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EvoDevo and Niche Construction: Building Bridges

KEVIN N. LALAND[1]*, JOHN ODLING-SMEE[2], AND SCOTT F. GILBERT[3]
[1]*School of Biology, St. Andrews University, Fife, United Kingdom*
[2]*School of Anthropology, Oxford University, Oxford, United Kingdom*
[3]*Department of Biology, Swarthmore College, Swarthmore, Pennsylvania*

ABSTRACT Evolutionary developmental biology and niche-construction theory have much in common, despite independent intellectual origins. Both place emphasis on the role of ontogenetic processes in evolution. The same historical events shaped them, and similar philosophical and sociological barriers hindered their respective advances. Both perspectives maintain that neo-Darwinism needs a theory of macroevolutionary variation and that such a theory can now be adduced from developmental biology. Some proponents of both EvoDevo and niche construction propose additional evolutionary mechanisms, and specify a key role for stable extra-genetic forms of inheritance. Similarly, proponents of each lay emphasis on ''reciprocal causation'' in the relationship between organism and environment. We illustrate here how EvoDevo and niche construction could gain ''added value'' from each other, and demonstrate how the niche-construction perspective potentially provides a useful conduit to integrate evolutionary and developmental biology. *J. Exp. Zool. (Mol. Dev. Evol.) 310B:549–566, 2008.*

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The field of evolutionary developmental biology (henceforth EvoDevo) maintains that all metazoans share a common ''tool kit'' of master regulatory genes that govern the formation and patterning of bodies (Gilbert et al., '96; Gilbert, 2003a,b; Carroll, 2005). However, there is more to EvoDevo than the differential expression of homologous genes; practitioners assert that developmental processes bias and constrain evolutionary pathways (Bonner, '81; Hall, '92; Raff, 2000; West-Eberhard, 2003; Arthur, 2004; Sultan, 2005, 2007; Müller, 2007). A subset of EvoDevo has given rise to ecological developmental biology, which stresses the roles of developmental plasticity in evolution, especially in the formation, preservation, and prevention of novelty. The focus is the ability of the developing organism to sense cues from its environment and to modify its development to become more fit in a particular habitat (Schlichting and Pigliucci, '98; Gilbert, 2001, 2003a; West-Eberhard, 2003, 2005; Müller, 2007).

EvoDevo has much in common with niche-construction theory, a fledgling branch of evolutionary biology that places emphasis on the capacity of organisms to modify sources of selection in their external environment and thereby act as codirectors of their own, and other species', evolution. Niche construction is defined as ''the process whereby organisms, through their metabolism, their activities and their choices, modify their own and/or each other's niches'' (Odling-Smee et al., 2003, p 419). The niche-construction perspective was introduced to evolutionary biology in the 1980s (Lewontin, '82, '83) and has recently gathered momentum (Odling-Smee, '88; Laland et al., '96, '99; Odling-Smee et al., '96, 2003; Lewontin, 2000; Oyama et al., 2001; Sterelny, 2003, 2007; Boni and Feldman, 2005; Donohue, 2005; Lehmann, 2008). Examples of niche construction include animals manufacturing nests, burrows, webs, and pupal cases, plants changing levels of atmospheric gases and modifying nutrient cycles, fungi decomposing organic matter, and bacteria fixing nutrients (Wcislo, '89; Jones et al.,

*Correspondence to: Kevin N. Laland, School of Biology, St. Andrews University, Bute Medical Building, Queen's Terrace, St. Andrews, Fife KY16 9TS, UK. E-mail: knl1@st-andrews.ac.uk

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'94, '97; Odling-Smee et al., 2003). Some organism-driven changes in environments persist as a legacy to modify selection on subsequent generations—an ''ecological inheritance'' (Odling-Smee, '88). The perspective is backed by theoretical population genetic and experimental findings that suggest that niche construction is evolutionarily consequential (Laland et al., '96, '99; Odling-Smee et al., 2003; Donohue, 2005; Silver and Di Paolo, 2006; Lehmann, 2008).

Ecological developmental biology looks at the converse relationship. It posits that environments, as well as genes, are sources of cues for the construction of phenotypes. Whereas classical evolutionary theory sees the organism as the key that has to fit into the environment's lock, both ecological developmental biology and niche construction see interactions between them. Niche construction emphasizes the ability of the organism to alter its environment; eco-devo emphasizes the ability of the environment to alter the developing organism.

Both EvoDevo and niche-construction theory maintain that refinements are required in the modern theory of evolution. Many EvoDevo practitioners believe that evolutionary theory is incomplete—or, at least, was at the time of the modern synthesis and that it is only becoming complete with the insights gained from their own field. Some EvoDevo advocates believe that additional mechanisms to those of neo-Darwinism, mechanisms involved with ontogeny, are required for a full understanding of evolution (Gilbert et al., '96; Raff, 2000; West-Eberhard, 2003, 2005; Amundson, 2005; Müller, 2007; Pigliucci, 2008). Similarly, some evolutionary biologists are explicit about the need to treat niche construction as both a developmental process and a cause of evolution (Odling-Smee et al., 2003). Both EvoDevo and niche-construction theory have ''complained'' about the synthesis' treatment of development as a ''black box'' (Hamburger, '80) that transforms naturally selected genes into functional phenotypes. Until very recently, attempts to show that the environment is critically involved in constructing phenotypes were marginalized (Gilbert, 2003b). Seemingly, evolutionary biologists have given themselves licence to ignore development:

> One consequence of Weismann's concept of the separation of the germline and soma was to make it possible to understand genetics, and hence evolution, without understanding development (Maynard-Smith, '82, p 6).

Fisher ('30), Dobzhansky ('37), and Mayr ('63) all make similar assertions. This treatment has engendered disquietness among many development biologists, who stress that one must understand how bodies are built in order to understand how the process of building bodies can be changed (Amundson, 2005; Carroll, 2005). EvoDevo advocates proclaim that neo-Darwinism had no theory of macroevolutionary variation, but that developmental biology can now supply this essential ingredient (see Gilbert et al., '96; Arthur, 2004; Minelli and Fusco, 2004; Müller and Newman, 2005a,b; Gilbert, 2006). Some development-minded evolutionists have argued that developmental processes constitute significant but neglected evolutionary mechanisms in their own right (Gould and Lewontin, '79; West-Eberhard, 2003). For instance, micro- and macroevolutionary patterns are viewed as shaped by developmental constraints (Gould and Lewontin, '79; Arthur, 2004), whereas developmental plasticity is perceived to provide phenotypic variants that can be later stabilized by genetic networks. ''Genes are followers, not leaders'' (West-Eberhard, 2003).

With striking parallels, advocates of niche construction suggest that it directs, regulates, and constrains the action of selection and is a source of evolutionary innovation (Odling-Smee et al., 2003; Sterelny, 2007). The only major difference between the arguments of these fields is in the domain (internal vs. external environment) in which they are deemed to act. Indeed, some researchers (Lewontin, '83, 2000; West-Eberhard, 2003, 2005; Jablonka and Lamb, 2005) and philosophers of biology (Griffiths and Gray, 2001, 2004; Oyama et al., 2001; Sterelny, 2007) have contributed to both debates. Representatives of both disciplines stress that developmental processes are neither predictable from the properties of genes nor well described as an unfolding genetic program (Lewontin, '82, '83; Nijhout, '99; Gilbert, 2001). Both suggest that functional, even complex, evolutionary innovations can occur without a long history of cumulative selection (Keys et al., '99; Odling-Smee et al., 2003; West-Eberhard, 2003, 2005; Müller and Newman, 2005a,b; Sterelny, 2007). In addition, representatives of both disciplines postulate that extragenetic forms of inheritance can be critical by affecting both normal development and evolutionary outcomes (Odling-Smee et al., 2003; Gilbert, 2004; Jablonka and Lamb, 2005). This has become a key point of emphasis of ecological developmental biology and developmental systems theory

(Gilbert, 2001, 2003a; Oyama et al., 2001; West-Eberhard, 2003). Given their shared agendas, we suggest that there is considerable potential for mutually enlightening interactions between the fields.

PHILOSOPHICAL BACKGROUND

The history of the relationship between the fields of developmental and evolutionary biology is well researched and multifaceted (Amundson, 2005; Laubichler and Maienschein, 2007a,b; Müller, 2007). The relationship involves not only just the marginalization of development, and hence niche construction, by evolutionary biology, but also the comparative neglect of evolutionary issues by developmental biologists. Our goal in revisiting these debates is not to apportion blame to particular camps, but to consider some philosophical issues that lie at the heart on the neo-Darwinian treatment of development (Lewontin, '83; Amundson, 2005), which reveal that the neglect of development and niche construction within evolutionary biology are historically linked.

Evolutionary causes

Responding to structuralist critics, evolutionary biologist and synthesis architect Mayr ('84, p. 126) wrote:

> All of the directions, controls and constraints of the developmental machinery are laid down in the blueprint of the DNA genotype as instructions or potentialities.

For Mayr, developmental processes cannot be regarded as independent causes of evolutionary events, as their characteristics, including their ability to control and constrain, are fully explained by the earlier natural selection of genes. If developmental processes direct evolutionary events, this is only the proximate manifestation of the ultimate cause, namely natural selection (Mayr, '61). The legacy of this view within contemporary evolutionary biology is that those aspects of development that have not been shaped by selection are perceived to play no evolutionary role, whereas those that have are viewed as mere vehicles for gene replication. Of course, the notion that developmental causation starts with the gene remains shared by many developmental biologists, including some advocates of EvoDevo. But this has only served to perpetuate the notion that developmental processes are not evolutionary causes, but merely products of selection.

The same conceptual barrier has hindered evolutionary biologists' acceptance of niche construction (Laland and Sterelny, 2006), which is also perceived to have no *independent* evolutionary significance (Dawkins, 2004). Here, niche-construction effects are treated as extended phenotypes, and extended phenotypes play the same role in evolutionary biology as ordinary phenotypes: affecting the replication of alleles responsible for those phenotypic effects (Dawkins, 2004). This stance attributes all causal significance associated with niche-constructing activity to genes or genotypes, and the ''ultimate cause'' (Mayr, '61) of such genes and their effects is earlier selection.

There are two major problems with this reasoning (Laland and Sterelny, 2006). First, although much niche construction is influenced by earlier selection, it does not follow that *all* evolutionarily consequential niche constructions are under genetic control. For instance, there are no genes for dairy farming (using ''genes for'' in the sense of Dawkins, '76), a cultural manifestation of human niche construction, yet in spite of the fact that this activity is not a product of natural selection, it has had a clear evolutionary consequence, leading to selection of the gene for lactose absorption (Durham, '91). Other human genes also appear to have been selected as a direct result of human cultural niche construction, including sickle cell *S* allele, G6PD, TNFSF5, CCR5, AGT, CYP3A and alleles coding for hemoglobin C and Duffy blood groups (Balter, 2005; Wang, 2006).

Logically identical examples are found in other species. Hundreds of species of mammals, birds, and fishes are now known to learn socially, with knowledge and skills spreading through populations, often modifying selection (Zentall and Galef, '88; Heyes and Galef, '96; Laland and Janik, 2006). Niche construction reliant on *asocial* learning is also sufficient to modify selection. For instance, Galapagos woodpecker finches create a woodpecker-like niche by learning to use a cactus spine or a similar implement to peck for insects under bark (Tebbich et al., 2001), selecting for a bill able to manipulate tools rather than the sharp, pointed bill characteristic of woodpeckers. The finch's capacity to use spines to grub for insects is not itself an adaptation and is not guaranteed by the presence of naturally selected genes: rather, the bird exploits a more general and flexible adaptation—the capacity to learn—to develop the

necessary skills. More generally, any mechanism of phenotypic plasticity in conjunction with reliably present signals from the environment can generate the same niche-constructing activity generation by generation, with evolutionary consequences, without that activity itself being an adaptation, or precisely specified by genes (West-Eberhard, 2003). Mechanisms other than mutation can underlie evolutionary novelties (Müller and Wagner, 2003).

Changes in niche construction that are not attributable to changes in genes, nor inherited genetically, can still trigger evolutionary episodes. Even a single isolated niche-constructing event can be evolutionarily consequential. Consider dispersal into a new environment, where descendants of the dispersing organisms will, for multiple generations, ''inherit'' modified selection. Moreover, niche-constructed resources and artifacts can last considerably longer than individual organisms, generating carry-over effects across generations. It is common for nests of birds and insects to outlive their builders (Hansell, '84). At the extreme, Watson ('67) describes a termite mound 20 m across, still occupied by *Macrotermes goliath*, on which archaeological remains dated at 700 years had been built! Additionally, social learning provides an alternative inheritance system (Zentall and Galef, '88; Fragaszy and Perry, 2003; Jablonka and Lamb, 2005), and there is strong empirical and theoretical support for the hypothesis that it, together with other forms of learning, is evolutionarily consequential (Kirkpatrick and Dugatkin, '94; Laland, '94; Lachlan and Slater, '99; Beltman et al., 2003, 2004; ten Cate and Rowe, 2007). These factors combine to render those changes in development that are not attributed to changes in genes, nonetheless capable of generating stable changes in selection pressures.

Although natural selection and niche construction are reciprocal interacting processes, they are logically distinct. Niche-constructing traits need not be products of earlier selection. The fact that some niche construction is not reducible to earlier selection illustrates that niche construction cannot legitimately be dismissed as a mere product of selection.

Similarly, for many developmental biologists, the cause of phenotypic characters includes not just genes but complex inductive interactions among various parts of the developing organism and the external environment (Gilbert, 2003a), often hierarchical in nature (Love, 2006). Differential gene expression is orchestrated by ''fields,'' ''organizers,'' ''niches,'' ''signalling pathways,'' often fed by external environmental cues, with each stage of development highly contingent on the state of the organism at the previous stage, and each cell's activity dependent on the state of neighboring cells (Gilbert et al., '96; Amundson, 2005; Kirschner and Gerhart, 2005).

Second, even if it were the case that the development were strictly under genetic control, it still need not follow that development be regarded as evolutionarily inconsequential. In many cases the ''controlling genes'' may themselves have been selected as a result of development-induced changes in the selective environment (Laland and Sterelny, 2006). For example, it is often assumed that the ultimate explanation for why earthworms modify soils is that selection has furnished them with genes for burrowing, tunnelling, and so forth. However, it is no more than a convention within evolutionary biology that natural selection should be regarded as the ultimate cause of such phenotypic characters, a convention that historians of science have dated to an article written by Mayr in 1961 (Amundson, 2005). From the perspective of a philosophical consideration of causation, one could well argue that the ultimate cause of the selection pressures that favored earthworm soil-processing adaptations is the earlier niche construction of ancestral worms, without which there would be no soil environment to act as a source of selection–reasoning that we would regard to be equally flawed (Odling-Smee et al., 2003). The convention brought to prominence by Mayr is not the only way to think about causation in biology. An equally tenable approach, which we advocate, is to adopt ''reciprocal causation'' in evolutionary explanations, in which the characteristics of organisms are regarded as caused by interacting bouts of selection and construction (Griffiths and Gray, 2001, 2004; Oyama et al., 2001; Odling-Smee et al., 2003; West-Eberhard, 2003; Laland and Sterelny, 2006; Oyama, 2006). One important ramification of reciprocal causation is that it is philosophically sound to argue that developmental processes can be evolutionarily causal, as they are not regarded as fully caused by earlier selection on genes.

Dichotomous thinking

Amundson (2005) identifies four dichotomies that lie at the heart of population genetic thinking, and that have been used to dismiss any active role

for development in evolution. These are: (i) genotype vs. phenotype (see Mayr, '84, quote above), (ii) germ line vs. soma (see Maynard-Smith, '82, quote above), (iii) proximate vs. ultimate (witness Mayr's, '92, response to structuralist critics), and (iv) typological thinking vs. population thinking (witness Wallace's, '86, critique of structuralist critics). These dichotomies remain prominent and widespread within many areas of contemporary biology (Amundson, 2005), and inadvertently act as barriers to the satisfactory integration of developmental and evolutionary biology. Nonetheless, the dichotomies have been under attack. Gilbert et al. ('96) argued that development is the necessary link between genotype and phenotype, and that the dichotomy between functional and evolutionary biology specifically left out development. Winsor (2007) has argued that the ''population thinking'' vs. ''essentialist thinking'' paradigm is a ''myth'' with ''scant basis in fact,'' and that it was proposed in order to confine the study of evolution within species boundaries and to have it require taxonomic skills. But undermining false dichotomies is not enough. As Amundson (2005) argues, ''*something new is needed*'': we suggest that this something new is reciprocal causation, as currently exemplified by niche construction and ecological developmental biology. Dichotomous thinking hinders any recognition of development as evolutionarily consequential, and such dichotomous thinking is undermined by niche construction.

(*i*) *Genotype/phenotype*: From the conventional evolutionary perspective, genotypes cause phenotypes, and the embryological causes that intervene between genotype and phenotype are irrelevant to the study of either heredity or evolution (Amundson, 2005). With niche construction, both evolution and development are perceived as interactive processes in which the developing organism is itself a codirector of change. Moreover, as in Schmalhausen's and West-Eberhard's hypotheses, the phenotype may be evolutionarily prior to the genotype.

Findings from developmental biology also undermine the view that genotype alone instructs the phenotype. For instance, bacteria play an essential component of the epigenetic inheritance system in many animals. Dedeine et al. (2001) have found that the females of the wasp *Asobara tabida* cannot make their oocytes without products being made from the Wolbachia bacteria stored in them: *A. tabida* treated with antibiotics were unable to produce mature eggs. Similarly, mammalian development is not complete without signals from symbiotic bacteria (Hooper et al., 2001; Xu and Gordon, 2003). Mice bred without gut bacteria have aberrant digestive systems and defective immune systems. The bacteria induce gene expression in intestinal epithelia, and these genes are responsible for activating the pathways that allow intestinal capillaries to form and permit lipids to be transported through the cells (Hooper et al., 2001; Stappenbeck et al., 2002). Without these microbes, mice lack the capillary vasculature of the intestinal villi; similarly, rabbits lack their gut-associated lymphoid tissue and therefore have numerous immune problems (Lanning et al., 2005; Mazmanian et al., 2005). Ley et al. (2006) have shown that human babies acquire their gut microbial communities from the vagina and the feces of their mothers early in life. Babies born through Caesarian section had an altered colonization pattern compared with vaginally delivered babies. More generally, symbiotic and commensal relations that affect development, and alter phenotypes and their fitnesses, are not well described by standard assumptions about genotype–phenotype relations.

(*ii*) *Germ line/soma*: From the conventional perspective, there is no direct feedback from soma to germ line (Weissman's barrier) and, accordingly, one does not need to know about development to understand evolution. Conversely, where it modifies selection, niche construction provides an indirect form of feedback from the soma to the germ line of descendant populations. Numerous examples are given above.

Similarly, we can see the accumulation of empirical support for epigenetic inheritance systems, which transmit phenotypes to daughter cells and even across generations (Bird, 2002; Jablonka and Lamb, 2005). For instance, the peloric form of the toadflax *Linaria* is caused by an epigenetically inherited methylation pattern, yet this phenotype has been stably inherited for over 200 years (Coen, '99; Cubas et al., '99). Methylation differences in the genes encoding certain enzymes in the rat liver are mediated by the protein content of the mother's diet during pregnancy. These enzymes help establish the rates of glucose utilization and fat production. Moreover, these methylation differences appear to be inherited (Lillycrop et al., 2005; Burdge et al., 2007). Pathological states can also be transmitted by methylation differences in DNA. Teratogenic factors from the environment can cause disease by altering methylation, and these altered methylation patterns can be stably

inherited (Anway et al., 2005; Chang et al., 2007). Although the notion that epigenetic variation can be transmitted across generations was controversial for decades, empirical data suggest that this may be a relatively common feature of development (Bird, 2007).

(*iii*) *Proximate/ultimate*: From the conventional perspective developmental processes play no independent causal role, as they are merely the proximate manifestation of the ultimate cause, namely natural selection. However, niche-construction theory emphasizes reciprocal causation, rendering developmental processes evolutionarily causal. Likewise, EvoDevo enthusiasts are now stressing reciprocal causation with the environment as a characteristic feature of development (Griffiths and Gray, 2001, 2004; Oyama et al., 2001; Gilbert, 2003a; West-Eberhard, 2005; Müller, 2007). This reciprocal causation also fits well with reciprocal induction in embryogenesis.

A good example is provided by the goldenrod gallfly, the female of which lays its eggs on the goldenrod, which hatch into caterpillars. When the caterpillars eat the goldenrod stem, the salivary proteins of the larvae induce cell proliferation in the goldenrod, thus forming a gall. The larva enters the gall and continues eating from within it. As winter approaches, the larva begins to produce sorbitol and trehalose sugars that act as an antifreeze. The trigger for this synthesis is not temperature but aromatic substances produced by the desiccating gall (Williams and Lee, 2005). Here, we see both reciprocal induction on the ecological level and reciprocal causation at the evolutionary level. The gallfly larva creates the niche by causing the plant to change its development. The niche provides not only nutrition but also the cue for the larva to change its development as winter approaches.

(*iv*) *Typological/population thinking*: From the conventional perspective there is an incompatibility between the typological thinking characteristic of much developmental biology and the population thinking characteristic of evolutionary biology. This dichotomy may have been emphasized by evolutionary biologists in order to ensure that natural selection within species (rather than the creation of higher taxa) became the conventional way of conceiving evolution (Winsor, 2007). However, in addition to convergent selection, divergent species can have similar forms owing to the inheritance of the same genes (viz. *Pax6*, *Otx*, and *Hox* genes in flies and humans). Thus, EvoDevo has proposed mechanisms for the ''continuity of type.'' Stressing the concept of reciprocal causation, below we argue that niche construction helps to explain the appearance of evolutionary change in ''bauplans'' or ''developmental types'' and explains in what respects developmental types might be regarded as evolutionarily causal.

Misleading metaphors

Lewontin ('82, '83) argues that the legacies of Darwin and Mendel include misleading metaphors that are responsible for certain difficulties in biology. Mendel's view of organisms as the manifestation of internal factors with their own laws translated into a postsynthesis metaphor in which ''ontogeny is seen as an *unfolding* of a form, already latent in the genes'' (Lewontin, '83, p 276). Although Darwin recognized organisms as constructors of their environment, and championed some marvellous examples of niche construction (e.g. earthworms, coral), his postsynthesis legacy became a view of organisms as passive objects molded by the external force of selection. Lewontin argues that it will not be possible to integrate developmental biology into evolution without the recognition that the organism is itself a cause, both of its own development and of its own selective environment.

Findings from developmental biology, among other disciplines, have rendered the metaphors of unfolding program, recipe, and blueprint untenable. The developing organism can have numerous different phenotypes depending upon the environment. The genotype does not usually encode the information for a particular phenotype, but for a repertoire of phenotypes. The environment will provide the specificity to elicit one of these phenotypes (at any particular time) from the available information provided by the genes (see Schlichting and Pigliucci, '98; Gilbert, 2001, 2004).

For instance, there are nutritional polyphenisms (where whether an ant is a huge reproductively active queen or a small worker whose ovaries have not formed depends on diet), temperature-induced polyphenisms (where the sex of numerous reptiles and the wing patterns of several butterfly species are temperature dependent), density-dependent polyphenisms (where parental density affects whether locusts have the solitary, green phenotype or the large, brown phenotype that swarms), and predator-induced polyphenism (wherein Daphnia, Hyla, and other organisms can change their pattern of development depending upon the

presence of cues from their major predators). In all these cases, the genome takes instructions from the environment.

Explanatory reference devices

Lewontin ('83) drew attention to the reference-device problem by summarizing standard evolutionary theory as

$$\frac{dO}{dt} = f(O,E) \quad (1a)$$

$$\frac{dE}{dt} = g(E). \quad (1b)$$

Evolutionary change, dO/dt, is assumed to depend on both organisms' states, O, and environmental states, E, but environmental change, dE/dt, is assumed to depend on environmental states only. With many caveats and complications (Odling-Smee et al., 2003), organisms are not generally regarded as causing significant changes in selective environments. For Lewontin, a better description is

$$\frac{dO}{dt} = f(O,E) \quad (2a)$$

$$\frac{dE}{dt} = g(O,E) \quad (2b)$$

where environmental change also depends on the environment-modifying activities of organisms.

Philosopher Godfrey-Smith ('96) drew attention to the same problem by describing standard evolutionary theory as "externalist," by which he meant that it uses the external environment as its explanatory reference device. It seeks to explain the internal properties of organisms, their adaptations, exclusively in terms of external properties, that is, sources of natural selection in external environments. Although differences in the properties of organisms (underpinning their differential ability to survive and reproduce) cause selection, these can only be evaluated relative to the local *external* environment, with different environments favoring different phenotypes. As a consequence, the phenotypes of organisms are viewed as sculpted by selection to become suited to external conditions.

The principal point that the conventional approach obscures is that organisms are active as well as reactive (Waddington, '69; Lewontin, '83). To stay alive organisms have to gain resources from their external environments by nonrandom work. They are compelled to choose and perturb specific components of their environments and, by doing so, they change some of the selection pressures in their environments. Lewontin's equation (2b) introduces an additional causal arrow into evolutionary biology, which Odling-Smee ('88) labelled as "niche construction."

The "reference-device problem," which Odling-Smee et al. (2003) regard as the principle obstacle to integrating evolution and development, is that the causal arrow in Equation (2b) representing niche construction points in the "wrong" direction, from organisms to environments, and hence is not compatible with the externalist assumption of standard evolutionary theory. It is therefore difficult for evolutionary biologists to describe changes in selection caused by niche construction as evolutionarily causal. Instead, standard evolutionary theory is forced by its own explanatory reference device to "explain away" all observed instances of niche construction as phenotypic, or extended phenotypic (Dawkins, '82), *consequences* of earlier selection. Standard evolutionary theory can recognize niche construction as a consequence of evolution, but not a cause. As a result, there exists extensive theory within evolutionary biology and evolutionary ecology concerned with how selection shapes the capacity of organisms to modify environmental states and construct artifacts, but little theory concerned with how niche construction modifies natural selection pressures, and the evolutionary ramifications of this capability.

The solution adopted by niche-construction theory was to change the explanatory reference device. Instead of describing the evolution of organisms relative to external environments, Odling-Smee et al. (2003) describe evolution relative to the "niches" or organism–environment interactive relationships. Because niches are defined by two-way interactions between organisms and their environments (Chase and Leibold, 2003), this step introduces an "interactionist" (Godfrey-Smith, '96) theory of evolution. The niche provides a neutral explanatory reference device for evolutionary theory, which can capture reciprocal causation. As a consequence, all developmental processes that modify the organism–environment relationship are recognized as evolutionarily causal.

CONSEQUENCES OF THE NICHE-CONSTRUCTION REVISION

Multiple consequences flow from the niche-construction revision (Odling-Smee et al., 2003).

Here we concentrate on those affecting the relationship between evolution and development.

Expanded inheritance

The niche-construction perspective adds a second general form of inheritance to the evolutionary process, stressing two legacies that organisms inherit from their ancestors, genes and modified selective environments (ecological inheritance). Unlike genetic inheritance, ecological inheritance is not a template copying system, and does not depend on replicators (Sterelny, 2001, 2005; Dawkins, 2004), but on organisms bequeathing altered environments to their offspring.

Population genetic models reveal that ecological inheritance generates some unusual evolutionary dynamics (Laland et al., '96, '99, 2001; Silver and Di Paolo, 2006). Populations evolving in response to features of the environment modified by their ancestors exhibit momentum effects (continuing to evolve in the same direction after selection has stopped or reversed), inertia effects (no noticeable evolutionary response to selection for a number of generations), opposite and sudden catastrophic responses to selection. Niche-constructing traits can drive themselves to fixation, by generating disequilibrium between niche-constructing alleles and alleles whose fitness depends on resources modified by niche construction (Silver and Di Paolo, 2006). Costly niche-constructing traits can be favored because of the benefits that will accrue to distant descendants (Lehmann, 2008).

A further way in which conceptions of inheritance should be expanded is through recognition of epigenetic inheritance systems, which have garnered considerable recent support (Bird, 2002, 2007; Jablonka and Lamb, 2005). The transmission of stably inherited traits through the epigenotype is thus a mechanism for stabilizing polyphenisms such that a juvenile is born already induced and does not spend developmental stages in the inappropriate condition. When the solitary locust undergoes its density-dependent transformation into the gregarious flying phase, it transfers this gregarious phenotype to its offspring through foamy secretions it deposits around the egg (Simpson and Sword, 2008).

Niche inheritance

The incorporation of ecological inheritance into evolutionary biology has consequences for development. It means that in each generation, offspring inherit a local selective environment that has, to an extent, previously been modified, or chosen, by its niche-constructing ancestors. The resulting dual inheritance implies that each offspring must actually inherit an initial organism–environment relationship. In standard evolutionary theory, the development of organisms begins with the inheritance of a ''start-up kit'' of genes: in niche-construction theory, it begins with the inheritance of a ''start-up niche.''

Minimally, parental actions and choices influence where and when offspring originate, and in many species, they ensure that some kind of resource package is also present. For example, phytophagous insects typically choose specific host plants on which to lay their eggs, which subsequently serve as food resources for their offspring. In birds and insects, where the egg is a major component of the ''start-up niche,'' yolk is provided for embryonic and larval nutrition. Moreover, many organisms provide protective chemicals in their start-up kit. These can include antibodies such that the young can survive before their immune systems mature, compounds that are poisonous or distasteful to predators, cues that induce subsequent dietary or host preferences, or even sun-blocks that protect transparent embryos and larvae from solar radiation (Dussourd et al., '88; Adams and Shick, 2001; Goldstone et al., 2006).

These niche-constructed components of the inherited niche frequently modify the range of developmental environments to which juveniles are exposed, and may subsequently transform the norms of reaction of the offspring's genes (Laland and Sterelny, 2006). Nests, burrows, mounds, and similar structures all tend to buffer environmental variation, making temperature, humidity, and exposure to wind and sunlight more uniform. For instance, when social insects build nests, they modify the mean, and reduce the range of temperature and humidity experienced by the developing larvae (Hansell, '84). Conversely, niche construction can result in exposure to a broader range of developmental conditions as a consequence of ancestral activities. If parents relocate to a novel environment, their offspring will be exposed to novel developmental conditions (see ''Innovation'').

As humans, we inhabit a bacterial environment, which reciprocally finds spaces to colonize within us. They are our environment, and we selectively become their environment. The intestinal microbe populations differ remarkably between newborn infants (except in twins), suggesting that our microbes originally depend on the accidental

population we encounter early in life. By a year of age, however, the microbiota of our gut approximates the standard population of the adult human gastrointestinal tract (Palmer et al., 2007).

Niche regulation in development

Lewontin ('83) described development from the conventional evolutionary viewpoint as an *unfolding* of a form already latent in the genes. From the niche-construction perspective, developing organisms do considerably more. First, niche construction is not only itself a developmental process, but one that is to some degree obligate. It follows that development will inevitably modify environments. Second, because they construct niches, developing organisms achieve a complementarity to their environments not only by responding to selection (as a population), but also by actively choosing and perturbing various components of their developmental environments, and by modifying them, often to suit themselves (as individuals). Third, niche construction is informed by all processes that inform development, including environmental experiences, learning processes in animals, and cultural processes in humans. Fourth, niche-constructing organisms introduce feedback in developmental as well as in evolutionary processes. Some of the ways in which a developing organism alters its environment are likely to feed back to affect later stages of its own development (Waddington, '59; Oyama et al., 2001).

These changes convert developmental processes from the mere unfolding of genetically guided programs to a process of active ''niche regulation,'' where organisms both respond to inputs from their developmental environments on the basis of inherited genes, and modify those same developmental environments by their niche-constructing outputs, based on genetically afforded (but not genetically determined) phenotypic plasticity. A major task for any developing organism becomes the active *regulation* of its inherited ''niche,'' in ways that keep its personal organism–environment relationship continuously adaptive, for the rest of its life. Thus, development closely resembles evolution (Odling-Smee, '88; Griffiths and Gray, 2004) in that they are both ''interactionist'' processes (Godfrey-Smith, '96), reliant on reciprocal causation. Both involve organisms responding to their selective environments, and modifying their selective environments by their niche-constructing activities.

A good example of an organism creating a niche and having the niche modify and permit the development of the organism is provided by the aforementioned goldenrod gallfly. Mammalian development provides another case *par excellence*. Mammalian embryos construct their niche by instructing the uterus to alter its cell cycles, its adhesion proteins, and by inducing angiogenesis and a barrier to the immune system. The fetus induces the decidua reaction in the uterus, causing the uterus to become a habitat for the developing organism. The uterus reciprocally helps induce the formation of the placental tissues of the embryo (Spencer et al., 2004; Ticconi et al., 2006).

How developmental niche regulation can contribute to evolution

If the way in which each organism in a population regulates its niche adds up stochastically, to become an evolutionarily significant ecological inheritance for a population, then it is possible for the developmental process of niche construction to influence the population's subsequent evolution. Genes (and associated cellular machinery, in stable environmental conditions) are the obvious reason why the consequences of the niche-constructing activities of individual organisms are likely to accumulate to become evolutionarily significant. If most individual organisms in a population inherit the same genes, such that most individuals express similar niche-constructing acts, then it is likely that these will modify the population's selective environment. Collectively, developing niche-constructing organisms in a population act as uni-directional ''biological pumps'' in their environments, provided that they constantly do the same things, to the same environmental components, generation after generation (Odling-Smee et al., 2003).

An example is the above-mentioned symbiosis between mammals and microbes. The mammalian gut depends on microbes for its differentiation, as the microbes induce the expression of numerous mammalian genes that are involved with blood vessel formation and immune system function. The critical species of microbes induce gene expression in the gut epithelia not to help the host, but to help themselves. One genus of microbes, *Bacteroides*, for instance, regulates gene expression in the Paneth cells of the mouse intestine, instructing them to produce several proteins. Among these *Bacteroides*-induced proteins are Angiogenin-4 and RegIIIγ, which prevent

the colonization of the intestine by other species of microbes. *Bacteroides* and *Escherichia coli* are impervious to this compound, whereas several pathogenic gram-positive bacteria (*Enterococcus faecalis* and *Listeria monocytogenes*) are wiped out by it (Hooper et al. 2003). RegIIIγ also prevents gram-positive bacteria from colonizing the gut (Cash et al., 2006). *Bacteroides fragilis* also produces polysaccharide A, which induces host T-cells to produce interleukin 10. This interleukin protects mice against ulcerative colitis induced by a pathogenic bacterium, *Helicobacter hepaticus* (Mazmanian et al., 2008). Microorganisms are modifying their niches, causing their environment to change in a manner that enhances their prospects for survival, with major consequences for bacterial and mammalian evolution. These are developmental examples of the reciprocal causation emphasized by niche construction.

DEVELOPMENTAL CONSTRAINTS AND INNOVATIONS

External and internal EMGAs

While discussing the impact of niche construction on ecosystems, Odling-Smee et al. (2003) introduce the concept of *environmentally mediated genotypic associations* or *EMGA*s. *EMGA*s are indirect but specific connections between genotypes mediated either by biotic or abiotic environmental components in the external environment, which are modified through niche construction. *EMGA*s associate genes in either a single population or multiple populations in ecosystems through gene products that alter the selective or developmental environmental contexts in which other genes are expressed. Where they arise, the expression of genetic information (and associated cellular machinery) by niche-constructing organisms in one population may affect the acquisition of genetic information in the same or a second population through the modification of natural selection. For instance, kelp reduces the impact of waves and currents, maintains water clarity, prevents sediment movement, influences siltation rates, and provides a habitat for numerous species (Estes, '95). Kelp genes are indirectly connected to the genes of a multitude of subtidal and intertidal creatures via *EMGA*s springing directly from this ecosystem engineering (Jones et al., '97) rather than trophic interactions, and accordingly kelp genes influence the expression of, and selection on, numerous genes in numerous species.

Odling-Smee et al. (2003) suggest that there may be a high degree of continuity between the logic of gene networks established by niche construction through *EMGA*s in ecosystems and the logic of gene networks established by interactions among genes inside developing organisms. In both cases, genes create products that affect the expression of, and potentially selection on, other genes in regulatory networks, and also respond to the products of other genes in their network. For instance, a gene may express a product, such as a transcription factor. The product either is itself, or indirectly leads to the manufacture of, an internal resource, informed by external cues and the cellular environment, which we can regard as an act of construction, or the first step in a constructive process, akin to niche construction outside the organism. The resource then switches on, or off, or regulates, the expression of other genes, in a network inside a developing organism, which we could think of as an internal *EMGA*. To the extent that the developmental products of this interactive process are fit, each gene constitutes a potential source of natural selection for other genes in the internal network, and each act of internal construction is a source of modified selection. We have already discussed several cases of symbiotic or commensal relationships where individuals of one species generate products that are expressed outside of their bodies to induce gene expression in another species, often with reciprocal feedback, and these too can be regarded as *EMGA*s.

It is too early to say to what extent the similarities between internal and external gene networks will prove instructive. However, we suggest that the parallels are sufficient to warrant serious consideration, because if real there are potential payoffs, in the form of insights into developmental processes.

Understanding developmental constraints

One major issue within EvoDevo, and a persistent source of controversy, is the notion of developmental constraints. At the heart of this issue lie a series of unanswered questions (Raff, 2000; Mabee, 2006): ''How do developmental constraints arise?,'' ''Do they bias the direction of evolution?,'' and ''What gene network properties promote resilience or enhance evolvability?''

Historically, the notion of ''constraint'' has been viewed as an alternative factor to selection in evolution (Gould and Lewontin, '79). Constraints were intrinsic qualities of the organism associated

with the rules of development or constructional principles. Yet unless one can specify the underlying mechanisms that prevent selection from acting, or reduce its potency, developmental constraints remain vaguely defined, poorly understood entities that lack explanatory power. Updating Waddington ('53), Schwenk and Wagner (2004) attempt to solve the paradox of developmental constraints by proposing that natural selection is resolvable into ''external'' and ''internal'' components. By external selection they mean the conventional sorting between variant organisms in populations. By internal natural selection they mean selection derived from the contemporary internal dynamics of a developing organism, that is, ''the characters interaction with other characters of a system within the internal milieu'' (p 395). They contrasted the conservative nature of internal selection with the often less conservative nature of external selection. The more integrated a system, the stronger the stabilizing internal selection acting on its constituent characters should be. For Schwenk and Wagner, internal selection is a mechanism underlying development constraints, as it limits the availability of phenotypic variation to subsequent external selection.

Schwenk and Wagner's formulation is attractive, because it provides a plausible and concrete mechanism by which developmental constraints could operate. However, as Schwenk and Wagner imply, the concept of internal selection alone may be insufficient to account fully for developmental constraints in organisms. In theory, niche construction too is resolvable into external and internal components. Conceivably, it may be useful to consider the expression of transcription factors by genes in the internal environments of developing organisms as consistent with the logic of ''internal niche construction,'' whether or not this is the best label to use.

The logical consistency of *EMGA*s in the external environments of populations of organisms, and gene networks in the internal environments of individual organisms, suggests that it may be possible to gain insights into developmental processes by considering interactions between the processes of natural selection and niche construction in external environments. For instance, observations of how niche construction constrains selection outside of the bodies of organisms might shed light on how internal niche construction might underpin developmental constraints by constraining internal selection.

We note a degree of functional similarity between the homeostatic mechanisms that regulate the internal physiology of the phenotype (waste excretion, osmotic pressure balance, salt–-water balance) and the externally expressed niche-constructing activities of organisms. Indeed, much niche construction (e.g. humidity and temperature regulation by bees, ants, and wasps through adjusting the slope of mounds, blocking and opening nest entrances, heating and cooling nests through muscular activity, placing water droplets on the surface, etc.) is a direct externally expressed functional equivalent of an internal regulatory process seen in other species (Turner, 2000). Odling-Smee et al. (2003) categorize such cases as *counteractive niche construction*, which they define as occurring when organisms either perturb their environments, or relocate in space, to neutralize some earlier change in selection pressures. Furthermore, it is apparent that virtually all gene expressions are highly regulated within bounds, according to external cues and internal signals. Might there be utility in regarding developmental processes in general as manifestations of bouts of internal construction and selection? Conceivably, developmental constraints may follow from internal constructive processes regulating internal conditions within suitable limits.

Many developmental biologists are already thinking along these lines. For instance, ''stem-cell niches'' are microenvironments maintained by specialized cells that nurture stem cells and enable them to maintain tissue homeostasis (Moore and Lemischka, 2006; Scadden, 2006). Stem-cell populations are established in specific anatomical locations that regulate how they will participate in tissue generation, maintenance, and repair. The construction of such internal niches is critical to normal development as they not only impose function on stem cells but also modulate that functionality, including under conditions of physiological challenge (Scadden, 2006). The concept of an extracellular matrix regulating primitive cells is long-standing and at least three examples now exist in mammalian stem-cell systems (Scadden, 2006), in the skin through β-1 integrins that help bind cells together, in the nervous system through tenascin-C activity that helps regulate cell proliferation, and in the blood where it regulates hematopoietic stem cells through osteopontin. These examples, which are probably just the tip of the iceberg of stem-cell-niche activity, suggest that matrix components provide localizing niche elements that can contribute stimulatory, or

impose inhibitory, influences on the stem-cell pool, regulating virtually all aspects of gene expression.

Could such regulatory activity be a source of constraint in the evolution of developing organisms? A consideration of the impact of niche construction in the external environment suggests a possible mechanism. Many animal constructions, such as nests, burrows, and pupal cases, are adaptive largely because they damp out environmental variability in conditions. For instance, insect, bird, and rodent nests and burrows are ideal for protecting the occupants from environmental extremes (Hansell, '84), and experiments have shown that the internal temperature is frequently appreciably damped relative to the external temperature (Noirot, '70; Hansell, '84, 2004). One consequence of this niche construction is a reduced intensity of selection on traits that constitute morphological adaptations to extreme environmental conditions. Moreover, animals do not just build regulatory structures, but act on them to reduce variability in the environmental conditions experienced. From the acorn-storing squirrel, to the wasp that cools her nest with droplets of water, counteractive niche construction is widespread (Odling-Smee et al., 2003). Such niche construction functions to negate a modified or fluctuating selection pressure, thereby reducing the intensity of selection.

A powerful example of how counteractive niche construction can cancel out external selection is provided by the earthworm. Paradoxically, selection favors physiological adaptation to an *aquatic* lifestyle in earthworms, in spite of the fact that these originally aquatic creatures moved onto land more than 50 million years ago. This is only possible because earthworm niche construction (tunnelling, burrowing, etc.) modifies the soil environment to reduce soil matrix potentials, allowing them to draw large amounts of water into their bodies (Turner, 2000). It is as if worms build their own swimming pools. Their niche construction has conserved selection pressures, in spite of the massive change from an aquatic to a terrestrial environment. The result is morphological stasis. Conceivably, internal regulatory activity may function in an analogous, or even directly equivalent, manner to stabilize internal selection.

There are currently several programs that are building bridges between evolutionary and developmental biology. These include the developmental genetic analysis of selectable variation within and between species (e.g. Abzhanov et al., 2004) and the combination of developmental genetics and comparative genomics to locate genes responsible for major evolutionary changes (e.g. Pollard et al., 2006). However, these genomic-level bridges may not be enough. Amundson (2005) lays stress on an incompatibility of EvoDevo and population genetics, stemming from the former's emphasis on ''bauplans'' and ''types'' (e.g. the vertebrate limb), a manifestation of typological thinking that has little accord with contemporary evolutionary thought. Evolutionary developmental biologists provide evidence that developmental types are real, useful, and evolving entities (Raff, '96; Hall, '99), but Amundson (p 236) asks: ''What could 'maintain and preserve' such an entity, distributed as it is across reproductively isolated populations?'' Internal selection within the embryo, as proposed by Waddington ('53) and Schwenk and Wagner (2004), might contribute to such constraints. A consideration of the effects of counteractive niche construction in the external environment suggests that one possible answer is stabilizing selection mediated by internal constructive regulatory processes. The structure of the termite *Cephalotermes rectangularis* mound is known to protect the occupants from extreme conditions (Noirot, '70), but the structure, or something like it, is common to many termite species (Hansell, '84), and the impact of this niche construction on selection is the same for probably thousands of reproductively isolated termite populations. The same point could be made with respect to bird's nests, which although diverse in form exhibit consistencies of structure that regulate and damp environmental conditions in equivalent ways among reproductively isolated populations. One could even describe ''the avian nest'' as a niche-constructed external ''bauplan.'' It is true that ''the avian nest'' does not evolve as a unitary entity, but consistent homologous or convergent patterns of niche construction generate consistent convergent selection and also expose consistent variants to selection. Conceivably, internal regulatory constructive processes preserve consistent selection pressures in a similar manner, to generate the appearance of some higher taxonomic-level ''force'' opposing directional selection.

Innovation

From the niche-construction perspective, with its emphasis on reciprocal causation, evolutionary change is not solely explained by changed selec-

tion, but also requires consideration of what causes these changes in selection pressures—and often the answer is the earlier niche construction of ancestral populations. Accordingly, the niche-construction perspective explicitly recognizes an additional process to natural selection, which could potentially be the source of directionality in evolutionary responses, namely the organism itself, and the changes it brings about in its selective environment. This means that, in addition to chance and natural selection, there is a third explicitly recognized source of evolutionary innovation, which occurs when gene-informed, directed, nonrandom, yet novel, acts of niche construction bring about consistent changes in environments.

If individuals select or manufacture a novel environment, they and their descendants will be exposed to novel selection and novel developmental conditions. Accordingly, the legacy of inherited niche-constructed effects, which includes not just altered developmental environments, but altered reaction norms, altered heritabilities, and altered responses to selection, can be a source of evolutionary and phenotypic novelty, as well as stability. Through what Odling-Smee et al. (2003) call *inceptive niche-construction* organisms may create new niches for themselves, for instance, through exploitation of a new resource, technological innovation, or relocation to a novel environment. For example, the effect of improved paper technology would appear to have had a massive effect on the geographic distribution, colony size, and social complexity of Polistinae wasps (Hansell, '93). In animals, learning can be a major source of evolutionary innovation (several examples are given in ''Evolutionary Causes''). Extensive documentation of inceptive niche construction can be found in Odling-Smee et al. (2003).

The much-discussed Baldwin effect can often be viewed as a special case of inceptive niche construction eliciting a bout of selection, the case in which the favored genetic variation is expressed in the same niche-constructing activity. Ever since Spalding (1873) and Baldwin (1896, '02), environmentally driven plasticity has been seen as a key to generate and propagate evolutionary novelties. This idea later came to be elaborated in the theories of genetic assimilation (Schmalhausen, '49; Waddington, '52, '56), where ''a phenotypic character initially produced only in response to some environmental influence becomes, through a process of selection, taken over by the genotype, so that it is formed even in the absence of the environmental influence that had first been necessary'' (King and Stanfield, '85). Genetic assimilation, which may or may not be a product of inceptive niche construction, has been routinely accomplished in laboratory selection experiments (see Pigliucci et al., 2006; Suzuki and Nijhout, 2006). In this process, previously hidden (cryptic) genetic variation becomes important for the selection or the regulation of that expression after an environmental stimulus overcomes the threshold for the expression of these phenotypes. Selection in the presence of this environmental factor enriches the gene pool for the cryptic alleles that would determine this trait, and eventually these alleles become so frequent that the trait appears even in the absence of the environmental stimulus (see Gibson and Hogness, '96; Rutherford and Lindquist, '98; Suzuki and Nijhout, 2008). In this way, a phenotypically plastic trait can be converted into a genetically fixed trait that is constantly produced under a wide range of environmental conditions.

Buttressing the idea that environmentally induced phenotypes could give rise to adaptive changes, temperature-induced phenocopies (environmentally induced phenotypes that resemble those produced by different alleles) were shown to mimic the genetically controlled phenotypes of related species living at different temperatures. Thus, Standfuss (1896) demonstrated that the heat-shocked phenocopy of the Swiss subspecies of *Iphiclides podalirius* resembled the normal form of the Sicilian subspecies. Similarly, heat shocking the central European form of *Papilio machon* produced some individuals that resembled those specimens more common in Syria or Turkey. More recent work (Goldschmidt, '38; Nijhout, '84; Chow and Chan, '99) confirms these observations. Diet can have similar effects; for instance, changes in cichlid *Cichlasoma citrinellum* diet produce transformations in cichlid pharyngeal jaw morphology, which precisely mimic forms fixed in numerous benthic and limnetic species (Meyer, '90). When these experimental observations from the EvoDevo literature are put together with the extensive data bank, collated in the niche-construction literature and elsewhere (e.g. Odling-Smee et al., 2003; Reader and Laland, 2003), of (many thousands of) examples of animals engaging in activities that modify or regulate the temperature that they and their offspring experience, selecting environments with specific temperature ranges, and adopting novel diets and feeding habits, often as a result of learning, their significance can be

truly appreciated. Evolutionary novelty may come about as a direct result of inceptive niche construction, which exposes developing organisms to new environments and resources, which are the self-induced environmental conditions that elicit novel phenotypes.

There are several variations on the theme that environmentally induced phenotypes are seen first, and that there is then selection for those phenotypes that are most adaptive (see Sollars et al., 2003; West-Eberhard, 2003; Suzuki and Nijhout, 2006; Young and Badyaev, 2007; Moczek, 2008). This process facilitates adaptive evolution because the novel phenotype is not ''random.'' The new trait is produced by an already organized, adaptively flexible phenotype whose responses have been tested by past selection (West-Eberhard, 2003). As Garson et al. (2003) note, although mutation is random, developmental parameters may ''account for some of the directionality in morphological evolution.'' Such developmental parameters include the expression of niche construction, which imposes consistent, directed changes on developmental and selective environments. The plausibility of such mechanisms is supported by the observation from the fossil record that evolutionary innovations are not randomly distributed in space and time (Jablonski, 2005).

In summary, evolutionary innovation may depend on systems-level developmental mechanisms, often responding to self-imposed, and not just autonomous, environmental induction, often epigenetic in origin, which may act before the mutations with which they become associated by selection, and which may generate novel phenotypes in response to organism-initiated transformations in developmental conditions.

What implications do such mechanisms have for understanding of the evolution of bauplans or developmental types? Inceptive niche construction frequently triggers bouts of directional selection, initiating evolutionary novelty. We expect related species frequently to engage in similar internal and external constructive processes, by virtue of shared genes, shared developmental systems, shared environments (partly owing to similarity in their self-constructed components), and overlapping niches. The taxon-wide convergent niche construction will generate convergent directional selection, leading to an evolutionary character ''theme,'' around which individual species exhibit variation. This potentially leads to the appearance of higher-level forces at work and evolutionary change in bauplans or ''developmental types.''

CONCLUDING REMARKS

Like others before us, we have drawn attention to conceptual barriers to the satisfactory integration of development and evolution (West-Eberhard, 2003; Amundson, 2005; Laubichler and Maienschein, 2007a,b; Sansom and Brandon, 2007). Yet, to quote Laubichler and Maienschein (2007a, p 21): ''The success of EvoDevo will crucially depend on whether its practitioners succeed in formulating an adequate conceptual and theoretical structure for the field.'' We suggest that the conventional evolutionary perspective fails to provide such a structure, because it can only accommodate a ''watered-down'' version of EvoDevo, a version that abandons any proposals for additional ontogenetic evolutionary mechanisms and that regards developmental constraints as manifestations of earlier selection. Most fundamentally, it is difficult to reconcile conventional evolutionary thinking with the view, common to both niche-construction theory and ecological developmental biology, that development must be regarded as an integral part of the evolutionary process. Conversely, niche-construction theory offers much brighter prospects for a satisfactory evolutionary synthesis with development, by building on its emphasis on the organism as a part cause of evolutionary change. Niche-construction theory allows for a synthesis that is consistent with the full gamut of EvoDevo mechanisms, and that does not compromise its conceptual tools. Given the remarkable parallels in their histories, conceptual framework, and goals, we suggest that there may be utility in building bridges between EvoDevo and niche-construction theory.

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