible that the molecular responses that lead to epigenomic repatterning—either to changes in DNA methylation and histone modifications or to DNA rearrangements during stress—were selected to deal with various hazards, including DNA damage, genomic parasites, infections, and physiological (nutritional, chemical, climatic) extremes. However, as in the example of the transposable elements and bacterial SOS systems, the mechanisms may have persisted also because of their variational, evolutionary effect, because of their contribution to evolvability. Hence not only their hereditary and evolutionary effects, but also the evolution of the mechanisms that lead to plasticity-based evolvability may have been intertwined.

The epigenetic mechanisms we discussed lead to evolvability in the two senses discussed by Wagner (2005). The first, simple, meaning—that of adding additional foci for selection is clearly exhibited by epigenetic heritable variation that can be developmentally induced. The second, more radical notion

of evolvability—the ability to produce hereditary *innovations*—was discussed here in the context of developmentally-induced large-scale genomic repatterning, or systemic mutations, which lead to macroevolutionary changes, including speciation. The epigenetic mechanisms involved in repatterning can be activated by both environmental and genomic stress, and lead to hereditary as well as ontogenetic changes.

The focus in this article on the similarity (indeed, the identity) of the molecular mechanisms underlying plasticity and evolvability is compatible with the developmental perspective that highlights the importance of the interactions between different types of inputs to development. The developmental perspective, however, runs deeper, as the examples discussed above show. The inescapable conclusion is that even heredity itself can be, and often is, developmentally constructed.

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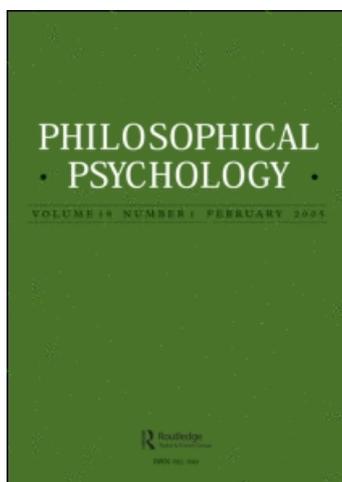
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A Plea for Human Nature

Edouard Machery

Philosophers of biology, such as David Hull and Michael Ghiselin, have argued that the notion of human nature is incompatible with modern evolutionary biology and they have recommended rejecting this notion. In this article, I rebut this argument: I show that an important notion of human nature is compatible with modern evolutionary biology.

Keywords: Evolutionary Psychology; Human Evolution; Human Nature; Species

The notion of human nature has fallen into disrepute in various quarters and a remarkable number of charges have been filed against it. Some social critics have alleged that it contributes to the justification of oppressive social norms (e.g., Haraway, 1989; Lewontin, Rose, & Kamin, 1984; for discussion, see Pinker, 2002), while some philosophers, such as Sartre, have challenged it on metaphysical grounds.¹ More seriously, prominent philosophers of biology have argued that the notion of human nature is incompatible with modern evolutionary biology. In a well-known article, David Hull writes (1986; see also Sober, 1980):

All the ingenuity which has been exercised trying to show that all human beings are essentially the same might be better used trying to explain why we must all be essentially the same in order to have such things as human rights. (...) Until this question is answered, I remain suspicious of continued claims about the existence and importance of human nature. (pp. 11–12)

Evolutionary biologist Michael Ghiselin concurs (1997):

What does evolution teach us about human nature? It tells us that human nature is a superstition. (p. 1)

And, more recently, David Buller has attacked evolutionary psychologists for endorsing the notion of human nature (2005):

The idea of a universal human nature is deeply antithetical to a truly evolutionary view of our species... A truly *evolutionary* psychology should abandon the quest

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for human nature and with it any attempt to discover universal laws of human psychology. (p. 419)

While some criticisms of the notion of human nature might well be justified, I argue in this article that many charges filed by philosophers of biology are unwarranted. Because Hull's (1986) article is the source of many attacks against the notion of human nature in the philosophy of biology, I focus exclusively on it here.² For the sake of space, I do not elaborate on the positive arguments for the notion of human nature.

Here is how I will proceed. In Section 1, I characterize in some detail two distinct notions of human nature—an essentialist notion and a nomological notion. In Section 2, I show that Hull's objections invalidate the essentialist notion of human nature, but not the nomological notion. In Section 3, I reply to two objections.

1. Two Notions of Human Nature

It is important to realize that there are many ways to construe human nature and that the arguments for and against human nature often bear on specific construals rather than on human nature in general. In this section, I describe two distinct construals.

According to the essentialist notion of human nature, human nature is the set of properties that are separately necessary and jointly sufficient for being a human. Furthermore, the properties that are part of human nature are typically thought to be distinctive of humans.

Philosophers and scientists have proposed numerous candidate properties for inclusion in human nature, so construed. In the *Discourse on Method*, Descartes argued that language, understood as the capacity to express and understand an infinite number of sentences, was distinctive of humans, in contrast to animals and machines (1637/1987):

We can certainly conceive of a machine so constructed that it utters words and even utter words which correspond to bodily actions causing a change in its organs (e.g., if you touch it in one spot it asks what you want of it, if you touch it in another, it cries out that you are hurting it, and so on). But, it is not conceivable that such a machine should produce different arrangements of words so as to give an appropriately meaningful answer to whatever is said in its presence, as the dullest of men can do. (p. 187)

Reason, morality, humor, and knowledge of death have also been proposed by philosophers and scientists as good candidates for belonging to human nature.

The essentialist notion of human nature is plausibly rooted in folk biology. Folk biology is the intuitive body of knowledge about animals, plants, biological properties, and biological events (death, disease, etc.) that people spontaneously rely on when they reason about biological matters. An important component of folk biology is the belief that each species is characterized by a distinctive set of properties, which develop endogenously and are transmitted across generations (Atran, 1990; Gelman & Wellman, 1991; Griffiths, Machery, & Linquist, submitted). Because humans are just one species, believing that humans share a set of necessary and

jointly sufficient properties, which set them apart from other species, seems to be a natural extension of this component.

Noteworthy, even if the essentialist notion of human nature is truly rooted in folk biology, it does not seem to be widespread across cultures and times, in contrast to other components of folk biology. When one looks at how people have thought about humans across cultures and times, one typically finds an emphasis on what distinguishes humans into distinct groups, rather than an emphasis on what is common to and distinctive of humans. To give a single example, during the last decades of the nineteenth century and the first decade of the twentieth century, European and American psychologists, sociologists, anthropologists, and biologists developed various pseudo-scientific racial classifications of humans that were allegedly grounded in what was then understood of human evolution (Degler, 1991).

The nomological notion of human nature stands in sharp contrast to the essentialist notion. According to this second notion, human nature is the set of properties that humans tend to possess as a result of the evolution of their species. According to this notion, being bipedal is part of human nature, because most humans are bipedal animals and because bipedalism is an outcome of the evolution of humans. The same is true of biparental investment in children, fear reactions to unexpected noise, or the capacity to speak. According to this construal, describing human nature is thus equivalent to what ornithologists do when they characterize the typical properties of birds in bird fieldguides.³

Although I do not have the space to elaborate on this point here, it is important to see that the nomological notion of human nature inverts the Aristotelian relation between nature and generalization. For Aristotle, the fact that humans have the same nature explains why many generalizations can be made about them (for a recent development of this idea, see Walsh, 2006). For me, on the contrary, the fact that many generalizations can be made about humans explain in which sense there is a human nature.

It is worth highlighting the contrast between the nomological notion and the essentialist notion of human nature. Most important, according to the former notion and in contrast to the latter notion, human nature does not define membership in the human species: the properties that are part of human nature are neither necessary nor jointly sufficient for being a human. Although biparental investment in children might be part of human nature (according to the nomological notion of human nature), membership in the human species has nothing to do with biparental investment in children.

Because the properties that belong to human nature are not definitional, they are not necessarily possessed by *all* humans. Although the capacity to speak is part of human nature, not all humans are able to speak, because the development of this capacity requires exposure to language. Not all humans have fear reactions, because the amygdala of some humans is impaired, following brain traumas or particular developmental trajectories. What is required of the properties that are part of human nature is that they be shared by *most* humans, as a result of a specific causal process—the evolution of humans.⁴ Relatedly, the properties that are part of human nature do

not have to be possessed *only* by humans. For instance, like humans, many animals react fearfully to unexpected noises. Finally, the properties that are part of human nature are not permanent; human nature might change.

Two additional points should be noted. First, nothing is said about the nature of the evolutionary processes in the proposed characterization of human nature. The traits that are part of human nature can be adaptations, by-products of adaptations, outcomes of developmental constraints, or neutral traits that have come to fixation by drift. In addition, human nature is not normative; there is nothing wrong in not having the properties that are part of human nature.

According to the nomological characterization of human nature, bimodal traits are not part of human nature (because they are not widely shared among humans in general). Thus, if males and females have different evolved mating psychologies, the properties of male and of female mating psychologies are not part of human nature. One could alternatively propose to include in human nature all the properties that humans have because of the evolution of their species, *whether these properties are shared by most humans or only by some subset of humans*. Then, males' and females' evolved mating psychologies would be part of human nature (supposing that they really differ). To support this alternative proposal, one could push the analogy between human nature and the description of birds in fieldguides, noting that fieldguides usually characterize a species with pictures of males, females, and members of geographic subspecies.⁵

Although this alternative proposal is perfectly coherent, I would like to resist it, because it is useful to have a notion that picks out the similarities between humans, particularly their psychological and behavioral similarities. Much of the social sciences attempt to characterize and explain differences between humans. Differential psychology (e.g., personality psychology) focuses on individual differences, while much of anthropology focuses on cultural differences. In addition, cultural and individual differences are more salient to common sense than similarities. The notion of human nature is thus a useful counterpoint to the widespread neglect of the similarities between humans.⁶

2. Hull's Arguments

Hull's (1986) main argument against the notion of human nature is straightforward:

Generations of philosophers have argued that all human beings are essentially the same, that is, *they share the same nature* . . . Periodically a biological species might be characterized by one or more characters which are both universally distributed among and limited to the organisms belonging to that species, but *such states of affairs are temporary, contingent and relatively rare*. (p. 3, my italics)

This argument is rooted in the understanding of the nature of species that emerges from Darwinian population thinking (Mayr, 1976). According to evolutionary biologists (Hull, 1986; Sober, 1980; Sterelny & Griffiths, 1999, chap. 9), membership in a given species is not defined by the possession of specific (intrinsic) properties. Thus, an animal is not a dog in virtue of having a given set of properties. As a result,

the biological notion of species does not entail that conspecifics share a set of (intrinsic) properties.

Furthermore, as Hull rightly emphasizes, evolutionary biologists insist on the variability of conspecifics. Because developmental conditions vary, it is biologically unlikely that all conspecifics share a given property, when in addition to being universal, this property is supposed to distinguish conspecifics from the members of other species. To focus on humans, it is hard to find any property that is both distinctive of humans and common to all humans. Furthermore, even if a property were both distinctive and universal, this state of affairs would be contingent. It would not be a necessary property for being a human.

The way biologists conceive of species clearly invalidates the essentialist notion of human nature, but certainly not the nomological notion of human nature described above, for according to the latter notion, the properties that constitute human nature are not definitional and they need not be universal among humans or distinctive of humans. Thus, Hull's argument leaves the nomological notion of human nature unscathed.

In addition to the argument summarized above, Hull also argues against a revision of the essentialist notion of human nature (1986):

Nor does it help to switch from traditional essences to *statistically characterized essences*. If the history of phenetic taxonomy has shown anything, it is that *organisms can be subdivided into species as Operational Taxonomic Units in indefinitely any ways* if all one looks at is covariation. (p. 11; my italics)

The reply Hull is considering here goes as follows. Rather than characterizing human nature as a set of properties possessed by all humans and only by humans, one might propose that human nature is the set of properties such that an animal is a human if and only if it possesses a sufficient number of these properties. According to this reply, the properties that constitute human nature need not be universal. Hence, this notion would be consistent with Hull's emphasis that it is biologically unlikely that all and only humans share a given trait. To counter this reply, Hull notes that it has turned out to be impossible to define the membership in species by means of clusters of properties. He concludes that appealing to a cluster of properties in order to flesh out the notion of human nature is of no help to defend this notion.

Like Hull's first argument, this second argument fails to invalidate the nomological notion of human nature. According to this notion, the properties that constitute human nature are not conditions of membership in the human species. Hence, whether or not one can define membership in the human species by means of a cluster of properties has no bearing whatsoever on the value of the nomological notion of human nature.

To summarize, Hull's arguments invalidate the essentialist notion of human nature and variants of this notion. Because species are historical entities and because conspecifics vary, one cannot characterize membership in the human species by means of a definition or by means of a cluster of properties. But, this conclusion does not mean that humans have no nature, if one construes human nature as those

properties that humans tend to possess as a result of the evolution of their species. This notion of human nature is consistent with the historical nature of species and with the variability of the traits possessed by conspecifics.

3. Two Objections

In the last section of this article, I consider two objections against the nomological notion of human nature. Both objections grant that some properties are common among humans as a result of the evolution of their species, but question the identification of human nature with this set of property.

3.1. A Canada-dry Notion?

One might first argue that the nomological notion of human nature is a “Canada-dry” notion: it looks like human nature, but it isn’t.⁷ For, the critic might go on, the nomological notion of human nature is unable to fulfill any of the roles that the traditional notion of human nature—the very notion attacked by Hull, Ghiselin, or Buller—was supposed to fulfill. According to this notion of human nature and in contrast to the essentialist notion, the properties that are part of human nature neither distinguish humans from other animals, nor define humans, nor determine what a normal human looks like. If this objection were correct, my criticism of Hull’s arguments would be a pyrrhic victory: I would have shown that there is a notion of human nature that is not invalidated by these arguments—just one that is not worth fighting for.

This objection ought to be resisted. The notion of human nature has played many roles in the history of philosophy and in the history of science. The nomological notion of human nature certainly fails to fulfill some of these roles, as noted by the present objection. But it does fulfill other roles, which have traditionally motivated the notion of human nature. Particularly, saying that humans have a nature entails that humans form a class that is of importance for biology. The members of this class tend to have some properties in common in virtue of evolutionary processes. Furthermore, saying that a given property, say a behavior, such as biparental investment, or a psychological trait, such as outgroup bias, belongs to human nature is to say that this trait is common among humans and that its occurrence among humans can be explained in evolutionary terms. This is also to say that some kinds of explanation for the occurrence of this trait among humans are inappropriate. Particularly, this is to reject any explanation to the effect that its occurrence is exclusively due to enculturation or to social learning.⁸

3.2. Do All Traits Belong to Human Nature?

The nomological notion of human nature makes sense only if not all properties of humans are part of human nature. This necessary condition is easily fulfilled because

many properties are not widespread among humans and, as a result, are not good candidates for being part of human nature. For instance, the belief that the son of a god died on a cross in Jerusalem is shared by (only) one human out of six and is thus not a good candidate for being part of human nature.

In addition to this easily fulfilled necessary condition, it should also be the case that not all properties that are common among humans are part of human nature. It might seem natural to argue that this condition is also easily fulfilled because not all properties that are common among humans are common because of some evolutionary processes. Among the common properties of humans, those that are not common because of some evolutionary processes are not part of human nature, according to the nomological notion of human nature. For example, the belief that water is wet is not part of human nature, in spite of being common, because this belief is not the result of some evolutionary processes. Rather, people learn that water is wet.

The second objection considered in Section 3 denies that one can tease apart in this manner those traits that are common among humans and that are part of human nature and those traits that are common among humans without being part of human nature. The reason is that evolutionary processes causally contribute to any property that is common to humans.⁹ To see this, consider again the belief that water is wet. People acquire this belief by experiencing the wetness of water or, to put it differently, they acquire this belief by individual learning. Now, people would not be able to form this belief if humans had not evolved the sense of touch and the capacity to form beliefs about the qualitative properties of substances. Because of the truth of this counterfactual, evolutionary processes seem to be among the causes of the belief that water is wet. Since this argument can be generalized to every property common among humans, it seems that human nature includes all these properties—suggesting that the nomological notion of human nature is too inclusive.

It is probably correct that evolutionary processes causally contribute to the existence of any trait that is common among humans. But only some of these traits can be explained by reference to evolutionary processes. That is, only some of them are the object of ultimate explanations. What distinguishes these traits from the traits that are not the object of ultimate explanations is that they have an evolutionary history. Saying that a trait has an evolutionary history is to say something stronger than the fact that it has perdured across generations. Humans have probably believed that water is wet for a very long time, although this belief has no evolutionary history. For this trait is not a modification of a distinct, more ancient trait. By contrast, human shame is probably a modification of an emotion that existed among the last common ancestors of humans and of the great apes (Fessler, 1999).

Thus, the second objection considered in Section 3 ought also to be resisted. By appealing to the notion of ultimate explanation, one can tease apart the traits that are part of human nature from those traits that are merely common among humans, in spite of evolutionary processes causally contributing to all human traits.

4. Conclusion

The significance of Hull's influential attack against the notion of human nature is limited. It decisively invalidates the essentialist notion of human nature, a notion that might be rooted in folk biology. However, it leaves the nomological notion of human nature entirely unscathed: humans have many properties in common as a result of the evolution of their species. Importantly, because this notion of human nature is probably the relevant one for understanding sociobiologists', such as E. O. Wilson, and evolutionary psychologists' interest in human nature, Hull's attack fails to undermine their scientific projects.

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Notes

- [1] Sartre (1958) wrote that “there is no human nature. Man first of all exists . . . and defines himself afterwards” (p. 28).
- [2] For a discussion of Buller's criticisms, see Machery and Barrett (2006).
- [3] I owe this analogy to Paul Griffiths.
- [4] For a related point, see Griffiths (1999). In contrast to Griffiths, however, I do not single out the relation of descent as the main source of generalizations among humans.
- [5] I am grateful to Paul Griffiths and Karola Stotz for pressing me on this point.
- [6] The nomological notion of human nature is thus defended on pragmatic grounds. I should note that I am strongly attracted by the alternative proposal sketched by Griffiths and by Stotz.
- [7] From an old French advertisement for the ginger-ale soda Canada Dry: “It looks like alcohol, it has the taste of alcohol, but isn't”.
- [8] This is of course not to deny that social learning, or indeed any other environmental influence, can be part of the explanation of the development of this trait.
- [9] I do not mean to suggest that evolutionary processes causally contribute only to those traits that are common among humans. I focus on traits that are common among humans, because being common among humans is a necessary condition for being part of human nature, according to the notion developed in this article.

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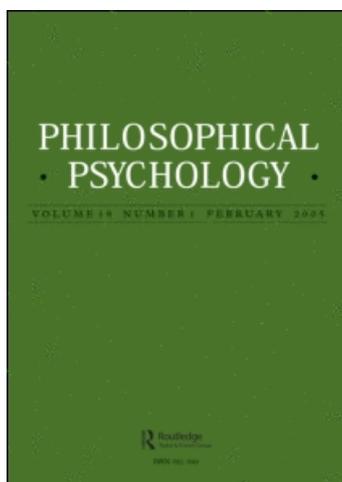
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Espousing Interactions and Fielding Reactions: Addressing Laypeople's Beliefs About Genetic Determinism

David S. Moore

Although biologists and philosophers of science generally agree that genes cannot determine the forms of biological and psychological traits, students, journalists, politicians, and other members of the general public nonetheless continue to embrace genetic determinism. This article identifies some of the concerns typically raised by individuals when they first encounter the systems perspective that biologists and philosophers of science now favor over genetic determinism, and uses arguments informed by that perspective to address those concerns. No definitive statements can yet be made about why genetic determinism has proven so resilient in the face of empirical evidence pointing up its deficiencies, but conveying the essential interdependence of 'nature' and 'nurture' to the general public will likely require deployment of the arguments that systems theorists ordinarily use to reject genetic determinism. In addition, the elaboration of new metaphors that focus attention on the dynamic nature of trait construction will likely prove valuable, because re-conceptualizing notions like 'genes' and 'nature' will probably be one of the most effective ways to help students and the general public abandon the genetic determinism that biologists now recognize as indefensible.

Keywords: Developmental Systems Theory; Gene-Environment Interaction; Genetic Determinism; Interactionism; Nature-Nurture

1. Introduction

Genetic determinism, in all of its various forms, has proven remarkably resilient in the face of both theoretical and empirical work showing it to be a poor way of explaining how biological and psychological characteristics emerge during development.

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It appears that merely comprehending what genes actually do does not necessarily lead to a rejection of genetic determinism, because in spite of evidence to the contrary, even some biologists continue to write as if developmental processes can be genetically determined (e.g., Gehring, 1998). There are likely several factors that contribute to most people's strongly held convictions that genes can deterministically produce at least *some* of our characteristics; this article will offer a noncomprehensive inventory of the concerns often raised by individuals when they first encounter the systems perspective championed by many philosophers of science (Griffiths & Gray, 2001; Moss, 2003; Robert, 2006; Stotz, 2006) and now ascendant in biology (Gilbert, 2001; Jablonka & Lamb, 2005; Lewontin, 2000; Nijhout, 2003).

Systems theorists advocate a probabilistic view of trait development that holds that all of our biological and psychological characteristics are co-constructed during development by genetic *and* environmental factors operating in collaboration with one another; this approach rejects the possibility that either type of factor is more agentive or provides developmental information that is more causally important than the other. In this article, after identifying particular questions often asked by those encountering the systems perspective for the first time, arguments informed by the systems viewpoint will be deployed to address these concerns. Those psychologists, laypeople, and biologists who embrace genetic determinism probably each have different reasons for doing so, but similar sorts of explanations are likely to be required when trying to disabuse these various populations of their deterministic ideas; nonetheless, the discussion below is intended to be particularly useful for psychologists.

The idea that genetic factors might be able to determine the form of biological and psychological traits has been with us since the beginning of modern theorizing about genes. Although Gregor Mendel did not use the word *genes* to name the 'heritable factors' that he inferred must be responsible for observed variations in his experimental pea plants, the notion of a deterministic 'germ plasm' had appeared in several late 19th century writings on biology—most notably in the work of August Weismann—and because of the close conceptual similarity between Mendel's 'heritable factors' and Weismann's deterministic 'germ plasm', it is little wonder that just a few decades later, Mendel's factors came to be thought of as deterministic 'genes'. T. H. Morgan's early 20th century discovery that genes are located on chromosomes eventually led to the development of the modern gene theory, which holds that genes are responsible for the development of inherited traits; this conclusion was based on the finding that the presence of particular genetic factors is highly correlated with the presence of particular traits. But even though such correlations do *not* support the contention that genes operate deterministically, modern gene theory nonetheless retained the genetic determinism that 19th century 'germ plasm' theorists relied on to explain the intergenerational transmission of evolutionarily adaptive characteristics. This sort of conceptualization continued to inform theoretical biology well past the middle of the 20th century, as biologists embraced François Jacob and Jacques Monod's operon model of how genes regulate development (Keller, 2000).

Deterministic theories in psychology, likewise, have a long history. Psychology was informed by theories of biology at its inception (Sulloway, 1992), but the popularity of the belief that evolutionary mechanisms—operating proximally through deterministic genes—can produce behaviors grew significantly with the publication of the works of Konrad Lorenz (e.g., Lorenz, 1965) and E. O. Wilson (e.g., Wilson, 1978) shortly after the middle of the last century. As there were in biology (Driesch, 1908, cited in Gottlieb, 1992; Sturtevant, 1915, cited in Carlson, 1966), there were behavioral scientists who understood from very early on that genetic-deterministic theories would ultimately fail to explain the origins of our psychological traits (Kuo, 1976; Lehrman, 1953; Schneirla, 1966); nonetheless, psychological theories rooted in the deterministic ideas of 19th century biology continue to be fashionable in branches of contemporary psychology such as evolutionary psychology and quantitative behavior genetics. The recent popularity of nativism in theories of language acquisition (Chomsky, 1975; Pinker, 1994, 2002), social cognition (Baron-Cohen, 1995; Cosmides, 1989), cognitive development (Spelke & Kinzler, 2007), and other domains illustrates how widely accepted deterministic accounts of psychological phenomena remain.

In spite of the popularity of these ideas in psychology, recent decades have seen the publication of overwhelming empirical support for the idea that genetic factors cannot deterministically cause either biological or psychological/behavioral characteristics (Stotz, 2006); this view has now become relatively mainstream among philosophers of biology. Contributing to the widespread acceptance of this idea have been data collected in several disparate fields, including developmental psychobiology, the neurosciences, embryology, and molecular biology. Neuroscientists, for example, have discovered that brain structure is more dependent on experience than was previously thought (Cohen et al., 1997; Greenough, Black, & Wallace, 1987; Pantev et al., 1998). Molecular biologists have determined that a gene can be spliced in several alternative ways as a function of the (nongenetic) cellular *contexts* in which the product of that gene will be constructed (Smith, Patton, & Nadal-Ginard, 1989). Embryologists have found that basic information about which pole of a developing organism will develop into the head and which will develop into the tail can be extracted from chemical gradients present in the cytoplasm—not in the genes—of the developing zygote (Gilbert, 2006; Wolpert, 1992). And developmental psychobiologists have learned that experiences early in development can have epigenetic—and inheritable—effects wherein particular genes in a newborn animal can be turned ‘on’ or ‘off’ as a function of the behavior of the animal’s mother (Harper, 2005; Meaney, 2007; Meaney & Szyf, 2005; Weaver et al., 2007). Consequently, it is extremely rare at this point to find a scientist willing to publicly endorse genetic determinism, because it is no longer tenable to argue that some genes can single-handedly cause the development of specific traits independently of the contexts in which development takes place.

Nevertheless, the now-discredited belief that genes can determine the form of some of our characteristics remains widely held by journalists, politicians, students, and the general public—and surprisingly, by many social scientists. This idea probably owes

its tenacity to different things in different people. For some, its persistence probably reflects no more than straightforward ignorance of the significant facts. For others, genetic determinism might retain some currency because of the apparent elegance of its simplicity; the emerging understanding of how genes interact with nongenetic factors during development is extremely complex, and so perhaps less appealing to these individuals. Yet other people might find it difficult to overcome a lesson learned at a young age, particularly when a deep appreciation of how traits really do develop is best obtained with exposure to empirical data from many diverse fields, including embryology, developmental psychobiology, the neurosciences, and developmental psychology (to name just a few). In the absence of relevant data, we cannot know how important each of these reasons is for different people, but anecdotally, it is clear that scientists trying to explain these ideas to their students often find them quite unwilling to believe that our biological and psychological characteristics *always* develop from the interactions of genetic and nongenetic factors.

To understand why genetic determinism is so tenacious, it will be helpful to consider some of the questions that naïve—but nonetheless resistant—audiences typically ask when first exposed to the essential facts of gene-environment interdependence. The following sections will each consider a question that such audiences commonly raise when encountering a systems viewpoint for the first time. While the questions themselves will serve to illuminate some of the major obstacles that hinder understanding of the fundamental interdependence of genetic and nongenetic factors during development, a presentation of the *arguments* that systems theorists ordinarily use to address these questions will aid those attempting to help students relinquish strongly held ideas about genetic determinism.

2. Several Questions Naive Audiences Typically Raise When Confronted With Gene-Environment Interactionism

2.1. *If I Look Like my Parents Regardless of the Environment in Which I Grew up, my Appearance Must Have Been Determined by my Genes, Right?*

One reason often given by those convinced that some traits must be genetically determined is the physical similarity of offspring to their parents. This objection to gene-environment interactionism typically involves references to the fact that children often resemble their biological parents even if they are raised by adoptive parents. As this argument is usually advanced, the physical characteristics of an adopted child who resembles her biological parents must be genetically determined, because the biological parents are thought to have contributed nothing to the child other than her genes. However, biologists now understand that parents provide several types of nongenetic ‘information’ to their offspring prior to birth (and certainly prior to adoption), some of which play important roles in the development of the offspring’s characteristics (Harper, 2005; Jablonka & Lamb, 2005). Examples of these types of ‘information’ include the patterns-of-distribution

of chemical factors in the ovum's cytoplasm, methylation patterns that affect the functioning of the DNA in the ovum and the sperm, and the specific chemical environment of the uterus (in which the characteristics of offspring first begin to develop), among others.

A common objection to this observation is that these types of 'information' do not, in most cases, account for the physical *differences* that give individual offspring their unique appearances; normal development usually entails very little cross-individual variability in either zygotes' internal structures or in the characteristics of their prenatal environments. Some evidence suggests that differences in such factors can account for behavioral differences between individuals (Meaney & Szyf, 2005; Weaver et al., 2007), but these data, while exceedingly important, ought not distract from the following general point: developmental factors that never vary—for example, the presence of oxygen or gravity in human developmental environments—while often ignored when people consider the origins of traits, are ignored in error.

For example, human developmental environments are always characterized by linguistic input that influences normal language development. The fact that such input is invariably present does not reduce the importance of this input in the development of linguistic competence. Of course, it remains the case that developmental factors that affect all individuals cannot account for the *differences* among those individuals; but just because a particular factor might not explain why you and I have different physical appearances does not mean that factor did not contribute importantly to the development of your (or my) physical appearance.

The importance of this point is so often lost on those who have never before considered these issues that some additional comment on it is warranted. At times, the argument is made that if a particular causal factor is always constant in a particular context, it can be safely ignored when assigning causation; although the presence of oxygen in a building is a necessary background condition for the building to burn, it is far more common to assign causal power in such a scenario to the arsonist striking a match than to the presence of the oxygen in the vicinity of the flame. This is because the presence of oxygen is usually *assumed*, because it is so invariable. This line of argument is not unreasonable; perhaps when a researcher refers to "the gene for trait X," she means to say—and is typically understood to be saying—that the gene in question causes trait X *assuming a particular (normal) developmental environment*. But such a position effectively encourages us *not* to study the developmental mechanisms by which genes interact with other factors to produce the trait in question. And in so doing, we are more likely to miss possible routes by which the development of the trait could be influenced, possibly with beneficial consequences.

For example, although most members of the medical community consider phenylketonuria (PKU) to be a genetic disorder (Cole, Cole, & Lightfoot, 2004)—because those with PKU have genetic constitutions that differ slightly from the rest of us—the fact remains that the disorder can be effectively treated with an *environmental* manipulation. If newborns at risk for PKU are fed a diet free of phenylalanine (an amino acid normally present in all human diets) the symptoms of PKU can be virtually eliminated. In this case, an understanding of the

developmental mechanics that produce the symptoms of PKU gave rise to a subsequently confirmed hypothesis about how to effectively treat the disorder by manipulating an environmental factor that is relatively constant in normal human developmental environments. Such are the benefits of remaining aware of the effects of such ‘invariable’ developmental factors on the emergence of our characteristics. If the goals of the behavioral and social sciences are pragmatic to the slightest degree, the causal roles of invariable ‘background’ factors in the development of our traits simply must not be ignored.

Of course, it should go without saying that an awareness of nongenetic contributions to development ought not blind us to the important roles *genetic* factors play in development; however, an explicit statement of this fact is necessary, because those offering explanations of development informed by systems theories are sometimes erroneously seen as denying the importance of genetic factors in development, simply because they insist on the importance of nongenetic factors as *contributors* to this process. If we want to know why two unrelated children raised by the same adoptive parents will typically resemble their own biological parents, we need to merely point out that genetic factors obviously *do* contribute in essential ways to the development of our characteristics. But developmental outcomes are not *pre-determined* by such contributions; nongenetic factors contribute to these outcomes in ways that are just as essential, even if it is difficult for us to see their effects when such factors are unvarying. Although the proverbial fish that has spent her entire life in the sea does not know that she lives in a watery environment (because she has never experienced anything else), it nonetheless remains the case that fish are as they are at least in part because of the peculiar qualities of water.

2.2. *If my Brother is Outgoing And I’m Shy Even Though Our Parents Treated us the Same Way, Our Personalities Must Have Been Determined by Our Genes, Right?*

Genetic-deterministic explanations for our characteristics seem to be readily accepted in some cases because of erroneous folk-beliefs regarding the environmental factors that contribute to those characteristics. If a ‘folk theory’ of personality holds that easily observable parental behaviors are the important nongenetic contributors to personality development, the subsequent discovery that there are no differences in such behaviors across parents might lead one to conclude that the important contributors to personality development are genetic. Of course, this is a fundamental error: in fact, *other* nongenetic contributors to personality development might be the important ones, and we cannot know in advance of developmental analysis if they are or are not. Time and again, developmental analyses have revealed that among the important influences on the development of particular characteristics are some that are, in the nomenclature of Gilbert Gottlieb (1991a), nonobvious; such contributing influences are factors that would not have been thought important prior to developmental analysis. The discovery that some of the nongenetic contributors to development are nonobvious means that until we understand exactly what factors are involved in the development of a particular characteristic—and to date, there are

virtually *no* psychological characteristics that develop in ways that are so well understood—we cannot effectively evaluate the extent to which two developmental environments are similar. Although it might *seem* as if the environment in which I developed is identical to the environment in which my brother developed, we cannot know if our environments were identical *with regards to the factors that really matter*, if we do not know in advance which factors actually are the ones that matter. The bottom line is that no two bodies can be raised in identical environments—even monozygotic twin fetuses in a single uterus are in slightly different positions relative to one another, and one of them will be born prior to the other—so the intuitions resulting from a casual observation that two individuals were reared in similar environments always ought to be considered suspect.¹

2.3. *Haven't Controlled Studies of Twins-Reared-Apart Proven That Genes Can Cause Traits?*

In the process of simplifying scientific reports so that they will be comprehensible to the general public, the popular press has contributed in significant ways to the widespread notion that genes can deterministically cause some of our traits. But the primary source of data that has contributed to this discredited notion has been the field of quantitative behavior genetics, which has drawn its most compelling conclusions from studies of fraternal and so-called 'identical' twins who have been adopted into 'different' environments. These sorts of studies have been the subject of several extensive critiques (Joseph, 2001, 2006; Lewontin, Rose, & Kamin, 1984), many of which have been decidedly negative. These cogent critiques need not be recited here, given their accessibility elsewhere. Rather, for the purposes of the present article, it is enough to note that the design of quantitative behavior genetics studies, by its very nature, is unable to demonstrate genetic determinism, even in those cases in which the general public is most convinced of its existence (e.g., in the case of eye color). This inability reflects the fact that such studies are always correlational, so the data they generate can at best reveal that variation in a particular factor (say, the presence or absence of a particular gene) is associated with variation in another factor (say, the presence or absence of a particular phenotypic characteristic). Of course, as all first year graduate students in the social sciences are aware, co-variation need never indicate that two factors are causally related; consequently, it is, frankly, far-fetched to think that these studies could even *lend support* to the hypothesis that particular genes determine the development of particular traits. In fact, quantitative behavior geneticists have conceded that their studies cannot be used to support claims of genetic determinism; as one leading evolutionary psychologist put it, no "sane biologist would ever dream of proposing" that human behavior is genetically determined (Pinker, 2002, p. 112). Nonetheless, when the results of studies of quantitative behavior genetics are passed on to journalists, textbook writers, students, and the general public, they are often misinterpreted in ways that perpetuate the idea that some of our characteristics can be genetically determined.²

Part of the trouble with the data collected by quantitative behavioral geneticists is that they are typically used to compute heritability statistics, extraordinarily misunderstood statistics that, despite their misleading name, do not reflect how *inheritable* various characteristics are (Block, 1995; Moore, 2001). Heritability statistics can only account for *variation* in characteristics across populations; they cannot explain the development of characteristics in individuals, and they do not measure the likelihood that a parental characteristic will also be present (i.e., develop) in descendant generations. Under certain controlled circumstances (e.g., animal husbandry projects), heritability statistics have proven useful, in large part because when developmental environments are strictly controlled—as they are whenever heritability statistics have any practical value—variations in developmental outcomes can be accounted for only by those factors that remain free to vary (i.e., genetic factors alone).³ However, heritability statistics have never been shown to have any predictive utility at all when applied to human beings and their offspring; this should not be surprising, given what we now understand about how environmental factors influence developmental outcomes.

One reason heritability statistics computed for human populations have proven useless is that they cannot be appropriately generalized beyond the study population that generated the statistics in the first place. Generalizing the results of a heritability study from a population of research participants to a much broader population requires confidence that the variation in developmental environments experienced by the broader population is no different than the variation in developmental environments experienced by the research participants. But, because we typically do not know which factors play important roles in the development of a particular characteristic (as discussed above), it is not possible to know if the variation in those factors is the same across the broader population as it was in the original population of research participants; the variation in developmental environments experienced by the research participants could be greater than, equal to, or less than the variation experienced by the broader population (Moore, 2006). Consequently, heritability statistics do not provide us with a useful measure of the extent to which a characteristic is influenced by genetic factors independently of environmental factors, our intuitions about it notwithstanding.

2.4. *I Learned in School That Eye Color is Genetically Determined; How Can This Satisfying and Predictively Useful Explanation be Wrong?*

One of the most widely disseminated insights in biology has been Gregor Mendel's interpretation of his pea-plant studies, brought to bear as an explanation of human eye color. Virtually any student with even a minimum of exposure to biology learned at some point in school that human beings have two alleles for eye color, the dominant brown allele typically being represented by a 'big B' and the recessive blue allele typically being represented by a 'little b'; this lesson has no doubt helped countless schoolchildren understand how a blue-eyed child might be born to two brown-eyed parents. This example has the obvious virtue of explaining people's

readily observed experiential data (i.e., their anecdotes) in a way that feels intuitively reasonable to them.

Unfortunately, although the Mendelian explanation of the intergenerational transmission of eye color is readily comprehensible and appears to have predictive utility, it remains the case that it is a misleading simplification of the actual developmental processes that give rise to eye colors. As early as 1915, Sturtevant wrote:

Although there is little that we can say as to the nature of Mendelian genes, we do know that they are not ‘determinants’ . . . The difference between normal red eyes and colorless (white) ones in *Drosophila* [fruit flies] is due to a difference in a single gene. Yet red is a very complex color, requiring the interaction of at least five (and probably of very many more) different genes for its production . . . we can then, in no sense identify a given gene with the red color of the eye, even though there is a single gene differentiating it from the colorless eye . . . all that we mean when we speak of a gene for pink eyes is, a gene which differentiates a pink eyed fly from a normal one—not a gene which produces pink eyes *per se*. (Carlson, 1966, p. 69)

Thus, the explanation proffered for eye color in schools around the world has never been one that biologists have meant to be understood as genetic-deterministic. Rather, the Mendelian explanation continues to be taught in schools because it has predictive utility, owing to the fact that many of the genetic and nongenetic factors that contribute to eye color during development are relatively constant from individual to individual.⁴ As noted above, a factor that does not vary during normal development might *appear* to make no contribution to development, simply because the factor does not contribute to observable *differences* in developmental outcomes. But just because a factor does not contribute to differences in an outcome does not mean that it does not contribute to the development of the outcome *per se*.

2.5. *But Don't Some Traits Develop Without Any Experiential Input at All? And Aren't Some Traits Impervious to Any Such Influences?*

The idea that genes can deterministically cause some of our traits is attractive in part because we have all noticed that some of our characteristics appear to develop without any experiential input. We often consider a characteristic to have been ‘nurtured’ if it has been *learned*, that is, if there are obvious experiences that an individual is required to have had in order to develop the characteristic. In contrast, some of our characteristics—for example, our basic body plans (i.e., two arms and two legs, etc.) or our secondary sexual characteristics (e.g., my facial hair)—do not appear to require any particular sort of experience for their development. Likewise, some traits seem impervious to environmental influence because they develop in all normal members of a species, seemingly independently of the environments in which those individuals are reared. In both cases, however, what appears to be the case at first glance turns out not to be the case once we

have studied the developmental mechanics that actually give rise to the traits in question.

To choose just two of many possible examples, Müller (2003) has written about the importance of experiencing particular mechanical forces during embryonic development in order to develop normal skeletal structures, and the work of Kaas (1991) and Merzenich (1998) with nonhuman primates and human beings has highlighted the important roles that normal sensory and motor experiences play in the development of the sensory and motor maps that characterize all normal human brains. In such cases, the experiences *required* for the development of these characteristics are nonobvious, perhaps because all normally developing individuals encounter the requisite experiential stimuli. If we define 'experience' very narrowly as 'learning', it is true that some of our characteristics do not require experience for their development. But such a definition is unnecessarily narrow, because there are many nongenetic factors that make essential contributions to development, and many of these would be considered 'environmental' by anyone's definition of that word (e.g., nutrition). If we define 'experience' more broadly as exposure to any nongenetic stimulus that can influence development, we can safely say that there are no traits that are impervious to experiential input.

As we have learned more about the biological mechanisms underlying the development of the structural and behavioral features of animals, it has become clear that just as genetic factors can only influence development by affecting cellular processes 'above' the level of the genes, all experiences must have their effects by influencing events in the body's cells, whether those experiences entail learning or not. Experiential factors are now known to influence gene expression through several mechanisms, including (but not limited to) those involving the actions of steroid hormones and those involving the actions of a class of genes known as immediate-early genes (IEGs). For example, testosterone levels change as a function of sexual experience, and hormones like testosterone are known to be able to diffuse across both cellular and nuclear membranes where—once they have been bound by specific receptors—they can bind with DNA to regulate gene expression (Yamamoto, 1985). In contrast, IEGs can be influenced even more directly by experience; IEGs located in the nuclei of neurons begin to be expressed only when specific experiences produce neural activity in those cells (Michel & Moore, 1995). As these examples make clear, experiences can influence development in many ways other than simply via 'learning', as it has traditionally been understood.

Although for many of our characteristics, we have not yet discovered which experiential inputs influence their development, persistent investigators have repeatedly found that characteristics previously thought impervious to experiential input *are*, in fact, influenced by an organism's experiences during development. In a classic demonstration of this phenomenon, Gottlieb (1981) demonstrated that mallard ducklings' natural tendency to move toward the source of the mallard maternal call—a tendency that had previously been described both as innate, and uninfluenced by experience—can be affected by particular sensory experiences; in fact, Gottlieb (1991b) was able to get mallard ducklings to move toward the source of

a chicken's call (a stimulus that normal mallard ducklings would not approach), simply by exposing them to particular auditory stimuli at the right time during embryonic development. Likewise, West and King have shown that a behavioral characteristic of male cowbirds previously thought to emerge from a species-specific innate template—namely the songs they sing during courtship rituals—is, in fact, importantly affected by the males' experiences. Specifically, female cowbirds influence the structure of developing male cowbirds' songs by responding to segments of their immature, variable vocalizations with a very specific, nonvocal, social behavior that ultimately shapes the songs into the stereotyped, potent form that is typical of mature males (West & King, 1988; King, West, & Goldstein, 2005). Given the number of characteristics that were once thought to develop in a way that is impervious to experiential input, but that have since been shown after extensive developmental analysis to be affected by such input, it is now the emerging consensus of developmental scientists that there are no traits whose development is impervious to experiential influence.

2.6. *Doesn't the Theory of Evolution Require Adaptive Characteristics to be Transmitted Across Generations by the Genes? Aren't Species-Typical, Adaptive Traits—Those That Develop in all Normal Members of a Species—Necessarily Caused by Genes Alone?*

Biologists' received view of evolution is known as the Modern Synthesis; this theory was produced by a collection of biologists writing in the first half of the 20th century, and was a remarkably successful fusion of Darwin's ideas about evolution and the theory of the gene that emerged following the scientific community's rediscovery in 1900 of Mendel's seminal work on heredity. In order to produce a comprehensive theory that actually worked, the architects of the Modern Synthesis felt compelled to exclude developmental processes from their theory, despite the fact that many of them—Theodosius Dobzhansky, Julian Huxley, and Sewall Wright, for example—recognized the central role that such processes rightly should play in a comprehensive theory of biology. One consequence of this decision was the subsequent definition of evolution in terms of changing gene frequencies across generations; the Modern Synthesis is a theory about genes and their role in evolution (Jablonka & Lamb, 1995). However, as more recent data have discredited the notion that genetic factors can deterministically cause traits (adaptive or otherwise), the Modern Synthesis has become an increasingly unsatisfactory theory of biology.⁵

Although consensus has not yet been reached on how the Modern Synthesis should be altered, it is clear that any theory that *requires* characteristics to be transmitted across generations via exclusively genetic mechanisms will ultimately fail, because genetic mechanisms alone are not capable of doing the work that such theories require of them. Therefore, one of the several revisions to the Modern Synthesis that must be implemented if it is to remain a viable theory of evolution would replace the strictly genetic transmission of adaptive traits with transmission mechanisms that

provide both the genetic *and* nongenetic resources that developing organisms require to build the traits that proved adaptive to their ancestors. Among the many theorists who have been trying to figure out how such mechanisms might operate are biologists (e.g., Jablonka & Lamb, 2005), philosophers of biology (e.g., Griffiths & Gray, 2001; Robert, 2006), and behavioral scientists (e.g., Lickliter & Berry, 1990; Lickliter & Honeycutt, 2003; West & King, 1987). These theorists acknowledge that nature has provided mechanisms by which organisms reliably develop adaptive characteristics during development; such species-typical, adaptive characteristics obviously play important roles in the evolutionary process. But it has become clear that such characteristics need not be genetically determined, in part because each species has evolved in a particular context—a developmental niche—and these contexts can now be *expected* to be reproduced during the development of descendant generations (Greenough et al., 1987). Thus, evolutionarily adaptive traits can be developmentally stable and universally acquired by members of a species, but still not be specified in genes that deterministically dictate phenotypic outcomes prior to the actual unfolding of development. In fact, it now appears that some genes *require* reliable developmental niches in order to be stably expressed across generations (K. Stotz, personal communication, January 28, 2008).

3. Why it is So Difficult to Convey the Fundamental Interdependence of Nature and Nurture to Students and the General Public

Beyond the reasons alluded to above, there are likely additional reasons that students, journalists, and social scientists unfamiliar with the current facts of biology are skeptical when they first hear that genes cannot determine the forms of even relatively simple biological characteristics. But given the questions that usually arise when laypeople learn of the nondeterministic nature of genetic influence, it is clear that the major sources of this skepticism include (1) previous exposure to teachers who asserted that genes *can* determine some characteristics (however incorrect those assertions might have been), (2) exposure to mass media reports about studies (including twin and adoption studies) that, when simplified, seem to support claims of genetic determinism, and (3) personal observations of the development of characteristics that appear to be unaffected by experiences. In contrast, it seems unlikely that simple ignorance of the relevant facts of biology should be considered the primary source of skepticism in this case, because if it was, then one ought to be able to convince a skeptic that genes cannot independently produce characteristics like hair color, alcoholism, height, or breast cancer by simply explaining what genetic factors *do* during development: that is, provide a portion of the information needed to sequence proteins. However, explaining that this is the one thing that genes actually can contribute to development generally does not lead immediately to acceptance of the idea that genes are unable to operate deterministically.

The resistance typically encountered by a scientist trying to explain how these processes work might be no different than the resistance physicists probably meet when they inform naïve listeners of the fundamental equivalence of matter and energy; as in that case, convincing listeners that genes do not operate deterministically would be difficult if they are ignorant of background information required to evaluate the relevant data. Similarly, although students can be taught about the limits to the conclusions one can appropriately draw when interpreting the correlational data generated in human twin studies, a deep appreciation of how little such studies can actually tell us about genetic contributions to our traits is more difficult to obtain; even professional social scientists occasionally jump to inappropriate causal conclusions on the basis of correlational data.

Two other features of this particular problem render it exceptionally difficult. First, even though biologists now *know* that genetic factors always interact with other factors to produce our characteristics, in most cases we are still poorly informed about the specific developmental mechanisms that produce those characteristics. Indeed, it is the very need to illuminate these developmental mechanisms that has motivated some of the more vocal critics of genetic determinism (Gottlieb, 1995; Lehrman, 1953; Lickliter & Honeycutt, 2003). Until such mechanisms have been elucidated, however, it is difficult to convince students to give up a sense of understanding (however ill-founded it might be) in favor of a more honest sense of ignorance; generally, students are discomfited by the very large gaps that still characterize scientific knowledge in this domain. Second, just as Albert Einstein was troubled by quantum mechanics because of its probabilistic nature—he famously expressed this uneasiness in a 1926 letter to Max Born, writing ‘I... am convinced that He [God] does not throw dice’—it is not particularly surprising that students would prefer deterministic theories to probabilistic theories. The fact that the developmental processes that give rise to our characteristics are fundamentally probabilistic (Gottlieb, 1998) renders them harder to understand for students more familiar with simple cause-effect relationships.

Ultimately, genetic determinism likely owes much of its endurance to the fact that it is so simple. However, although many philosophers and scientists consider simpler theories to be preferable to more complex theories, all other things being equal (Sober, 1981), a truly complex reality requires a complex theory, even if such a theory might initially be less appealing to those exposed to it. Unfortunately, it is not yet clear how we might best attempt to convey the essential interdependence of nature and nurture to students and the general public.

That having been said, some of the new concepts and metaphors introduced recently by theorists concerned with these issues (Moss, 2003; Stotz, 2006; Griffiths & Stotz, 2006) might be useful tools in this effort. Metaphors can powerfully influence how we think about complex phenomena; for example, if one thinks of a genome as analogous to a recipe, one is more likely to focus on matters of process than if one thinks of the genome as analogous to a blueprint. Many systems theorists remain dissatisfied with ‘recipe’ metaphors for the genome as they were with ‘blueprint’ metaphors for the genome, but it is clear from this example how the use of different

metaphors can influence our conceptualizations. In addition to those theorists who have suggested new metaphors for re-conceptualizing the genome, other theorists (e.g., Moore, 2001; Thelen & Smith, 1994) have proposed metaphors specifically designed to dispense with the outworn and obstructive dichotomies (e.g., nature vs. nurture, gene vs. environment, etc.) that we inherited from our intellectual ancestors at the end of the 19th century. In much the same way as a ‘recipe’ metaphor for the genome draws attention to the *processes* driving development, these metaphors serve to focus attention on the fundamental interdependence of ‘nature’ and ‘nurture’, an interdependence that runs so deep that the very distinction between the two ideas is rendered suspect. Perhaps re-conceptualizing notions like ‘genes’ and ‘nature’ will finally enable students and the general public to abandon the genetic determinism that biologists now recognize as indefensible. But regardless, effective education about these matters will no doubt improve as we come to understand why belief in genetic determinism has persisted so long in the face of evidence indicating that it is not a constructive way to think about the emergence of biological and psychological characteristics in development.

Notes

- [1] Strong evidence that no two bodies ever develop identically—even if they are reared in identical environments—has been generated in studies of a relatively simple type of worm, the nematode *Caenorhabditis elegans*. Such studies have determined that genetically identical worms reared in identical environments can nonetheless develop different behavioral characteristics. As Gilbert & Jorgensen note, “organisms with the same inheritance . . . and the same environment . . . still [wind up with] behavioral differences as a result of chance events during development” (1998, p. 263). If differences like these can characterize such relatively simple animals reared under such controlled conditions, we ought to be very suspicious indeed of our intuitions that differences between human individuals reared in ‘the same environment’ must have been caused by differences in their genes.
- [2] To give just one of many possible examples, The New York Times reported in a 2005 article that “some researchers believe there is likely to be a genetic component of homosexuality because of its concordance among twins” (Wade, 2005). Although studies of twins are not able to produce valid conclusions about the extent to which genetic factors contribute to the development of particular characteristics (Moore, 2001, 2006), such studies were presented to the public in this article in the context of a sentence that began ‘If sexual orientation has a genetic cause . . .,’ as if it might actually be possible for genetic factors to determine sexual orientation.
- [3] Of course, the converse is true as well: when genetic variation in a population is virtually absent, variations in developmental outcomes can be accounted for only by environmental factors. This feature of heritability has given rise to some heritability estimates that strike most people as remarkably counterintuitive. For example, although no one would question the importance of the role that genetic factors play in the development of 5-fingered hands in human beings, the heritability of this characteristic is nonetheless extremely low (Block, 1995). This result reflects the fact that variations in finger numbers across human populations are better accounted for by variations in the frequencies of industrial accidents than by variations in the frequencies of particular genes (because there is very little variation

across human populations in the frequencies of genes that contribute to the numbers of fingers on our hands).

- [4] It is worth noting here that it is not merely a happy coincidence that the nongenetic factors required for the development of some of our characteristics are so common in our developmental environments. Developmental environments are *typically* reproduced in each generation, ensuring that the presence of these nongenetic factors is relatively stable across generations. Griffiths and Gray (1994) have identified several types of 'developmental resources' that are reliably reproduced in the developmental environments of successive generations, including (among others) parental, population-generated, and persistent resources. For example, the homes that parents build prior to the birth of their offspring impose important structure on the offspring's developmental environments; while enhancing an offspring's survival prospects, such parental resources also ensure that offspring are exposed to a limited portion of the stimulation the world has to offer, a portion that is, in the words of Lickliter and Berry (1990) "structured, organized, and specific to the organism." Likewise, cultural artifacts (e.g., spoken language in human beings) are present in the developmental environments of successive generations of organisms, and resources such as these, generated not by particular parents but by a *population* of ancestors, can structure the environment in ways that contribute importantly to the development of adaptive characteristics in individuals. Finally, environmental factors that are not actively produced by organisms at all but that are—by virtue of where a particular species reproduces—persistently present in those organisms' developmental environments can also provide successive generations with stable nongenetic information that contributes to the development of species-typical characteristics. For example, the National Academy of Sciences (1999) reports that 'the larvae of the fly *Drosophila carcinophila* can develop only in specialized grooves beneath the flaps of the third pair of oral appendages of a land crab that is found only on certain Caribbean islands;' clearly, extremely specific developmental environments can, in effect, be passed from generation to generation in a way that ensures the stable transmission-across-generations of nongenetic developmental information.
- [5] Scientists dissatisfied with the Modern Synthesis have identified a variety of problems with the theory, only one of which is its underlying assumptions regarding the genetic determination of adaptive characteristics. Some of the most trenchant critiques of the Modern Synthesis have emerged from Evolutionary Developmental Biology, the branch of biology sometimes known as 'evo-devo'. For instance, evo-devo researchers have argued that the Modern Synthesis does a poor job of explaining how novel adaptive characteristics of animals emerge in the first place, a question that arguably should be at the center of any theory purporting to explain evolution. However, given the focus of this article on genetic determinism, a broader critique of the Modern Synthesis that considers such concerns is beyond the scope of this work.

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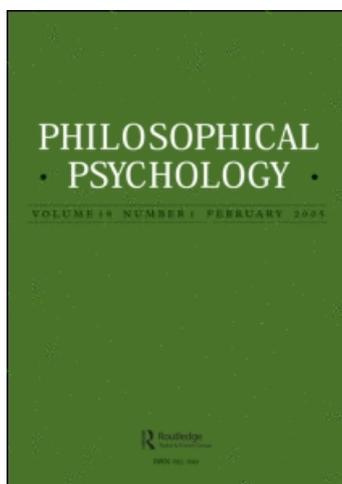
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Nature and Nurture in the Development of Social Smiling

Susan Jones

Research on the origins of human social smiling is presented as a case study of how a species-specific, species-typical behavior may emerge from thousands of momentary events in which a continuously changing biological organism acts to make, respond to, and learn from its experience.

Keywords: Emergent Behavior; Infants; Social Smiling

Human social smiling satisfies the traditional criteria for identifying innate behaviors. It appears to be species-specific. Other great apes do sometimes contract their cheek muscles to raise the corners of their lips, but the lack of response from other members of the species suggests that such ‘smiles’ have no signal value for chimpanzees. Human social smiling is universal—the only exceptions being individuals with considerable damage to the central nervous system—and appears to have a universal meaning (Ekman, 1973). Smiling is present in the newborn (Messinger et al., 2002; Wolff, 1963). It is in fact present in the *blind* newborn, and so requires no social experience or observational learning. The infant’s first smiles are not exactly like mature smiles, but within weeks of birth, typically-developing infants look their social partners in the eye and grin. All of these observations support the idea that social smiling is an innate behavior in humans.

A closer look, however, adds complexity to the picture. The newborn’s smile is not really a social behavior—the infant first smiles only during sleep, then only when drowsy (Emde, Gainsbauer, & Harmon, 1976; Messinger et al., 2002; Wolff, 1963). When waking smiles begin, about 4–6 weeks after birth, the infant

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smiles in response to a range of human and non-human, living and non-living, internal and external stimuli, and she does not distinguish among them. She may smile at a parent at one moment, and a moment later smile equally broadly at a wind-blown curtain (Wolff, 1963). The common characteristic of these stimuli for smile production seems to be that they induce a rapid change in the infant's state of arousal—almost a startle—that has been called an 'arousal jag' (Berlyne, 1967; Sroufe & Waters, 1976).

In adults, smiling is widely interpreted as an expression of happiness. Undoubtedly, smiles sometimes accompany feelings of joy; but in reality most smiles appear to be produced as emotionally neutral social responses. You are likely to smile at a number of people today, though it is not likely that they will all make you happy. Thus, there are two general trends in the development of smiling; the elaboration of the largely endogenous, reflex-like smiling of the newborn into correlates and signals of positive emotion; and the emergence of the socially controlled and voluntary smiling most common in adulthood. We know little about the processes that produce these changes, but it is the job of developmental science to discover them.

Say 'developmental science' to most people and they will reply 'Nature versus nurture'. Researchers have generally avoided this bald dichotomy, attempting instead to find a way to express the interactional/transactional quality of the relations between what seemed to be biologically-based, species-typical behavioral tendencies and the effects of the individual organism's experience in the world. In recent years, some very elegant research (especially work with non-human species) has shown more clearly than ever before that the organism's species-typical features and its individual pre- and postnatal experiences are inextricably intertwined in the mechanisms that underlie behavior, and that produce developmental change over longer periods of time (see Blumberg, 2005 for multiple examples). We have learned that behaviors that appear to be evidence for the inheritance of complex behaviors often have more straightforward explanations. For example, Blumberg has shown that infant rats 'cry'—that is, produce high-pitched vocalizations—when out of the nest and cold, not because they want to be rescued, but because the cold causes a slowing of the heart, which results in an abdominal compression reaction to increase the rate at which blood returns to the heart, and incidentally results in high-pitched vocalizations (Blumberg, Sokoloff, Kirby, & Kent, 2000). This is not the purpose-built 'attachment' system it was thought to be, but it is good enough: mother rats respond to the pups' cries by returning them to the nest, where the warmth of their siblings increases their heart rate and the vocalizations stop.

We have also learned that developmental change is not maturation: it does not happen automatically on a pre-set schedule. Instead, development takes place in moment to moment transactions between the organism and its environment—though the changes may be measurable only on larger time scales (Thelen & Smith, 1994). With each momentary transaction, the organism may change, and thus come to the next transaction a slightly different creature.

My data on changes in smile production in human infants, though incomplete, provide an example.

Many years ago, my students and I began to look for the emergence of social smiling in infants (Jones & Raag, 1989; Jones, Collins, & Hong, 1991). We posed the question this way: Is infant smile production affected by whether or not there is an audience for that facial signal? Two social psychologists, Kraut and Johnston (1979) had shown such an 'audience effect' in adults. For example, bowlers who scored a strike or a spare did not begin to smile when they saw the pins drop. Instead, they smiled when they turned to face their bowling companions.

We set up an infant analogue to the bowling alley to ask whether infants' smiles were similarly socially elicited or directed. Infants were seated before an array of wall-mounted toys with their mothers seated several feet behind them. The toys could be manipulated in various ways: they opened and closed, spun, beeped and rattled, and the infants smiled when they did. Our question was whether the infants would smile at the toys that provided the arousal, or would turn and smile at their mothers behind them. We also had mothers spend some of the time watching their infants and some of the time reading a magazine, to see whether infants were sensitive to whether their 'audience' was in fact paying attention and available to receive their social signals.

Eighteen-month-old infants in this situation showed an audience effect on smile production much like that seen in adults. They smiled at toys that spun or rattled, but also turned to smile at their attentive mothers. When mothers were not paying attention, smiling in their direction dropped precipitously; but there were no separate indications that the infants were less happy than before: infants continued to play with and smile at the toys, and when a friendly and attentive stranger sat in, infants in the Inattentive Mother condition transferred their smiling to the stranger (Jones & Raag, 1989). It appeared, then, that by 18 months of age, social smiling was already in place and distinct from reflexive or emotional smiling.

In our next studies, we put considerably younger infants—10-month-olds—into the same play situation, and examined their smiling in the direction of their mothers and at the toys (Jones et al., 1991). Interestingly, 10-month-old infants looked at their mothers with an average frequency very similar to that observed in 18-month-olds—that is, a little less than twice in each minute. This was just the mean, however. Smiling did not occur at regular intervals. Instead, in both age groups, smiles occurred in clusters, or 'runs'.

As with 18-month-olds, we found that the 10-month-old infants directed more smiles to an attentive mother than to a mother who was reading and not making eye contact. And also as with the older infants, there was nothing in the 10-month-olds' other behaviors—their concentration on toy play, their smiling at the toys, their vocalizations, or the timing of their looks toward their mothers—to suggest that they were disturbed by their mothers' inattentiveness. They simply reduced their smiling in the mothers' direction (Jones et al., 1991).

We distinguished between smiles that began before and after the infant turned her head in her mother's direction, and found that, when the mother was not paying attention, the infant continued to turn to look at her (though she *was* not paying attention), and continued to smile when he saw his reading mother *after* turning his head. The infant also directed *more* smiles toward the toys when his mother was not paying attention. However, he stopped turning those already-smiling faces away from the toys and toward his preoccupied mother (Jones et al., 1991).

We labeled smiles to mother that began before the child turned toward his mother 'anticipatory smiles' because they appeared to begin in anticipation of the social contact to come. I should stress that the anticipation we referred to was temporal, not cognitive or emotional. The same behavior had elsewhere been called 'affective sharing' (Baldwin & Moses, 1996; Waters, Wippman, & Sroufe, 1979), but we rejected this label because it assumed a rather sophisticated function for young infants' smiles. To suggest that young infants cognitively or emotionally anticipate social contact would go equally far beyond the data.

The finding that anticipatory smiles, in particular, were reduced when mothers were not paying attention suggested that, by 10 months of age, these smiles were already voluntarily controlled social signals. This impression was strengthened in a 3rd study when we found that those infants in the 8–12 months age range who produced anticipatory smiles (about one-third of 8-month-olds; about three-fourths of 12-month-olds) were the same infants who had begun to produce other communicative behaviors like pointing and reaching for objects, and the vocal equivalents of these manual actions (Jones & Hong, 2001).

In short, it appears that, by 10 months of age, anticipatory smiling is one of a group of emergent communicative behaviors; that it is at least partially dependent on the infant's appraisal of the social context; and that it is at least partially under voluntary control.

Still in search of the age of emergence of voluntary social smiling, we next put 8-month-old infants into the same play situation with their mothers seated behind them (Jones & Hong, 2005). The 8-month-olds also looked at their mothers on average a little less than twice in each minute, and smiled during many of those looks. These infants, however, produced virtually no anticipatory smiles. It appears, then, that anticipatory smiling emerges between 8 and 10 months of age. Moreover, because anticipatory smiling is the only kind of smiling for which there is evidence of some social context effects and evidence of voluntary control, it may be accurate to say that truly social smiling emerges between 8 and 10 months. But what brings about this change in the conditions for smile production?

We assume that developmental change occurs in moment-to-moment interactions between the infant as he is (has come to be) at that instant, and small scale events that he produces and/or experiences. The infant comes to each small event as the combined product of all of his biological potential as it has been realized in a succession of other events in other moments. He is slightly changed by each event, so that at any moment in time, he is the cumulative product of all of his biological and experiential history. Thus, we expect that large

changes in smile production—such as the changes that bring smiling under social control—will be the cumulative effect of changes on a much smaller, smile-by-smile scale. It follows, then, that the factors that make smiling more social across infancy should be there to be discovered among the factors that make infants at specific ages smile specific smiles.

The 8-month-olds did smile at the toys and they did smile during looks to their mothers, but only when the mothers were paying attention. So clearly, something about the attentive mothers was a causal force in infants' smile production during looks to mothers. However, as with the older infants, no infant behaviors other than smiling differed in the periods of attentive and inattentive mothers. Infants played as long and with apparently equally concentration in each part of the experiment. They smiled as much at the toys, looked equally often in their mothers' direction, and in both periods produced no unhappy vocalizations. In short, there was no evidence of broad differences in emotional state when mothers paid attention versus when they did not. Clearly, then, the different rates of smiling could not be explained in terms of differences in happiness. So we looked instead for an explanation of the different rates of smiling among the moment-to-moment differences in mothers' and infants' behaviors that distinguished between infant looks that included smiling and those that ended without the infant's having smiled.

For this comparison, we obtained larger samples of smiles and looks from a new group of 8-month-olds who spent 10 min in the playroom, with their mothers paying attention throughout. About half of the looks these infants directed toward their mothers included an infant smile. Note, however, that the looks with and without smiles were not randomly mixed across the play session. Instead, as in older infants, smiles—including smiles during looks toward the mother and smiles at the toys—tended to occur in runs.

To determine whether mothers were eliciting smiles by doing specific behaviors, we compared the things that mothers did during infant looks with and without infant smiles. Specifically, we coded everything mothers did throughout each infant look with no smile, and compared those behaviors with what mothers did during infants' looks with smiles *before* the infant's smile occurred. We found nothing to explain why infants smiled during some looks and not during others. Mothers consistently responded to more than 80% of their infants' looks whether or not the infant smiled. Mothers' responses in both cases were mostly smiles. They showed a small tendency to talk more and nod more during infant looks without smiles, but there was nothing that mothers did more commonly before an infant smile than during a non-smiling infant look.

On the other hand, the previous study had shown that, if mothers were not looking at all, their infants did not smile. So the sight of the attentive mother, or perhaps the establishment of eye contact, or perhaps *any* behavior on the mother's part, seemed to be a condition for infant smile production.

We next looked at what the infants were doing in the 3 s just before turning to look at their mothers and either smiling or not smiling. Here there was one large difference: infants who were engaged in active toy play just before turning were

more likely to smile at their mothers than infants who were just looking at the toys, or just beginning or ending a particular activity before they looked. This finding, and the fact that smiles during looks to mother clustered with smiles directed toward the toys, suggested that arousal from toy play was necessary, though not sufficient, condition for smile production.

In summary: we found that a number of factors appeared to converge to determine whether or not infants smiled at their mothers during play.

- First, we found that infants' rate of looking toward their mothers during play was quite stable across different groups at the same age (8 months), and across ages from 8 to 18 months. This finding suggests that this kind of monitoring behavior might be universal in infants, and might be regulated by its own, perhaps attachment related mechanism. The stable periodicity of infants' looking to mothers makes this behavior worthy of study in its own right.
- We found that infants did not smile at the mere sight of the mother. Instead, it appeared that eye contact or some behavioral response from the mother was usually a necessary condition for infant smiling.
- We found that mothers almost always responded to infants' looks, and that more than 80% of the time the mothers' response was a smile. However, infants only smiled during about half of their looks to mother. So a smile from the mother was not sufficient to elicit an infant smile. More generally, since no maternal behavior was more common before infant smiles than during looks with no smiles, it appeared that no particular maternal behavior was linked in these studies to infant smiling. However, because the infants did not smile at unresponsive mothers, it appears that some behavior on the mother's part was a necessary condition for infant smiling.
- Finally, we found that active play on the infant's part predicted smile production. So active play appeared to predispose the infant to smile during a look at his mother. However, active play was not the whole story, since infants typically did not begin to smile until they turned and saw their attentive mothers do something.

In short, the data suggest that three events came together in particular moments in time to make the 'social' smiles that infants produced while looking at their mothers. These events were: (1) heightened arousal due to active play; (2) regularly-timed looks toward the mother; and (3) a social response from the mother during the infant's look. How did these events interact?

The fact that smiles directed to toys and to mothers were clustered rather than distributed evenly across the experimental period suggests that infants' arousal levels due to engagement with and feedback from the toys waxed and waned across that period. At times, arousal levels rose to the point where a sound or motion of the toy could create an arousal jag sufficient to make the infant smile. At other times, arousal hovered below threshold. However, if the infant looked at his mother, and she responded, the stimulation provided by her behavior produced a large enough spike in arousal to take the infant above the threshold for smiling. At still other times,

the infant's level of arousal from play was so far below threshold that when he looked at his mother, the increment in arousal produced by her response was not enough to raise the infant's arousal above the threshold for smiling. Those looks, then, were smile-less.

The question we started with was how anticipatory smiles—those in which the infant turns an already-smiling face toward the mother—might develop between 8 and 10 months. The evidence from our studies of 8-month-olds' smile production during play provides a plausible answer. The high levels of arousal from toy play apparently carried over into many of the 8-month-olds' looks toward their mothers, because the infants smiled during 50% of those looks. Mothers smiled at infants during looks even more often—more than 80% of the time. After repeated occurrences of these pairings, by the laws of association, the infant's arousal would come to be associated with looking toward the mother, and with the sight of the mother's smile. The infant's and mother's smiles would also be directly associated. Once associated, the occurrence of either member of any of these pairings would evoke the other member of the pairing. Thus, for the infant, arousal during play would evoke a memory of the mother's smile, which would provide *additional* arousal, either because the mother's smile was an arousing stimulus, or because it was directly associated with the infant's smiling. In either case, the additional arousal would (sometimes) take the infant over the threshold for smile production. So the infant would smile without actually seeing the mother. Arousal would also evoke looking—so the infant would turn an already smiling face toward the mother. The result would be an anticipatory smile.

This associative learning account suggests that infants would turn anticipatory smiles toward their mothers during toy play even when the mother was not paying attention. However, if infants respond with reduced pleasure to the sight of the inattentive mother, their baseline level of pleasurable arousal will be lower, and memories of the smiling mother will not add sufficient arousal to take them over the threshold for smile production. In this case, infants should produce anticipatory smiles at the beginning of the period of mothers' inattention but stop producing anticipatory smiles as this condition continues—and this is in fact what we see. The alternative—that infants as young as 10 months of age can remember (throughout extended play intervals) that their mother is reading, and know this means she will not see their smiling signal, and conclude therefore that a smile is not worth producing—seems to us less plausible than the account I have given. This means that we will have to reconsider our previous belief that the mother's inattention has no effect on her infant's state.

The account of how infant's endogenously controlled smiles come increasingly under social control is obviously incomplete. But it seems to me that we have already moved a long way away from the statement that human social smiling is innate.

As it stands, the account combines 'nature'—for example, the arousal response of the nervous system—with 'nurture'—for example, the acquired associations between the arousal response and specific stimuli. However, each of these components can be broken down into smaller and smaller sub-components, which,

like fractals, resemble one another at each level of reduction. Each subcomponent will also have a history, and it is most likely that each history will also combine elements of nature and nurture. As one crude example, consider the arousal from toy play that is associated in this account with both smiling and looking behavior. The history that leads the infant to arousing interaction with the toys includes the development of sitting upright, perception of the toys and their affordances for action, accurate reaching, and positioning of the hand, as well as the development of a self-regulating system that acts to maintain levels of arousal between under- and over-stimulation—and, I am sure, much more. Each of these subcomponents of arousal from toy play, to the extent that it has been studied, has been shown to develop via the interplay of the infant's biological status and input from the environment.

What all such studies tell us is that each history of each contributing component of any single emergent behavior is likely to be a story of a continuously changing biological organism exercising his current capacities for action, thought and emotion in each moment, to make, respond to, and learn from his experience. At all levels of analysis, then, this is likely to be the story of development.

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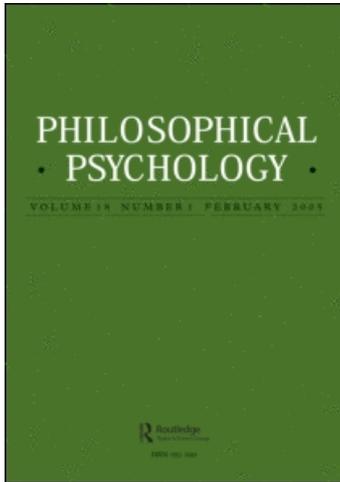
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The Ingredients for a Postgenomic Synthesis of Nature and Nurture

Karola Stotz

This paper serves as an introduction to the special issue on “Reconciling Nature and Nurture in Behavior and Cognition Research” and sets its agenda to resolve the ‘interactionist’ dichotomy of nature as the genetic, and stable, factors of development, and nurture as the environmental, and plastic influences. In contrast to this received view it promotes the idea that all traits, no matter how developmentally fixed or universal they seem, contingently develop out of a single-cell state through the interaction of a multitude of developmental resources that defies any easy, dichotomous separation. It goes on to analyze the necessary ingredients for such a radical, epigenetic account of development, heredity and evolution: 1. A detailed understanding of the epigenetic nature of the regulatory mechanisms of gene expression; 2. The systematical questioning of preconceptions of ‘explanatory’ categories of behavior, such as ‘innate’ or ‘programmed’; 3. Especially in psychological research the integration of the concepts of ‘development’ and ‘learning’, and a richer classification of the concept of ‘environment’ in the production of behavior; 4. A fuller understanding of the nature of inheritance that transcends the restriction to the genetic material as the sole hereditary unit, and the study of the process of developmental niche construction; and last 5. Taking serious the role of ecology in development and evolution. I hope that an accomplishment of the above task will then lead to a ‘postgenomic’ synthesis of nature and nurture that conceptualizes ‘nature’ as the natural phenotypic outcome ‘nurtured’ by the natural developmental process leading to it.

Keywords: Developmental Niche Construction; Environment; Experience; Extended Inheritance; Gene Expression; Molecular Epigenesis; Nature–Nurture Controversy; Postgenomic Synthesis

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1. Introduction

A scientific understanding of the nature and history of living beings depends crucially on our understanding of the most basic biological processes that brought them about: development. Since ancient times, this process has captured the imagination of scholars but has eluded a satisfactory explanation or consistent framework until today. From the beginning, the main problem in the interpretation of development has been the question of whether organisms are the result of the emergence of structures and processes not entirely predictable from the undifferentiated properties of the embryo, or whether they merely unfold or mature out of something preformed or predetermined from the beginning. The term development with its literal meaning of ‘unfolding’ unfortunately suggests this latter interpretation. Today’s received view of development attempts to reconcile both visions: a (multicellular) organism begins as one cell packed with ‘innate’ information of how to build the phenotype, from which the final form emerges in interaction with the ‘acquired’ influences from the environment.

This ‘interactionist consensus’, however, perpetuates the nature–nurture debate by maintaining its inherent dichotomy. Despite being declared dead many times, this debate is alive and well today in the dichotomy of nature as the genetic, and stable, factors of development, and nurture as the environmental and plastic influences (Kitcher, 2001). The term *nature* is applied to those traits that seem genetically determined, fixed in their final form and are present in all cultures, as in discussion about human nature; the term *nurture*, on the other hand, implies variable rearing conditions, including human culture. In contrast to this received view, I want to promote the idea that all traits, no matter how developmentally fixed or universal they seem, contingently develop out of a single-cell state through the interaction of a multitude of developmental resources that defies any easy, dichotomous separation.

One of the foremost aims of a new conception of development is therefore to challenge the widely held view that the physiological or behavioral phenotype derives from *either* nature *or* nurture, or from *both* nature *and* nurture. Both the exclusive and the additive model make no biological sense whatsoever, since no genetic factor can properly be studied independent of, or just in addition to, the environment. The same is true for the environment, which in itself is a concept that includes a wide variety of very different causes and factors, from the genomic environment of a gene, over its chromatin packaging and cellular context, up to ecological, social, and cultural influences upon the whole organism. The message of this article will be that the familiar dichotomies, of which many are so fond, stand in the way when attempting to study and understand development. Those different dichotomies, such as innate–acquired, inherited–learned, gene–environment, biology–culture, and nature–nurture, are not just inappropriate labels in themselves but they do not map neatly onto each other: *genes* do not equal innate, biology, or nature, and neither does the environment stand for acquired, culture, or nurture. So-called innate traits include effects of the organism’s extended inheritance of

epigenetic factors, which are reliably reproduced with the help of ontogenetic niche construction. As a matter of fact, no developmental factor coincides with either nature or nurture, or so I contend. Instead, I advocate new and scientifically more useful distinctions between developmental resources, and ultimately promote the understanding of ‘nature’ as the natural phenotypic outcome ‘nurtured’ by the natural developmental process leading to it.

The papers of this issue are the outcome of an international symposium on “Reconciling nature and nurture in behavior and cognition research” in March 2007 at Indiana University, organized by Colin Allen and myself and funded by Indiana University. Part of its objective was to explore interdisciplinary frontiers in this controversy that may as well promise new insights into the human condition and the idea of ‘human nature’ (see the papers by Robert and Machery in this issue). It was not our intent to have the speakers, who came from different sub-disciplines of cognitive science (including philosophy and biology), merely debate why a certain behavior or cognitive competence is due to either nature or nurture, but instead to use the symposium as an opportunity to reflect on the empirical, semantic, conceptual, methodological/epistemological and metaphysical issues that may help to *resolve* this unhealthy debate. The symposium, we hoped, would provide the perfect venue to think aloud about new directions that current research should take and how the proposed directions could be integrated. The current issue is the outcome of these reflections.

To resolve the nature–nurture debate with a newly emerging view of development several distinct but related sub-problems need to be addressed, which I shall introduce and discuss in this article:

1. An understanding of development requires a deep knowledge not only of the sequences of the genome but also of their regulated expression. A realistic view of gene activation is of pivotal importance since better than any other developmental process it manifests in detail the intricate interaction between genetic material and other developmental factors (Stotz, 2006a, 2006b). In addition, a fully mechanistic picture guards against conflating explanations of the role of genes in development with an explanation of the complete process of development.
2. We need to systematically question preconceptions of ‘explanatory’ categories of behavior, such as innate, acquired, genetically determined or programmed, or even just ascriptions such as ‘genetic’ trait or disease, all of which obscure the necessity of investigating developmental processes in order to gain insight into the actual mechanisms of behavior (see Moore, this issue). In addition such preconceptions are prone to commit the ‘phylogenetic fallacy’, which conflates evolutionary and developmental explanations. The classical research technique to divide the ‘innate’ from the ‘acquired’ are so-called ‘deprivation experiments’, in which the exposure of the developing organism to certain—mostly ‘obvious’—environmental parameters are controlled. However, it does not provide evidence for some general property of ‘independence of the environment’. For instance, restricted housing of cowbirds reveals innate

- artifacts without illuminating actual developmental pathways (see West and King, this issue; Griffiths and Machery, this issue; but also Weinberg and Mallon, this issue).
3. Especially in psychological research, the concepts of ‘development’ and ‘learning’ need to be integrated instead of being studied in isolation and by distinct research traditions (see for instance Jones, this issue; Moore, this issue). This involves a richer classification of the influence of the environment starting with basic environmental influences, e.g., of gene expression or cellular behavior, over low-level sensory processes and real individual experience, to full-fledged individual and social learning (Stotz & Allen, submitted). Careful investigations of the origin of behavior demonstrate the need to distinguish between bioavailability as opposed to simple exposure to stimulation. The distinction is between what an animal has the capacity to do as opposed to how social/perceptual systems function to gate what is available to be learned (see for instance West & King, this issue).
 4. We further require a complete understanding of the nature of inheritance that transcends the restriction to the genetic material as the sole hereditary unit. Instead, heredity must be more widely understood as the processes providing transgenerational stability through the reliable availability of developmental resources in the next generation either through its transmission or reproduction. This includes maternal and paternal (parental) effects, epigenetic factors in a narrow and wide sense, behavioral, cultural, and symbolic inheritance systems. Many of these processes come together to form the ontogenetic niche for the offspring (see West and King, this issue; Lamm and Jablonka, this issue; Alberts, this issue).
 5. Ideas such as (developmental) niche construction and adaptive phenotypic plasticity, and the discussion of the difference between mere exposure to stimulation versus bioavailability suggest that ecological validity will be an indispensable factor for studying development and evolution, and how both processes interact with each other. The long history of reliance on restricted investigative methods in combination with highly insensitive model organisms has given genetic explanation unwarranted dominance by masking the prevalence of nonlinear interactive effects between a multitude of developmental resources (see West and King, this issue; Robert, this issue). Also, a wider understanding of inheritance that often relies on the provisioning by organisms underscores the importance of development for answering evolutionary questions (Lamm and Jablonka, this issue).
 6. A new epigenetic understanding of development encompassing the organism in its developmental niche takes seriously the idea that all traits, even those conceived as ‘innate’, have to develop out of a single-cell state through the interaction between genetic and other resources of development. Such a view should ultimately resolve the dichotomy between preformationism and epigenesis, and instead provide us with a real postgenomic synthesis of development, evolution, and heredity.

2. Molecular Epigenesis

A true appreciation of development will never emerge without a focus on the genome and its regulation by the environment, and it is precisely this field of biology that most forcefully demonstrates that the mere presence of a genetic variant, in all but the extreme cases, is not sufficient to explain variation at the level of the phenotype. . . . It is not the mere presence of a gene that is of functional importance, but rather its expression. [. . .] The structure of the genome highlights the importance of gene-environment interaction. (Meaney, 2004, p. 5)

Genuine understanding of development depends on a knowledge not merely of the sequence of the genome, but of the regulated differential expression of these sequences. Genetic activity is involved in most biological processes, and so are nongenetic activities. Explanations that list only interacting genes are vacuous, or at the very least one-sided and incomplete. Postgenomic biology has brought with it a new conception of the ‘reactive genome’—rather than the active gene—which is activated and regulated by cellular processes that include signals from the internal and external environment (Stotz, 2006a, 2006b). This is not the place to report in detail results that have only very recently come to light concerning the mind-numbing complexities of the expression of genes during development; instead a few examples should suffice. The last decade of whole-genome sequencing led to the formulation of the so-called *N*-value paradox that the number of genes does not increase to match increases in organismal complexity. Instead, the ratio of noncoding DNA rises, and so does the number of functional, regulatory roles played by noncoding DNA and RNA that help to translate, with the active help of instructive environmental signals, sequential information encoded in the genome into developmental complexity (Mattick, 2004). In other words, the more complex an organism, the more complex the expression of its limited number of coding sequences. This lends support to Michael Meaney’s conclusion that what is of particular importance during development is not the *existence* of some genes but their differential time- and tissue-dependent *expression*. In the last two decades development has become equated with *differential gene expression*, but what is hidden behind this equation is the complex network of molecules other than DNA (such as proteins and metabolites), cellular structures, three-dimensional cellular assemblages, and other higher-level structures that control or are otherwise involved not only in the differential expression of genes but in a wide range of other developmental processes decoupled from the direct influence of DNA sequences.

In eukaryotes, DNA is part of a densely packed chromatin structure, which allows it to fit neatly into the nucleus, but which is also a major mechanism to control gene expression. The DNA’s weak chemical bond to the histone proteins, around which it is tightly wrapped to form nucleosomes like beads on a string, needs to be broken down in order to free the DNA molecule to undergo new bonds with transcription factors. Hence, the default position of DNA in eukaryotes is no expression unless expression is activated. Several large complexes of transcription factors and several other accessory proteins such as chromatin remodeling factors are needed in order to

proceed with the transcription of a stretch of DNA. Beyond the activation of DNA, an ever-expanding array of processing and targeting mechanisms are coming into play that not only determine the final gene product but also amplify the repertoire of protein products specified through the eukaryotic genome. We have to understand that genes are not straightforward, structurally- or functionally-defined entities, or even mixed functional–structural entities. Instead, genes are “things an organism can do with its genome” (Stotz, 2006b, p. 905): they are ways in which cells utilize available template resources to create biomolecules that are needed in a specific place at a specific time. The same DNA sequence potentially leads to a large number of different gene products and the need for a rare product calls for the assembly of novel mRNA sequences. Hence, the information for a product is not simply encoded in the DNA sequence but has to be read into that sequence by mechanisms that go beyond the sequence itself. Certain coding sequences, plus regulatory and intronic sequences, are targeted by transcription, splicing, and editing factors (proteins and functional RNAs), which in turn are cued by specific environmental signals. Regulatory mechanisms determine not only whether a sequence is transcribed, but where transcription starts and ends; how much of the sequence will be transcribed; which coding and noncoding regions will be spliced out; how and in which order the remaining coding sequences will be reassembled; which nucleotides will be substituted, deleted, or inserted; and whether and how the remaining sequence will be translated. Many of these mechanisms do not simply produce alternative protein-coding transcripts. A sequence may be transcribed into several parallel coding and noncoding, transcripts. The factors that interactively regulate genomic expression are far from mere background conditions or supportive environment; rather, they are on a par with genetic information since they co-specify the linear sequence of the gene product together with the target DNA sequence. Networks of genome regulation including several different kinds of gene products and instructional environmental resources specify a range of products from a gene through the *selective use* of nucleotide sequence information and, more radically, the *creation* of nucleotide sequence information. This thesis of “molecular epigenesis” argues that even at the molecular level no strict preformationism is warranted since gene products are not specified through DNA sequences alone (Stotz, 2006a).

I would again like to stress the importance of environmental factors in most mechanisms of gene expression. Even though one might argue that most work is done by proteins and other gene products, it generally holds for all eukaryotes that

In the absence of their respective inducing signal, transcriptional regulators tend not to be found in the nucleus with (in the case of activators) their activating regions free to work. Rather, activating regions are masked . . . or . . . the regulators are maintained outside of the nucleus, until the inducing signal is detected. (Ptashne & Gann, 2002, p. 67)

Many genes require for their differential activation and selection the integration of a proper combination of several environmental signals, and this combination of signals together with the presence of a particular combinations of activational factors

controls the exact sequence which will be transcribed and how much should be transcribed. It will also affect co-transcriptional processes such as alternative splicing and RNA editing. The 'same' genes can therefore be expressed in many distinctive ways by different set of signals and activators through "regulated recruitment" and "combinatorial control" (Ptashne & Gann, 2002, p. 7).

These complicating factors of gene expression are not the only reason why it is important not to regard development as *nothing but* gene action and activation. Genes have an important role in development, but their role can be properly understood only within the larger system that holds controlling influence over them. Jason Scott Robert summarizes this attitude:

To take development seriously is to take development as our primary explanandum, to resist the substitution of genetic metaphors for developmental mechanisms... The translation of embryology's hard problem (how a specific organism arises from a single, relatively homogenous cell) into a problem about gene action and activation generates explanations at the level of genes; but these explanations solve (or, rather, begin to solve) the subsidiary problem of the role of genes in development, not the problem of development as such. . . . There is indeed good reason to believe that genetics reduces to development, and not the other way around. (Robert, 2004, p. 22)

3. The Reconceptualization of 'Explanatory' Concepts and Categories of Behavior

This section attempts to analyze a few overused concepts, dichotomies, metaphors, and shorthand formulations that are commonly used in the explanation of behavior. It claims that these, instead of being useful characterizations of behavior or shorthand classificatory schemes, they sidestep deep explanatory analyses of developmental processes and therefore prevent useful and necessary further research into the nature and origin of characteristics or traits that we want to explain. To name just a few of such explanatory concepts: Nature–nurture; innateness; interaction; information; program; inheritance; gene action; maturation; genes-and-environment. I advocate here the replacement of these placeholders by real explanations with specified mechanisms of developmental interaction.

The main problem with all allegedly explanatory categories and concept of behavior, such as instinctive, learned, or genetically programmed, is that they block further investigations into the real ontogenetic and evolutionary causes of a behavior just by their very nature of purporting to explain while really doing nothing but labeling it. After careful and often arduous empirical investigation, all apparently 'innate' processes operating to regulate behavior have turned out to involve epigenetic or experiential factors (Blumberg, 2005). As Paul Griffiths has argued, the vernacular concept of innateness can imply three different and unrelated things, namely the developmental fixity (noninvolvement of experience), species-typicality or universality, and adaptedness, or normativity, of a trait (Griffiths, 2002; see also Griffiths & Machery, this issue; Weinberg & Mallon, this issue). All three of these are

sometimes equated with genetic determination. Besides the fact that I want to argue against the existence of any genetically determined trait; a deeper investigation of these three characteristics is able to show their relative independence of each other (Griffiths, Machery, & Linquist, submitted). Evolutionary adaptations need not be developmentally fixed, independent of life experience, and hard to change, but can instead be phenotypically plastic as is the case with many highly environmentally sensitive polyphenisms, distinct phenotypes that are elicited by different environmental conditions (see Section 5). Neither do adaptations need to be species-typical nor universal, since they can result from frequency-dependent selection, where a trait is only adaptive if a certain percentage of the population carries it. Species-typical or universal traits are not necessarily the result of natural selection but can be dictated by strong physical or developmental constraints that render them hard or even impossible to change. This has for instance been shown by many examples uncovered by the new 'physicoevolutionary' approach, or by research into the homologies of organisms (Gilbert, 2003; Newman, 2003). Last but not least, universality need not be and often is not due to the developmental fixity or experience-independence of a trait. It may be, and often is due to the reliability of certain experiences, which the organism needs to make in order to develop a trait. Song learning in many bird species is a case in point. In some species of birds, such as the brown-headed cowbird, all birds of a population sing the same song (while in many others the songs of individuals may differ substantially, such as in the Australian Lyre bird, or the Indian Common Mynah). While such instances have formerly been taken as support for the genetic determination of song 'learning', we now know that all individuals have to be exposed to other members of their species in order to acquire their population-specific song. The story in cowbirds, which are nest parasites and are therefore not even raised by their own parents or even a member of their own species, is even more complicated and intriguing than with birds which copy the song from their parents, but the details of how they acquire their song need not interest us here. Suffice it to say that cowbirds nevertheless always learn to sing the particular dialect of the population they belong to because of the reliability with which they meet, recognize, and flock with members of their own species and are therefore exposed to the right stimulating experience when maturing (Freeberg, West, King, Duncan, & Sengelaub, 2002; West, King, & Duff, 1990).

The use of metaphors can be understood as another form of stand-in for a full-fledged explanation in causal terms. A metaphor is a rhetorical trope that enhances a description of a subject through the application of implicit and explicit attributes from a well-known subject taken from a different domain. The use of a metaphor 'tacitly involves and assumes as valid all the familiar logical implications, consequences and interrelations between the concepts used as metaphors', and the described concept (Kurakin, 2005, p. 46). A famous example is the ascription of properties of a 'program' to genes, the genome or the genotype. Since the beginning of molecular genetics coincided with the beginning of the informational and computational era, this seemed to be a natural move. The postgenomic era, however, brought with it an insight in the structural complexity of the genome and the

heterogeneity of the genetic material to which a computational and also a classico-mechanistic interpretation seem illfitted. More appropriate seems

An alternative conceptualization of cell differentiation and development . . . where the developing organism is viewed as a dynamic self-organizing system of adaptive interacting agents. This alternative interpretation appears to be more consistent with a probabilistic nature of gene expression and the phenomena of cell plasticity, and is coterminous with the novel emerging image of the cell as a self-organizing molecular system. (Kurakin, 2005, p. 46)

The program metaphor, however, has stuck. This metaphor inspired generations of biologists to describe whole cellular or organismic behavior in terms of gene networks and programs (Luscombe et al., 2004; Weber, Polen, Heuveling, Wendisch, & Hengge, 2005; Wilkins, 2008). In his philosophical gloss on this kind of biological work, Alexander Rosenberg claims that the development of *Drosophila* can be exhaustedly described in a '*Drosophila* developmental program' following "Boolean switching rules in a small number of relatively simple linear programs." Rosenberg adds: "It bears emphasis that I do not mean this claim to be metaphorical. As I shall illustrate and then argue, the genes literally program the construction of the *Drosophila* embryo in the way the software in a robot program the welding of the chassis of an automobile" (Rosenberg, 2006, pp. 61–62). Rosenberg disregards the fact that models of gene networks such as the ones reproduced in his book are in reality only a convenient shorthand for the elucidation of functional co-dependencies of genes, and an intentional simplification of the reality of these dependencies. There is no mention that this shortcut collapses a multi-molecular network, which includes genes, regulatory DNA sequences, a large variety of gene products, intra- and extra-environmental signals, and the contingent history of the cell, onto a single dimension of structural and regulatory (protein-coding) genes. This is deliberate, because it makes the genome *appear* to constitute a program (with the exception that genetic 'programs' rarely ever crash like computer programs!). It also has to be acknowledged that Boolean models offer only a rather crude representation of real world gene networks since they can only describe discrete, instead of continuous changes in the cell (Schlitt & Brazma, 2006).

The time- and tissue-dependent activation, selection and even creation of the relevant nucleic acid sequences from the 'same gene' requires, among other necessary factors, instructional environmental resources. The regulatory network integrates many different aspects of cell activity (transport, cytoplasmic enzyme activities, and energy metabolism) into the transcriptional and post-transcriptional decision. This makes it literally impossible to separate physiology from genomic regulation in any living cells (Shapiro, 1999). The common assumption of predetermination inherent in many such descriptions of gene action begs the question of what determines changes in a sequence of activities; it is always the model rather than the reality which seems to suggest a dictatorial rather than a democratic vision of cell action. I want to argue that what appears as a 'program' is constituted *a posteriori* by a network of interactions within the whole cell.

Of course there are no hard-core genetic determinists around, who would seriously dispute the necessity of any ‘environment’ for the production of a trait. A more common version defends genetic determinism against a background of a ‘normal’ or ‘standard’ environment in which a gene is tightly correlated with a behavioral trait. In many such cases elucidating the details about a ‘normal’ environment—instead of just taking it for granted—would have shown how much the organism or its parents must invest in order to reliably provide the stable environmental resources that allow for a predictable pattern of gene expression (i.e., a tight correlation between gene and trait). I will turn to the importance of developmental niche construction in Section 5. Here I only use the notion to provide further support for the idea that similar to a self-organized ant colony, agency is located neither in the genome nor in the environment but in the organization of all factors in an intricate network.

4. Understanding and Integrating Development, Learning, Experience and Environment

In the last decade, it has become fashionable for cognitive comparative psychologists to study animal behavior in an ‘integrated’ fashion to account for both the ‘innate’ and the ‘acquired’. I argue that these studies of animal learning against an evolutionary background, instead of really integrating the concepts of ‘nature’ and ‘nurture’, rather cement this old dichotomy. They combine empty nativist interpretations of behavior systems with blatantly environmentalist explanations of behavior acquisition. While in some areas of biology, interest in the relationship between behavior and development has surged through topics such as parental effects, extragenetic inheritance, and phenotypic plasticity, which has gone almost completely unnoticed in the study of animal behavior in comparative psychology and is frequently ignored in (cognitive) ethology too. Reasons for this may include the traditional focus on the function of behavior in its *species-specific* form in *adult* animals, which can favor a preformationist or deterministic conception of development, or generally the separation of psychology from biology. In psychology, the process of learning is often set against the maturational unfolding of the young to the adult instead of being understood as part and parcel of behavioral development, either as a process that drives or explains certain developments, or a process influenced by other developmental processes. One of the necessary prerequisites to the integration of nature with nurture is to clarify the relationship between the concepts of learning and development, and to investigate whether and how both concepts can be usefully deployed in the study of animal behavior. This will require the full integration of the concept of learning into a much wider concept of individual experience, or if this term is itself already understood as a higher-order sensory process only applicable to higher organisms, then another more basic concept such as sensation or sensing (Ginsburg & Jablonka, 2007).

What has all this talk about biological development to do with cognitive or behavioral phenomena? The symbolic approach of 'good old-fashioned artificial intelligence' (GOFAI) (Haugeland, 1985) that sees a principled distinction between the cognitive and the noncognitive, or the mind and the body, investigates cognitive processes as if they were disembodied and decoupled from the world and consisting of symbolic manipulation of abstract and enduring mental representations of events in the world. This representational stance cannot or would not deal with the emergence of such symbolic representation out of biological processes, and therefore usually goes hand in hand with a *nativist* understanding of development. In contrast, the embodiment and dynamical systems paradigm understands cognition as emergent, embodied, embedded, situated, and softly assembled action, and attempts to break down the distinction between sensorimotor activity and cognition. In other words, this view is much more conducive to the epigenetic understanding of life and history proposed by the developmental systems perspective that refuses to partition the phenotype into genetic, morphological, psychological, and social levels. These two related perspectives both of which focus on explanations of how novel properties can emerge, complement each other: they not only investigate 'behavior' as the *product* of development but also as the *process* through which development takes place. On the one hand, developmental systems theory grounds cognition in developmental processes. On the other hand, dynamical systems theory attempts to ground development in cognitive processes.

Questions about which species are capable of what forms of learning are typically treated as if organisms come to the task as fully-formed representatives of their species. Thus questions about, for example, the imitative capacities of primates rarely take individual development into account (Jones, 2005, see also Jones, this issue). In fact, it is widely believed on the basis of nondevelopmental studies that nonhuman primates are not capable of genuine imitation. But the importance of development is underscored by experiential findings with human-reared or enculturated apes that show the differential effects of enculturation in human socio-cultural environments on the development of a whole range of capacities in great apes. Among those are many that are theoretically consigned to humans alone, such as mental representational capacities and a whole range of social cognitive capacities like intentional understanding, empathy, and 'true imitation' (Bering, 2004; Call & Tomasello, 1996; Furlong, Boose, & Boyson, 2008; Tomasello & Call, 2004). In a recent paper, Povinelli and collaborators (2008) strongly urge taking the discontinuities between humans and apes more seriously, especially those regarding higher-order, systematic, relational capabilities of a physical symbol system. However, just as with most of traditional cognitive science, they confound cultural symbolic achievements with individual cognitive competencies. By not allowing any explicit role for learning and development, their core rationale for claiming a discontinuity between human and on-human primates rests on a hybrid symbolic-connectionist, formal model of cognition, LISA. Such models are criticized by an embodied stance as a quite unrealistic model of cognitive growth (McGonigle & Chalmers, 2008; Penn, Holyoak, & Povinelli, 2008).

In most of the work within comparative psychology, the basic classificatory scheme is methodological and not tied to recognizing the shortcomings of the operationalism underlying the traditional classification scheme. For instance, Grau and Joynes argue for a ‘neurofunctionalist’ approach, which seeks to classify learning in terms of both neural mechanisms and adaptive function. Their results with rat spinal cords suggest that even in the spinal cord, ‘experience’ has lasting effects on the capacity of neurons to respond adaptively to future environmental conditions (Grau & Joynes, 2005). The basic cellular mechanisms for learning and memory are highly conserved between invertebrates and vertebrates (Burrell & Sahley, 2001) and may even go further back in evolutionary history. Furthermore, the NMDA receptors involved in the synaptic plasticity of neurons use proteins for binding amino acids that are highly conserved from bacteria (Kuryatov, Laube, Betz, & Kuhse, 1994). Even the simplest organisms, bacteria, respond differently to similar configurations of cues in their surroundings on the basis of their specific life experiences. But the concept of bacterial learning may be not more than a philosophical abstraction; do bacteria *really* learn? The answer you give, of course, depends very much on your definitions of learning and experience. Not if learning is restricted to organisms with nervous systems that connect sensory to motor systems, and that extract from the environment information for action (behavior narrowly defined). Possibly yes, if ‘environment’ is understood as the source of a “quite heterogeneous mix of resources called experience” extracted by a wide variety of means, only one of which is sensory, and if knowledge and means for behavior derive from more than what is known to the senses (Moore, 2003, p. 350).

Central to the project of synthesizing development and learning is to identify types or token of epigenetic interaction, the role of experience and learning in the development of particular traits and in development in general, and the role of development in the phenomenon of learning. We need to ground the process of learning in development, and development in cognitive processes. As Samuelson and Smith have noted, “coupling the dynamics of perceiving and remembering with the dynamics of development will lead us to a more complete theory of knowledge and its development” (Samuelson & Smith, 2000, p. 98). From a psychobiological perspective, learning appears as a category within an overall framework of development as the lifelong, adaptive construction of the organism-environment system. Taking the idea of phenotypic plasticity seriously may lead to a conception of development as a lifelong process of ‘learning’ or ‘acquiring’ an adaptive mode of living in a partially constructed environment. And learning as the acquisition of novel behavior and gain of knowledge about the environment becomes synonymous with developing. In a systems view of development, learning is certainly just one among many processes in which experience influences behavior. This new synthesis should help to overcome the age-old dualism between the innate and the learned. Something may not be learned in the strict sense but it is still acquired in the sense that some environmental factors will have played a pivotal role in its origin. A trait may be learned and is still reliably reproduced generation after generation. This is not to say

that there are no differences between developmental trajectories. It is to call for the development of scientifically more fruitful distinctions.

5. From Extended Inheritance to Ontogenetic Niche Construction

The triumph of the reductionist path, from the instrumental particularization of heredity, through the hardening of the particles as material genes, to the resolution of the heredity material in molecular terms, could not, in the final analysis, provide the answer to the plight of inheritance. Heredity is a property immanent to living systems and needs the perspective of the life sciences. (Falk, 2000, p. 339)

Transgenerational stability need not rely on the faithful transmission of DNA alone. Natural selection selects for adaptive traits or phenotypes, which are always derived from the nonlinear interaction among a range of diverse developmental resources. Their organization frequently exhibits phenotypic plasticity, a capacity that allows the organism to react adaptively to different environmental conditions (Pigliucci, 2001; West-Eberhard, 2003). The stable inheritance of this adaptive phenotype depends on the reliable transmission of all the necessary developmental factors across generations. In other words, phenotypic plasticity relies on a stable 'developmental niche' which is faithfully reconstructed by the species, the parent and the organism itself (West & King, 1987). The subject of selection is the whole developmental system (Oyama, Griffiths, & Gray, 2001b).

Inheritance is the reliable availability of developmental resources for and in successive generations either through transmission or reconstruction. The rise of classical genetics produced the theory of the genetic material in the germ line as the only factors faithfully transmitted from parent to offspring; inheritance became synonymous with genetic inheritance. In Section 2, I have argued for the thesis of molecular epigenesis: "Even for the clearest examples of molecular genes such as those traditionally thought to specify polypeptide sequence, epigenetic change ensures that nucleotide sequence alone is not sufficient to predict whether a polypeptide product will be produced or, if it is, what the resulting sequence of amino acids will be" (Burian, 2004, p. 60). Regulatory mechanisms of genome expression amplify the literal coding sequence of the 'reactive genome' by providing additional sequence specificity to the literal DNA sequence; this extends the range of constitutive epigenesis all the way down to the molecular level of sequence determination. Inheritance systems have evolved to make the transmission of crucial information from parents to offspring more reliable and enhance the offspring's fitness. However, it is not so much the particular gene you inherit that counts, but when, where and how a particular sequence is transcribed or translated by the higher order network of gene regulation that controls the time- and tissue-dependent expression of genes. As Matt Ridley has remarked, "the more we lift the lid on the human genome, the more vulnerable to experience genes appear to be" (Ridley, 2003, p. 3). Evolution's answer to this plight of inheritance, or the parents' answer to assert a more reliable influence on the fitness of their offspring, was to provide more than

just genetic resources to the next generation, to construct a stable 'niche for the genome'. West and King were one of the first to urge: "Ask not what's inside the genes you inherited, but what the genes you inherited are inside of" (West & King, 1987, p. 552). Looking at the enormous complexity of gene expression of eukaryotes that reveals a very flexible and reactive genome open to many intra- and extra-organismal environmental influences, "it was simply a matter of time before some systems found ways to manage aspects of their own developmental environment" (Lucas, 2006). In other words, many aspects of experience have evolutionary explanations, an insight shared by some of the most recent and some of the oldest contributors to evolutionary thought:

To the extent that there exists heritable variation among mothers in their ability to discern high-quality mates, pick an appropriate host to place seeds or eggs, or provide protection from predators, and so on, such traits are expected to evolve in much the same way as any other trait subject to the inevitable consequences of Darwinian natural selection. (Mousseau & Fox, 1998, p. *v*)

We live from birth to death in a world of persons and things which is in large measure what it is because of what has been done and transmitted from previous human activities. When this fact is ignored, experience is treated as if it were something which goes on exclusively inside an individual's body and mind. It ought not to be necessary to say that experience does not occur in a vacuum. There are sources outside an individual which give rise to experience. (Dewey, 1938/1963, p. 39)

The ontogenetic niche comprises all molecular, cellular, ecological and social circumstances inherited by the organism and includes all developmental factors that are reliably and dependably provided from one generation to the next. All these resources are indispensable for the successful reproduction of a developmental system. The construction of a developmental niche relies heavily on the extragenetic or extended inheritance of developmental resources. The great variety of inherited resources are made reliably available through epigenetic, behavioral, cultural and symbolic inheritance 'channels' (Jablonka & Lamb, 2005). These channels include maternal and paternal (parental) effects, which are defined as the causal influence of the parental phenotype, or the environment the parents' experience, on offspring phenotype. Such effects are completely independent of the genes contributed to the offspring, and can also not be reduced to the influence of parental genes or RNAs, even though they can and do play a role in many instances. Parental effects are comprised of differential resource allocation either through egg size and composition, placental nutrition, or nursing; preference induction (oviposition, imprinting on food, habitat, and mates); mate choice; and the nonfacultative and facultative imprinting of genes and reprogramming of gene expression through chromatin remodeling and DNA methylation (Jablonka & Lamb, 2005; Mousseau & Fox, 1998). The cytoplasmic chemical gradients plus the messenger RNA and transcription factors, all of which are inherited with the mother's egg, give the influence of the offspring's gene expression a head start, but as the examples above show the mother's control over the fetus' environment does not stop there. Even after

birth rearing practices, such as the licking of pups by rat mothers; the facilitation of offspring experience through the creation of opportunities; and various forms of social learning continue to influence gene expression levels and other developmental processes. Parental activity can facilitate, guide and entrench social learning, which in the case of humans and higher animals falls under the rubric of the cultural and even symbolic ‘transmission of information’.

There have been repeated attempts to reduce all of these mechanisms to the action of inherited or parent-of-origin genes, so that ultimately the real causes are all genetic. This special pleading fails in the light of the discovery that development relies less on the existence of genes in an organism than on the regulated expression of these genes, which ultimately depends on a host of environmental factors. Wherever there are genes there are extragenetic factors necessary for their regulated expression.

I have called the design-like control of the next generation’s developmental environment extended inheritance or ontogenetic niche construction. What all of the above cases of inheritance through environment construction have in common is making the transmission of crucial information more reliable. And while some of the above mechanisms have at first sight not much in common with the construction of epistemic structures by an extended mind, in the latter cases of behavioral, ecological and cultural inheritance the biological shades smoothly into the cognitive.

As Lamm and Jablonka have pointed out, epigenetic inheritance, just like genetic inheritance, is not just about reliability, stability and fixity, it can also lead to ‘transgenerationally extended plasticity, and developmentally-induced heritable epigenetic variations provide additional foci for selection’ beyond genetically-induced heritable variations (Lamm & Jablonka, this issue).

6. Ecological Evolutionary Developmental Biology

The nature–nurture dichotomy is not reduced to the field of developmental biology and psychology but plays an important role in our conception of the process of evolution. One might even say that it was the very separation of nurture (germ line, genes) from nature (soma, environment, individual development) that cemented the exclusion of developmental biology from the Modern Synthesis of Evolution. The last two decades saw a growing interest in questions that the received view was not able to address, such as questions about patterns and processes of phenotypic evolution, and the origin of evolutionary novelty and innovation. A new synthesis of evolutionary developmental biology (evo-devo) began to form. From the developmental systems perspective, the elucidation of extended processes of inheritance made it clear that the reason to exclude so-called processes of nurture or individual development—for not producing heritable variation—no longer holds. Increasingly now, one hears that in order to situate a synthesis of development and evolution ‘in the real world’ the role of ecology needs to be fully integrated as well (Gilbert, 2001).

The rise of the new science of *Entwicklungsmechanik* (developmental mechanics) in the late nineteenth and beginning of the twentieth century regarded the anatomical

tradition, with its evolutionary context and its methods of observation of developing organisms in their natural context, old-fashioned and unscientific, and completely rejected any (at that time regarded as) mystical ideas of epigenesis. The new mantra of experimentation with its new methodology of manipulating the animal in controlled laboratory settings brought the discipline of embryology, now called developmental biology, from the sea shore to the laboratory. Against this background we have to understand the emerging ‘model organism’ approach. To make animals constantly available and as uniform as possible, and the scientist independent from the dictate of seasonal availability and natural variability, laboratories started to breed their own animals. This constrained the choice of organism, which “must be selected for the inability of their development to be influenced by specific environmental cues.” In other words, “the influence of...environmental sources of phenotypic diversity were progressively eliminated under the physiological context of embryology” (Gilbert, 2003, p. 88f).

While the physiological tradition favored the whole organism at the expense of the environment, the newly emerging genetics, especially molecular genetics, focused on genes at the expense of the organism. Also, the paradigm model organism of genetic research, *Drosophila*, showed such a remarkable robustness against the limited scale of environmental variables in the lab that the original conceptualization of genes in the Norm-of-Reaction approach shifted soon to genes as the only marker of phenotypic variation (Collins, Gilbert, Laubichler, & Müller, 2007). Both research traditions discounted and dispensed with the environment, the former as the external niche of the organism and the latter as the internal cellular niche of the genes and their expression. This shows an unexpected parallelism to the so-called ‘environmentalist’ movement in psychology that emerged at the same time. By moving the study of animal behavior and learning from the field and mere observational approaches into a laboratory that allowed for rigorous testing and experimentation, the behaviorist tradition dispensed with both the variety of organisms—after all, it presupposed the generality of learning mechanisms—and their natural habitat in favor of uniform organisms and controlled (environmental) test conditions.

Recently, this exclusion of the natural environment from experimental studies in evolutionary, developmental and also behavioral studies has been criticized, most notably by calls for an ‘ecological developmental biology’ or ‘Eco-Devo’ (Gilbert, 2001) and West and King’s call for a ‘Developmental Ecology’ (West, 2003; West & King, this issue). These criticisms included concerns about the use of model organisms and their limited generalizability for the interpretation of research results (see Robert, this issue). Together with growing research into parental effects, these approaches have inspired a flood of new observations and experiments cementing the influential role of ecological context in development and evolution. West and King’s surprising results in their extended research of the development of cowbirds warn us how a neglect of the natural, social conditions of flock living can mislead us in our interpretation of the developmental causes of behavior. Maternal effect research has produced many examples of how environmental conditions in the parental generation can exert an influence on the development of many successive generations

(Gilbert, 2001; Mousseau & Fox, 1998; West et al., 1990; see references in West & King, this issue).

In summary, we can identify three reasons for the exclusion of development from the modern synthesis:

- (a) The misconception of development as the mere unfolding or maturation of the organism out of its genetic 'blueprint' or program;
- (b) The reduction of inheritance and transgenerational stability of traits to the transmission of genetic 'information', and
- (c) The neglect of the problem of evolutionary novelty, the so-called 'arrival of the fittest'. Taken together, these attitudes have not paid serious enough attention to the necessary and sufficient conditions for the process of adaptation by natural selection to take place: the origin of reliably reproducing developmental systems. In other words, evolutionary biology has hitherto failed to address the very possibility of evolution through the variability, adaptability and evolvability of phenotypes.

The last decade has seen the emergence of multiple approaches that focus on the active role of organisms and their development in evolution. These accounts are Developmental Systems Theory (Oyama et al., 2001b), Extended Inheritance (Jablonka & Lamb, 2005), evolutionary developmental biology (Evo-Devo) (Hall, 2000) and developmental evolution (Wagner, Chiu, & Laubichler, 2000), ecological developmental biology or Eco-Devo (Gilbert, 2001), phenotypic and developmental plasticity (Pigliucci, 2001; West-Eberhard, 2003), and Niche Construction (Odling-Smee, Laland, & Feldman, 2003). I believe that the concept of *developmental niche construction* has the power to integrate many if not most of the ideas laid down in the other approaches. The central idea behind developmental niche construction, and developmental systems theory, is the developmental system. It unifies many of the pressing questions and ideas mentioned above: the developmental system as the subject of evolution and their forces; the developmental system as the producer of evolutionary innovations; the interdependency and codetermination of the organism and its niche; the developmental system as the provider of many different interdependent channels of inheritance that reliably make available the necessary developmental resources for the reproduction of successive generations of developmental systems. Research in the process of developmental niche construction can elucidate three main evolutionary questions:

- (a) The *origin of a trait* by introducing new epigenetic resources for variation and innovation beyond mutation and recombination and describing how developmental processes situated in their ecological niche can produce novel phenotypes;
- (b) The *spread of a trait* by showing in detail how organisms or their parental generation co-construct a selective environment; and
- (c) The *maintenance of a trait* through processes of transgenerational stability of variation that extend the inheritance through the transmission of genetic

material with the reliable availability of necessary developmental resources through multiple mechanisms of reproduction or transmission.

We now have all necessary ingredients for a real postmodern or postgenomic synthesis of development, heredity, and evolution.

7. A Postgenomic Synthesis: An Epigenetic Understanding of Development

The ‘century of the gene’ (Keller, 2000) spawned a new and more sophisticated preformationism, with the homunculus as the preformed ‘form’ of the organism replaced by the ‘information’ to make an organism encoded in the genome. This modern consensus accepts the emergence of qualitative change in development, which it explains with the preformed inherited genetic program as a materialized vital force directing the epigenesis of the organism out of a seemingly homogenous mass. Hence, the new conception is rather a kind of ‘animistic’ predeterminism, where genes ‘program’ outcomes. True to the spirit of today’s interactionism, the mainstream ‘modern consensus’ can be “standardly construed as the epigenesis of something preformed in the DNA” (Robert, 2004, p. 34). Instead of avoiding the unscientific dangers of both preformation and vitalist epigenesis, however, it combines the shortcomings of these age-old ideas and rests ultimately on an unrealistic conception of genes and gene action.

I want to promote what others have called ‘probabilistic, contingent, or constitutive epigenesis’ in its place, a systems view that understands development as an epigenetic process of qualitative change based on the orderly emergence of novel behavioral traits during development without recourse to either an internal or external, pre-existing plan. Taking development seriously is demanded by its contingent nature due to the immense importance of experiential factors at all stages of development, via the environmental regulation of gene expression. This ranges from the chemically facilitated emergence of new behavior (like sucking in rats, see Alberts’ paper, this issue), to individual learning of new behaviors through various processes including trial and error and social learning (such as the emulation or real imitation of new solutions to problems, as shown by the tool use in chimp and crows), and includes ‘ultra-social’ learning through cultural participation, instruction, and formal schooling (as in the acquisition of language) (Gottlieb, 2001; Herrmann, Call, Hernandez-Lloreda, Hare, & Tomasello, 2007; Michel & Moore, 1995; Oyama et al., 2001b; Robert, 2004).

What a new account of development really has to accomplish is not just to go beyond these vexed dichotomies such as innate and learned, but to provide a framework that integrates a complex set of heterogeneous factors into a system of developmental resources all of which are reliably reproduced in succeeding generations of a developmental system but none of which really belong alone to either ‘gene’, ‘organism’, or ‘environment’ (the famous “Triple Helix” of Lewontin, 2000). Its contextualization of genes should obviate “even naïve temptations toward gene/environment dichotomies, and... will open up a very rich area of empirical

investigations to examination and conceptualization in developmental-system terms. . . . Ultimately, such a view should work towards “overcoming inner/outer dichotomies in favor of self-organizing, causally reciprocal systems of interaction” (Moss, 2001, p. 85). Developmental Systems Theory (DST), an alternative approach to the integration of evolution, development, and inheritance, provides just such a framework and its conception of development is basically the one promoted in this article (for a short introduction in its central tenets see Oyama, Griffiths, & Gray, 2001a).

The important systems features of such a view are the rejection of dichotomous description of behavior in favor of a full analysis in terms of continuing interaction between, and the joint determination by, heterogeneous developmental resources. Learning may be involved but only as part of an overall concept of experience which includes less obvious contributions, such as self-stimulation. An important part of such an analysis implies seeing behavior as belonging to the organism’s overall anatomical and physiological make-up. A dynamical systems view of locomotor development exemplifies such an approach very well by including the growth of muscles and the infant’s strength in an account of behavioral coordination of movement (e.g., Thelen, 1995). Other important features are the context sensitivity and developmental contingency of any factor, including genetic factors; the distributed control of development upon its heterogeneous resources, and the acknowledgement of the role played by the developmental system to control its further development; extending the idea of inheritance to include factors other than DNA, including factors formerly thought of as ‘environmental’ or ‘experiential’ if they are reliably reproduced or ‘passed on’ for succeeding generations; and last but not least the reconceptualization of development (and evolution) as the interactive construction in a thoroughly epigenetic account of development that “never sidesteps the task of explaining how a developmental outcome is produced” (Oyama et al., 2001a: 4).

Alleged explanatory categories of behavior such as ‘innate’, ‘acquired’, ‘programmed’, ‘hard-wired’, or ‘instinctive’ do not really explain the origin of the behavior. Worse, by their presumptuous nature they preclude further investigation into the real causes of the trait, which are never just genetic or environmental but are necessarily ‘epigenetic’ by nature. This broad conception of epigenesis is expressed succinctly by Eva Jablonka:

Epigenetics . . . focuses on the general organizational principles of developmental systems, on the phenotypic accommodation processes underlying plasticity and canalization, on differentiation and cellular heredity, on learning and memory mechanisms. Epigenetics includes the study of the transmission of subsequent generations of developmentally-derived differences between individuals, thereby acknowledging the developmental aspect of heredity” (Jablonka, personal communication, cited in Gottlieb, 2001)

The last decade has witnessed enormous scientific advances in genomics, systems biology, social neuroscience, evolutionary, and ecological and developmental biology (‘evo-devo’, ‘eco-devo’, phenotypic plasticity, niche construction, extragenetic

inheritance, developmental systems theory). They challenge over gene-centered/predeterministic and environmentalist explanations of behavior. Nature and nurture do not interact as if they were separate entities, with nature as the *a priori* plan being separated from concrete living and nurture being the means for modifying nature's plan through experience. Every trait develops out of the nonlinear interactions among a range of very diverse developmental resources that cannot be usefully divided into genetic and nongenetic resources. It starts with the environmental regulation of gene expression, continues over a range of experiences beneath the skin and above the gene, through stages of sensory and social learning in vertebrates, to the exquisitely sensitive learning capacities of the human brain. 'Nurture' is this ongoing process of development, while 'nature' is the natural outcome of the organism-environment-system (Oyama, 1999).

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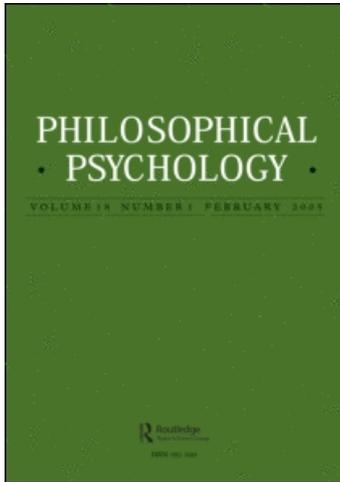
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Deconstructing Innate Illusions: Reflections on Nature-Nurture-Niche From an Unlikely Source

Meredith J. West and Andrew P. King

Despite great advances in understanding genetic mechanisms, there still exists a bias toward equating genes with innate modules that determine important developmental events. But genes are equally relevant to understanding developmental plasticity shaped by ecological events. In other words, the term ‘genetic inheritance’ does not specify ontogenetic mechanisms. Here we present a case history of a species assumed to be under the control of prespecified genetic wiring to direct critical behavioral events such as communication and mating. We show, however, that exogenetic processes stemming from the species’ ontogenetic niche provide an alternative view of the flexibility of development especially with respect to behavioral performance.

Keywords: Brown-Headed Cowbirds; Communication; Development; Learning; Nature–Nurture; Ontogenetic Niche

1. Goals and Background

The focus here is on exogenetic inheritance: the idea that organisms inherit parent’s genes but also parents, peers, and places they inhabit (West & King, 1987). This is not a new idea—in fact, it is a very simple and old idea but it does not seem to be able to compete for relevance on a level ground with ideas about genetic inheritance. In this essay we will connect the nature–nurture concept to exogenetic inheritance, as it is relevant to present day research on the development of behavior. We will take a case history approach by discussing brown-headed cowbirds, a brood parasite with a long history of eliciting developmental questions about species and mate recognition

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(Lehrman, 1974; Mayr, 1974). We examine these same questions and show how the idea of an ontogenetic niche provides answers to the above questions while the concept of an assumed innate mate recognition system provides a widely popular but empty conceptual framework.

At a behavioral level, the ontogenetic niche is the set of ecological and social circumstances inherited by individuals. Exogenetic heredity can be highly reliable, probable and stable over generations, e.g., inheriting conspecifics is as dependable as inheriting genes. Sociality is an engine of exogenetic mechanisms such as niche creation and cultural transmission, which can adapt rapidly to changing ecological circumstances, a critical attribute of successful species. The best way to see these effects is to look at the life history of a species where development and origins of reproductive behavior are well understood. In fact, too often critical behaviors like reproduction are not studied developmentally and consequently the function and origins of behaviors are misunderstood which we believe contributes to all the common habit of relying on innate explanations (West, King, & Arberg, 1988; West, King, & White, 2003).

2. The Cowbird: A Wired Species?

The species we chose to study would seem to be a natural for innate explanations. Cowbirds (*Molothrus ater*) are brood parasites, which means that the young ones are raised by a foster species and have no reliable early contact with their parents (Friedmann, 1929). Thus, the young cowbird faces a chance of learning the “wrong” species typical behaviors leading to lower reproductive fitness. Evolutionary theorists have argued that cowbirds must be a hard-wired species immune to postnatal experience to avoid mis-mating. Even strong proponents of nurture have suggested that the cowbird would need an innate safety net to prevent mating errors (Lehrman, 1974; Mayr, 1974). The origin of these views followed the emotionally charged era post-WWII about innate and learned origins of behavior, e.g., Lehrman vs. Lorenz debate. But the argument was not just an esoteric science debate but was seen as having impact on public policy. The nature–nurture debate continued with the rise in the model of early experience and sensitive periods: an extreme example at the time was how to “inoculate” the young against effects of early social deprivation (e.g., ‘Head Start’). At the same time, the emerging study of bird song was then seen as an exemplar of an interactionist view between nature and nurture. The thinking was extended to many areas including a growing interest in language origins and the comparative role of innate modules vs. learning. Against this backdrop we started our investigation of the North American cowbird.

3. Assumptions and Methods

Our study of the development of song and mate recognition was rooted in the assumption that sociality was a basic mechanism for development. We viewed studies

that documented song development in nonsocial contexts as uninterpretable with respect to communicative development. Small cages or sound attenuating chambers were the typical laboratory environment but they structured, restricted, or eliminated social interactions. Such isolate housing and the reliance on isolate song supported the dominant view of complete social isolation as being capable of revealing the innate blueprint (Searcy & Marler, 1987). But, in all of our studies, housing conditions were a critical dimension, and while we housed birds in different social contexts from sound attenuation chambers to flight cages to large aviaries, we never housed birds alone. The first studies were done using a pair or triad of young cowbird males or females or with different companions including nonconspecifics. During the 9-month period a male takes to develop his song, we recorded vocal development as well as the social interactions with his companions who could be other males or females of different ages or nonconspecifics. In the spring, we would record the final vocal repertoire of the male and measure the female perception and reaction to song. For females, comparable developmental studies looked at the effects of vocal and social companions on the development of her preferences.

4. Species Identification

Our first study of the role of early experience in the development of reproductive behavior in cowbirds asked whether hand-raised males and females would develop typical songs and preferences without conspecific experience. Thus, during their first year we raised males and females from the egg with nonconspecifics. In the spring, we played back the males' song to the females. We discovered that while the males' songs were in many cases atypical for the species, these songs, when played back to the females, caused them to adopt a copulatory solicitation postures (King & West, 1977) (Figure 1). Thus, the naive females, when they were about



Figure 1. Female copulatory response to song.

1-year-old and the first time in their life they heard cowbird song, revealed the songs' functional properties. It seemed we had discovered the perfect innate safety net where mate recognition did not require conspecific experience. We called the playback procedure as the female bioassay and used it to measure male song quality. In addition, many developmental studies went beyond just measuring song quality by female preferences for song by playback, we also measured actual courtship and reproductive success in breeding aviaries and correlated performance across the two contexts (West, King, & Eastzer, 1981).

In light of the discovery of a lock and key reflex for species and mate recognition, we turned our attention to the role that experience might play in macrogeographic variation of song across the three subspecies of cowbirds (Friedmann, 1929). We were especially interested in the subspecies borders where we suspected song learning might play a role in incipient speciation (King & West, 1990). Because the cowbird's range extends throughout all of North America and is classified as three distinct subspecies, this permitted us to look for experiential differences against different genetic backgrounds. The female bioassay of male song took on great importance because it allowed us to do microphyletic comparative studies. It also allowed us to use our own experiential probes to examine behavioral plasticity. We found a range of phenotypes many of which could be culturally transmitted.

5. Investigations of Communicative Plasticity

We carried out studies to investigate the developmental origins of male vocal plasticity. We raised juvenile *Molothrus a. ater* males from North Carolina with adult *M. a. obscurus* males and females from Texas. The *M. ater* males became bilingual adding lexical elements to their songs that were not produced by their natal population. We recorded the breeding season songs of the bilingual males and played them back to *M. ater* and *M. obscurus* females to measure their song preferences by the frequency of copulatory solicitation postures. We found that these males were functionally bilingual, as the playback females preferred the song variants from their natal population. We also found that in mating tests that females copulated more with males that were singing more of the females' local song variant. Thus, male vocal development was highly flexible at the macrogeographic level (West & King, 1985; West, King, & Harrocks, 1983).

After showing that juvenile males could become bilingual, we asked if adult females from the *M. ater* and *M. obscurus* populations could also modify their song preferences at the macrogeographic level. To do this, we housed adult males from NC with adult TX females and adult males from TX with adult NC females. We discovered that the nonsinging females did not change their preferences for their natal song but that the males they were housed with modified their song structures to try to appeal to the distant population females' song preference. Because the females did not sing, this finding was a complete surprise. At the time, current

theories of song learning were based on song copying and nonvocal social stimulation was not believed to play a role in vocal development (King & West, 1988; King, West, & Eastzer, 1980). We followed this study by raising juvenile males from NC with adult females from TX or local females from NC. We videotaped their social interactions during development to try to account for the females' influence on male song.

We discovered that the females used social cues in reaction to male song to shape the males' vocal behavior (West & King, 1988). While the female can use a variety of social cues, we were led to one particular female behavior by the males' excited reaction and approach to female movement during his singing. Specifically, we discovered that to some songs females would rapidly move a wing, a movement that appeared to be a precursor to a female copulatory solicitation posture. We called this behavior a 'wing stroke' and found that the songs that a female wing stroked to were likely to be repeated and retained in the males' repertoire. We also played back songs that elicited wing strokes, and those that did not, to a group of females and found that the songs that elicited wing strokes were the most effective elicitor of the copulatory posture. These data were important because they implicated nonimitative social stimulation in song learning for the first time (King, West, & Goldstein, 2005; Smith, King, & West, 2000).

We proposed that song learning was a matter of both copying songs from other males as well as of contingency learning based on social feedback from females. To begin the process, males produced a generic alphabet of sounds in their babbling or subsong. They elected to keep sounds based on the nature of social feedback from both males and females. Thus, during development young males produce original songs that are a consequence of vocal experimentation and their consequences as well as copies of songs they have heard. The choices of copied songs that will be retained are a consequence of the social feedback they elicit. Thus, the male song development system was open to both social and vocal stimulation at the macrogeographic level.

The next logical question with respect to male vocal plasticity was: what was the limit of this plasticity. Thus, we asked whether males housed with conspecific or nonconspecifics would be open to learning at the species level. To answer this question, we housed wild caught juvenile cowbird males with canaries, or as a control condition, with adult female cowbirds during the males' first year. We found that the males interacted with their companions by singing to them. In the case of the canary-housed cowbirds, they sang along with their companion. By spring of their first year, the males had incorporated elements of the canary trills into their crystallized songs. By contrast, the males housed with female cowbirds developed their songs faster and crystallized sooner (King, West, & Freeberg, 1996).

6. Investigations of Communicative Pragmatics

At the beginning of the breeding season, we carried out two tests of species recognition. First, we housed all of the canary-housed males in one large flight cage

and all of the female-housed males in another identical cage. After the birds had habituated to their surroundings, in a choice test, we introduced them, one at a time, into a neutral flight cage that contained both unfamiliar females and canaries. We measured approach, spatial proximity, and singing in the new surroundings. The female-housed birds did what we expected: they ignored the canaries and sang to the females who showed an interest in their overtures by allowing the males to approach and sing to them. By contrast, when the canary-housed males were introduced to the choice test, their response was a surprise as they ignored the female cowbirds and approached and sang to the canaries. These new canaries were not accustomed to being courted by cowbirds and so they uniformly retreated when males sang to them. This created a situation where the male cowbirds were constantly pursuing the canaries around the cage and trying to sing to them. While this was going on, the female cowbirds who were also present as part of the choice test were completely ignored by the males to the point where a female cowbird would occasionally adopt a copulatory solicitation posture to the song of a male but the male appeared not to notice. We found this to be an extraordinary finding because it seemed obvious that if there was any merit to the idea of innate mate recognition, the males' prior social experiences should have no influence on his ability to recognize and mate with a solicitous conspecific female (West, King, & Freeberg, 1996).

We carried out a second test of mate recognition in a socially more complex setting. For this test we used large outdoor aviaries that housed adult male and female cowbirds from local and distant populations along with canaries and with adult and juvenile starlings. We introduced the canary and female-housed males in small groups to these flocks and measured social interactions between the males and females. We found the female-housed males did not show the necessary social skills to consistently interest female cowbirds, preferring to sing to one another or undirected song. We had recorded the male's songs and played them back to an independent group of females and found that the female-housed males' songs were higher quality song than the canary-housed songs. This was not surprising in that the canary-housed males incorporated canary elements in their song. The high-quality song of the female-housed males explains why they did occasionally get a female to adopt a copulatory solicitation posture. However, as we had seen in the cage setting, the males generally ignored the female overtures. The males appeared to have failed to learn that courtship requires persistence and sustained attentional focus. After this test revealed male inexperience, we introduced experienced adult males as models to see if the young males could quickly learn to court by observing adults. The introduction of the adult males produced no change in the juvenile male behavior (Freeberg, King, & West, 1995; West, King & Freeberg, 1996).

We also introduced the canary-housed males to these flocks using the same procedures. As in the previous test, the canary-housed males continued to pursue and sing to the canaries. The canaries showed no reciprocal interest spending most of their time flying away from the males. Interestingly, the canary-housed males showed persistence in their pursuit of the canaries that the female-housed males did not show

in their actions toward female cowbirds. We believe this is explained by the fact that during development the canary-housed males had to work harder to sing at a close distance to their canary companions than did the female-housed males and so in the mating competence test were accustomed to having to chase and court. In that all of the cowbirds in this study were wild caught at around 50 days, we know that early experience with conspecifics did not guarantee the appropriate outcome. Taken as a whole it was clear that for male cowbirds, there was no hardwired system that insured mate recognition even at the species level. Thus, the earlier discovery that the males could produce a functionally effective song without conspecific experience was clearly not the end of the story as it was now apparent that the males needed social experience to know how to use their songs. Evolution had apparently selected a developmental system for the males that was heavily dependent on a socially rich ontogenetic niche. But, what about female cowbirds? Perhaps the innate safety net resides in the female, as they are the gatekeepers of reproductive outcome.

7. Female Song Perception

We had looked at female cowbird song preferences a decade before the aviary experiment described above. We had used triads of females housed together or with males from local and distant populations in sound chambers; we examined whether we could modify female preferences for male song. We used the female bioassay and actual mating tests to look for evidence of modifiability at both the micro- and macrogeographic levels. In all of these studies, we found that females preferred the songs of their natal population. In fact, the lack of evidence of female modifiability was striking (King & West, 1983). For example, even when females were housed with the same male for many months, in playback tests, they never showed any preference or aversion to the song of their companion when compared to unfamiliar males. Finally, we hybridized North Carolina *M. ater* females with Texas *M. obscurus* males. Many of the young ones died which seemed to suggest a genetic incompatibility, but one hybrid female survived and we raised her with other females in the absence of male contact. In the spring, we played back *M. ater* and *M. obscurus* song to her and she preferred *M. ater* song, her mother's natal preference, by a 3:1 margin. So, the evidence seemed overwhelming at the time that evolution had selected females to be the gatekeepers of reproduction and given them a genetic template to guide their perception (West, King, & Freeberg, 1997). This seemed to make evolutionary sense: if females retained their preferences, males interacting with them would be socially shaped toward effective song.

In sum, the ontogenetic niche for males and females is highly plastic with respect to mate recognition for the males while females appeared to be exemplars of the innate modules thought to be necessary for a brood parasite. The scale of the male incompetence was in two parts: song structure (e.g., learned canary song) and song use (e.g., what, when and to whom to sing). The incompetence seen in aviary-flock setting was not seen in the flight cage, so we were missing something by ignoring the

most social aspect of their niche, flock living. So the question becomes how do cowbirds learn when pragmatically challenged in more realistic ecologies?

8. Learning and the Ontogenetic Niche

Most work on social learning, however, occurs in labs where animals are individually housed and passively exposed to selected stimulation. By creating flocks that allow choices, the birds select stimulation and thereby opportunities to learn. As an example, a young male songbird may not have access to hearing adult song because adults do not sing when juveniles are close by and do not affiliate with juveniles. What do birds learn when the flock controls what is available to be learned? Said another way, what information is bio-available and how does an animal get access to it? Public information could be something so simple as degree of sex or age assortment, i.e., the presence of 'flock signatures.' Private information is embedded such that information must go through a social 'gateway', e.g., female wing strokes hinge on males singing to her first but that depends on his interest and ability to attend to her prior to singing. To give an example of a social gateway, male cowbirds in conventional restricted housing learn the songs they hear whether they are exposed through tape or live tutors. But what happens in flocks? We studied flocks of 20–25 birds in two adjacent aviaries separated only by a wire wall. In these aviaries, the birds could see and hear individuals in the other aviary as readily as in their own. To our surprise, we found no song sharing or social influences across aviaries in the 16 flocks we studied in this way (King, White, & West, 2003a; White, Gros-Louis, King, Papakhian, & West, 2007; White, King, Cole, & West, 2002). Thus, seeing and hearing did not define psychological borders, but social interaction did. This finding demonstrated the need to consider bioavailability as opposed to simple exposure to stimulation. The finding also showed that more is not always better as males copied less, and were more selective, in the flock setting than in restricted housing. The distinction is between what an animal has the capacity to do as opposed to how social/perceptual systems function to gate what is available to be learned. Restricted housing does not activate the systems responsible for ecologically relevant learning.

9. Experiential Knock Outs in Context

This finding led to a series of studies that applied principles of neo-phenogenesis to create 'experiential' knock outs (EKO's), a phenotype whose early social environment has been biased based on knowledge of natural ecological variation. We used EKO's to understand the role of social structure of a flock to guide learning. For example, we knocked out the presence of adult males in flocks composed of juvenile males and both classes (juvenile & adult) of females. We discovered atypical fall, winter, and spring behavior: few male–male song exchanges, no male affiliation with other males or females. The EKO's became self-imposed social isolates that sang frequently but in an undirected manner. The single largest effect in the first year is that they avoided

aggressive encounters apparently a consequence of their social isolation within the flock. During mating they showed little courtship or mate guarding, no fighting, no song exchanges with other males, and no genetic or social monogamy (Gros-Louis, White, King, & West, 2006; White, King, & West, 2002a). As we followed these birds beyond their first year, the lack of aggression proved to be a stable phenotype that persists for years which shows the robustness of ecologically relevant social learning. Thus, juvenile males are dependent on social interaction with adult males and females for social competence. The nature of flock structure becomes the safety net to guide male learning as it defines what is bioavailable (West, King & White, 2003).

We also looked at the role of flock structure with respect to female perception of song. We had found no evidence of female preference modifiability in our previous work. All of our studies on female perception had been done in restricted settings, a context we now saw asking a question about such a narrow dimension of behavior so as to limit inferences about basic developmental systems. Specifically, if bioavailability is shaping male development, the same might be true for females? To test this idea, we studied females in flocks where they could exercise more control over their exposure to male behavior. The first question we asked was whether female song preferences could be influenced by social context. We created *no male* EKO juvenile and adult females, who were housed together without males in a large aviary for 8 months prior to the breeding season. The females could see and hear wild males outside of their aviary. Wild males could land on the aviary and sing to individual females but the females could strictly regulate male access by moving away. In the spring, using the female bioassay we tested their preferences for local or distant song from two other subspecies of cowbirds. We discovered that we had erased the females' natal song preferences! This was completely contrary to what we had found in restricted housing. Erasing natal preferences in adult, wild caught females, may well be the first such demonstration of such malleability in song birds. This was an especially compelling finding in which some of the distant song variants were lexically very different from the natal song of their population. Thus, while males housed with canaries could incorporate elements of canary song the females obviously has a similar plasticity when studied in ecologically plausible settings (West, White, & King, 2003).

We then went on to ask the question whether we could build in a preference for certain song variants when females were in flocks, i.e., could we see constructive effects? To this end just prior to breeding, we both tape and live tutored flock females with specific natal songs and found that both types of exposure led to stable preferences for specific song types. Thus, females are able to re-set song preferences every year. The obvious difference between the flock setting and the restricted housing studies was that in the flocks many more females were in social contact with one another. In restricted housing, we frequently used pairs or triads of females with a single male or tape tutored. The evidence suggests that females observe other female reactions to song (e.g., wing strokes) and that the social dynamics among the females unlocks learning. Thus, as is the case with the males, it appears that the social structure is stimulating female learning for different song variants but also acting

as a safety net to constrain the underlying plasticity of potential preferences (King, West, & White, 2003; West, King, White, Gros-Louis, & Freed-Brown, 2006).

10. Pragmatic Learning in a Flock

Changing to a dynamic setting as in the flock manipulations revealed the importance of an animal's ability to engage a social context to obtain relevant information, information not available in restricted settings. Several examples illustrate this point. In restricted housing, when a male sings to a female she cannot leave, the best she can do is to move away a short distance but this still allows the male to sing to her. The male in this situation does not have to learn to be strategic in his approach or his attention to a female. In the flock setting, if the male simply runs up to a female to sing she is likely to leave before he ever has a chance to sing. In the flock, the approaching male must attend to subtle female social signals and modulate his approach in accordance with what she will permit without departing. This male is then learning social pragmatics of the how, when and where the song should be used. He is also learning to sustain his attentional focus to be able to pick up in the female signals. The same general principles apply when singing to other males who may react aggressively if their social skills are low. We know from examining the neural structures of males housed with interactive vs. noninteractive females during their first year that the males with socially interactive females develop a larger neural volume of nucleus rotundus in the visual thalamus than males with less interactive females. The nucleus rotundus is thought to mediate the perception of form and motion. Thus, during development the nature of the social environment is literally shaping the neural ability of the brain to process information. This is important because it shows that the neural development is also dependent of exogenetic influences (Freeberg, West, King, Duncan, & Sengelaub, 2002).

The story is the same for females. Restricted housing is generally justified as revealing basic innate capacity without distracting influences. For female cowbirds restricted housing reveals innate artifacts without illumination actual developmental pathways. Specifically, females need to be motivated by being part of a larger female social group and be sensitive to the social reactions of that group to modify their evaluation of male behavior. For example, in an all female flock composed of both juvenile and adults the presence of male song will trigger, in real time, rapid segregation of the flock by age (Gros-Louis, White, King, & West, 2003). The segregation will dissipate in minutes if no song is present. The effect can be produced with the presence of a singing male or by playback of song. By contrast, the presence of nonsinging males produces no segregation. During the period when a group of females are reacting to song in this way some females will wing stroke and the wing stroking elicits the attention of other females. Thus, it appears that the collective social context is the key to stimulating female preference learning. The collective social context necessary for this learning is not available in simpler housing in pairs or triads.

11. The Safety Net

For this parasitic species, it is clear that evolution has trusted an exogenetic system to transmit information vital to reproduction from one generation to the next. The 'safety net' is the social structure of the flock. The ontogenetic niche can be considered at several levels, but at the most basic level, an individual's niche is defined by his or her status or position within the flock. This position defines what is available to be culturally transmitted or learned throughout the lifespan. Consider the malleability of adult female song preferences at the macrogeographic level and the fact that the male's song is for the female. Female sociality/song preferences are the major determiners of flock organization and the goal of male behavior is to meet those preferences (Freed-Brown, King, Miller, & West, 2006; King, White West, 2003). This developmental system is designed to be open as ecologically possible and thus immediately sensitive to ecological change. This system is the antithesis of a closed developmental system. Is this system unique because of the parasitic habit? The answer is that we do not know because comparable experimental studies of non-parasitic songbirds have not been carried out.

Thus theoretically, to most people, this little brown bird occupies the status of the annoying gadfly. The cowbirds' rules of sociality are stinging rejections of accepted beliefs about songbird development by virtue of their use of nonauditory vocal learning and the ability of males and females to communicate about communicating. The cowbird findings also fly in the face of accepted rules about the role of early experience. We see it as a model for the dynamics of developmental growth where function alters function, within and across generations.

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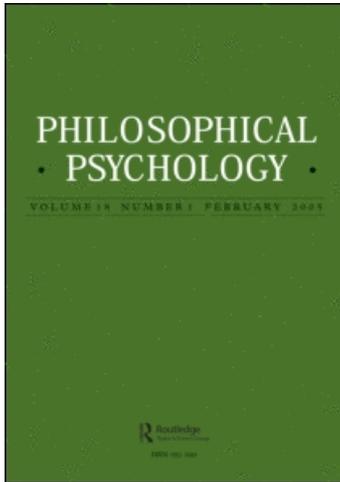
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Innateness, Canalization, and 'Biologizing the Mind'

Paul E. Griffiths and Edouard Machery

This article examines and rejects the claim that 'innateness is canalization'. Waddington's concept of canalization is distinguished from the narrower concept of environmental canalization with which it is often confused. Evidence is presented that the concept of environmental canalization is not an accurate analysis of the existing concept of innateness. The strategy of 'biologizing the mind' by treating psychological or behavioral traits as if they were environmentally canalized physiological traits is criticized using data from developmental psychobiology. It is concluded that identifying innateness with environmental canalization can only result in adding unhelpful associations from 'folkbiology' to the relatively precise idea of canalization.

Keywords: Canalization; Folk Concepts; Innateness

1. Is Innateness Canalization?

Ariew has published an influential series of papers (1996, 1999, 2006) arguing that the concept of an innate trait can be explicated using the idea of 'developmental canalization'. This concept was introduced by the influential mid-twentieth century embryologist and theoretical biologist Waddington (1940, 1942, 1957, 1959). Waddington's developmental canalization was part of a broader vision of how an organism develops from the fertilized egg. The entire collection of genes and their interactions makes up a 'developmental system' (Waddington, 1952) that produces a phenotype. Many features of the phenotype are explained by the dynamical properties of that developmental system as a whole, rather than by the influence of one or a few specific alleles. Thus, for example, Waddington sought to explain one

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of the major biological discoveries of his day—the fact that extreme phenotypic uniformity can be observed in many wild populations despite extensive genetic variation in those same populations—by appealing to the global dynamics of developmental systems. A genetically canalized developmental system takes development to the same endpoint from many different genetic starting points. The development of wild-type phenotypes can thus be buffered against genetic variation. Waddington represented this idea with his famous ‘developmental landscape’ (Figure 1).

The ‘developmental landscape’ is a representation of development as a system whose parameters are genetic loci and whose state space is a set of phenotypic states. The state space is depicted as a surface, each point of which represents a phenotype.

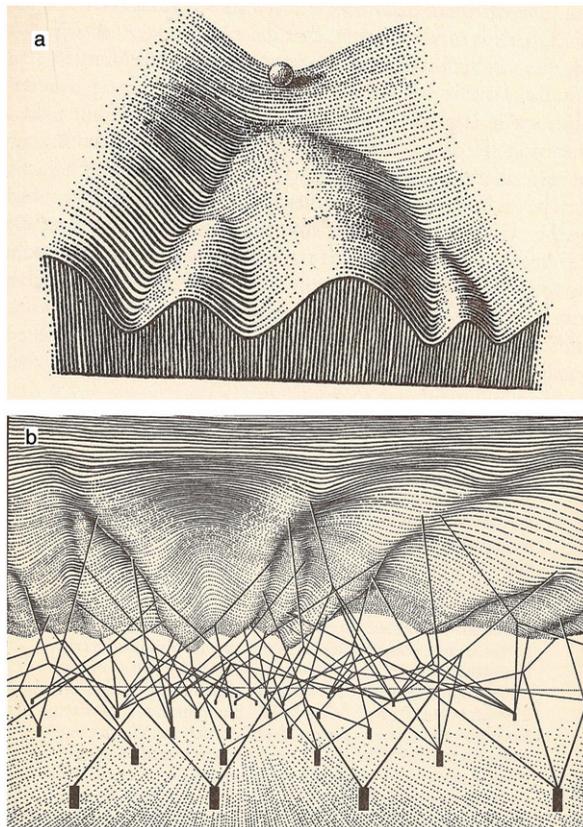


Figure 1. Waddington’s ‘developmental landscape’. (a) The developmental trajectory of the organism, represented by the rolling ball, is determined by a landscape representing the developmental dynamics of the organism. (b) The shape of this landscape is determined by genes, represented as pegs pulling the landscape into shape via strings, and by epistatic interactions between genes, represented by connections between the strings. From Waddington (1957, p. 36).

The genetic parameters are depicted as pegs that pull on the surface and thus determine its contours. Epistatic interactions between genetic loci are represented by links between the strings by which those loci pull on the surface. The development of the organism is represented by the trajectory over the surface of a ball which passes through a series of phenotypic states as it rolls 'downhill' from conception to death. Waddington intended this diagram to make vivid the idea that the effect of a change at one genetic locus depends upon the states of all the other genetic loci, since it is all the loci together which determine the shape of the landscape. Some genetic changes, such as those which affect the tops of inaccessible 'hills,' will have no effect on development. Other changes of the same intrinsic genomic magnitude which affect the entrance of a valley or 'canal' will have a massive effect on development. The phenotypic impact of a genetic change is not proportional to the magnitude of the genomic change, but depends on the structure of the developmental system. Furthermore, the phenotypic difference produced by a genetic difference is not explained by that genetic difference in itself, but by how that change interacts with the rest of the developmental system. This picture retains considerable validity in the light of contemporary developmental genetics.

In various places, Ariew seems to identify canalization with insensitivity to the environment: "Waddington called the process of buffering against environmental cues 'canalization'" (Ariew, 2006, p. 10). But this is only part of what Waddington meant by "canalization", as, we believe, Ariew actually understands. It is clear from Waddington's presentation of the 'developmental landscape' that 'canalization' refers to buffering against both genetic and environmental parameters. Indeed, in many places Waddington's primary concern is with buffering against genetic variation. But if we suppose that some of the 'pegs' in the model are environmental factors, rather than genetic loci, then we can indeed define separate notions of 'environmental canalization' and 'genetic canalization'. A phenotypic outcome is environmentally canalized if those features of the surface which direct development to that endpoint are relatively insensitive to the manipulation of the environmental parameters. A phenotypic outcome is genetically canalized if those features of the surface which direct development to that endpoint are relatively insensitive to the manipulation of the genetic parameters. Ariew proposes to identify innateness with *environmental* canalization. Innateness-as-canalization is a matter of degree. A trait is more innate if the more environmental parameters its development is buffered against and the wider the range of variation in those parameters against which it is buffered.

2. Canalization is a Poor Conceptual Analysis of Innateness

One can interpret Ariew's characterization of innateness as canalization in two distinct ways. Ariew could be proposing an analysis of the concept of innateness or he could be proposing to identify innateness with environmental canalization because this identification provides the basis for a promising scientific

research strategy. In this section, we focus on the first interpretation of Ariew's project. We argue that Ariew's characterization of innateness is not an adequate conceptual analysis. In the remainder of this article, we turn to the second interpretation of Ariew's project.

So, could Ariew's characterization of innateness as canalization be a satisfying conceptual analysis of the concept of innateness? An example shows the answer to be 'no'. Consider the penile reflexes of the rat. Celia Moore has shown that the spinal cord nuclei of male rats differ from those of female rats in ways that allow the male to use his penis during copulation (Moore, 1984, 1992). These neural differences result from differences in gene expression in the developing spinal cord of the rat pup, which in turn result from differences in the amount of licking of the genital area by the mother, which in turn results from greater expression in male pups of a chemical that elicits maternal licking. According to Ariew's (2006) characterization of innateness as canalization, these experiments show that the rat's ability to copulate is not innate:

Distinguish between two reasons why the trait appears invariantly in an environmental range: the first, because an environmental condition is developmentally required yet is found everywhere the system develops; the second, because the system develops independently of the environmental condition. Innateness should be identified with the second sort of invariance, not the first. (p. 10)

The rat's ability to copulate depends on an environmental condition (maternal licking) that is found everywhere the system develops and so, according to Ariew, it is not innate. But intuitively the rat's ability to copulate is innate. It constitutes a counterexample to Ariew's proposal, understood as a conceptual analysis of the concept of innateness. To put the same point differently, the example shows that environmental canalization is not a necessary and sufficient condition for something to seem intuitively innate.

Why does this example strike us as being innate? A companion paper, written in collaboration with Stefan Linquist, suggests an explanation (Griffiths, Machery, & Linquist, submitted). In this article, we used experimental methods to describe systematically the vernacular concept of innateness. We hypothesized that when people folk decide whether a trait (a behavior, an organ, a psychological disposition, etc.) is innate, they focus on the three following properties of this trait:

1. Fixity: the trait is hard to change; its development is insensitive to environmental inputs in development; its development appears goal-directed, or resistant to perturbation.
2. Typicality: the trait is part of what it is to be an organism *of that kind*; every individual has it, or every individual that is not malformed, or every individual of a certain age, sex, or other natural subcategory.
3. Teleology: this is how the organism is *meant* to develop; to lack the innate trait is to be malformed; environments that disrupt the development of this trait are themselves abnormal.

Furthermore, we hypothesized that these three properties influence people's decision about the innateness of a given trait in an additive manner. That is, the typicality, fixity, and teleology of a given trait should increase people's likelihood of judging that this trait is innate independently of each other.

To test this empirical hypothesis, we developed eight vignettes describing the song of eight different bird species (see Griffiths, Machery and Linquist, submitted). These eight vignettes systematically varied whether the trait was species-typical, whether its development was dependent on the environment, and whether it had a biological purpose. For instance, one of the eight vignettes was the following:

Birdsong is one of the most intensively studied aspects of animal behavior. Since the 1950s scientists have used recordings and sound spectrograms to uncover the structure and function of birdsong. Neuroscientists have investigated in great detail the areas of the brain that allow birds to develop and produce their songs.

Other scientists have done ecological fieldwork to study what role song plays in the lives of different birds.

The Alder Flycatcher (*Empidonax alnorum*) is a migratory neo-tropical bird which breeds in southern Canada and the northern USA. Studies on the Alder Flycatcher show that the song an adult male produces does not depend on which songs they hear when they are young. Studies also show that different males in this species sing different songs. Furthermore, close observations of these birds reveal that the males' song attracts mates and helps to defend their territory. Scientists therefore agree that the bird's song has a real function, like the heart in humans.

On a 7-point scale, 1 meaning strongly disagree and 7 meaning strongly agree, how would you respond to the following statement?

"The song of the male Alder Flycatcher is innate."

Subjects were each given one of these vignettes and were asked to judge on a 7-point scale whether bird song was innate for the species described in the vignette. Subjects' answers were then statistically analyzed to determine whether typicality, fixity, and teleology additively influence their judgment about innateness, as we hypothesized.

Consistent with our hypothesis, we found that typicality and fixity significantly predicted the folk's judgments about innateness and that their influence on people's judgments was additive. In addition, teleology was a marginally significant predictor of these judgments. We interpret our experimental findings as showing that folk judgments about the innateness of a trait are independently influenced by at least two properties of the trait: its typicality, its fixity, and possibly its teleology. We plan to replicate and extend these findings in future research.

Our interpretation of these results explains why the rat's ability to copulate strikes us as innate and why Ariew's characterization of innateness as canalization cannot be an adequate conceptual analysis of the concept of innateness. Let us consider these two points in turn. We judge the rat's ability to copulate to be innate because it is both species-typical and has an evolved purpose. Although the development of this

ability depends on very specific (and unexpected on *a priori* grounds) environmental stimuli, its typicality and its teleology are sufficient to tip us to judge that it is innate. Furthermore, because intuitive judgments about the innateness of a trait are influenced by whether a trait is typical, fixed, and teleological, any putative conceptual analysis that identifies innateness with only one of these three properties will fall prey of intuitive counterexamples (for a more detailed version of this argument see Griffiths, Machery and Linquist, submitted). For instance, consider a proposal to analyze innateness as species-typicality. Because judgments about innateness are influenced by typicality, fixity, and teleology, people should be willing to judge some traits which are not typical but which are fixed and functional to be innate. These judgments would constitute counterexamples to the proposed conceptual analysis of innateness. Understood as a conceptual analysis, Ariew's characterization of innateness as canalization fails for exactly this reason. It picks out one of the three properties—namely fixity—that influences folk judgments about innateness. As a result, it falls prey of intuitive counterexamples that make salient two other properties influencing folk judgments about innateness—namely typicality and teleology.

3. Biologising the Mind

Ariew's most recent presentation of innateness-as-canalization states clearly that it is not intended as an analysis of the concept of innateness, but instead is a way to embody and recommend a particular scientific research strategy (Ariew, 2006). The strategy is that of modeling the development of a psychological trait on the development of some paradigmatically 'biological' trait such as a limb or an internal organ. Ariew argues that the distinctive feature of the development of prototypically biological traits is their independence of the environment, so that using 'innate' to mean 'environmentally canalized' as he originally proposed embodies this research strategy. He calls the strategy "biologising the mind" and cites the linguist Noam Chomsky and the philosopher and cognitive scientist Jerry Fodor as examples of scientists who have used it (Ariew, 2006, p. 1). His analysis should be accepted, he argues, not because it captures the pre-scientific meaning of 'innate', but because the research strategy it embodies has been a successful one. In the same vein, Mohammed Ali Khalidi has recently defended his own, very different, analysis of innateness with the suggestion that it is "presumptuous for philosophers to advocate rejecting a theoretical concept that continues to play an important role in some area of science" (Khalidi, 2007, p. 93). Our response is that far from facilitating the study of psychological development the innateness concept actually obstructs it. In saying this we are not presumptuous philosophers flying in the face of scientific success, but instead are drawing the attention of fellow philosophers such as Ariew and Khalidi to a long and substantial tradition of scientific criticism of the research strategy they have identified.¹ The analysis should be rejected

because it embodies a research strategy that has been subjected to a powerful scientific critique.

According to Ariew, the term 'innate' should be used to mean something like 'study the development of this trait on the model of legs and livers', with the understanding that this means the trait develops independently of many of the details of the environment. Ariew accepts that a full causal understanding of development will not reveal any clear distinction between the 'innate' and the 'acquired', but argues that it is useful to have this blunt, high-level classification to direct research. A similar view of innateness has long been defended by the eminent student of birdsong development (Peter Marler):

[in comparing the calls of song sparrows and swamp sparrows] we find ourselves confirming what Lehrman (1953) asserted 50 years ago, that in the long run classifying behavior as 'learned' or 'innate' is not conceptually productive; however, it is not clear whether this is worth getting upset about. The habit of labeling behaviors in this fashion is so deep-rooted that we will probably never succeed in eradicating it. And it does have some value in placing behaviors on a lability continuum, with some behaviors more 'nurture dependent,' more changeable and variable, and others more 'nature dependent,' more stereotyped and resistant to change. (Marler, 2004, p. 31)

It is therefore unsurprising that Ariew's main example comes from Sober's (1998) discussion of innateness, which in turn derives from the work of Marler and his collaborators. Some birds, Sober informs us, require exposure to their own, species-specific song in order to acquire that song, others require exposure to some song or other, but not the specific song which they will eventually sing, and yet others will develop normal song when raised in isolation. Ariew places these three cases along his continuum of greater or lesser degrees of environmental canalization. He suggests that even if there is no sharp distinction between innate and acquired, what he calls the innate/triggered/acquired distinction can play a useful heuristic role. Traits that are independent of salient environmental parameters to a similar degree are usefully regarded as being of the same kind for various scientific purposes, despite the many differences of detail between them (Ariew, 2006, p. 7).

Placing traits along such a continuum might, indeed, be heuristically useful if this particular axis of variation in the developmental patterns of birdsong were the only one, or was one of a few common axes of variation, or if there were some correlation between different axes of variation. But neither of these conditions is met. The 'ontogenetic niche' for birdsong is multi-dimensional and idiosyncratic. For example, the Brown-headed Cowbird, *Molothrus ater* is a brood-parasite, and so cowbirds are not raised by their own species. The species-typical song of the male cowbird develops without experience of that song, placing at the 'innate' end of Ariew's continuum. But despite this, cowbird song is heavily influenced by experience and cowbirds have regional dialects, the classic signature of 'learned' song. It is impossible to do justice here to the decades of research of Meredith West and Andrew King and their collaborators into the ontogenetic niche that supports

cowbird song development (e.g., West & King, 1988, this issue; West, King, White, Gros-Louis, & Freed-Brown, 2006), but amongst other processes, males performance is strongly influenced by feedback from female cowbirds, whose wing stroking and gaping displays in response to the songs they prefer are strong reinforcers of male song, and female song preferences are themselves subject to social influences. The strategy of placing traits along a continuum of independence of the environment founders on the fact that there is no continuum, but rather a high-dimensional space whose axes are often specific to one or a few species. Even when the same axes can be applied to different species, so that canalization of a character in two species can be meaningfully compared, there is no systematic tendency for traits that are at the 'innate' end of one axis to be at the 'innate' end of the other. Instead, something that is 'innate' with respect to one axis can be 'acquired' with respect to another. For example, while female cowbirds have a large influence on male song the development of song is independent of the responses of female conspecifics in the intensively studied group of North American sparrow species which are probably the inspiration for Ariew's description of an 'acquired' song. Which of the two species has the 'innate' song and which has the 'acquired' song depends on whether we concentrate on the developmental role of male conspecifics or that of female conspecifics.

Ariew's strategy for 'biologicing the mind' has the same weakness as the classical deprivation experiment (Lehrman, 1953; Tinbergen, 1963). Deprivation experiments, in which animals are raised without any obvious source of environmental information to guide their development, were originally interpreted as demonstrating that traits which survive deprivation are 'innate'. Thus, for example, a bird might be raised without exposure to species-specific song and nevertheless produce that song as an adult, or a rat might copulate successfully the very first time it meets a receptive female. The problem with this interpretation, as we see from the facts about cowbird and rat development given above, is that there is no such thing as raising an animal without an environment, only raising it without access to some specific aspect of the environment. The deprivation experiment is a way to show that chosen aspects of the environment do not affect certain traits. But it does not provide evidence for some general property of 'independence of the environment' (for more on the proper use of the deprivation experiment see Michel & Moore, 1995).

If we examine the examples that Ariew provides of the successes of 'biologicing the mind', such as Chomskyan nativism about language development, or the development of birdsong without exposure to adult models, it is evident that what has been shown is that some prominent environmental factor does not affect the development of the trait. It is simply not valid to infer from this that the traits in question have some general property of insensitivity to the environment. Developmental fixity, environmental canalization and their relatives are not general properties of traits, so that insensitivity to one variable implies insensitivity to others. Instead, they are the result of specific feedback mechanisms that buffer the development of the trait against specific sources of variation. One of the lessons of

50 years of developmental psychobiology is that detailed research on the development of a trait almost always reveals that it depends on critical parameter values in the developmental niche.² Developmental stability is achieved, not by making development insensitive to that parameter, but by stabilizing the parameter at the right value. In many species, this stabilization takes the form of parental care. We have already described one role of parental care in the sexual development of the male rat. One of the most spectacular achievements of developmental science in recent years has been Michael Meaney and collaborators' detailed account of the role of parental care in the development of temperament in rats. The BALBc strain of laboratory rat is "highly fearful, and maze dull... these animals show increased endocrine and behavioral responses to stress, they are hyperactive and show profound learning and memory deficits that are associated with, among things, impaired hippocampal development" (Meaney, 2001a, p. 57). Cross-fostering BALBc pups to mothers of the more laid-back C57 strain removes the differences between the two strains. Meaney and collaborators have shown in extraordinary detail how the amount of licking and arched-back nursing which pups receive from the mother regulates gene expression so as to direct the development of the pup's brain (Meaney, 2001b). In wild rats, these behaviors, which are heavily influenced by the mother's stress levels, may convey important information to the pup about the quality of its environment, and 'preset' stress metabolism to match that environment.

Meaney and collaborators' work reinforces another lesson of developmental psychobiology, which is the relevant aspects of the ontogenetic niche that are 'non-obvious'. We might guess that the rat pup will respond to indicators of environmental quality, and we might guess that the mother's behavior is a useful source of information on this topic, but we would surely not guess that the information resides in whether the mother arches her back during suckling. The concept of innateness is an anti-heuristic which encourages researchers to check the obvious sources of environmental input, and then to stop looking. This flies in the face of everything we have learnt from 50 years of developmental psychobiology including, importantly, the past few years in which those lessons have been extended to the molecular level. Prototypically biological traits develop in a highly specific ontogenetic niche (West & King, 1987).

4. Innateness and Human Psychological Development

Ariew's characterization of innateness is not merely a poor research strategy for studying the development of rat and birds. It is also misleading when studying human psychological and behavioral development. To illustrate this point, we will compare Ariew's characterization of innateness as canalization with the poverty of the stimulus arguments that have been developed throughout cognitive science.

Chomsky introduced this type of argument to support the conclusion that children are innately endowed with a tacit knowledge of the grammatical principles that govern any human language—what Chomsky calls Universal Grammar (Chomsky, 1967, 1975). In substance, poverty of the stimulus arguments start by describing a given psychological trait—children’s knowledge of the syntax of a language such as English in Chomsky’s case. Poverty of the stimulus arguments contend that children do not have access to the evidence that would be required to acquire this psychological trait by learning. The relevant evidence may be absent from children’s environment, its presence may be unreliable, or the environment may provide children with misleading information (for instance, when adults use ungrammatical sentences). Poverty of the stimulus arguments conclude that the relevant psychological trait is not learned and therefore that it is innate.³ In addition to children’s syntactic knowledge, this kind of argument has also been used to argue for the innateness of our moral sense (Dwyer, 1999; Hauser, 2006; Mikhail, 2000).

An example will make clear the gist of these arguments. Consider the case of children’s knowledge of the constituent structure of noun phrases, such as ‘the red ball’ (Lidz & Waxman, 2004; Lidz, Waxman, & Freedman, 2003, see also Akhtar, Callanan, Pullum, & Scholz, 2004; Regier & Gahl, 2004; Tomasello, 2004). One can at least form two distinct hypotheses about the constituent structure of ‘the red ball’:

1. [[the][red][ball]]
2. [[the][[red][ball]]]

According to hypothesis 2, but not according to hypothesis 1, ‘red ball’ is a constituent of the noun phrase ‘the red ball.’ 2 is clearly the correct constituent structure of noun phrases for adult speakers, as is shown by the fact that the pronoun ‘one’ can refer anaphorically to the constituent [red ball] (Lidz et al., 2003):

1. I’ll play with this red ball and you can play with that one.

By analyzing a corpus of adults’ utterances to children, Lidz and Waxman have argued that the linguistic environment of children does not contain the evidence that would be needed to discriminate constituent structure (1) from constituent structure (2). Still, evidence suggests that 18-month-old babies know that (2) is the right constituent structure of noun phrases. Since they could not have learned it from the evidence they are exposed to, the constituent structure (2) must be innately specified in their innate universal grammar—or so Lidz and Waxman conclude.

An extensive literature focuses on the cogency of poverty of the stimulus arguments (see particularly Cowie, 1999; Laurence & Margolis, 2001; Piattelli-Palmarini, 1980; Pullum & Scholz, 2002; Putnam, 1967; Seidenberg, 1997). But, for present purposes, what matters is the similarity between the notion of innateness at stake in poverty of the stimulus arguments and Arieuw’s characterization

of innateness as canalization. Poverty of the stimulus arguments essentially claim that the development of a trait does not depend on the presence of specific environmental stimuli. Thus, according to Lidz and Waxman, to know the constituent structure of noun phrases such as ‘the red ball,’ children do not need to be exposed to linguistic constructions distinguishing constituent structure (2) from constituent structure (1). This conclusion of poverty of the stimulus arguments is in line with Ariew’s characterization of innateness, for, as we saw above, Ariew contends that in some cases, “the system develops independently of the environmental condition” and that “innateness should be identified with . . . [this] sort of invariance” (Ariew, 2006, p. 10).

Now, it is important to keep in mind that poverty of the stimulus arguments do not show that the development of a trait is independent from the environment. Rather, if successful, they show that it is independent from a *specific* environmental condition. For instance, if successful, Lidz and Waxman’s poverty of the stimulus argument shows that the acquisition of the constituent structure of noun phrases does not depend on exposure to linguistic constructions distinguishing structure 1 from structure 2. This is perfectly consistent with the development of children’s knowledge of the constituent structure of noun phrases being contingent upon the presence of very specific stimuli in the environment of children, in exactly the same way the development of rats’ ability to copulate is contingent upon the mother licking the pups’ genital area. Furthermore, the stimuli required for the development of the trait under consideration might well be non-obvious. That is, they might be such that one cannot predict on *a priori* grounds that the development of this trait depends on their presence in children’s environment.

As far as we know, evidence is lacking to assess whether the development of children’s syntactic knowledge of a given language, of their mind reading capacity, or of their moral sense depends on the presence of specific non-obvious stimuli in their environment. This is partly due to the fact that for obvious ethical reasons, one cannot conduct with children the kind of experiments done with rat pups. The success in locating environmental tuning parameters in species where controlled experimentation is ethical should caution us against placing any weight on the mere absence of evidence in humans. Moreover, recent research on mimicry in human neonates has produced just this sort of evidence. Mimicry is the disposition to imitate perceived gestures and bodily movements (including facial expressions). Numerous psychologists and neuropsychologists have contended that mimicry is an innate disposition that is present at birth (for a review, see Butterworth, 1999; Meltzoff & Moore, 1977). However, Susan Jones’ recent work suggests that mimicry might depend in subtle ways on parents mimicking the child and highlighting their mimicking with sounds (reported in Jones, in press; Jones & Yoshida, 2006). Jones and Yoshida conducted a longitudinal study of mimicry in a female infant named Yo (from 3 to 18 months). At the beginning of the study, Yo’s motor patterns were uncorrelated with her mother’s motor patterns, while her mother repeatedly mimicked Yo’s action patterns. When Yo started mimicking (in the second half of her first year), she did not mimick every motor pattern of her mother. Rather, as

Jones puts it, “[t]he actions matched were behaviors (a) that Yo had spontaneously and repeatedly produced at home, on her own; (b) that Yo’s parents had imitated; and (c) that produced or were accompanied by sounds” (Jones, in press). This suggests that the development of mimicry may be contingent upon a parent mimicking her child’s motor patterns and associating her mimicking with sounds (for further evidence, see Jones, in press).

Because the development of psychological or behavioral traits typically depends on non-obvious environmental stimuli, even when this development is buffered from the presence or absence of other environmental stimuli, Ariew’s strategy for biologicizing the mind is misleading. Rather than focusing developmental psychologists’ attention on the environmental conditions that are necessary for or that contribute to development, calling a trait innate would typically lead psychologists to believe that some trait, such as children’s syntactic knowledge or their disposition to mimic, is globally insensitive to the environment. This would prevent developmental psychologists from properly studying the development of the relevant trait. That Ariew’s proposed research strategy that might have such an effect is not a far-fetched possibility. In her discussion of mimicry, Jones makes a similar point:

Despite its wide interest and relevance, the development of imitation has not been the subject of a great deal of research. This may be because it is widely accepted among developmentalists that the ability to imitate is innate, and that much of the underlying mechanism is inherited. (Jones, in press)

This is of course not to say that the notion of canalization has no role to play in studying the development of a psychological and behavioral trait. Developmental psychology should inquire whether the development of a trait is buffered from variation in specific aspects of the environment. For instance, the development of the knowledge of constituent structure of noun phrases seems to be buffered from the variation in the availability in children’s linguistic environment of linguistic constructions that would discriminate constituent structure 1 from constituent structure 2. Rather, the point of our discussion is that canalization is always relative—traits are canalized with respect to a particular environmental condition. Identifying canalization with the older idea of innateness, as is proposed by Ariew, is likely to obfuscate this important fact and to result in erroneous conceptions of development and misguided research strategies to study development.

5. Canalization relative to specific parameters

Canalization is commonly described as if it were a single property of a trait, albeit one that comes in degrees. But this assumes that canalization is assessed in terms of the dynamical structure of Waddington’s ‘developmental system’ as a whole. As a theoretical biologist Waddington was free to stipulate that the ‘pegs’ in his model include all the Mendelian loci in the genome and all the environmental factors

that affect development, just as he was free to treat the two-dimensional surface which the pegs determine as a representation of a hyperspace with one axis for every way in which the phenotype can differ. In the context of this idealized model, Waddington was able to talk of the canalization of some phenotype in general, that is, relative to the entire set of developmental parameters, or its canalization relative to all and only the genetic parameters, or to all and only the environmental parameters. But this is a luxury not available to biologists who study real organisms. We can realistically look forward to the day when the systems biology of single-celled model organisms like the yeast *Saccharomyces cerevisiae* will allow the construction of something approaching Waddington's model from actual data, but doing this for songbirds or human children is still the stuff of science fiction.

We have discussed above how the experimental evidence produced for environmental canalization typically examines whether a trait varies in response to one or a few salient environmental parameter. Obviously, showing that a trait is insensitive to changes in one or a few 'pegs' in Waddington's diagram is very far from showing that it is canalized in general. Moreover, different traits are said to be canalized because they fail to respond to *different* environmental manipulations. Classifying traits together as environmentally canalized because they each show evidence of insensitivity to *some* environmental factor is hardly better than classifying them together because one is not very large, another is not very rigid, and a third is not very heavily pigmented. If the idea of canalization is to be taken seriously then it must either be relativized to specific parameters or it must be assessed in the light of a fairly complete model of the development of a trait. The second of these options is impractical and so the claims about canalization that have an actual scientific basis are usually relativized ones: grammar is canalized relative to the corpus of sentences to which children are exposed and the song of *Melospiza melodia* is canalized relative to the corpus of birdsong heard in the Song Sparrow's first season.

6. Innateness: You Just Know It Makes Sense

We suspect that a common response to these strictures about canalization will be that they are merely nitpicking. The limited evidence that is cited in support of claims of innateness may not formally imply that other environmental variables are irrelevant, but it shows the fundamentally 'biological' nature of these traits, and so only some sort of environmentalist prejudice makes us demand direct evidence of their independence of other variables. The fundamental idea behind 'biologizing the mind' is that there is some association between being part of an animal's biological nature and developing independently of the environment. This idea has deep roots, but we do not think they are deep *scientific* roots.

We have argued elsewhere that the idea of an innate trait is simply one expression of a folkbiological theory of the 'natures' of living things (Griffiths, 2002; Griffiths,

Machery & Linquist, submitted). Just as there are commonsense ideas about physical objects and the forces acting on them ('folk physics'), so there are commonsense ideas about biology, or 'folkbiology' (Medin & Atran, 1999). It is part of folkbiology that some traits are expressions of the inner nature of animals and plants, whilst other traits result from the influence of the environment. The idea that living things have inner natures that make them the kind of organism that they are is closely linked to the idea of heredity. Hereditary traits are those which form part of the inner nature that is passed on from an organism to its descendants. Despite the valiant efforts of early geneticists to combat this idea by distinguishing the inherited genotype from the phenotype that emerges as a result of the interaction of genotype and environment (e.g., Johannsen, 1911), the folkbiological picture that Schaffner (1998) has described as the inheritance of 'traitunculi' has persisted and even been strengthened by the well-publicized successes of genetics.

For all its intuitive appeal, the idea that traits which are part of an organism's biological nature develop independently of the environment is not well-supported. The direct evidence against it can be found in textbooks on psychobiological development (e.g., Michel & Moore, 1995), and we have given a few examples above. But it is also theoretically unsupported. An evolutionary perspective suggests that organisms will make use of whatever environmental regularities are available and that they will not pay the costs of buffering against variation in developmental parameters unless those parameters vary in a way that makes buffering cost-effective. This is why human beings and their primate relatives have lost the ability to synthesize vitamin C that their ancestors possessed and rely on environmental sources of this chemical (Jukes & King, 1975). If it is more cost-effective to stabilize an environmental parameter than to build (or to maintain) a mechanism that buffers against variation in that parameter, organisms will do just that. This is presumably why, as the evidence increasingly shows, intelligence and many aspects of general health depend to some degree upon substances provided in the mother's milk. These principles are not merely applicable to early development, but continue to apply throughout an organism's life cycle. One of Konrad Lorenz's insights which he initiated the modern study of animal behavior in the 1930s was that animals do not need the abstract instincts like 'parenting' or 'sociability' in which many of Lorenz's contemporaries believed. Instead, they rely on regularities in the ecological context in which their lineage has evolved to structure relatively simple responses into coherent and appropriate sequences of behavior.

One important aim of philosophical analysis should be to highlight problems that arise from the conceptual framework within which a topic is being treated. In the philosophy of science this often means highlighting assumptions which persist from pre-scientific thought, as opposed to emerging from the science itself. The concept of innateness is a classic example. The distinction between innate and acquired characteristics was not invented by Francis Galton in 1874, but has existed in some form or another as long as people have thought about heredity. It is a highly intuitive distinction and has survived a half-century of scientific critique because of the gut-feeling that there must be something to it. It is time that we freed

ourselves from the idea of an innate characteristic just as we have freed ourselves from the idea that any projectile must eventually run out of force and fall to earth. We need to do this, not to embrace some form of environmentalism, since the idea of an acquired characteristic is as monstrous as its twin (Stotz & Allen, submitted), but to make space for a more realistic picture of the development of biologically significant traits.

Notes

- [1] For example, Bateson (1991); Bateson and Martin (1999); Gottlieb (1981, 1997); Hinde (1966, 1968); Johnston (1987); Lehrman (1953, 1970); Lickliter and Berry (1990); Michel and Moore (1995); Moore (2001); West and King (1987, this issue); West, King, and Duff (1990); West, King, and White (2003). For historical accounts of this critique and its influence, see Gottlieb (2001); Griffiths (2008); Johnston (2001).
- [2] It is probably worthwhile pre-empting the idea that traits can be split into sub-traits some of which will turn out not to rely on these parameters and thus remain 'innate'. This suggestion was first made by Konrad Lorenz in the 1930s in response to evidence of the 'fine tuning' of supposedly innate behaviors by experience. For example, the anterior-posterior axis of a head-movement might be an innate 'fixed action pattern' despite the movement in the lateral plane which accompanies it depending on interaction with the environment. However, while it is certainly possible to identify some elements of a complex behavior pattern that survive a particular deprivation experiment, this does not establish that those elements are independent of other parameters, and Lorenz later abandoned this idea (Lorenz, 1965). A brief account of how this idea fails in specific cases of birdsong development can be found in Marler (2004).
- [3] Various types of poverty of the stimulus arguments have been distinguished in the recent literature (Cowie, 1999; Pullum & Scholz, 2002). We neglect this complication here.

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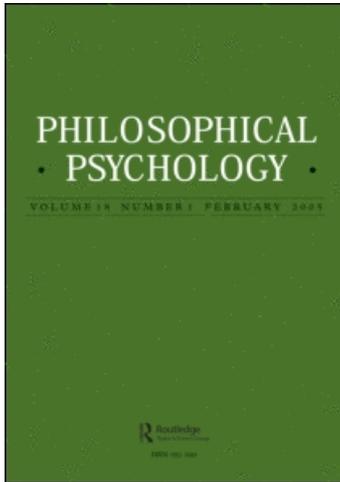
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Living with Innateness (and Environmental Dependence Too)

Jonathan M. Weinberg and Ron Mallon

Griffiths and Machery contend that the concept of innateness should be dispensed with in the sciences. We contend that, once that concept is properly understood as what we have called ‘closed process invariance’, it is still of significant use in the sciences, especially cognitive science.

Keywords: Canalisation; Cognitive Science; Innateness; Invariance

Critics of innateness like Griffiths and Machery (this issue) would have us dispense with the scientific use of the concept of innateness entirely. We find ourselves much more sanguine. The concept of innateness continues to organize and motivate vast amounts of research in the various sciences of the mind, and we see no reason to think that organizing such projects around the concept of innateness is fundamentally mistaken. Instead, we think it is possible and valuable to offer an account of what innateness is in the context of this research, and recently we have done just that (Mallon & Weinberg, 2006).

Here is how we will proceed. In the first section, we’ll set out what we take the problem-space to be, and briefly recapitulate our own solution. In the second section, we will discuss Ariew’s canalization account and how Griffiths and Machery, and perhaps Ariew himself, go astray, applying that lesson in the third section to Griffiths and Machery’s arguments about the dimensionality of developmental invariance. Finally, in the fourth section we address their claims that the concept of innateness serves as some sort of ‘anti-heuristic’ for productive research.

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1. Innateness as Closed Process Invariance

Our account of innateness follows recent theorists such as André Ariew and Richard Samuels in adopting what we have called an “invariance-plus” strategy: a trait’s invariant development across some relevant range of environments is a necessary but not sufficient condition for its being innate. For each theorist, a second condition that yields sufficiency concerns the way in which the trait develops. For each of us, this second condition must solve the *problem of liberalism*: it must rule out traits that are invariantly acquired but that, according to shared usage on both sides of the extant scientific and philosophical debates over innateness, ought not to count as innate. For example, suppose that the trait *believing that water is wet* is acquired by every normal human adult across the many differences in their developmental environments, but that the acquisition process by which it is acquired is general learning. An account of innateness ought not count such a trait as innate, because it is standard in the target literature to consider a trait’s being learned to be inconsistent with its being innate.¹ Invariant development alone will not do this because (by hypothesis) the trait is invariantly learned.

Ariew’s, Samuels’s, and our accounts diverge as how best to address this problem. Our own account, *closed process invariantism*, has it that trait-producing processes in an organism vary along a dimension we label ‘open’ and ‘closed.’ The idea is that trait-producing processes vary in the size of the array of traits that they can produce in that organism, depending on which particular history they have with the world. For example, the basic bipedal body plan does not admit of many variants; if a person develops limbs at all, they are overwhelmingly likely to develop them in this one way. Because the process that produces the body plan tends to produce one (or only a very few) outcomes across a range of developmental environments, this process is rather closed. In contrast, the language acquisition device is prepared to give a person competence in any of a very wide set of languages (though, importantly for Chomskyan linguistics, sharply less than all logically possible languages), and this process is therefore open. Openness and closedness are thus matters of degree.² The category of innateness, we claim, distinguishes between traits that develop invariantly because they are the product of a closed system, and traits that develop invariantly because they are the product of an open system which happens to be getting invariant input from the environment; only the former count as innate. Our account thus holds that a trait *t* is innate in an organism *O* to the extent that:

- (i) *O* would develop *t* across the range of normal environments (*invariance condition*); and
- (ii) the proximal cause of *O*’s development of *t* is by a closed process or processes (*closed process condition*).

It is a feature of our account to recognize that two different evaluations are needed: the first is designed to limit our attention to traits that are invariantly acquired across normal environments, and the second is to focus on the sort of mechanism that does

the acquisition. It is the failure to recognize this distinction, both by Ariew and by Griffiths and Machery that makes the prospects for innateness seem so dire.

2. Ariew, Canalization, and 'Invariance-Plus' Accounts

Ariew holds that the concept of innateness might be usefully identified with environmental canalization. Griffiths and Machery (this issue) begin by claiming that Ariew's account of innateness as environmental canalization (understood as a conceptual analysis) cannot accommodate Celia Moore's (1992) research on the neural bases of copulatory reflexes among male rats. They write:

The spinal cord nuclei of male rats differ from those of female rats in ways that allow the male to use his penis during copulation (Moore, 1984, 1992). These neural differences result from differences in gene expression in the developing spinal cord of the rat pup, which in turn result from differences in the amount of licking of the genital area by the mother, which in turn results from greater expression in male pups of a chemical that elicits maternal licking. (p. 398)

Let's assume with Griffiths and Machery that the maternal genital licking behavior is a robustly present factor in rat pups' normal environment. Now, as Griffiths and Machery note, Ariew is not going to let that fact automatically count that penile reflex as innate, for Ariew (2006) holds that we should

Distinguish between two reasons why the trait appears invariantly in an environmental range: the first, because an environmental condition is developmentally required yet is found everywhere the system develops; the second, because the system develops independently of the environmental condition. Innateness should be identified with the second sort of invariance, not the first. (p. 10)

From this claim, and the fact that the rat's penile reflex depends on features that are "developmentally required yet found everywhere the system develops," Griffiths and Machery conclude that "according to innateness-as-canalisation these experiments show that the rat's ability to copulate is not innate... But, intuitively, the rat's ability to copulate is innate." (p. 398) Thus Ariew's view fails as a conceptual analysis of innateness.

Something, however, is very wrong here, and it is a mistake that animates much of Griffiths and Machery's paper. We can see this simply by reflecting that one does not need to consider the fascinating work on maternal licking to find an environmental parameter on which rat penile reflexes depend. We could choose a rather ordinary environmental input, like the presence of water, to make Griffiths and Machery's point: because the presence of water is developmentally required yet found everywhere the system develops, Griffiths and Machery's way of reasoning from Ariew's account would have it entail that traits that depend upon the presence of water are thereby not innate. But surely that is a mistake about what an invariance-based theory of innateness should be committed to.³

Consider how distinguishing the question of invariant development from the question of how to solve the problem of liberalism allows our own account to handle the case of rats' penile reflexes. In particular, our first, invariance condition limits our attribution of innateness to traits that develop across normal environments. Because normal environments exclude the ingenious environments produced in the course of Celia Moore's experiments, the failure of rats to develop normal penile reflexes in such environments is not directly relevant to whether those reflexes are innate. This sort of restriction is hardly *ad hoc* since arguably what we are trying to do in characterizing a scientifically useful concept of innateness is characterize a concept of something that figures in a trait's natural development, development independent of our manipulations and interventions in it.⁴ Moreover, it is a restriction that is explicitly part of our own account of innateness, and that of Richard Samuels, and we see no reason why Ariew might not modify his account to adopt it as well.⁵ Of course, all these accounts still disagree about how to handle the problem of liberalism, but as long as we recognize that whatever environmental assessments are required to handle that problem are distinct from those involved in assessing invariant development *tout court*, we can effect this sort of solution.

Like Ariew, we hold that our account of innateness is not hostage to folk intuitions about innateness. Our project concerns a scientific concept as it plays a key role in structuring a bit of scientific discourse, and thus we have at most a secondary interest in the particular psychological structure in any particular scientists' heads, and no relevant interest whatsoever in what innateness concept is in the heads of the folk. (We of course agree that that question is of independent interest to such projects as understanding folk biology.) So, for our purposes, it would only matter if the nativist/empiricist debates, as manifested in the scientific literature, clearly registered the judgment that rat penile reflexes are innate, or something that would clearly entail that result, and we are unaware of any consensus view on that question. But we take it that, nonetheless, there is an easy way for accounts of innateness to accommodate the judgment that these rat penile reflexes are innate: recognize that invariance accounts of innateness must (and indeed, do) allow reliance on stable environmental features of normal environments. When we do, we arrive at the alleged intuitive conclusion: the reflexes are candidates to be innate because the stable developmental mechanisms that produce them occur across the range of normal environments, and the manipulations Moore produces in her lab are outside that normal range.⁶

3. The Dimension(s) of Invariance

As will be obvious from the above, we agree that the idea of development occurring completely independent of the environment is an incoherent notion, for every trait depends on *lots* of factors in the environment. So the idea of developmental invariance must be interpreted differently than one of complete independence from

the environment. It seems that Griffiths and Machery think that the only way for an invariance theorist to progress against this background is to identify invariant development one factor at a time. This is very odd, given that the idea of identifying invariant development and innateness is surely not to identify innateness with just *any* flat norm of reaction, but rather to identify it with development in a broad range of environments *that vary along many dimensions simultaneously*. If we think about cowbirds and sparrows in such a way, we get that male cowbird songs and North American sparrow songs have substantial acquired elements, as evinced by their variation across, for example, a normal range of actual environments. Our own account tries to make good sense of invariance by separating the claim of invariant development across a broad range of normal environments from whatever additional condition solves the problem of liberalism. But just pointing out this alternate possibility is sufficient to undermine Griffiths and Machery's assumption that invariance claims can only be understood once relativized to particular dimensions.

This metaphysical issue about the multi-dimensionality of developmental space seems curiously mixed up with an epistemic one concerning deprivation experiments. Griffiths and Machery write: "The deprivation experiment is a way to show that chosen aspects of the environment do not affect certain traits. But it does not provide evidence for some general property of 'independence of the environment'" (p. 402).⁷ We agree that this is the case if "independence of the environment" is understood as the absurd denial that the environment has a role to play in development, but, again, no one wants to deny *that*. Now, Griffiths and Machery may mean simply that such experiments do not provide *conclusive* evidence (we note that they write, in the following paragraph, of a proposed nativist inference from such experimental results being 'simply not valid'), in which case we will of course agree, since no single experiment is ever *conclusive* evidence of much of anything. But if they mean (as we think they do) that such deprivation experiments provide *no evidence whatsoever* for claims that go beyond the particular controlled aspects of the environment, then we think their mistake has recurred. As no invariantist would need to be interested in a claim about what an animal would do in the absence of any environment (since, as we've said, a great deal of environmental particulars are fixed by the restriction to only normal environmental trajectories), seeing that a trait can develop even in a number of significantly impoverished atypical environments is pretty good—if, again, non-conclusive—evidence that the trait will develop across a very large set of the normal ones.

The 'invariance-plus' framework opens up a yet further and more substantive line of response. For it may be that deprivation experiments are supposed to speak not just to the question of invariant development, but also to the exact nature of the mechanism that acquires the trait.

If we interpret deprivation arguments as addressing such a 'plus' condition, then we can grant to Griffiths and Machery that it is not at all implausible that any particular trait (for example, the constituent structure of noun phrases) might require some non-obvious environmental factor to be present in order to develop.

But what is rather more unlikely is that the organism has a developmental process that can produce a significant *range* of such traits, *each one* of which depends on a different, nonobvious environmental factor. We are not claiming that this is impossible, but no such claim is needed in order for deprivation experiments to provide good-but-nondemonstrative evidence for nativist claims. When a deprivation experiment indicates that a trait can develop under significantly impoverished environmental conditions, that narrows the possible range of different environmental trajectories that could possibly produce a wide range of different traits. It is still possible, of course, and we agree with Griffiths and Machery that it would be a mistake for theorists to take the results of any one or small set of deprivation experiments to be anything like conclusive evidence for an innateness claim. It may be that an open process is indeed involved, but one that is sensitive to variations in the environment that the researchers have not yet been clever enough to discern.

This brings us to the last of Griffiths and Machery's arguments that we wish to respond to: does the very idea of innateness somehow inhibit research into such matters?

4. What Kind of Heuristic is *Innate*?

A key part of Griffiths and Machery's argument for eliminating *innate* from scientific discourse is their claim that it functions as a sort of negative research heuristic, foreclosing what should be fruitful lines of investigation into the complex and subtle ways in which the environment is involved in the development of the trait. They write that "the concept of innateness is an anti-heuristic which encourages researchers to check the obvious sources of environmental input, and then to stop looking" (p. 403). But one must distinguish between innateness claims that have been established, and those that have merely been proffered. If an innateness claim has been established for a particular trait, then *of course* it should lead researchers to stop looking for ways in which that trait is acquired, say, by learning. More generally, for any concept C, the claim that some *a* is C will, if accepted, discourage all sorts of research into propositions inconsistent with *a*'s being C. We use what we take to be true as a guide for what further research should—or should not—be done. So it can't be a problem with the concept of innateness that were some particular innateness claim to have been established, people would then do a lot less looking for evidence that that claim is false.

Rather, if there is to be a problem with *innate* serving as an anti-heuristic, it must be because of what happens when researchers propose or investigate an innateness claim. Griffiths and Machery's formulation suggests that researchers may fail to recognize what it really would take to establish an innateness claim—such researchers are not looking sufficiently far past their own noses, they are claiming, in looking for ways in which a trait's development may depend on environmental factors. The proponent of an invariantist concept of innateness has several moves available here. First, as we argued above, many logically possible environmental variations are just

not counted as relevant to the claim in question, especially when those variations are not biologically very likely, perhaps because the factors in question are reliably part of the extended inheritance of the organism. So it may be fine for someone investigating an innateness claim to ignore a large number of environmental dependencies, even while someone else who wants to investigate more closely the particular path of that trait's development may appropriately examine those same factors. Thus at least some of the cases Griffiths and Machery envision are ones in which those investigating an innateness claim are perfectly reasonable in not attending to some "sources of environmental input".

Second, invariantist approaches to innateness are typically graded accounts: traits can develop in a greater or lesser portion of the relevant environments. If the environmental contributions are subtle enough to be so easily missed, then they may also vary only in so small a fraction of the relevant environments, that we are facing at worst a case where researchers endorse a slightly stronger innateness claim than they really have a right to. So we think that Griffiths and Machery may be miscategorizing at least some cases that look to them like this alleged "anti-heuristic" in action.

Nonetheless, we would acknowledge that at least sometimes nativist researchers may miss truly substantial but non-obvious environmental contributions to development. For example, it had seemed to Chomskyan linguists that children were lacking a particular kind of environmental input, namely, evidence as to which utterances were grammatically incorrect. This has been called the "poverty of negative stimulus." Even if the child language learner does not receive much explicit negative stimulus, however, she might well be able to extract such information from other, more subtle aspects of their verbal environment. For example, when someone stops a sentence in mid-stream, and then starts it over with a slight change, or when they repeat a sentence the language-learner just uttered with a slight change, this kind of evidence from 'recasts' indicates that the initial utterance is likely to be incorrect (see, e.g., Demetras, Post & Snow, 1986; Bohannon & Stanowicz, 1988). Noting the existence of this potential environmental resource for the language learner is not enough, by itself, to show that Chomskyan nativism is wrong, or even that the poverty of negative stimulus argument is mistaken, since it may be that, even though this kind of evidence is out there, there is not enough of it, or children are not sufficiently sensitive to it (see Crain & Pietroski, 2001; though see also Saxton et al., 2005). Our point here is just to take an uncontroversial instance in which nativists overlooked a potential environmental resource that the relevant developing organism (the language-learning child) might draw upon. And we expect that there are others like them.

On inspection, though, there does not seem to be any deep problem about such cases—nothing about them that calls for the large-caliber philosophical artillery of eliminating the very concept that figured in the research in the first place. Nativist claims have not led to a research anti-heuristic, and much to the contrary, the proffered claims by the linguistic nativists have clearly spurred on their empiricist interlocutors to find the holes in their arguments, and to do so at least in part by

looking closely at the organism's environment. Our diagnosis of where Griffiths and Machery have gone wrong here is that their picture of research concerning innateness has only included the nativists, when in fact the arguments of nativists (just like the claims of any other set of scientists) have to be understood in the context of their debate with their scientific rivals. When evaluating the heuristic/anti-heuristic status of a concept, we would suggest that the correct unit of analysis is that of a *debate* or a *literature*, and not of a researcher or set of collaborating researchers. And when one does so, "innate" seems to have a pretty good track record, at least in cognitive science.⁸

We think that Griffiths and Machery are mis-identifying, and over-philosophizing, the source of their ire. They frame their arguments as an attack on the very concept of innateness, but we would suggest that a better way to understand what is really vexing them is that nativists sometimes, perhaps even frequently, prematurely endorse innateness claims as established. A better expression of their worries would be: there are many fewer innate traits than a lot of nativists think there are, and many of the arguments that people have used to justify innateness claims are less good than their proponents have thought. We would find their concerns, were they expressed in such first-order terms, to be not only eminently defensible views (if not necessarily correct, for the reasons we have been presenting here), but they would also thereby contribute to an area in which philosophers can really play a substantive role as players in an ongoing scientific debate. Griffiths and Machery would be better off running their arguments as straightforwardly anti-nativist arguments. Why turn into philosophy that which continues to work so well as science?

Notes

- [1] See, e.g., Samuels (2002) on what he terms the 'Fundamental Conceptual Constraint', which he extracts from the debates over innateness in cognitive science: "If a cognitive structure is innate, then it is not learned. This condition is so widely accepted among cognitive scientists and its role in inference to and from innateness claims so pervasive, that any account of innateness that cannot accommodate it is surely unsatisfactory." (p. 236)
- [2] As is invariance itself.
- [3] It is now widely recognized that the idea that anything develops independently from its environment is a fantasy. For example, Ariew (2006) "every case of trait growth involves both genes and environments . . . if innateness refers to what the genes do alone then nothing is innate" (p. 3). Indeed, as far as we know, no one in the recent literature endorses a view that some traits are "independent of the environment" or "insensitive to the environment" in the sense that their development does not depend on the environment. Nonetheless, it is unclear how to interpretively reconcile Ariew's account of canalization with his recognition that environmental interaction is ubiquitous. As we argue in our earlier paper, Ariew's account generally suffers from insufficient articulation of what it is that distinguishes truly innate/canalized traits from those that are universally acquired. By constructing a clear second condition that makes this distinction, we believe that our account avoids this pitfall, and makes clear how an invariantist can cleanly disown the peculiar entailments that Griffiths and Machery attribute to the view in their paper.

- [4] While explaining the sense in which scientific manipulations are not ‘natural’ is no doubt a difficult philosophical puzzle, that such a distinction is needed is uncontroversial. Lots of science is concerned with the world as it is when we are not looking.
- [5] Indeed, Samuels adds such a condition to his account precisely in order to rule out peculiar scientific manipulations of the organism.
- [6] Of course, Griffiths and Machery have another explanation of this intuition, that it is rooted in intuitions about species typicality or functionality, an explanation they (along with Stefan Lindquist) have produced experimental evidence for (Griffiths, Machery, Lindquist under review). Because we hardly have the space to consider this work with the care it deserves here, it will have to suffice to say we are skeptical of their alternate explanation of the intuition of the trait’s innateness, and satisfied with our own.
- [7] Immediately before this passage, they write that ‘that there is no such thing as raising an animal without an environment, only raising it without access to some specific aspect of the environment,’ but this contrast is clearly false: one can raise an animal without access to *lots and lots and lots* of specific aspects of a standard environment. Many birdsong experiments do not do this because they are interested in studying the effects of specific causal factors. But part of our interest in, say, the linguistic development of ‘wild child’ cases (e.g., Curtiss, 1989) has to do with how drastically impoverished these environments are. (And indeed, the difficulty of drawing conclusive inferences about the relevant factors that accompany these cases is so great precisely because the deprivation is so profound.)

It should also be emphasized that these studies are often relevant primarily for their exploration of the pattern of what capacities are or are not spared under different conditions of deprivation, in order to argue for double dissociation claims, critical period claims, and the like. These arguments are especially important for mooting the applicability of the “plus” condition of any “invariance-plus” account of innateness to any particular case.

- [8] If its track record over in biology is radically different, that would be a fact calling out for explanation. The closed process invariance account suggests one possible hypothesis: that psychology has generally been more concerned than biology has with open trait-producing processes. If so, there may thus be more fruitful debates to be had within psychology about whether any given trait is innate.

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The Comparative Biology of Human Nature

Jason Scott Robert

Model organismism—the over-reliance on model organisms without sufficient attention to the adequacy of the models—continues to hobble our understanding of human brains and behaviors. I outline the problem of model organismism in contemporary biology and biomedicine, and discuss the virtues of a genuinely comparative biology for understanding ourselves, our evolutionary history, and our place in nature.

Keywords: Model Organisms; Nature vs. Nurture; Neuroscience

1. Introduction

The sun has long since set on the heyday of the nature–nurture controversy. There are virtually no hard-line genetic or socio-environmental determinists anymore. Geneticists and social scientists both recognize the context-sensitivity of gene action and activation, and the multiple determination of phenotypes through the interactions of biological and social factors. Indeed, a kind of compromise position has emerged, an ‘interactionist consensus’ according to which “nature *versus* nurture” has been jettisoned in favor of “nature *via* nurture” (Ridley, 2003) or, slightly more happily, “nature *and* nurture in complex interplay” (e.g., Gray, 1992; Oyama, 2000; Kitcher, 2001; Moore, 2002; Robert, 2003, 2004a; Rutter, 2006). Yet despite this apparent breakthrough, pressing and challenging tasks remain in contemporary biology and psychology. Chief among them are the need to unpack the interactionist consensus and to interrogate the mechanisms of organismal and behavioral development and their evolution (Robert, 2004a, in press-a).

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In this essay, I explore one persistent obstacle for moving beyond the sterile sequelae of the nature–nurture disputes in understanding human beings, and especially human brains: the over-reliance on model organisms. I will briefly discuss the limits of model organism-based human biology, and then elaborate the promise of a more comparative approach for shedding light on ourselves, our history, and our relationships with other animals. This is a programmatic exercise in the practical philosophy of science—and specifically neuroscience, neuropsychology, and psychobiology—which I hope will serve to provoke further and more detailed studies at the leading edge of contemporary science.

2. Studying Human Nature by Studying Nonhuman Animals

In November 2006, the National Academy of Sciences sponsored a prestigious Sackler Colloquium on *The New Comparative Biology of Human Nature*. The aims were 2-fold, one primary and substantive and the other secondary and more pragmatic. The primary, substantive aim was to explore the benefits and limitations of model organisms in understanding the human brain, and so to determine the desirability and even the necessity for comparative studies (especially with our closest living relatives). The secondary, more pragmatic aim was to assess levels of scientific support for large-scale comparative research with chimpanzees, which might require overturning the National Institutes of Health’s moratorium on captive breeding of chimpanzees. The format of the meeting was as follows: (1) frame the topic from the perspective of the history and philosophy of science; (2) solicit scientific presentations focused on the justification of research programs (why model organisms? why *this* model organism? why comparative approaches? why *these* particular comparisons?); and (3) generate philosophical, historical, and scientific reflection on (2) on the basis of (1). I was a plenary speaker at this meeting, along with my colleague Jane Maienschein, as well as Cheryl Logan of the University of North Carolina at Greensboro; we were primarily responsible for (1), that is, for framing the issues historically and philosophically.

Unfortunately, despite significant interest in the driving questions we laid out, and despite the participation of excellent scientists, the colloquium was not as successful as we had hoped in meeting either of the articulated aims. Though the framing session proceeded as planned, the ensuing scientific presentations failed to address the key questions, but rather, for the most part, focused on the researchers’ own particular experimental data. Instead of *reflecting on* and *justifying* their research programs/approaches/techniques/choices, the presenters mainly *described* their findings. This had two negative effects for the colloquium: first, the primary, substantive aim simply was not met; second and accordingly, there was little motivation to support the more pragmatic aim, as no one was able to explain why more comparative research is a good thing for science or for society. (That said, the science presented was interesting and first-rate. I learned a lot at this meeting. But the content of the presentations was not what we had expected.)

One plausible hypothesis to explain the failings of this colloquium is that, beyond their particular research paradigm, scientists may be insufficiently equipped to ask and answer conceptual, epistemological, and methodological questions about their research program—at least in this kind of forum. This hypothesis, if true, would be somewhat worrisome, given that much of this research is supported through publicly-funded grants. Another plausible hypothesis is that Maienschein, Logan, and I failed to motivate the discussion, or simply failed to elucidate the sheer importance of the discussion for contemporary biology and biomedicine. In case that were so, my brief here is to make the argument more strongly.

3. Model Organismism in Biology and Biomedicine

Biologists, historians, and philosophers have generated a large and fascinating literature on the use of experimental creatures (rodents, mammals, fish, bacteria, viruses, plants, and so on) as ‘model organisms’ (e.g., Ankeny, 2000, 2001; Bolker, 1995; Bolker & Raff, 1997; Burian, 1993; Creager, 2001; Gest, 1995; Gilbert, 2001; Gilbert & Jorgensen, 1998; Jenner & Wills, 2007; Kohler, 1994; Logan, 2002; Preuss, 2000; Rader, 2004; Robert, 2004b; Saikkonen, Lehtonen, Helander, Koricheva, & Faeth, 2006; Schaffner, 1998). There are a variety of uses of ‘model’ at play in these and related discussions. For instance, models may be ‘real’ or theoretical—the latter would include mathematical models, for instance. Here, ‘real’ models are of interest: physical instantiations of target phenomena. Moreover, a ‘disease model’ may be quite distinct from a ‘model of synapse formation’ or a ‘model of limb development.’ Though both are designed to facilitate experimentation and generalization, their epistemological and methodological roles differ. The former is meant to serve as a proxy, as a living replica (however inaccurate) of some disease process in humans; to cure the disease model is (putatively) to go some way toward curing the disease. The latter are meant instead as a kind of prototype or archetype for understanding normal function or behavior, whether in humans, mammals, vertebrates, or whatever. Also, not all experimental animals are animal models, and not all animal models are model organisms—the latter honorific tends to be applied only to those officially sanctioned by the National Institutes of Health or other funders and around which a model ‘system’ has grown (including research networks, literatures, databases, and so on).

In developmental biology, for instance, only a few experimental animals have achieved feature performer status as model organisms, including mice, frogs, chicks, and zebrafish. Rats are especially popular in psychology, fruit flies and nematodes in genetics and molecular and cellular biology, and so on. While there are all sorts of contingent historical reasons for the establishment of these particular animals, there are also contingent epistemological reasons at work.

First, model organisms thrive in laboratory contexts and are generally inexpensive to raise and maintain—they are *lab-friendly*. Second, model organisms yield to laboratory analysis—they are experimentally *tractable*. Third, model organisms are *standard*—the organisms that everyone uses. These features help to explain the

pragmatic advantages afforded by model organism research. But they also introduce a range of distinct disadvantages, including developmental simplicity that does not always translate well to more developmentally complex organisms. Indeed, model organisms are tailor-made (literally custom-produced) for analysis, and are “both selected and selectively fashioned in order to make experiments work” (Robert, 2004b, p. 1008). Accordingly, they may exhibit characteristics (mechanisms, morphologies, behaviors) that are available on demand in the lab and yet nonexistent in nature. Also, these standardized characteristics that are reliably elicited on demand tend not to evince any of the variation naturally exhibited by other organisms. Moreover, where the organisms selected as model organisms are not developmentally simple, they may nonetheless be developmentally *different* in interesting ways—and this may be the reason they were anointed in the first place (Bolker, 1995).

Additionally, while the continuity of material resources across labs makes it much easier for scientists to replicate (or disconfirm) each others’ results, this also introduces a kind of insularity and even one-dimensionality into contemporary biology: scientists who use model organisms are limited to studying only those aspects of biology that are reproducible in those particular organisms—at the expense of studying everything else in nature. Sometimes, general lessons can be extracted, but any scientific inference on the basis of model organism research must be cautiously drawn. If the model organism—or even all model organisms taken together—were a model of everything of interest, then this would be less of an issue. But no model organism is a *model* model in this sense, and the current set of model organisms is phylogenetically skewed enough to cast serious doubt on the capacity of model organism-based research to reveal the intricacies of genetics, development, or evolution in any comprehensive way. (See Jenner & Wills, 2007 for a demonstration of the uneven phylogenetic distribution of the six major model organisms in developmental biology.)

As indicated above, remarks such as these are not new under the sun. And yet *model organismism* persists as the guiding framework of contemporary biomedicine and some parts of biology, too. Why that should be the case is a fascinating sociological and epistemological question, to which future research must be squarely directed. For now, let us stipulate that model organismism refers to the research methodology according to which scientists use model organisms in experimental research to accomplish pragmatic aims—making experiments work—in the absence of adequate attention to whether those experiments hook on to anything in the world, let alone the phenomenon of experimental interest. When we forget the limits of model organisms in explaining, say, development, we are prone to errors in inference that are both subtle and powerful in biasing our understanding. Of course, not every model organism experimentalist is a model organismist. But, it seems, plenty of them are.

How does model organismism matter to the debates about nature and nurture? First, it encourages a potentially inappropriate reductionism (not just methodological but epistemological and even ontological) about development, according to which development is nothing but gene activation, whether in *E. coli*, elephants,

or you and me (Robert, 2003; see also Robert, 2004a). This is because the animals are selected and bred to be robust in the laboratory and so insensitive to environmental factors; they are specifically designed to exhibit genetically manipulable characters on demand (e.g., Gilbert & Jorgensen, 1998). Second, it places undue emphasis on the putative similarity between organisms—and the putative universality of developmental mechanisms—as against the differences between organisms that might justify another approach. Third, and as a result of these first two influences, it biases not just what we do know but even what we *can* know by validating some research questions and approaches and invalidating others. In particular, one key fallout of model organismism is the subtle translation of the original problem or question into a new question that is tractable via model organisms but non-identical to the problem or question of interest (see Robert, 2003; cf Wimsatt, 1986). In the next section, I explore how this happens in the special case of model organismal approaches to human disease, but the lesson is more general than that.

4. Model Organismism in Practice: The Case of Parkinson's Disease

Consider the case of Parkinson's Disease (PD). Cell transplant research for PD is premised on the notion that a hallmark feature of PD—the loss of dopaminergic neurons in the substantia nigra—may potentially be mediated by the transfer of new dopaminergic neurons, or of other cells that would promote the production or prevent the loss of dopaminergic neurons. While some clinical studies of cell transplant research for PD have been undertaken with fetal ventral mesencephalic cells harvested from aborted fetuses, the results have been equivocal (Freed et al., 2001; Olanow et al., 2003; cf Mendez et al. 2002, 2005). As this research continues, a new paradigm is emerging: using neural stem cells derived from embryonic or other sources. As part of the Model Systems Strategic Research Network funded by the Canadian Stem Cell Network, Françoise Baylis and I convened a series of collaborative workshops with stem cell researchers interested in cell transplant research. We focused on PD given its perceived status as 'low-hanging fruit' in the domain of neural stem cell research. Beyond the intricacies and challenges of cell transplantation, a more basic concern came into focus through the workshops: How well is PD understood on the basis of nonhuman animal models of the disease? And how is our current understanding of PD in humans biased by our animal models?

PD researchers primarily use variants of two animal models, both of which happen also to be model organisms: the 6-hydroxydopamine (6-OHDA) rat model and the 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) nonhuman primate model, which is typically a rhesus macaque (Emborg, 2004). While there are other animal models in use in PD research, rat and macaque models have pride of place. We hosted collaborative workshops first with those who study 6-OHDA rat models, and then with those who study MPTP macaque models. The results were interesting, though not at all surprising: at the former workshop, investigators

emphasized that we already have proof of principle in primates that rodent models are suitable, and perhaps that there is no need for non-human primate models; at the latter workshop, investigators emphasized the distinct limitations of rodent models, and insisted on the propriety—indeed, the necessity—of nonhuman primate research. The *differences* between humans and both rodents and nonhuman primates were only rarely emphasized, perhaps because of the ongoing penchant to anthropomorphize inappropriately (Povinelli, 2004) and to indiscriminately lump together nonhomologous behaviors and traits (Robert, 2004a).

At both workshops, and in our own deliberations, we explored the further possibility that neither the rodent nor the primate model of PD is particularly good. What these models model or, rather, approximate, is a subset of the motor symptoms of PD induced primarily via acute lesion. That is, they model some of the motor deficits of traumatically-induced parkinsonism, a motor phenotype that looks like PD in some respects but may be fundamentally different. PD in humans is (a) more than just these motor deficits, including as well cognitive and affective components, and (b) chronic and degenerative, not acute and static. Accordingly, if cell transplantation ‘works’ in rats or macaques to ‘cure’ some motor symptoms, we may not thereby be licensed to infer anything about cell transplantation in humans as a treatment for PD. That is, treating parkinsonism in these animal models is simply not comparable, let alone equivalent, to treating PD in humans.

Some symptoms of parkinsonism are resolvable via model organisms and these particular animal models are a case in point. But they bias our understanding and skew the research enterprise. For the task was never to resolve some symptoms of parkinsonism; it was, instead, to understand, treat, and even cure PD. Phenotypic parkinsonism in model organisms is not equivalent to PD in humans, and we should not delude ourselves into believing that the models resolve the original problem (PD) rather than resolving something else (parkinsonism). And yet, with research energy focused on these at-best partial models of the disease, the complexities of human PD are ignored—which helps to explain the lack of dramatic progress toward better treatments and cures for PD (Philips, 2004).

5. Beyond Model Organismism

Differences between species tend to be ignored and similarities tend to be overemphasized within model organismism. These twin tendencies suggest that comparisons are implicit and limited rather than explicit and more comprehensive. Where the quest for universal mechanisms is undertaken in the absence of a similar quest for (evolved) specializations, there are obvious limits to what we will learn. The presumption of similarity is dangerous, blinding us to the importance of understanding difference—the very essence of life.¹ Todd Preuss raises an interesting problem in this regard:

The ubiquity of variation seems to pose a challenge to animal research, for if every species is unique, how can we hope to learn anything about humans by studying

other animals? I suggest that this dilemma is more apparent than real. The fact that variation is extensive does not mean that there are no important cross-species commonalities. After all, rats do possess many features of cortical organization found widely among mammals. The problem is that we cannot be sure that any *particular* feature of rats is a widespread feature of mammalian organization by studying rats alone. (Preuss, 2000, p. 295)

But neither will comparing rats with mice or monkeys, given that all of these models have often been selected for experimentation for extra-scientific reasons, and on the assumption of fundamental similarity. Barely comparative biology is the most that model organismism typically affords; here, various model organisms (or their genomes) may be compared with one another, against the backdrop of the assumption of universal sameness.

An interesting feature of some scientific discussions of the model organismal approach in understanding life is the repeated reference to Hans Krebs' invocation of the 'August Krogh principle' that "for many problems there is an animal on which it can be most conveniently studied" as a justification for model organismism (Krebs, 1975; for discussion, see especially Gest (1995) and Jørgensen (2001)). Although many scientists (especially but not exclusively physiologists) interpret this as model organismal *carte blanche*—to study autism in voles, neural development in worms, or anything whatsoever in mice—they have also often either misinterpreted the principle or restated it so as to misrepresent its meaning (as intended by Krebs or by Krogh). Jørgensen (2001) cites telling examples, such as Feder and Watt's (1992) assertion that Krogh maintained that 'for every biological question is an organism best suited to its solution' and Randall, Burggren, and French's (1997) more elaborate (and fundamentally misleading) claim that:

One of the reasons for Krogh's extraordinary success as a physiologist was his uncanny ability to choose just the right experimental animal with which to test his hypotheses. His view was that for every defined physiological problem, there was an optimally suited animal that would most efficiently yield an answer. (Randall, Burggren, & French, 1997, p. 15)

Jørgensen returns to Krogh's 1929 address to the International Physiology Congress to show instead that Krogh was not only not a model organismist, but was a comparative physiologist to the core: for Krogh, understanding 'the essential characteristics of matter in the living state' will require 'the study of the vital functions in all their aspects throughout the myriads of organisms'; moreover, he maintained, "we will find out before very long the essential mechanisms of mammalian kidney function, but the general problem of excretion can be solved only when excretory organs are studied wherever we find them and in all their essential modifications" (Krogh, 1929, as cited by Jørgensen, 2001, pp. 59–60).

This is, of course, not to deny that certain organisms really are well-suited to particular lines of inquiry, problem agendas, or experimental designs. But no organism is uniquely suited to all of these, and whether any particular organism is well-suited in any particular situation depends entirely on the details of that situation: What do we already know, what do we want to know, and how are we

going to go about knowing it? As Krebs (1975) pointed out 30 years ago, 'A general lesson to be learned from these considerations is the importance of looking out for a good experimental material when trying to tackle a specific biological problem'. And yet that general lesson has not been universally well learned, as scientists too often alter the question to suit the model rather than selecting an appropriate model to suit the question.

How else to explain the continued penchant not only for a small number of model organisms, but also resistance to the establishment of new models, and even attempts to further narrow the number of sanctioned model organisms within the biomedical research enterprise? With regard to the latter, consider the limitation of 'nonhuman primate' research to macaque monkeys (and, where budget is an issue, to marmosets), and also the rise of transgenically humanized mice as putatively universal models for all biological and biomedical phenomena of interest (Shultz, Ishikawa, & Greiner, 2007; cf Rissman, 2005). In the background, there are ethical concerns at play, especially regarding invasive research with many animals, not least with nonhuman primates—though it is often unclear why noninvasive experimental or even observational research with nonhuman animals should be in question. While ethical deliberations, and political pressure, may eventually decide the limits of scientific experimentation, there are at present more scientific options than mouse, macaque, marmoset, man, or bust.

In her excellent study of the limits of model organisms in developmental biology, Jessica Bolker argues that the assumption of universality 'has consequences at two levels':

At the level of data accumulation, we lack knowledge of the existing diversity in developmental patterns and processes. At the conceptual level, our ignorance of developmental variability and diversity leads to an overly deterministic view of development, and to a concomitant narrowing of focus to proximate, internal mechanisms. (Bolker, 1995, p. 453)

Sound inferences are, accordingly, unlikely at best. The dearth of comparative data, coupled with genetic reductionism and ignorance of the total ecology of the genome—that is, that there is more to an organism than its genome, and that the genome has meaning only in developmental and environmental context—suggests that our understanding of fundamental biology based on model organisms is not only incomplete but fundamentally biased (Robert, 2004a; see also West & King, 1987, on the 'ontogenetic niche').²

6. Toward a More Comparative Biology and Biomedicine

Mere comparisons are not enough to move us beyond this lacuna. For comparing apples with apples, or with oranges or watermelons, tells us nothing about other kinds of fruit, let alone vegetables, legumes, or anything else under the sun. Only a rigorously comparative biology and biomedicine, operating at multiple levels of

organization and analysis, and grounded in both ecological context and in evolutionary considerations about relatedness and divergence, can begin to shed adequate light on life itself, and on our peculiarly human form of it.

While it is of course possible to make comparisons absent evolutionary hypotheses and phylogenetic frameworks, this is not advisable as a way forward in biology and biomedicine. Neither is it advisable simply to make assumptions about evolutionary history (say, about similarity or universality of forms or functions). Taking evolution seriously is critical here (in press-a, in press-b). So too is taking development seriously (Robert, 2004a).

In the specific context of the brain sciences, Povinelli (2004) laments the current status of comparative neuroscience:

Comparisons of the brains of humans and apes have traditionally been limited to gross considerations such as size and surface features (such as lobes and sulcus patterns). Remarkably, the details of the internal organization of human and great ape brain systems and structures have been largely ignored, in part because it's so difficult to study these brains, but also because most neuroscientists have frequently assumed that despite great differences in size, all mammalian brains are organized pretty much the same. (Povinelli, 2004, p. 31)

Continuing in the same vein, Preuss (1995, 2000, 2006) is similarly concerned about the limits of our current understanding. The abstract of his chapter in a key textbook of cognitive neurosciences exemplifies this perspective:

Traditionally, many neuroscientists have supposed that all mammals possess variants of the same brain which differ only in size and degree of elaboration. Under this model, the brains of nonhuman species can be treated as simplified versions or models of the human brain. However, there is evidence that mammalian cerebral organization is much more variable than is commonly acknowledged. The diversity of mammalian brain organization implies that neuroscientists can make better inferences about human brain organization by comparing multiple species chosen based on their evolutionary relationships to humans, than by studying individual 'model' or 'representative' species. The existence of neural diversity also suggests that nonhuman species have evolved cognitive specializations that are absent in humans. (Preuss, 1995, p. 1227)

Accordingly, as Gilbert Gottlieb and Robert Lickliter have observed, it is “dubious that nonhuman primates can serve as models for the most distinctive of human cognitive abilities, any more than we could serve as good models for their distinctive traits” (Gottlieb & Lickliter, 2004, p. 317).

A rigorously comparative approach to neuroscience would overcome the limits of model organismism by beginning with an assumption of neural diversity across mammals and other animals, and by selecting a broad range of experimental organisms for study on the basis of phylogeny and not just ideology, convention, or laboratory-friendliness. The question—not the research material—would drive the investigation. Detailed analysis would be undertaken at various levels of organization across multiple disciplines, and under systematically serially varied ecological and

developmental conditions. Experimental designs would challenge key assumptions, query orthodoxies, and yield results that would reveal the virtues and biases of our current knowledge base in the neurosciences.

My claims here are not intended as a condemnation, but rather as a challenge: to reinvent the biological and biomedical research enterprise so as to generate genuine understanding of human and nonhuman-animal nature, cognition, development, and evolution, in sickness and in health. The challenge is, obviously, an immense one. Sometimes, model organisms or other animal models will prove particularly useful to the task at hand; not all research with model organisms is necessarily problematic. But where such research presumes rather than demonstrates similarity (or dissimilarity), constrains the research agenda, or otherwise biases our knowledge, a different approach is in order—as many others before me have presciently but vainly maintained. The nature of this different approach remains to be fully elucidated, in part empirically, in part conceptually, in part methodologically, but also in part ethically. For if, at the end of the day, there is no substitute for research with our closest nonhuman primate relatives or, indeed, with humans ourselves, then the *moral* and not merely epistemic justification of scientific research will take on ever greater significance.

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Notes

- [1] Similarity and difference are, of course, complex concepts. Judgments of similarity or difference are a function of the resolution and the presumed background for comparison. Things that appear dramatically similar from a distance may appear totally different up close; things that appear dramatically different against one background may appear significantly similar to each other against another. Indeed, how like or unlike entities actually are may be indeterminate, subject to variation based not only on resolution and background but also methods and research strategies. This is not a problem unique to model organismism and comparative neuroscience, but rather a generic philosophical issue. Thanks to Karola Stotz for discussion of these issues (personal communication, February 27, 2008).
- [2] This last point is especially important, though not unique to model organismism: any laboratory experiment involves simplification of the developmental (rearing) context, and this, too, may bias our knowledge base. Biases may occur where the simplifications are noted for the record but ignored for interpretive purposes (Robert, 2003; Robert, 2004a), or when the experimental set-up appears benign but turns out to be problematic. As an example of the latter, Gilbert (2001) reports on research with methoprene, a juvenile hormone

mimicking substance present in pesticides. The pesticides appear entirely harmless when lab organisms (in this case, *Xenopus* eggs) are exposed to them. But methoprene functions as a teratogen when exposed to sunlight, generating deformities in tadpoles in the wild. So, what we thought was true under laboratory conditions turns out to be false under ecologically realistic conditions. How widespread or significant such biases remains to be fully explored, but initial evidence suggests the need for correctives (Gilbert, 2001; West & King, 1987).

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