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More on how and why: cause and effect in biology revisited

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Abstract In 1961, Ernst Mayr published a highly influential article on the nature of causation in biology, in which he distinguished between *proximate* and *ultimate* causes. Mayr argued that proximate causes (e.g. physiological factors) and ultimate causes (e.g. natural selection) addressed distinct 'how' and 'why' questions and were not competing alternatives. That distinction retains explanatory value today. However, the adoption of Mayr's heuristic led to the widespread belief that ontogenetic processes are irrelevant to evolutionary questions, a belief that has (1) hindered progress within evolutionary biology, (2) forged divisions between evolutionary biology and adjacent disciplines and (3) obstructed several contemporary debates in biology. Here we expand on our earlier (Laland et al. in Science 334:1512–1516, 2011) argument that Mayr's dichotomous formulation has now run its useful course, and that evolutionary biology would be better served by a concept of reciprocal causation, in which causation is perceived to cycle through biological systems recursively. We further suggest that a newer evolutionary synthesis is unlikely to emerge without this change in thinking about causation.

Keywords Niche construction · Nongenetic inheritance · Evo-devo · Cultural evolution

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Proximate and ultimate causation

Just over 50 years ago, Ernst Mayr's classic paper on causation ('Cause and Effect in Biology' 1961) was published in the leading journal *Science*. Aided by his standing as one of the world's leading evolutionary biologists and architect of the modern Synthetic theory of evolution, Mayr's article had a massive influence in shaping how most biologists understand causality.

In this article, Mayr distinguished 'proximate' from 'ultimate' causes. Proximate causes are immediate, mechanical influences on a trait—they explain *how* internal (e.g. hormonal) and external (e.g. temperature, day length) factors combine to elicit or generate the character. Conversely, ultimate causes are historical explanations—they explain *why* an organism has one trait rather than another. Mayr presented ultimate explanations as invoking natural selection, but other evolutionary processes, such as drift, may also be important (Ariew 2003). While the proximate-ultimate distinction had been made earlier (Baker 1938; Lack 1954), it was Mayr's (1961) article that led to its widespread acceptance, and Mayr was its outstanding proponent (Mayr 1974, 1984, 1992, 1993).

Mayr argued that different types of biologist were interested in different kinds of causal account. He maintained that physiologists, molecular and cellular biologists (which Mayr collectively labeled 'functional biologists') are interested in showing *how* systems work, and hence concentrate on proximate causes. In contrast, evolutionary biologists are interested in *why* history has produced one system rather than another, and hence they trade in ultimate causes.

To illustrate his argument Mayr deployed the example of avian migration. To fully comprehend migration, we need to understand both why birds migrate (its selective advantage) and how they migrate (how they time their migrations, how they navigate, etc.). The answers are complementary, and not competing, a position that we endorse. The value of a logical distinction between answers to how and why questions is now widely recognized (Sherman 1988; Dewsbury 1999; MacDougall-Shackleton 2011).

Our issues with Mayr's stance relate less to the utility of a proximate-ultimate distinction, and more to practical and conceptual problems associated with its implementation. Below we show that distinguishing between proximate and ultimate processes is not always as straightforward as it might appear, as whether a process is characterized as proximate or ultimate depends critically on the conceptual framework of the researcher. We also take issue with Mayr's corollary to the proximate-ultimate dichotomy, which infers from it that ultimate hypotheses cannot invoke developmental processes.

In the intervening years since its publication, Mayr's account of causation has become so cannonized in biological thought that most contemporary biologists regard it as a fundamental truth about the nature of causation rather than as just a useful way to structure and isolate targets of scientific investigation. In this paper we will, firstly, reiterate our argument that the status of Mayr's distinction plays a role in many of the ongoing debates in contemporary biological research (Laland et al. 2011). Secondly, we will expand on our previous arguments for why proximate causes also should feature in ultimate explanations. This goes beyond the recognition

that ontogenetic processes can impose constraints on the action of selection, or that proximate and ultimate processes interact, to recognize developmental processes as a source of evolutionary novelty, an initiator of evolutionary episodes, and a co-director of patterns of evolutionary change. Finally, we will argue that, because of problems with its implementation, this dichotomous framework is now actually impeding progress in several areas in biology, and outline an alternative causal framework that encompasses Mayr's scheme as a special case.

The proximate-ultimate distinction in contemporary debates

In spite of the overarching acceptance of the proximate-ultimate dichotomy by the wider biological community, over the years a steady stream of biologists, psychologists and philosophers have dissented from Mayr's portrayal of causation (Waddington 1962; Francis 1990; Hogan 1994; Dewsbury 1999; Watt 2000; Oyama et al. 2001; Ariew 2003; West-Eberhard 2003; Amundson 2005; Thierry 2005; Jablonka and Lamb 2005; Laland et al. 2008; Hogan and Bolhuis 2009). In our earlier treatment we pointed out that several major debates within contemporary biology revolve around different notions of causation, with acceptance or rejection of Mayr's position often at the heart of the controversies (Laland et al. 2011; see below). The disputes share a common pattern. Some believe that interaction and feedback processes traditionally characterized as 'proximate' are relevant to 'ultimate' evolutionary questions too, whilst the more traditional majority adopt Mayr's stance. The more radical group sometimes explicitly identifies the proximate-ultimate dichotomy as a conceptual barrier to scientific progress (Watt 2000; West-Eberhard 2003; Amundson 2005; Thierry 2005; Jablonka and Lamb 2005; Laland et al. 2008). For instance, Mary-Jane West-Eberhard (2003) has argued that the proximate-ultimate dichotomy has hindered the integration of evolutionary and developmental biology. She writes (p. 11): 'The proximate-ultimate distinction has given rise to a new confusion, namely, a belief that proximate causes of phenotypic variation have nothing to do with ultimate, evolutionary explanation'. Of course, evolutionarily minded researchers often consider proximate factors as part of their studies, and are well aware that addressing mechanistic questions can improve understanding of functional questions (e.g. Davies et al. 2012), but West-Eberhard's point goes beyond this. The concern here is that the proximate-ultimate distinction has discouraged consideration of the manner in which ontogenetic processes can set the evolutionary agenda, for instance, by introducing innovations, channeling variation, or initiating evolutionary episodes through modifying selection pressures (see also e.g. Gottlieb 1992 for a similar argument). More frequently, this difference in conceptual frameworks goes unrecognized, with opposing sides seemingly frustrated that they are talking past each other, instead of to each other. The distinction itself can be one of the contested issues, since a failure to respect Mayr's dichotomy is sometimes taken as a fatal flaw in views of adaptive evolution and a sign of confusion (e.g. Dawkins 2004; de Jong 2005; West et al. 2011; Scott-Phillips et al. 2011; Dickins and Rahman 2012).

In our previous work, we identified four contemporary debates that revolve around views of Mayr's scheme (Laland et al. 2011). In highlighting them, below, we do not wish to imply that different views of causation are the sole issue at stake, but rather we seek to draw attention to the common thread that links these seemingly separate conflicts, which we subsequently use to illustrate the problems that have resulted from Mayr's proximate-ultimate dichotomy.

Evolutionary developmental biology

In evolutionary developmental biology developmental processes are seen by many as not only outcomes of genetic programs molded by natural selection, but also as having an impact on the rate or direction of evolution. Although most biologists would acknowledge the importance of developmental constraints in evolution (sensu Maynard-Smith et al. 1985), some proponents of a developmental perspective go further and argue, firstly, that lineage differences in developmental lability (rather than natural selection) may explain differences in rates of evolutionary diversification and, secondly, that environmentally induced phenotypic variation has evolutionary potential, for instance, as a source of innovation (Moczek et al. 2011). If so, much of adaptive evolutionary change may have its origin in plastic responses to novel environments, later followed by genetic changes that stabilize and fine-tune those phenotypes, rather than the other way around (Baldwin 1896; West-Eberhard 2003; Piersma and van Gils 2011). The intellectual history of these arguments goes back to early criticism of natural selection as a creative force (e.g. Mivart 1871) and they share the fundamental feature that the origin of adaptations in development becomes part of an evolutionary explanation. In the Modern Synthesis, the 'origin question' was reduced to mutations with small and random effects on the phenotype, and substantial resistance remains to ascribing to development an important role in evolution (e.g. de Jong and Crozier 2003; de Jong 2005; Haig 2007).

Niche construction

Whereas the argument from development focuses on the *origin of adaptations*, recent work at the interface of ecology and evolution emphasizes the importance of addressing the *origin of selection*. Specifically, the concept of niche construction emphasizes how organisms modify their environment and hence selective regimes. Here the environment-altering (niche-constructing) phenotypic traits of organisms are portrayed as coevolving with other traits in the same population whose fitness is a function of organism-modified factors in the environment. For instance, earthworms change the structure and chemistry of the soils in which they live and thereby modify selection acting back on themselves, thereby influencing the fitness consequences of their water-balance organs (Turner 2000; Odling-Smee et al. 2003). The 'ultimate explanation' of the earthworm soil-processing behavior is selection stemming from a soil environment, but a substantial cause of the soil environment is the niche-constructing activity of ancestral earthworms. Thus, even though the selective environment is primarily abiotic, it can be viewed as evolving dynamically, in a manner akin to co-evolution.

Human cooperation

The importance of both plasticity and niche construction is perhaps most easily appreciated for our own species. For example, one group of researchers offers cultural evolution and gene-culture coevolutionary explanations for the evolution of human cooperation (Boyd and Richerson 1985; Boyd et al. 2003; Fehr and Fischbacher 2003; Henrich 2004). This work often relies on formal models that embrace multilevel selection (Boyd and Richerson 1985; Boyd et al. 2003; Henrich 2004; Nowak et al. 2010; Nowak and Highfield 2011), and on explanations that stress ontogenetic processes such as imitation, teaching, or strong reciprocity as causally involved in shaping phenotypes available for selection or as sources of selection. An alternative approach emphasizes the importance of inclusive fitness and insists that cultural transmission (often integral to multilevel-selection models of cooperation) be treated as an aspect of proximate, not evolutionary, biology. Accordingly, the former research has been criticized because the 'approach mixes up two different questions (how and why)' (West et al. 2011, p. 243). For West et al., Fehr, Boyd, Richerson, and others misconceive a proximate hypothesis as a solution to an ultimate question. By this mindset, strong reciprocity or cultural evolution are not answers to the question 'Why cooperate?', because such answers invoke a selection pressure, and the selection pressure must logically precede the selected mechanism. However, those criticised view matters differently, and argue that proximate mechanisms modify the selection acting on individuals and thus must feature in evolutionary explanations (Boyd and Richerson 1985; Gintis 2003; Fehr and Fischbacher 2003; Richerson and Boyd 2005; Efferson et al. 2008; Sterelny, in press). From this perspective, social learning creates developmental bias and/or shapes natural selection pressures, allowing genetic and cultural variation to coevolve, and developmentally plastic responses to trigger novel evolutionary episodes. For instance, strong reciprocity could create or modify the selection pressures that favour cooperation if norm-based preferences, perhaps exploiting more ancient predispositions, generate selective feedback.

Cultural evolution

This difference in how researchers treat the proximate-ultimate distinction seems to be common to ongoing debates over the dynamics of cultural change. Aspects of human cognition and society—features of language, reading, writing, norms, cooperative behavior, institutions, and technology—can be regarded as fashioned by a process of 'cultural evolution', encompassing generations of social learning, teaching, and innovation (Cavalli-Sforza and Feldman 1981; Boyd and Richerson 1985; Jablonka and Lamb 2005; Plotkin 2010; Blute 2010; Mesoudi 2011; Pagel 2012; Sterelny 2012). This is not the same as natural selection acting on cultural variation according to the dictates of biological fitness: here phenotypic change occurs through the differential retention and spread of acquired knowledge, rather than through differences in the inclusive fitness of human bearers, although the two processes may interact. Where cultural evolution mimics biological evolution in

generating the cultural equivalent of adaptation, cultural evolution is itself a historical process responsible for character design.

This difference in perspective is also manifest in debates between evolutionary psychologists and cultural evolutionists (Laland and Brown 2011). Evolutionary psychologists often characterize cultural influences on development as operating like a (proximate) switch (like the buttons on a jukebox) to shift behavior and cognition from one pre-established program to another, with each context-dependent strategy fashioned by natural selection (Tooby and Cosmides 1992). In contrast, cultural evolutionists view culture as a historical knowledge-gaining process and therefore as a legitimate source of ultimate explanations for acquired human characters (Cavalli-Sforza and Feldman 1981; Boyd and Richerson 1985; Mesoudi 2011).

Key to these debates is the extent to which the naturally selected genes underlying these learning mechanisms constrain and specify the content of what is learned. Evolutionary psychologists (and other nativists) assume that natural selection fashions both *how* and *what* humans learn, such that cultural transmission is 'under genetic control' (e.g. Dickins and Rahman 2012). In contrast, cultural evolutionists conceive of more open-ended learning facility with only limited constraints on content, such that the design features of, say, cars, computers or languages are hugely *under*-specified by considerations of biological fitness. If the former position is correct cultural evolution is best regarded as a proximate mechanism; conversely, if the latter is correct then cultural evolution provides a second class of ultimate explanations.

Problems with the proximate-ultimate causation distinction

We draw attention to these disputes not to evaluate them, which of course also would require consideration of many other important aspects, but to consider how acceptance of Mayr's scheme may have shaped, and may continue to shape, research in the biological sciences. Given that opposing conceptions of causation are common to all of these debates, it is informative to view the different perspectives within a broader context of evolutionary theory.

Unidirectional causation

Mayr's chosen example of avian migration fits his model of causation well, for several reasons. First, migration is clearly an evolved behavior with a selective explanation. Second, migration is probably not the ancestral condition of bird life, leaving the baseline conditions relatively unambiguous. Third, migration is a response to autonomous, independent features of the environment (i.e. the seasons), features that are not changed by the act of migration. Fourth, researchers can comprehend the physiology of migration without understanding the selection pressures that favoured that physiology. On this view developmental processes feature only in proximate causal accounts.

This line of reasoning seems to justify Mayr's (1984, 1992) stance that researchers could understand evolution without understanding development, a belief that subsequently became part of the conceptual framework inherited by generations of biologists together with Mayr's model of causation. Mayr championed the view that evolutionary biologists could legitimately jump from genotypes to fitnesses, whilst ignoring all the biology in-between. In addition, bird migration seemingly evolves through a *unidirectional* or *linear* causal process, as the organism is shaped by selection to 'match' features in an autonomous external environment. Causation is viewed as starting in the environment, and ending with a changed organism; here there is no need to explain the selective environment.

Reciprocal causation

Other examples do not fit Mayr's model of causation so well. Here it is useful to contrast Mayr's dichotomous model of causation with what we have termed reciprocal causation (Laland and Sterelny 2006; Laland et al. 2011; see Figure 1 in Laland et al. 2011 for a graphical illustration). When a trait evolves through intersexual selection, for instance, the source of selection is itself an evolving character. A peacock's tail evolves through mating preferences in peahens, and those preferences coevolve with the male trait. The 'ultimate explanation' for the male trait is the prior existence of female preferences, proximately manifest in differential peahen mate-choice decisions—decisions modified by individual experiences throughout the peahen's development. Likewise, the 'ultimate explanation' for the peahens' mating preferences is the prior existence of variation in the peacock's tail associated with fitness.

There are two important points here. First, in this example, causation is *reciprocal*. This means that 'ultimate explanations' must include an account of the sources of selection (what caused the selective environment?) when they are modified by the evolutionary process itself. Second, here developmental processes become relevant to evolutionary accounts as they contribute to the patterns of selection on the male and female phenotypes. Proximate mechanisms both shape and respond to selection, allowing developmental processes to feature in both proximate and ultimate explanations. Further, these sources of selection cannot always be reduced to genes that have been previously selected, as it is the environmentally co-determined phenotypes that are the immediate causes of selection in co-evolution, not the genotypes.

Reciprocal causation in contemporary evolutionary biology

Contemporary evolutionary biology recognizes many other cases where the source of selection on a character is coevolving with it—for example, many instances of coevolution, habitat selection, social evolution, frequency-dependent selection, as well as indirect genetic and maternal effects—are therefore better captured by a model of reciprocal than unidirectional causation. Likewise 'adaptive dynamics' theory can be viewed as a more ecologically informed outgrowth of evolutionary game theory that captures feedback between evolutionary and ecological processes

(Waxman and Gavrilets 2005). It might appear, then, that reciprocal causation is already the dominant conception within evolutionary biology, and that unidirectional causation is solely of historical interest. A closer look reveals that many, perhaps most, such analyses, for instance, of coevolution, frequency-dependent or habitat selection, are conducted at a level (e.g. genetic, demographic) that edits out any consideration of ontogeny. These studies capture a core structural feature of reciprocal causation—namely, selective feedback—but typically without truly embracing its full ramifications; specifically, without recognizing that this means that developmental processes can initiate and co-direct evolutionary outcomes. In all such cases, acquired characters are potentially sources of novel variation and selection (e.g. Bateson 1988; Gottlieb 1992; ten Cate 2000; Odling-Smee et al. 2003; West-Eberhard 2003; Jablonka and Lamb 2005; Cornwallis and Uller 2010; Bateson and Gluckman 2011; Piersma and van Gils 2011).

Evolutionary innovation and developmental bias

Reciprocal causation makes explicit the fact that the origin of each new evolutionary episode is ambiguous. For instance, in peahen mate choice the cycle of causation could have begun with prior phenotypic preferences, or with fitness differences in a trait. Either way, such a cycle can originate as an expression of phenotypic plasticity, in which case developmental processes are needed to explain the *origin* of evolutionary change. The same point holds more generally. Phenotypic plasticity is now known to be ubiquitous, and there is growing evidence that it is evolutionarily consequential (reviewed in Odling-Smee et al. 2003; West-Eberhard 2003; Jablonka and Lamb 2005; Pfennig et al. 2010; Day and Bonduriansky 2011; Moczek et al. 2011; Uller 2012). Environmentally induced phenotypes can have evolutionary potential if there is heritable variation in the extent to which individuals maintain a functional phenotype. This means that adaptive evolution often begins with phenotypic accommodation of novel environments—generating the adaptive variation necessary for selection—followed by genetic changes as a result of selection on genetically variable accommodations. Thus, in some instances, at least, following West-Eberhard (2003): 'Genes may be followers, not leaders in evolution' (see also Gottlieb 1992; Chudek and Henrich 2011; Piersma and van Gils 2011). For example, phenotypic accommodation of environmental conditions during egg formation and maternal effects facilitated rapid adaptive population divergence in sexual size dimorphism during range expansion of the house finch in North America (Badyaev 2009).

More generally, a full description of evolutionary change needs to explain how novel adaptations arose from existing structures, whether they were random with respect to past, current and future function, and how those changes were inherited and became entrenched in individual development (Badyaev 2011). However, in one of the main conceptual shifts in evolutionary biology after Darwin, the architects of the Modern Synthesis avoided this question altogether by simply treating random mutation as the sole cause of adaptive phenotypic variation (see Schmalhausen 1949 for an exception). Indeed, Mayr's dissociation of proximate and ultimate causation was an important contributor to the Modern Synthesis's

separation of developmental and evolutionary biology (Amundson 2005) because it justified treating development as the unfolding of a genetic programme:

> 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 (Mayr 1984, p. 126).

Under Mayr's scheme, the origin of adaptations was simply removed as an explanandum from the evolutionary biology agenda.

If phenotypes were indeed tightly regulated by genes, with new variants arising solely by random mutation in gene codes, then black-boxing development might be a reasonable stance for evolutionists. However, the contingency of development suggests that most evolutionary change comes from reorganization of existing structures rather than de novo production of adaptive variation. Developmental pathways could therefore restrict novel variation to particular axes in morphospace. For example, patterns of evolutionary changes in dentition in mammals can largely be explained by a shared regulation of tooth development that allows variation to be expressed only in certain directions (Kavanagh et al. 2007; Harjunmaa et al. 2012). Such 'developmental bias' is potentially widespread in nature (Arthur 2004; Brakefield 2006; Muller 2007). On this view, if the proximate biology of a lineage makes some variants more likely to arise than others, these proximate mechanisms help construct evolutionary pathways and should therefore be considered as partial causes of patterns of biological diversity (West-Eberhard 2003; Arthur 2004; Brakefield 2006; Muller 2007; Olson 2012).

Reciprocal causation and selective feedback

The evolutionary implications of environmentally induced phenotypes are not restricted to their effect on the origins of innovations. Recent research emphasizes how non-genetic inheritance and niche construction can sustain or repeatedly induce changed selective regimes, thus allowing joint evolution of organism and environment. Niche construction theory (Lewontin 1982, 1983; Odling-Smee et al. 2003), like related developmental systems approaches (Waddington 1969; Gottlieb 1992; Griffiths and Gray 1994; Oyama et al. 2001), exemplifies a reciprocal view of the interaction of proximate and ultimate factors, with researchers stressing how organisms modify environmental states, and bias natural selection. Here the environment-altering phenotypic traits of organisms are portrayed as coevolving with other traits whose fitness is affected by organism-modified factors in the environment.

Niche-construction advocates view phenotypes, and the environmental modifications they cause, as underdetermined by genes. Organism-derived environmental modifications do not flow solely from the biological adaptations of organisms, but also stems from their developmental plasticity, their byproducts, and their acquired characters. They are often caused by the cumulative output of multiple individuals, in multiple species (Odling-Smee et al. 2003; Laland and Sterelny 2006; Erwin 2008; Erwin and Tweedt 2012). In hominins, cultural practices have apparently generated selection for alleles that aid the digestion of starch, carbohydrates, proteins, and other novel features of our post-agricultural diets (Laland et al. 2010;

Richerson et al. 2010). This began at least with the controlled use of fire and cooking in *H. erectus* (Berna et al. 2012), and extends to the more recent planting of crops, the domestication of animals, the processing of food, and living in cities in *H. sapiens.* Cultural practices also select for alleles that confer resistance to diseases (e.g. malaria and smallpox) inadvertently promoted by these practices (Durham 1991; Ewald 1994; Laland et al. 2010). Features of the environment manufactured or modified by organisms can accumulate over generations to affect selection on descendant populations, a legacy known as 'ecological inheritance' (Odling-Smee et al. 1996, 2003; Laland et al. 1996, 1999; Lehmann 2007, 2008). At the extreme, phenomena such as sediment bioturbation or the accumulation of shell beds can accumulate over geological time, modulating macro-evolutionary patterns and diversity (Erwin 2008; Erwin and Tweedt 2012). Moreover, these changes spill over to drive evolution in other species (Post and Palkovacs 2009). Once, again, 'proximate', niche-constructing activities in phenotypes, and 'ultimate', natural selective factors in environments, interact, in series.

Tinbergen's four questions

Shortly after Mayr proposed his scheme, the Nobel Prize winning ethologist, Niko Tinbergen, came up with a related conceptual framework, which many authors regard as superseding, or extending, Mayr's account. Tinbergen (1963) argued that a complete understanding of a character required four 'questions' or 'problems' to be addressed: *What is its immediate cause?*, *How does it develop?*, *What is its adaptive significance?* and *From what did it evolve?* Most commonly, these four questions go by the labels of *causation*, *development*, *function* and *evolution*. Tinbergen presented his four questions as independent and ungrouped, and did not refer to Mayr's proximate-ultimate dichotomy. However, subsequently a tradition grew amongst animal behaviourists of linking the two schemes (Klopfer and Hailman 1972a, b; Alcock 1975): causation and development were characterized as proximate, or how, questions, and function and evolution described as ultimate, or why, questions (Dewsbury 1999).

There is a sense in which Tinbergen's framework might be regarded as superior to Mayr's, as it clearly delineates function and evolutionary history. It also avoids the term 'ultimate' which can mislead, since to some readers it implies 'first cause', 'most important cause' or 'initiator of a causal chain', which has led some commentators to prefer the term 'distal' to 'ultimate' (Francis 1990; Dewsbury 1999; Sober 2000). However, because Tinbergen's four questions have been widely interpreted as mapping onto the proximate-ultimate dichotomy cleanly, a general reading of Tinbergen's scheme potentially suffers from the same problems as Mayr's. There is still the temptation to regard both development and immediate causation of a trait as irrelevant to understanding its function and evolutionary history. Consider, for illustration, the aforementioned disputes over cultural evolution, which can be cast as addressing either the development question, or the evolution question, depending on perspective. Tinbergen's four questions do not resolve our concerns about the separation of proximate and ultimate causation whilst answers to his four questions continue to be regarded as independent. To his

credit, unlike Mayr, who forcefully advocated the separating of proximate and ultimate causation, Tinbergen (1963) did not make a strict separation of his questions necessary, and actively encouraged that they should all be asked for a given phenotype. However, Tinbergen left the fundamental question—How should the four be integrated?—unanswered, which resulted in each largely being treated as independent lines of investigation (Bolhuis and Verhulst 2009; Shettleworth 2010), much like Mayr's view of proximate and ultimate causes.

Identifying proximate and ultimate causes is not straight-forward

Mayr's concern that proximate and ultimate explanations should not be regarded as alternatives remains valid and valuable. There is no doubt that proximate explanations cannot replace, or render unnecessary, ultimate explanations (although this mistake is sometimes made). However, what the above considerations establish is that determining what is 'proximate' and what is 'ultimate' is not simply a matter of clear thinking, but invokes commitment to a specific philosophical stance. In principle, researchers could propose alternative hypotheses that incorporate different processes, and judge between them on the basis of empirical or theoretical evidence in a given instance. However, whether the featured processes will be characterized as proximate or ultimate will depend on the conceptual framework of the researcher, whilst the assumptions underlying their conceptual framework will constrain the set of hypotheses that are deemed to be competing.

Our argument does not give license for mechanistic-minded researchers to ignore legitimate evolutionary questions, nor does it justify juxtaposing proximate and ultimate explanations in cases where there are no differences in how causation is conceived, or when alternative conceptions agree on what is proximate and ultimate. However, before passing judgment on these matters, researchers would be wise to reflect on whether what might appear to be a proximate mechanism can form part of an ultimate explanation from another standpoint; and specifically to consider whether reciprocal causation is invoked.

We particularly stress that Mayr's corollary, that ultimate hypotheses cannot invoke developmental processes, is highly problematic (Laland et al. 2011). As shown above, developmental processes frequently play some role in explaining why characters possess the properties that they do, and are central to the historical processes that explain their current state. When researchers regard the proximate/ultimate distinction as a barrier to the satisfactory integration of evolution and development (e.g. Amundson 2005; West-Eberhard 2003; Laland et al. 2008), this is largely because of the widespread, but mistaken, tendency to treat the exclusion of proximate mechanisms from answers to why-questions as logically following from the proximate-ultimate distinction itself.

Academic disciplines as cause and context 'clubs'

It would seem that Mayr and the other architects of the modern synthesis 'black boxed' development. Such 'black boxing' or 'screening off' of a phenomenon, and

exiling its study to an adjacent discipline, is often initially useful. It is only a problem if and when it becomes dogma. We argue this has happened with Mayr's distinction. An initially useful heuristic (i.e. that proximate and ultimate explanations are not competing alternatives) has degenerated into a convention (e.g. where developmental processes are seen as irrelevant to evolution), leading to unhelpful divisions between academic fields of enquiry (e.g. the weak link between evolutionary and developmental biology; Amundson 2005).

More generally, it would seem that discipline-based scientific fields frequently emerge that by default do not treat potentially relevant phenomena as causes, leading to the neglect of relevant processes, in this case that do indeed contribute to evolutionary change or stasis. In one sense this might be regarded as inevitable, or perhaps even desirable. In the struggle to understand a devastatingly complex and changing world, scientists utilise conceptual tools that render their business more manageable. They assume that, at least for the kinds of questions in which they are interested, with their associated temporal and spatial scales, it is reasonable to treat certain processes, and certain kinds of variation, as relatively unimportant. This allows them to hold certain aspects of the world constant, to treat them as 'context', and to explore the causal structure of the phenomena of interest relative to that context. All scientists do this—although we suspect, few do so deliberately; rather these fundamental assumptions are commonly accepted with little reflection. Accordingly, scientific disciplines effectively become 'clubs' in which likeminded researchers share some consensus over what is, and what is not, reasonably treated as cause and context. Mayr, by separating proximate from ultimate causation, legitimised screening off ontogenetic processes by evolutionary biologists. Ontogeny was someone else's problem. This also helped legitimise the view that researchers could understand the theoretical basis of evolution through population genetics alone (Watt 2000), a point to which we return below.

The externalism of evolutionary biology

Godfrey-Smith (1996) describes evolutionary biology as 'externalist', by which he means that the adaptations of organisms are described relative to the characteristics of *external* environments. Mayr's position, which exemplifies that of the Modern Synthesis, was 'externalist' by assuming that the selective environments of organisms pre-exist the biological traits of interest, are little affected by the activities of organisms, and to all intents and purposes, are fixed. It is a moot point as to how prevalent this externalism is in contemporary evolutionary biology, particularly as several subfields of evolutionary biology that are more interactionist in conception, such as sexual selection, coevolution, and maternal effects, have exhibited considerable recent growth. Nonetheless, the externalist stance can still be regarded as the 'default' position for many (Godfrey-Smith 1996; Odling-Smee et al. 2003).

Regardless of its history, a major consequence of this externalist assumption is that it hinders the environment-altering activities of organisms from being treated as evolutionarily causal (Odling-Smee et al. 2003). Darwin's natural selection is fully compatible with the externalist assumption because the 'causal arrow' of natural

selection points in the 'right' direction, from environments, to organisms, and so it is natural to describe natural selection as causing adaptations. However, the causal arrow of niche construction points in the 'wrong' direction, from organisms to their environments. Similarly, the causal arrow in an evolutionary scenario involving developmental bias points not solely from the environment, but also from the organism. The externalist assumption thus makes it more difficult to describe developmental processes as co-causal in evolution. Instead, standard evolutionary theory is encouraged to explain all instances of niche construction, and instances of developmental bias, as nothing but phenotypic, or possibly extended phenotypic, consequences of prior natural selection. This is not to imply that evolutionary biology does not recognize that organisms modify environmental states; rather, such organism-driven change is recognized but treated as merely a product or effect of natural selection. Thus, the possibility that developing phenotypes might contribute anything to evolution other than their differential survival and reproduction, is ruled out a priori. Standard evolutionary theory can recognise that plastic phenotypes are capable of fine-tuning their adaptations during their development, and may, thereby, affect their fitness. But it struggles to recognize that phenotypic plasticity can ever drive, or co-cause, evolution, through generating innovation, biasing variation, or imposing directionality on evolutionary trajectories. This externalism is a core assumption that causes problems for evolutionary biology and hinders integration of evolution with adjacent disciplines.

Problems for evolutionary biology

The primary problem derived from the externalist stance, reinforced by the proximate-ultimate causation dichotomy, for evolutionary biology itself is that it has hindered the full recognition of the pathways by which organisms' ontogenetic processes affect evolutionary processes. Two points that we have already made are that the modern synthetic theory has been slow to recognize the significance of both developmental bias and niche construction. An equally pressing third concern, voiced by some evolutionary biologists, is that variability has been treated as fixed, with little attention being given towards evolution of the genotype-phenotype map itself (Antonovics 1987; Wagner and Altenberg 1996; Watt 2000, 2004; Hansen 2011). Ward Watt (2000, 2004) describes the synthesis as 'amechanistic', by which he means it legitimised neglect of the processes underlying genotype to phenotype (and phenotype to fitness) translations. Likewise Janis Antonovics (1987, p. 326) complains that 'the Synthesis placed restrictive notions on the conceptual richness and depth of evolutionary biology as a science'.

Watt (2000) describes how the architects of the Synthetic theory screened off developmental and physiological processes through specific assumptions about the nature of genetic variation and its relations to phenotypes, fitness and selection. Fisher (1958) made additive genetic variation central to his view of natural selection. He argued that genetic variation supporting small changes in structure or process was likely to have additive, interchangeable effects, but that large heritable changes in phenotypes were unlikely to be adaptive. Simpson (1949) and Mayr (1958) made similar arguments. This led to the view that the total heritable variation

in the character, not the specific nature of the variants, was important, reinforcing the neglect of mechanism. The use of quantitative genetic theory, which also focuses on the analysis of additive genetic variation, has supported this viewpoint. Moreover, the emphasis on micro-variationism, and on genetic variation as additive (genes are 'good mixers', Mayr 1958), precipitated the strong adaptationist stance that is widespread within evolutionary biology today. If traits are underpinned by lots of genes of small effect that combine seamlessly and additively then there is little to stop selection from optimizing phenotypes (Parker and Maynard-Smith 1990). Much contemporary adaptationist thinking relies on similar reasoning.

In fact, there is now considerable empirical evidence that challenges the assumptions of micro-variationism and additivity: adaptive genetic variants show a wide range of magnitudes of phenotypic effects and widespread epistatic interactions, across a broad range of taxa (Watt 2000, 2004; Stern 2010). With hindsight, it can now be seen that many traits seem to be regulated by a few genes at which alleles of major effect segregate. At the same time, the processes of development have in some cases proven to be more general and robust across phyla than anticipated at the time of the Synthesis (West-Eberhard 2003; Arthur 2004; Brakefield 2006; Muller 2007; Stern 2010). These observations support the view that developmental processes can structure phenotypic variation and hence contribute to differences in evolvability between traits and taxa.

It is not clear to what extent notions of causation fed into these key assumptions of microvariationism and additivity, or merely reinforced them, but the proximate-ultimate dichotomy is certainly highly consistent with this conceptual framework. Mayr (1961, 1980) promoted a sub-division of the biological sciences in which the genotype to phenotype translation was studied by non-evolutionary biologists:

> The functional biologist is interested in the phenotype and its development resulting from the translation of the genetic program within the framework of the environment of the specific individual. It is this interaction between the translation of the genetic program and the environment that we refer to as proximate causes. The evolutionist is interested in the origin of the genotype, in the historical reasons of antecedent adaptation and speciation responsible for the particular genetic program that exists. This analysis deals with ultimate causes (Mayr 1980, p. 9).

These assumptions collectively have undermined evolutionary biology's ability to understand the causes of fitness differences between phenotypes, both by discouraging investigation of the developmental processes responsible for the origins of the phenotypic differences, and by discouraging consideration of the phenotype-environment interactions the construct those fitness differences (Watt 2000; Badyaev 2011).

Mayr's unidirectional characterization of causation encourages focus on single cause–effect relations within systems rather than on broader trends, feedback cycles, or the tracing of causal influences throughout systems (Odling-Smee et al. 2003; Amundson 2005; Laland et al. 2008). Except in the case of direct coevolutionary interactions (e.g. predator–prey, host–parasite), evolutionary biologists have generally been content to study the evolution of each focal species in isolation,

often content to demonstrate the mere occurrence of natural selection (Endler 1986). Endler encouraged the investigation of functional variation in phenotypes and their interactions with ecological variables. Recent developments notwithstanding (Post and Palkovacs 2009), resistance to incorporating ecological processes into evolutionary analyses continues to hinder investigations into the interaction of ecological and evolutionary processes, and of how that interaction generates eco-evolutionary dynamics, and macro-evolutionary trends (Erwin 2008; Laland et al. 2008).

Problems for adjacent disciplines

Developmental biology

The longstanding tension between developmental and evolutionary biology in part reflects evolutionary biologists' treatment of development as a 'black box' (e.g. Mayr 1980; Maynard-Smith 1982), which precludes any causal role for the organism in constructing both selective environments and variants subject to selection (Amundson 2005):

> The clarification of the biochemical mechanism by which the genetic program is translated into the phenotype tells us absolutely nothing about the steps by which natural selection has built up the particular genetic program (Mayr 1980, pp. 9–10).
>
> 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 1982, p. 6).

We have already noted that black-boxing masks developmental bias. Mayr's view of development as an unfolding of information encoded in the genome is equally problematic. By encouraging evolutionary biologists to adopt this view, Mayr's stance created divisions between evolutionary and developmental biology. For developmental biologists, the metaphors of genotype as 'program' or 'blueprint' proved inconsistent with the dynamic, reciprocal nature of development and inheritance emerging from their studies (see e.g. Gottlieb 1992; Hood et al. 2010; Pigliucci 2010). Developing organisms both modify gene expression and modify developmental environments, generating feedback in the processes of ontogeny that invoke organism-environment relationships and non-genetic inheritance as causes of species-typical, invariant, phenotypes typically seen as 'genetically determined' by evolutionary biologists (Oyama 1985; Gottlieb 1992; Oyama et al. 2001; Gilbert 2003; Jablonka and Lamb 2005; Lickliter and Harshaw 2010; Bateson and Gluckman 2011; Uller 2012).

Mayr's proximate/ultimate distinction builds on an incorrect view of development that fails to address the origin of characters. Here as elsewhere, a reciprocal model of causation may prove more useful than a unidirectional characterization, because it is better placed to accommodate the insights of developmentally minded evolutionists.

Ecology

Ecology is a divided discipline, with separate (evolution-embracing) population/community and (evolution-ignoring) ecosystem approaches, in part because contemporary evolutionary biology does not provide tools to capture the ways organisms engineer habitat, control and regulate flows of energy and matter, and drive state changes in abiota (O'Neill et al. 1986; Odling-Smee et al. 2003). Many ecologists recognize an inherent problem in integrating their discipline:

> Recently there has been a fractionation of ecology into its various specialities, thus, potentially at least, reducing the power of the field as a whole to integrate and synthesize to the fullest extent (Likens 1995).
> Ecologists who study populations do not talk as much as they should to those who are interested in the complexities of ecosystems (Ehrlich 1986).
> In general, we do not understand how to link organismal activities, population dynamics, and community assemblages to ecosystem processes, and there is little general theory that relates ecosystem properties to the activities, dynamics, and assemblages of species (Jones and Lawton 1995).
> Evolutionary and ecosystem ecologists have been talking past one another for almost a generation (Hagen 1992).

These divisions can be seen to follow, in part, from the disciplinary assumptions that, firstly, ecological and evolutionary processes operate on different time scales and, secondly, that understanding ecological change was outside the remit of evolutionary biology. The latter assumption was that, since evolutionary biologists are primarily interested in organismal change rather than environmental change, it is legitimate to treat environments as just context. These fictions were convenient for both fields: it meant that the manifest changes that organisms brought about in environments could be ignored by evolutionary biologists as potentially ecologically important, but unlikely to sum up over temporal and spatial scales to be also evolutionarily important. Conversely, functional and ecosystem ecologists could regard evolution as something that had happened in the past, often long ago, and as unlikely to greatly affect the distribution of species, or ecological dynamics, on the shorter timescales of ecological processes.

For instance, the separation out of ecological and evolutionary timescales legitimised the neglect of evolutionary processes by ecosystem ecologists. Conversely, another group of ecologists—population and community ecologists, with their focus on co-evolutionary and food web dynamics, were forced to incorporate evolution into their thinking, leading to a division within ecology. At the same time, the treatment of the environment as context by evolutionists led population and community ecologists to reduce ecosystems to their biotic components (O'Neill et al. 1986). For ecologists working in this sub-discipline: 'The biota *are* the ecosystem…. The biota may interact with the abiotic environment, but the environment is largely viewed as the backdrop or context within which biotic interactions occur' (O'Neill et al. 1986, pp. 8–9). In contrast, ecosystem (or process-functional) ecologists, with their focus on how energy and

matter flows through ecosystems, and biogeochemical cycles, could not afford to neglect abiota, reinforcing the division.

It would seem that ecology 'missed out on the Modern Synthesis' (Pigliucci and Muller 2010), and although it subsequently cultured an evolutionary wing, known as 'evolutionary ecology' (e.g. Ford 1964; Roughgarden 1979), the broader discipline remains separated from evolutionary biology in important respects. Recently, the recognition that ecological and evolutionary processes operate on overlapping timescales has led some ecologists to a more dynamic conception of eco-evolutionary causation (Pelletier et al. 2009; Post and Palkovacs 2009; Loreau 2010). This eco-evolutionary dynamics literature explicitly recognises reciprocal causation, with ecological processes both shaped by and triggering bouts of selection. However, authors rarely explicitly reject the standard interpretation of the proximate-ultimate distinction.

Behavioural ecology

In their core textbook, Davies et al. (2012, p. 22) state 'Behavioural ecology aims to understand how behaviour evolves in relation to ecological conditions'. It is perhaps surprising, then, that fellow behaviour ecologist Deborah Gordon (2011a) should complain that the fields of behavioural ecology and ecology are not well-integrated. This claim is striking given that many behavioural ecologists dedicate much effort to collecting ecological data but, as noted above, the links of evolution (of which behavioural ecology is a sub-discipline) to ecosystem ecology are very weak. Gordon was particularly concerned that contemporary animal behaviourists underemphasize behavioural and ecological variation, and advocates greater attention to natural history and ecology. However, she also maintains that ecology, whilst recognizing the existence of variation, does not currently adequately capture both the fluid, dynamic nature of the variation and the manner in which behaviour constructs ecological context (i.e. niche construction).

Gordon's concerns reflect the differential screening off of different aspects of the field by ecology and evolution. Behavioural ecologists, like evolutionary biologists in general, often regard ecological environments as context, where, for instance, different (conceptually pre-existing) distributions of food or resources select for different patterns of behaviour. The active role of behaviour in constructing those resource distributions receives comparatively little attention, except in some special cases. McNamara and Houston (2009) also lament the lack of attention to variation, both at the behavioural level and in the selective environment, and show that this variation can be self-reinforcing in generating selective feedback (McNamara et al. 2009; see also Fawcett et al. 2012), an example of reciprocal causation. Even those unsympathetic to Gordon's argument (Westneat 2011, p. 234) acknowledge that: 'few studies have assessed individual variation in plasticity or variation in the effects of plasticity on fitness' (see Dingermanse et al. 2009 for exceptions). Behavioural ecology has thus far paid comparatively little attention to the way in which behaviour modifies ecological conditions, in a way that feeds back to affect its subsequent evolution (Duckworth 2009; Cornwallis and Uller 2010). Once again, we see that progress depends on fully recognizing the reciprocal influence of agent

and context, and witness reciprocal causation being advocated as a vehicle to break down disciplinary conventions (Gordon 2011b).

McNamara and Houston (1980, 2009) and Fawcett et al. (2012) make related points, in stressing that 'behaviour is determined by mechanisms that are not optimal in every circumstance', and that because of this it is necessary to integrate the study of function and mechanism. They emphasize how psychological and perceptual mechanisms mediate the expression of adaptive behaviour, leading to behaviour that is often not globally optimal, and undermining the 'phenotypic gambit' that genetic architecture and other aspects of mechanism can be safely ignored. Importantly, this does not simply mean that mechanism act as 'constraints', but rather that by modelling evolution of the mechanisms by which phenotypes develop (e.g. the learning rules), rather than the phenotypes themselves, we can come to novel insights about evolution (McNamara and Houston 2009; Fawcett et al. 2012). These arguments mirror the concerns of developmental biologists, who maintain that the mechanisms of development evolve, not phenotypes, and that those mechanisms can affect the evolutionary process by biasing variation and contributing to the origin of adaptations (e.g. Lickliter and Harshaw 2010). Indeed, cognitive and perceptual mechanisms can be viewed as introducing a form of developmental bias, and may need to be considered in evolutionary analyses (McNamara and Houston 2009; Fawcett et al. 2012).

The human sciences

The primary working assumption that screened off much of the human sciences from evolutionary biology was that human culture, like development, is solely a 'proximate' causal process, and can therefore be dealt with in the same way that development is dealt with by mainstream evolutionary biology. That assumption constrained much of human sociobiology, and contemporary evolutionary psychology, to treat human behaviour, cognition and culture as no different from any other aspect of phenotypic plasticity (Wilson 1975; Tooby and Cosmides 1989). However, this stance alienated most human scientists (Sahlins 1976; Kuper 2000; Layton 2010; Laland and Brown 2011).

While evolutionary analyses in the human sciences are becoming more common, they remain a minority approach, and frequently evoke considerable hostility (Kuper 2000; Bloch 2000; Ehrlich and Feldman 2003; Layton 2010). Critics of evolutionary anthropology and archaeology dwell specifically on the failure of the fields to recognize humans as actively constructing their environments (e.g. Ingold 2007; Fuentes 2009; Layton 2010). Evolutionary psychology is criticised for its emphasis on 'universal' evolved psychological mechanisms that fail adequately to capture human behavioural diversity (Ehrlich and Feldman 2003; Brown et al. 2009, 2011; Layton 2010; Henrich et al. 2010), its failure to recognize the diversity of human selective environments (Foley 1996), and because it treats humans as passive victims of selection (Laland and Brown 2006; Layton 2010; Bolhuis et al. 2011), assumptions that follow directly from the proximate-ultimate dichotomy. But there are recent positive developments, with the growing influence of cultural evolution, gene-culture coevolution and niche construction theory, with their explicit advocacy

of reciprocal causation, within anthropology, archaeology, and other human sciences (Boyd and Richerson 1985; Richerson and Boyd 2005; Smith 2007a, b; Fuentes 2009; Bickerton 2009; O'Brien and Laland 2012; see special editions of journals edited by Pyne and Riel-Salvatore 2010, and Kendal et al. 2011).

The reciprocal causation revision

Given that we believe that the proximate-ultimate dichotomy has both scientific limitations (it constrains understanding evolutionary change) and social problems (it reduces communication between researchers of different background), what do we offer as an alternative? The conceptual revisions necessary to fully integrate evolutionary biology with developmental biology, or ecology, or to provide a satisfactory evolutionary framework for the human sciences, require a different way of thinking about evolutionary causation. We need a view that no longer treats ecological and developmental environments as context, that explicitly recognizes organisms as part constructors of environmental states, and that views such construction, and its legacy over time, as part of the evolutionary process (Lewontin 1983; Gottlieb 1992; Odling-Smee et al. 2003; West-Eberhard 2003; Jablonka and Lamb 2005). This leads to a view of development as source of origin of adaptations (West-Eberhard 2003), niches as dynamic, evolving entities (Odling-Smee et al. 2003), of ecosystems as governed by engineering as well as food webs (Jones and Lawton 1995), and of ecological and evolutionary processes as interwoven in space and time (Pelletier et al. 2009; Post and Palkovacs 2009; Loreau 2010). In Godfrey-Smith's (1996) terms, it requires an *interactionist* rather than an externalist theory, in which organismal (and, for that matter, environmental) change is described in relation to a relativistic and dynamic niche concept, which can only be specified relative to an organism, rather than to a pre-established environment. It requires a reciprocal, rather than dichotomous, model of biological causation, in which ontogenetic processes and nongenetic inheritance are not dismissed as irrelevant or under genetic control but recognized as evolutionary causes (West-Eberhard 2003; Jablonka and Lamb 2005; Laland and Sterelny 2006; Laland et al. 2011; Uller 2012).

Niche construction can serve as an example because, in origin, niche construction is a developmental process. It depends on the environment-altering activities of phenotypes during ontogeny. If the consequences of these activities accumulate, in the form of modified natural selection pressures in environments, as they are frequently likely to do (Odling-Smee 2010), then in conjunction with natural selection, niche construction becomes a cause of evolution. To echo West Eberhard's (2003) phrase that genes may be 'followers not leaders', natural selection pressures too may be 'followers not leaders' in evolution. Whenever a natural selection pressure is 'caused' by prior niche construction, its subsequent evolutionary consequences will follow niche construction. This perspective connects a developmental process, niche construction, to evolution, and insists that the adaptations of organisms may be co-caused by natural selection and niche construction. Neither development nor ecology can be black-boxed. Likewise, a reciprocal causation model provides an evolutionary framework for the human sciences that allows for feedback encompassed in dynamic cycles of cultural

evolution, gene-culture coevolution and organism-environment coevolution, and where plasticity drives evolutionary outcomes. As these forms of feedback are increasingly acknowledged to be important (Oyama et al. 2001; West-Eberhard 2003; Jablonka and Lamb 2005; Richerson and Boyd 2005; Layton 2010), the reciprocal causation framework offers much promise, and, more generally, may help to promote an integrative systems approach by highlighting causal connections between processes interacting across a range of spatio-temporal scales.

This is not to suggest that all biological phenomena are reciprocally caused but rather that the unidirectional selection emphasized by Mayr should be viewed as a special case of reciprocal causation where feedback is negligible. Nonetheless, reciprocal causation should now be regarded as the norm, rather than the exception. Although reciprocal causation does feature in many biological explanations, it is typically viewed as applicable solely to special cases (e.g. sexual selection, coevolution), and the ramification that developmental processes become relevant to evolutionary accounts remains underappreciated. In contrast, we suggest that reciprocal causation is a very general property of biological systems (Laland et al. 2011), which means that development is of general relevance to evolution. However, in making this claim we wish to emphasize that ultimately it is an empirical issue which causal arrows will prove significant for understanding evolution in a given case. It may turn out that there are models, with good fit to the data, in which, for instance, fitnesses can be directly attributed to genotypes, without incorporating the intermediate variable of phenotypic traits, or that environmental states are independent of genotypes. Our concern is that potential causal influences should not be ruled out a priori. While screening off certain processes will be necessary for model building and empirical tractability (Levins 1966), acceptance of pluralism in conceptual frameworks and the recognition that exclusion of certain phenomena are pragmatic stances rather than inherent truths are vital.

Prediction, induction and teleology

Mayr (1961) argued that a key element of causality is the prediction of future events, and reciprocal causation is relevant here too. Darwinian evolution, based as it is on variation, heritability and the struggle to survive and reproduce, comprises a kind of natural process by which populations of organisms accumulate information (Campbell 1974; Odling-Smee 1983; Dennett 1995; Hull et al. 2001). Inherited naturally selected genes must accumulate information about whatever past adaptive successes were expressed by those same genes in earlier generations relative to the selective environments of their ancestors (e.g. Leimar et al. 2006; Frank 2009; Shea et al. 2011). As there is no guarantee that past environments will resemble present conditions, the re-use of inherited genetic information that relates to past environments, and its re-expression by phenotypes in the present environment, amounts to an inductive gamble that the selective environments of contemporary organisms are sufficiently similar to the selective environments of their ancestors to make whatever was adaptive before, adaptive again (Slobodkin and Rapoport 1974; Odling-Smee 1988). Organisms, including humans, don't need to know anything for

certain; life is reliant on endless probabilistic inductive gambles. The fact that all species go extinct shows that such induction is not fool proof, but the fact that countless species survive for hundred of thousands or millions of years shows that the inductive gamble has nonetheless been highly effective for individuals.

But given that there is no guarantee that the future will resemble the past, why is this inductive gamble so effective, and why is inductive reasoning so prevalent? We suggest the predictive power of the genome is actually only part of the story. In no small part it is also because organisms are active agents who do work on their environments, transforming its state in reliable, predictable and often homeostatic ways (Odling-Smee et al. 2003). The external world is likely to be capricious but the *selective environment* is what matters to evolving organisms and if the selective environment retains some constancy across generations (or is changed in predictable ways), inductive gambles are more likely to pay off. This means that we would expect evolutionarily successful organisms to transform their selective environment in predictable ways (Odling-Smee et al. 2003); effectively, to 'load the dice' as well as predicting the outcome of the roll. The predictability of the selective environment, then, relates to the capacity of organisms to maintain environments within suitable (i.e. adaptive) ranges through their homeostatic activities, that is though 'counteractive' niche construction (Odling-Smee et al. 2003). For instance, bird's nests, termite mounds and mammal burrows experience reduced variation in temperature, and higher mean temperatures, than the external environment (Odling-Smee et al. 2003). The same point holds for constructed developmental environments (Badyaev and Uller 2009). (This argument is captured, to a degree, by the extended phenotype concept, but becomes much more powerful with the recognition that selective environments can be inherited.) Counteractive niche construction is likely generally to be adaptive, since it acts to increase the chances that organisms and their descendants will remain within their tolerance spaces. While it has been recognized at least since Hume (1910[1748]) that past observations themselves do not establish the validity of inductive reasoning (see Howson 2000, for a contemporary evaluation), the effectiveness of that reasoning (i.e. the likelihood that such reasoning will lead to accurate predictions) is nonetheless a function of the temporal consistency of environmental conditions. To the extent that organisms are able to control those conditions to enhance environmental consistency they are able to enhance the effectiveness of their inductive gamble. Mayr saw the inductive lottery as fair; whilst, as we envisage it, the lottery is rigged by the incumbents.

The ability of organisms to predict the environment also depends on 'selective' processes operating at other levels (e.g. learning, culture), which are also knowledge-gaining processes (Plotkin and Odling-Smee 1981; Plotkin 1994, 2010; Shea et al. 2011), and which further enhance the efficacy of plastic responses. These knowledge-gaining aspects of plasticity fine-tune the predictive power of inductive reasoning, allowing organisms to function effectively in a dynamic and only weakly predictable world. Ultimately, they enhance absolute fitness (McNamara and Dall 2010). Moreover, this capacity for learning and social transmission enhances the potency of organisms' niche construction, often in ways that further incrementing the predictability of their world.

Mayr (1961) also characterised the interpretation of teleological—that is goal-directed—phenomena as a component of causality. The problem of teleology (Aristotle's final cause, in the sense of apparent purposiveness, or design) was, according to Mayr, solved by Darwin. For Mayr organisms can function in a goal-directed manner, and exhibit design, solely because natural selection has programmed such functionality into their genomes. Once again, Mayr's unidirectional conception of causation is manifest. However, this solution attributes too much to selection. The observed adaptive fit, or hand-in-glove complementarity, between organism and environment can come about through two interacting processes, not one: natural selection or through the active construction of ecological or developmental environments suited to an organism and/or their descendants. There is no clearer indication of the fact that evolutionary biologists tend to assume that the former process dominates than that the same term—adaptation—is used to describe both the *process* by which natural selection brings about such complementarity and the end *product*. In contrast, a reciprocal causation framework views the routes to this fittedness as a 'two-way street' (Odling-Smee et al. 2003).

Conclusions

We have argued that the proximate-ultimate dichotomous model of causation, while intuitively appealing and *prima facie* helpful, has associated with it considerable drawbacks. Of particular concern are the suggestions that it hinders progress within the evolutionary sciences, that it creates divisions between academic disciplines and that it is inconsistent with much data. The commonalities of the above debates also raise rich issues concerning the history and philosophy of science, for instance, over the ways conceptual frameworks channel thinking and hinder paradigm shifts. The manner in which biologists think about causality has acted like a meta-theoretical conceptual framework to stabilize the dominant scientific paradigm. It may also have contributed to a series of corollary assumptions consistent with the causal model, for instance the architects of the Synthesis' conception of the genome as 'program', and their emphasis on microvariationism and additivity (Watt 2000). Violation of the proximate-ultimate distinction became a theme in Mayr's rejection of the claims of developmentally minded critics of the Synthesis (Mayr 1984, 1992), and similar arguments continue to be made today (e.g. Dickins and Rahman 2012), reflecting that the dichotomy has become a serious hurdle for integration of biological knowledge. It is only in recent years that talk of a new evolutionary paradigm has gathered momentum (Watt 2000; West-Eberhard 2003; Jablonka and Lamb 2005; Pigliucci and Muller 2010), and it is far from certain that such a frame-shift is imminent. We suggest that such a revision will not, indeed cannot, occur without a change in thinking about causation; the very notion that a shift is necessary is intimately linked to clarifying these issues. The time has come to replace the proximate-ultimate dichotomy with a new conception of biological causation, and we propose 'reciprocal causation' as a suitable framework.

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