On the origins of novelty in development and evolution

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Summary
The origin of novel traits is what draws many to evolutionary biology, yet our understanding of the mechanisms that underlie the genesis of novelty remains limited. Here I review definitions of novelty including its relationship to homology. I then discuss how ontogenetic perspectives may allow us to move beyond current roadblocks in our understanding of the mechanics of innovation. Specifically, I explore the roles of canalization, plasticity and threshold responses during development in generating a reservoir of cryptic genetic variation free to drift and accumulate in natural populations. Environmental or genetic perturbations that exceed the buffering capacity of development can then release this variation, and, through evolution by genetic accommodation, result in rapid diversification, recurrence of lost phenotypes as well as the origins of novel features. I conclude that, in our quest to understand the nature of innovation, the nature of development deserves to take center stage. ***BioEssays*** **30:432–447, 2008.**

Introduction

Evolutionary innovation is the spark that ignites much of biological thinking. Starting with biology lessons in school, organisms and their unique features provide the foundation for our understanding of evolutionary relationships across the organismal world, as well as our affinities within that world. Evolutionary novelties are central to many biological disciplines, most obviously in any *-ology* devoted to the biological distinctness of particular taxonomic groups, or phylogenetic systematics where novelties help define apomorphies, and thus relatedness. Less obvious, evolutionary novelties abound in virtually all other areas of biology from developmental biology to animal behavior, from pattern formation in zebra fish to the song of song birds, where the majority of work focuses on traits that in their exact configuration are unique to a tiny subset of organisms, with the hope to draw inferences applicable to a greater taxonomic whole. Here, evolutionary novelties become tools employed to study phenomena of more general significance, rather than foci in and of themselves. But I would argue that it is the uniqueness of particular traits, shapes, parts, displays or patterns that is responsible for inspiring scientists to study their biology in the first place, just like it inspires and motivates much of public interest in biological research in general. Given its importance and pervasiveness, the processes underlying evolutionary innovation are, however, remarkably poorly understood, which leaves us at a surprising conundrum: while biologists have made great progress over the past century and a half in understanding how existing traits diversify, we have made relatively little progress in understanding how novel traits come into being in the first place.[1] In fact, the terminology itself is ambiguous, and exactly what constitutes "innovation" in evolution, or what qualifies as an evolutionary "novelty", is subject of much debate.[2–4] Here I begin with a brief review of definitions of novelty and their implications and limitations. I then discuss how more recent ontogenetic perspectives may allow us to move beyond current roadblocks in our understanding of the mechanics of innovation in nature. Specifically, I highlight multiple avenues of innovation, all of which emerge as properties of how development links genotypes to phenotypes, and both to the environment. But to embark on any of this, we first need to work our way through some definitions of novelty. Where, exactly, does novelty begin?

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Evolutionary novelties: we know one when we see one?

Definitions of novelty abound, and this essay will not be able to do them all justice (for excellent reviews see Refs 2,3). Instead, I highlight three particularly influential definitions of novelty to point out the challenges and limitations that such definitions face when attempting to move beyond an intuitive understanding of what constitutes a novel trait to a more quantitative metric of novelty, and ultimately towards an understanding of the mechanisms of evolutionary innovation.

Ernst Mayr[5] (p. 351) defined novelty as "any newly acquired structure or property that permits the assumption of a new function". The notion of equating novel traits with novel functions, dating back to Lamarck[6] and Darwin,[7] holds tremendous intuitive appeal. Clearly, organisms capable of performing a function that others can't, involving a trait present in them but absent in others, make the assignment of novelty seemingly straightforward. Such novelty is obvious, for

example, in the exquisitely diverse wing patterns of butterflies and moths (Fig. 1A) generated by the arrangement of colored scales produced by wing epithelial cells.[8] Such wing patterns are restricted to the Lepidoptera, where they play important roles in mate recognition and predator defense.[9–11] Among butterflies and moths, these patters have permitted wing surfaces to attain functions that they previously did not have, hence it clearly makes sense to consider them novel traits. By the same logic, the abdominal bioluminescent organ, or lantern, of fireflies (Lampyridae) should qualify as a novelty (Fig. 1B). The adult lantern spans the ventral portion of two abdominal segments and emits a bright, species-specific light signal using luciferase-catalized oxidation of luciferin to oxiluciferin and energy in the form of light.[12] Light emission is used by adults for mate recognition and by some species for prey attraction.[13,14] The developmental origin and regulation of lanterns are largely unknown, though preliminary observation suggest an involvement of appendage-patterning genes and possibly abdominal Hox genes in establishing location and identity of the organ (Stansbury and Moczek, unpublished data). Adult abdominal lanterns, and the complex flash patterns that they produce, are unique to members of the family Lampyridae and their status as a novelty should be indisputable. A third example are the abdominal claspers of male sepsid flies (Fig. 2): highly diverse, paired, jointed appendages used to stimulate females during copulation.[15]

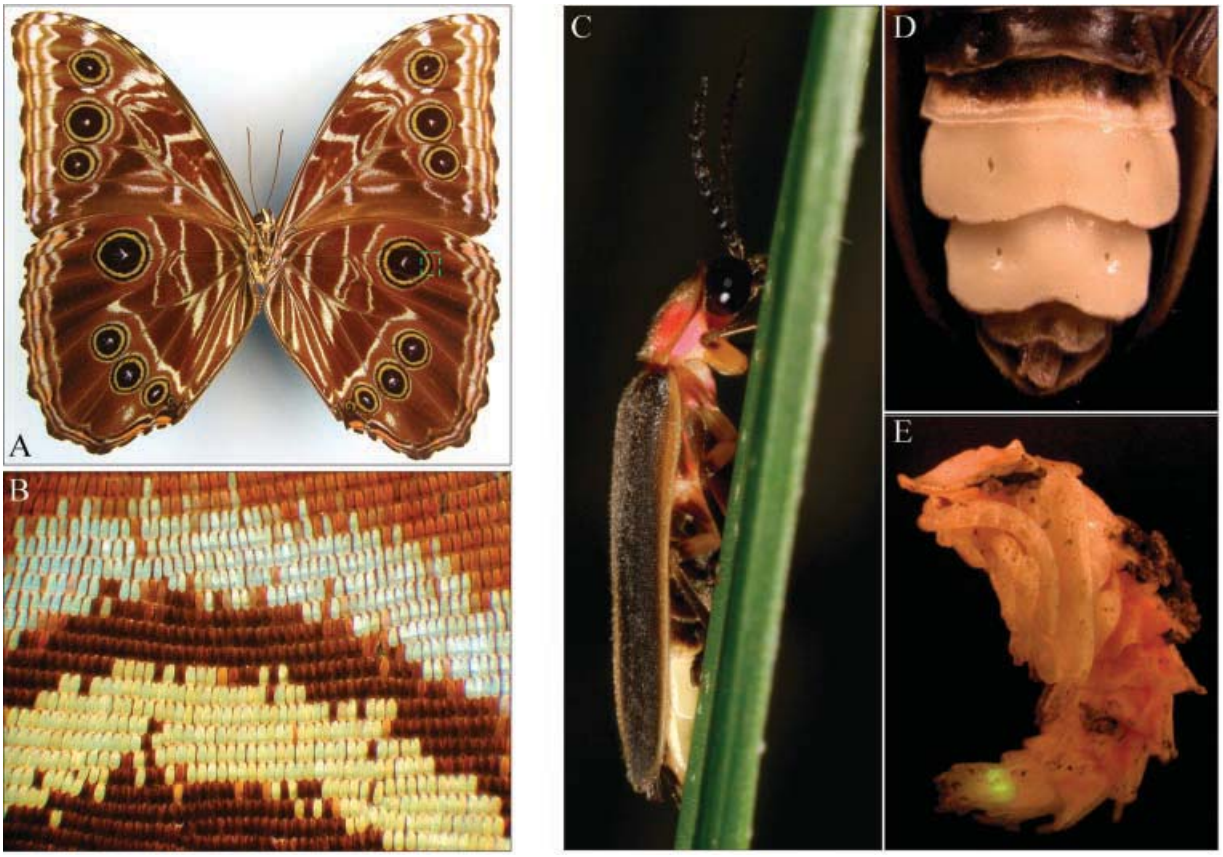

Figure 1. Two examples of novelties that lack obvious homology to other traits. **A,B:** Butterfly wing patterns function in species recognition and predator defense. Pattern elements may include lines, chevrons, semi-circles, ellipses or spots. **A:** Underside of the wings of *Morpho deidamia*. **B:** Closeup of part of one of the eye spots (indicated by boxed area). Patterns arise through the coordinated, two-dimensional arrangement of colored scales. Each scale is the product of a single, epidermal cell, which undergoes programmed cell death near the end of wing development. While scales are homologous to the setae produced on the wings of other insects, the wing patterns of butterflies and moths lack obvious homology outside the Lepidoptera. Images courtesy of H. F. Nijhout. **C–E:** Beetles in the family Lampyridae (fireflies, glow worms, lightning bugs) are famous for their bioluminescent displays. Adults use light flashes to attract mates and sometimes prey. The light-emitting organ, or lantern, is located at the ventral abdomen and varies in size depending on species and sex. Other life stages, including egg, larva and pupa also emit light, though location and size of the lantern, as well as flash pattern, differ across life stages. Shown here are **C:** an adult male *Photinus*, **D:** a close up of the male lantern, and **E:** a glowing *Photinus* pupa. All images by the author.

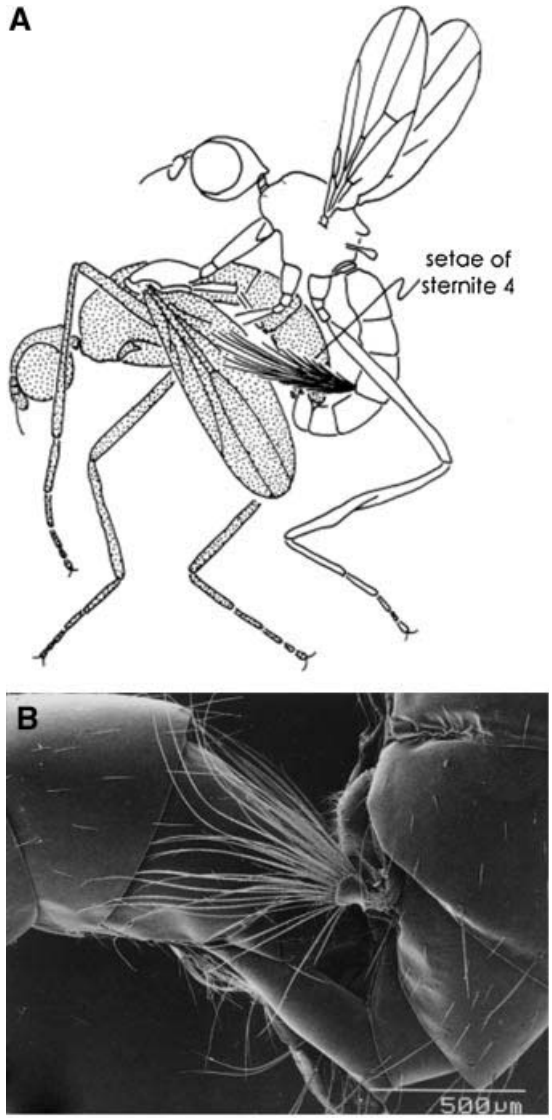

Figure 2. A: Male sepsid flies, such as *Themira leachi* shown here, use a pair of clasping appendages to stimulate females during copulation. Claspers are jointed, moveable and innervated, and develop from the fourth abdominal segment, which normally does not bear appendages in insects. Unlike other Dipteran appendages, which develop from imaginal discs, claspers appear to originate from histoblasts.[112] Claspers do not occur in females, and clasper morphology differs widely between species. **B:** A scanning electron micrograph of the lateral abdomen of a male (right) and female (left) *Palaeosepsis* during copulation. Note left male clasper contacting the female abdomen. Images modified after William G. Eberhard 2001. Multiple origins of a major novelty: moveable abdominal lobes in male sepsid flies (Diptera: Sepsidae), and the question of developmental constraints. Evolution Develop 3:206–222. Blackwell Publishing, and used with permission.

Such appendages are absent in most other families of flies, and they equip sepsid flies with a novel function that they would otherwise be unable to carry out to the same degree. Again, we know novelty when we see it! This, however, is about where the ease and simplicity of a novel function-based definition of evolutionary novelty ends, and its limitations begin. Three in particular are worth pointing out. First, linking novel traits to novel functions often carries with it the implication that the new trait evolved *because of* the new function that it carries out, that selection somehow favored the origin of the new trait because its new function was advantageous. A novel trait thus originates due to a new function that it provides, which favors the evolution of the new trait, setting up a dangerous circularity of argument. Second, while selection is likely to play a major role in honing novel traits and optimizing their function, it is difficult to see how selection could have played a role in the origin of novel traits and functions. For selection to play a role, there must have been heritable variation for both trait and function, but if that were the case the trait under consideration could no longer be considered a novelty. Put another way, selection cannot act on traits that do not yet exist, and therefore cannot directly cause novelty.[16] Lastly, a novel function-based definition of evolutionary novelty excludes the possibility that evolutionary innovation may still occur in the absence of obvious functional gain, or inversely, that much functional diversification may be feasible with little to no innovation. We will later encounter a situation that illustrates precisely this situation. For now let us note that a novel function-based definition of novelty captures without doubt important aspects evolutionary innovation, but ultimately leaves no obvious framework for exploring the mechanisms by which novel features, whether with or without conveying a novel function, first originate.

A second definition was proposed by Müller,[2] who defined novelty as "a qualitatively new structure with a discontinuous origin, marking a relatively abrupt deviation from the ancestral condition". In many ways, this definition again captures what most of us intuitively assume must be true for novel features, and it opens the way for traits to qualify as novel without necessarily conveying a new function, as long as they represent qualitative and discontinuous departures from the previous range of variation. Apart from the three examples introduced above, other examples of novelty might now include the narwhal's tooth (Fig. 3), which, though technically just a tooth, has taken on a size, shape and, possibly, function that most would agree mark an "abrupt deviation from the ancestral condition" as it evolved into what is believed to be a superb temperature-, salinity- and particle flow-sensing organ.[17] Another example might include the labial capture mask of dragonfly nymphs (Fig. 4), an unparalleled, highly modified version of the insect labium, which permits efficient capture of aquatic invertebrate and vertebrate prey.[18] Similarly, the elaborate, ornately sculpted and often bizarrely

Figure 3. The immature stages (najads) of dragonflies and damselflies possess a unique and extreme modification of the labium, or ''lower'' lip of insects. In these organisms, the labium has given rise to a prehensile labial mask used for prey capture. This labial capture mask is normally kept folded underneath the head, but can unfold rapidly during prey capture, allowing dragonfly najads to capture a wide range of vertebrate and invertebrate prey. The scanning electron micrographs shown here are courtesy of Wilfried Wichard and modified after Wichard et al. (2002). Biological Atlas of Aquatic Insects. Apollo Books.

shaped pronota of membracid treehoppers (Fig. 5) should qualify as novel as their sizes and shapes are well outside anything known from other insect orders.[19] Or do they? After all, the pronotal outgrowth of membracid treehoppers are really just elaborations of the pronotum, and even though often extreme in shape and size, they don't have to be, and many Membracidae have perfectly reasonable pronota, making it difficult to determine exactly where the ''abrupt deviation from the ancestral condition'' begins. But then the same could be said of a dragonfly's capture mask, which is ultimately just an elaboration, albeit an extreme, of the labium, a mouthpart present in one shape or form in almost all insects. And if we toss the labial capture mask back across the novelty divide, what should happen to the narwhal's tooth, this nearly 3-meter long, spiraling monster of an incisor densely covered in nerve openings: just another tooth? Unfortunately, Müller's definition leaves us with little help to determine where quantitative variation ends and qualitative distinctness begins. How much deviation from the ancestral condition is enough? How different is novel?

This is where a third definition, proposed by Müller and Wagner,[3] seeks to fill the void. Focusing on morphological novelties, they propose a two-part requirement. ''A morphological novelty is a structure that is neither homologous to any structure in the ancestral species or homonomous to any other structure in the same organism'' (p. 243). In other words, novelty begins where homology and serial homology (homonomy) end. This definition leaves unscathed the novelty status of butterfly wing patterns, which lack homology to other traits outside the Lepidoptera. While the scales making up these patterns are derived from, and homologous to, setae (hair) found on the wings of the sister order Trichoptera, the patterns that they help compose are unique to the wing surfaces of butterflies and moths, lacking any natural affinity to other traits outside the Lepidoptera.[8] Similarly, the lanterns of fireflies and the flash patterns that they emit are likely to pass this hurdle, lacking even remotely any homology to other traits in insects or arthropods. But this may be the only two of our examples that manage to survive these stringent requirements. Down go the pronota of membracid tree hoppers, dragon fly capture masks and the narwhal's tooth, as all can be unambiguously homologized to preexisting structures in ancestral species. In addition, the abdominal claspers of sepsid flies are also unlikely to retain their status as a novelty. Ancestral arthropods expressed paired appendages from every single segment, including those that have since evolved into the clasper-bearing segments of extant sepsids.[20,21] And even though these segments have lost their appendages long before claspers appeared, one could argue that their presence is still reflective of ancestral homology. What we once considered a novelty may now merely be a recurrence. Lastly, even if one disputes the latter, it is hard to overcome the second requirement: lack of serial homology. After all, claspers are just another paired appendage on another segment, just like antennae, mouthparts, legs, genitalia.

Excluding homology and homonomy thus sets a very stringent standard for assigning novelty, one that from our preliminary list of examples only two, butterfly wing patterns and firefly lanterns, are able to meet without ambiguity. We

Figure 4. The adaptive significance, if any, of the narwhal's (*Monodon monoceros*) tooth has been the subject of much speculation. **Top:** The tooth, a highly modified incisor, extends from the left upper jaw of males only. It measures up to 2.7 metres in length, spirals counterclockwise (as viewed from the animal) and is surprisingly bendable (photo courtesy of Glenn Williams, Narwhal Tusk Research). **Bottom:** Recent studies identified that millions of small openings connect the outer surface of the tooth to nerve cells in its core, consistent with the hypothesis that the tooth functions as a sensory organ involved in detecting subtle changes in temperature, pressure, salinity and particle gradients (photo courtesy of Joseph Meehan, Narwhal Tusk Research).

thus have overcome a major problem associated with the previous two definitions and now established a clear cut off. At the same time, however, I would argue that we have created a new problem: our definition of novelty now only becomes as strong as our definition of homology. After all, one begins where the other ends. But exactly where does homology end? As it turns out, the concept of homology is complex, now more than ever,[22–24] and it may behoove us to take a closer look at what exactly constitutes homology and on what level of biological organization, in the hope to better be able to characterize what does not, and what we might then legitimately call novel.

Novelty begins where homology ends?

As with novelty, many definitions of homology exist, and again this essay will only be able to touch on a few of them (for excellent recent reviews see Refs 22–26). Homology is an ancient concept, and one that much like novelty holds great intuitive appeal. The first precise definition of homology is credited to Owen[27] who described a homolog as ''the same organ in different animals under every variety of form and function''. The first pair of legs of flies, beetles and mantids, for instance, clearly are the *same* organ in different animals, as are the first thoracic vertebrate in mice, monkeys and whales. Homonomy, or serial homology, then refers to the presence of the *same* organ in different places of the same organism: first, second, third leg pairs as well as antenna, and mouth-parts of insects are all considered serial homologs, the same basic organ found in different segments along the body, just like the first thoracic vertebrate is a serial homolog of the first cervical vertebrate, the second caudal vertebrate etc. But what exactly do we mean by *sameness*? In most definitions of homology, sameness first and foremost implies the presence of the organ in the common ancestor of two organisms under consideration.[22–26] The thoracic legs of flies, beetles and mantids are homologous because thoracic legs existed in the common ancestor of all insect orders, including these three. Clearly, homologs and serial homologs are not meant to be identical, instead they are understood to be modifications, or *versions* of the ''same'', ancestral organ in descendant lineages, or in different locations of the same organism. Such *sameness* may be very obvious in cases such as vertebrates or insect appendages, but what criteria exist that would allow us to identify whether two organs that have diverged more drastically from each other are actually versions of the ''same'' ancestral organ, and thus need to be considered homologs? Owen[27] early on emphasized the importance of *relative position*, i.e. exactly where in an organisms a focal organ was located, including its connections to neighboring parts, with the idea that homologs share similar locations and connections. The German systematist Remane[28] popularized two additional criteria, *special quality*, and the existence of *intermediate forms*. Special quality (sometimes called specific quality, or special attributes) refers to any particular aspect or characteristic of an organ under consideration that contributes to its distinctness, with the idea that homologous organs are likely to retain such ''special qualities'' and may thus be recognizable by their presence. Special quality can mean anything, from developmental properties to chemical composition and has often been criticized for its imprecision. Lastly, the third criterion, the existence of intermediate forms either during ontogeny, or in fossils, extant organisms or hybrids permits the identification of sameness even among highly divergent organs: homologous organs may appear far more similar early in ontogeny than later, and intermediate forms in fossils or extant species may facilitate the establishment of correspondence, and ultimately homology, between otherwise widely divergent structures. With the advent of molecular evolutionary biology and evo-devo, homologues are now also

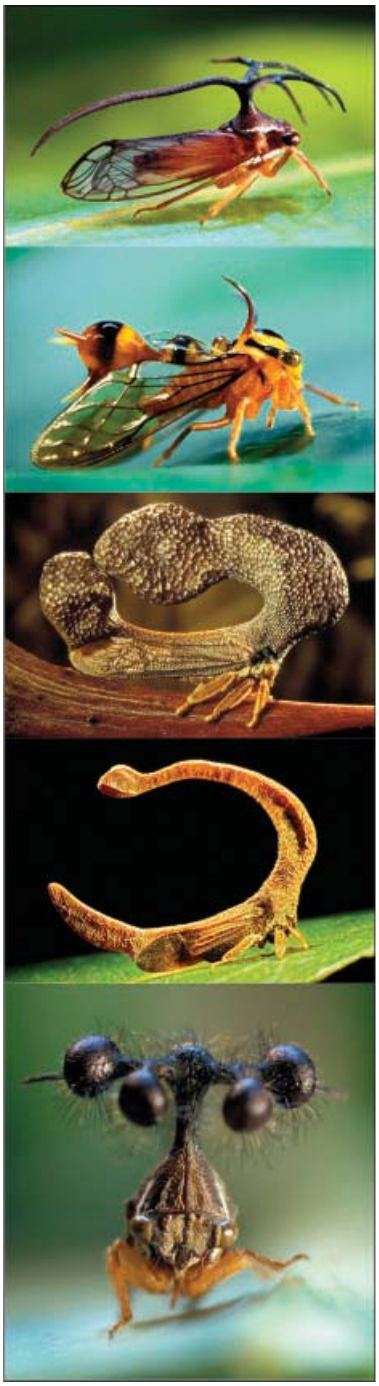

Figure 5. Treehopper in the family Membracidae are famous for their often bizarre elaborations of the pronotum. The adaptive significance of these elaborations is poorly understood, though some appear to mimic thorns, wasps or ants and thus may function in predator evasion. The species shown here are, from top to bottom: *Umbelligerus peruviensis, Heteronotus maculatus, Cladonota benitezi*, and *Bocydium globulare*. All photographs are courtesy of Patrick Landmann.

increasingly postulated (or rejected) on the basis of gene expression data, the idea being that homologous structures can be expected to express the same genes during development, in similar temporal and spatial patterns, a criterion that could probably be considered as one of Remane's special qualities. Needless to say, much debate exists as to the legitimacy of any of these criteria, or even their collective nature, in establishing homology.[22–26] Ever since Remane's special qualities, homology assignments no longer just consider the final adult version of traits but include the entire ontogeny of traits as well, and no longer consider traits as a whole but explore them as the sum of their parts on every level of biological organization from gene expression to cell identity to organ architecture. It is here where homology assignments become far more complex and ambiguous,[22,25] but at the same time far more interesting. And as introduced in the next section, it is here also where I would argue that novelty can rightfully reclaim ground prematurely vacated, ground that the study of evolutionary innovation needs to focus on if we ever want to understand the origins of novel features.

Why homology is like an onion

Over the decades, evolutionary developmental biology has made many critical contributions to our understanding of the origin of phenotypic diversity, two of which are particularly relevant to this context. The first, and arguably most celebrated, contribution of evo-devo is the observation that the extraordinary phenotypic diversity that exists on the level of organisms and their parts is not paralleled by a corresponding diversity in genetic and developmental mechanisms, at least not as initially envisioned.[29] Instead, the developmental genetic underpinnings of phenotypic diversity are remarkably conserved, and highly divergent organisms rely on the same patterning mechanisms to instruct the development of very different, and clearly non-homologous, organs and structures. For example, the transcription factor *Distal-less* plays a critical role in establishing proximodistal axis polarity across phyla, from the siphons of tunicates to the tube feet of sea stars and the segmental appendages of arthropods.[30] Similarly, programmed cell death involves on one side a highly conserved genetic and cellular machinery, which on the other is employed in a myriad of developmental contexts, from the removal of tissue between fingers and toes and the resorption of the tadpole's tail to the sculpting of pupal into adult features during holometabolous insect development and the destruction of infected or cancerous cells.[31] Clearly, a vast number of nonhomologous traits rely on much of the same developmental genetic machinery to build certain aspects of themselves. While the organs that result from the participation of these structures clearly do not reflect shared ancestry, many of the developmental processes that participate in their making may in fact be the *same* trait in different organisms. Proximodistal

axis formation and programmed cell death are ancient developmental mechanisms, and it makes perfect evolutionary sense that descendant lineages re-utilized them in a wide variety of developmental contexts. We may be able to integrate this complication by considering this a kind of *partial* homology,[32–34] evident on one level of biological organization in the formation of two organs, such as use of the same gene, patterning process, cell types etc, but absent at others. Homology thus becomes more like an onion, layered, and with core layers being able to reflect homology while surface layers may not.

One might argue that partial homology in the above sense may imply that traits that are homologous on the level of the entire organ, such as the legs of flies and beetles, must have maintained homology across lower levels of organization as well. Comparative developmental biologists have long pointed out that this need not be the case;[35–37] however, it took the advent of modern evo-devo to document how frequently indeed phenotypes remain seemingly conserved while their underlying developmental and genetic mechanisms may undergo at times striking divergence.[38] For example, while the legs of adult flies form from imaginal discs specified during late embryonic development, which then grow during most of larval development as two-dimensional invaginations into the larval body, and functionally require the expression of the morphogen *decapentaplegic* (*dpp*) for initial specification,[39] the ''same'' legs of adult beetles do not form until very late in larval development, grow as three-dimensional evaginations into the space vacated between larval epidermis and cuticle during epidermal detachment,[40] and do not require *dpp* expression for normal development.[41] Similarly, the development of the ''same'' vulva in two nematode genera relies on remarkably divergent developmental and genetic mechanisms.[42] Both genera derive their vulva from ventral epidermal cells that express the homeotic gene *lin-39*, but differ in the fate of adjacent non-vulval cells. In *Caenorhabditis*, non-vulval cells fuse with the epidermis, whereas in *Pristionchus*, those ''same'' cells undergo programmed cell death. More importantly, even though *lin-39* mutants are vulvaless in both genera, failure of vulva formation occurs for different reasons: epidermal cell fusion of presumptive vulval cells in the case of *Caenorhabditis*, and programmed cell death in the case of *Pristionchus*.[42] Same organ, same gene, but different mechanisms. Similar differences exist in the mechanisms underlying sex determination in different families of flies[43,44] or the determination of mating type in yeast.[45,46] In each case, the developmental and genetic machinery underlying clearly homologous traits has diverged, at times significantly, despite remarkable conservation of the resulting phenotype, a phenomenon that Weiss and Fullerton[47] termed *phenogenetic drift* and True and Haag[48] referred to as *developmental systems drift* (see both references for many additional examples).

Recently, Wagner[49] sought to integrate these and related observation into a developmental genetic definition of homology, and argued that genetic regulatory networks underlying the expression of particular characters can be separated into networks that give characters their *identity*, and networks, or parts thereof, that determine character *state*. He then proposed that the homology of morphological characters is brought about, and reflected in, the historical continuity of character identity networks. While the function of character state genes is labile, that of character identity genes remains preserved, reflecting the developmental genetic counterpart to morphological homology. A particularly illustrative example supporting such a distinction involves the role of the Hox gene *Ultrabithorax (ubx)* in the regulation of hind-wing *identity* across insect orders that differ dramatically in hind-wing *state*, from the membranous hind wings of beetles to the reduced, club-shaped halteres of flies.[50–52] While hind-wing states changed across these orders, hind-wing identity has remained under the control of *ubx*. A distinction between genes responsible for character identity versus character state thus represents an interesting new perspective, even though its general applicability remains to be determined. Particular challenges may arise from the difficulty in assigning identity to characters where this is less obvious, or separating precisely where identity ends and state begins and, in such cases, resisting the temptation to declare any network conservation one might observe across taxa as indicative of shared character identity. Regardless of whether Wagner's distinction will prove useful, divergence in the mechanisms underlying an otherwise conserved phenotype introduces a peculiar kind of novelty, one that is clearly not reflective of ancestral conditions, yet may at least initially be little more than a divergence in the ''details''. Combined, the recruitment of ancestral developmental mechanisms into ''new'' developmental contexts on one side, and the divergence of developmental mechanisms underlying homologous traits on the other, therefore blur the line between exactly what we can call homologous, and why, and what we might consider novel. The implications of such blurring are, however, substantial. For starters, no two traits are likely to be ever completely homologous, or non-homologous, causing homology and novelty assignments to lose their discreteness and instead to become a matter of degree. Such assignments then risk turning into arbitrary cutoffs along what ultimately is a continuum of differentiation, with homology emerging simply as the expected outcome of descent with modification[23] and novelty beginning where our ability to trace the ancestry of a given trait ends. If correct, the most-productive and honest definition of novelty may then be an operational one, as proposed by Wilkins[53] who distinguished between novelties arising from extensions of known developmental processes and novelties that arise from as of yet unknown processes. Secondly, the above observations raise the possibility that the initial steps that eventually lead to

what we recognize as major evolutionary innovation may be brought about by highly conserved developmental processes operating in subtly altered contexts, and may take place well within the confines of strict homology. Exactly how such early evolutionary innovation may be initiated and elaborated is therefore the subject of the next section. For now, let us accept that what will move us forward is likely not another definition of novelty—or homology—but instead to try to be as explicit as possible about exactly what level of biological organization we are considering, and what traits we are comparing. We may differ in exactly where we draw the line beyond which novelty begins, but chances are we may learn from those who draw it elsewhere. So lets move on from exactly where novelty begins, to *how* it begins.

The origins of *new*: innovation in a pre-Mendelian world

In a series of publications, Newman and Müller[54–56] argued that the mechanisms of evolutionary innovation changed dramatically as the history of life moved from the origin of bauplans to the origin and diversification of their details. Their idea is based at least in part on the postulate that the tight correlation between an organism's form and ontogeny on one side, and its genotype on the other, represents a highly derived state, and one that existed to a much lesser degree during early metazoan evolution. Newman and Müller envision a "pre-Mendelian world" of living organisms characterized by only a loose connection between genotypes and phenotypes, with a single genotype having the capacity to generate many different phenotypes, and the same phenotype originating from many different genotypes. In such a world, organismal "form" would be primarily determined by the biochemical and biophysical properties of cells and cell aggregates and their products, with the potential to generate cell layers, cell differentiation, compartmentalization and segmentation, all in the absence of the kind of tight developmental genetic programming found in extant organisms. According to Newman and Müller, such programming mechanisms may have evolved secondarily and *after* major metazoan body plans were established to promote reliability and repeatability of development, ultimately leading to a "Mendelian world" characterized by a tight matching between genotype, development and phenotype that we see today. Clearly this proposal holds great appeal, at least in part because it would provide an interesting explanation as to why an explosion of form may have been possible early in the history of life, but not since (see also Refs 57,58). But regardless of the possible contributions of a pre-Mendelian world to the diversity of life on earth, much remarkable evolutionary innovation seems to have occurred ever since, including all examples of possible novelties introduced above. In the sections below, I will try to argue that much of this innovation did not occur *despite* an increasingly tight correlation between genotype, development and phenotype, but has been made possible *because* of it.

Innovation as a byproduct

Earlier I introduced what is often referred to as the "selection paradox", the notion that for selection to bring about novel features and their functions directly there needs to be heritable variation for both trait and function in a population, which if it exists renders the trait no longer novel. Put another way, selection can not act on something that does not yet exist. A possible resolution of this conflict is as old as evolutionary biology itself, with the basic notion being formulated by Darwin[7] who, at least remotely, considered the possibility that complex characters may "have originated from quite secondary causes, independently of natural selection" (p. 196). The nature of these *secondary causes* has been explored in great detail by many subsequent publications,[2–4,59] and the remainder of this essay will therefore concentrate on two types of explanation of particular significance.

The first is *exaptation*, the notion that many novel features may have originated for reasons unrelated to their present-day expression and function.[60] Exaptation allows us at least in part to circumvent some of the difficulties associated with the origin of novelty, because we no longer need to invoke selection, or for that manner, any other evolutionary mechanism, to have initiated a given trait of interest alongside its function. Instead, selection or neutral processes already generated a trait, which now finds itself in a previously unencountered, *novel*, selective environment in which it happens to fulfill an adaptive function, and this new selective environment then shapes and elaborates the trait accordingly.[2–4,61] The only requirement is that a given trait becomes exposed to novel selective environments, i.e. traits expressed ephemerally during development may have less of a chance to be exposed to new contexts compared to traits expressed throughout. Here, a special role is likely to be played by behavior, which in the broadest sense can be understood as the means by which organisms interact with their biotic and abiotic environment. The greater the degree and frequency of such interactions, the larger the number of qualitatively different selective environments encountered by a given trait, and hence opportunities for traits to find themselves providing novel functions by chance. Many of our previous examples can easily be fit into such a scenario. Incisors originated well before the appearance of Cetaceans, and originated for reasons completely unrelated to those that ultimately shaped morphology and function of the left incisor of the narwhal.[62] Likewise, the pronotum is the dorsal plate of the first thoracic segment of every insect. Its existence is deeply rooted in whatever forces originally shaped the basic bauplan of insects,[21] which likely bear little resemblance to the forces that eventually shaped the elaborate pronota of Membracid treehoppers.

A particularly illuminating example, and one that has not yet appeared in this essay, are the horns of scarab beetles (Fig. 6). Beetle horns are major cuticular projections of the head and thorax and in regions in which insects normally do not produce outgrowths.[63,64] Used as weapons in male combat over females, horns are similar, and often larger, in size than more traditional appendages such as antennae, mouthparts or legs, but lack muscles, nerves or joints. However, they are not simply modified antennae, mouthparts or legs, which instead still exist alongside horns in the same organisms, and beetle horns can thus not be homologized to other structures in a straightforward manner.[65] At the same time, however, the development of beetle horns appears to rely to a large extent on the exact same processes employed during the development of more traditional appendages, from timing of epidermal outbudding to the establishment of proximodistal axis polarity to sculpting via programmed cell death.[66] As such beetle horns clearly embody something new, except it seems for all the parts and processes needed to make them. But what makes beetle horns, in particular those emanating from the first thoracic segment, especially interesting for the current discussion, is that, apart from understanding how they are made during development and how they function in their current behavioral context, we are also beginning to understand the selective environment that facilitated the actual origin of these outgrowths, alongside the events that may have enabled these traits to appear in a new selective environment. Here, by happenstance, they found themselves providing a novel function, setting the stage for one of the most dramatic radiations of secondary sexual traits found in the animal kingdom.

Figure 6. Beetle horns are projections of the dorsal head and thorax, and in regions in which insects normally do not produce any outgrowths. Beetle horns commonly far exceed the size and mass of regular appendages such as legs and mouthparts, but unlike them lack muscles, joints and nerves. Adults use horns as weapons in male combat over females, however, pupae appear to use at least some horn types to aid in eclosion from the larval cuticle during the larval-to-pupal molt. Shown here are the horns of *Trypoxylus dichotomus* (top left), *Phanaeus imperator* (top right), and *Golofa eacus* (bottom). All photographs by the author.

Specifically, recent studies on the genus *Onthophagus* found that many species go through the trouble of growing and patterning pronotal horns during larval and pupal development without converting these immature horns into adult structures.[67] Instead they reabsorb them via programmed cell death, leaving no trace of the former existence of these structures.[66] In fact, among 19 species sampled thus far, only four converted pupal into adult horns, and only in one sex, while all others resorbed their horns prior to turning adult, raising the question as to the adaptive significance, if any, of transient horn expression. Experimental approaches subsequently revealed that pupal horns actually play a crucial role during the larval-to-pupal molt and the shedding of the larval head capsule, and phylogenetic analysis suggests that this pupal molting function of horns may have preceded that of the adult counterparts, and that ancestrally pupal horns were always resorbed prior to the adult molt.[67] If correct, this suggests that the origin of *adult* horns could have been the result of a simple failure to remove otherwise pupal-specific projections through programmed cell death, and a survey of the available literature suggests that such events indeed occur in natural populations at a frequency high enough to be detected by entomologists. Even though such an outgrowth would initially have been rather small, behavioral studies have shown that, if used in the context of a fight, even very small increases in horn length carry with them significant increases in fighting success and fitness.[68–70] Behavioral studies have also shown that aggressive fighting behavior is widespread among beetles and occurs well outside horned taxa. Possession of adult horns is therefore not a prerequisite for fighting, instead male beetles most likely fought each other well before the first adult horn surfaced, creating a selective environment in which the first pupal horn that failed to be removed before the adult molt could have provided an immediate fitness advantage. Pronotal beetle horns may thus be a good example of a novelty that arose as an exaptation from traits originally selected for providing a completely different function, during a completely different stage of development. Traits such as these horns also illustrate how little evolutionary innovation may sometimes be needed to allow *old* traits to acquire *new* functions.

But, if we are honest, we also have to admit that an exaptation-based origin of novelty only provides an incomplete resolution of the selection paradox, because many of the major

questions are merely pushed one step back in time. For example, while the origin of adult horns may now well be solved, it remains an open question as to how pupal horns, alongside their molting function, originated, and the same logic applies to our previous examples as well. However, what the beetle horn example does illustrate is how development has the capacity to expose traits to selective environments that they previously did not encounter. As we will see in the next section, development's ability to release phenotypic variation into novel contexts is not a new idea, but one whose significance, including for the origin of novel traits through exaptation and otherwise, we are only beginning to appreciate.

Innovation through developmental capacitance and genetic accommodation

Developmental capacitance can be defined as the ability of developmental processes to act as capacitors of genetic variation, shielding it from being visible to selection under some circumstances, but capable of releasing it under others.[71] Developmental capacitance arises from the canalizing nature of many developmental processes, i.e. their ability to buffer the production of a certain phenotype against variation in genetic and environmental inputs,[72,73] and may reach extremes in at least some, though more likely, many cases. A particularly interesting example is the arthropod segmentation network, one of the best-understood developmental regulatory networks. Von Dassow et al.[74] simulated this network and studied its dynamics and robustness in response to perturbations. The main observations were that the network was remarkably robust against perturbations and capable of absorbing orders of magnitude of changes in parameter values without altering the final phenotypic output. Despite this tremendous canalization, new phenotypes eventually emerged as parameter perturbations exceeded certain thresholds. This example is illuminating for primarily two reasons. On one side, it shows how developmental processes, at least in theory, have the capacity to receive a wide range of genetic inputs yet still produce the same phenotype. Put another way, if genetic variation for components of such developmental processes would arise within a population, much of it would be selectively neutral, free to accumulate, and drift. As such, developmental capacitance may be considered one of the causes of the phenomenon of developmental systems drift introduced earlier. On the other, it shows that, once particular thresholds are exceeded, new phenotypes might emerge.[2,3] Putting the two together, exceeding such threshold values may not just reveal a new phenotype, but *heritable variation* in the expression of this phenotype in a population as well. Selection would then be able to act on this variation and shape a novel phenotype.

What is the evidence for the evolutionary capacitance of this sort, and what are the kinds of mechanisms that would permit developmental processes to release genetic variation previously held in a cryptic state? To answer these questions, it may be useful to remind ourselves that canalization buffers against genetic and environmental perturbations. Both genetic and environmental perturbations that exceed the buffering capacity of a given developmental process should therefore also have the ability to expose cryptic genetic variation. Several classic, and a growing number of elegant recent studies suggest that this is indeed the case. In 1953 Waddington[75] showed that a brief exposure to ether during the egg stage of *Drosophila* causes the resulting adult flies to exhibit varying degrees of the so-called *bithorax* phenotype, that is the identity of the third thoracic segment was more or less subtly changed to resemble that of the second thoracic segment.[75] Most importantly, some of this variation appeared to be selectable and the frequency and intensity of *bithorax* phenotypes responded quickly to artificial selection. Remarkably, *bithorax* phenotypes eventually became expressed even in the absence of the originally inducing ether treatment. This experiment was replicated 40 years later[76] with similar outcome and, more importantly, was able to implicate a polymorphism at the *ultrabithorax* gene as the genomic region that permitted the accumulation of genetic variation held in a cryptic state under normal conditions, but made visible to selection under ether treatment. In another important study, Rutherford and Lindquist[77] showed that genetic or pharmacological impairment of the *heat shock protein 90* (*Hsp90*) lead to the release of substantial amounts of cryptic genetic variation capable of fueling rapid responses to artificial selection. Functional *Hsp90* proteins act as chaperones and correct incomplete or faulty folding of other proteins.[78] These proteins include many signal transducers in a variety of important developmental pathways.[79–81] As such *Hsp90* buffers the organism against environment (such as temperature)-induced errors in protein folding, but also against genetic variation in a population. When *Hsp90* function is compromised, this variation is exposed and becomes selectable, and the resulting phenotypes persist even after the originally releasing mutation is lost from the laboratory population.

Canalization of this sort is often juxtaposed to phenotypic or developmental *plasticity*, commonly defined as a single genotype's ability to produce different phenotypes in response to changes in environmental conditions.[82] If canalization and plasticity were indeed opposites, plasticity should counteract development's ability to act as a capacitor for genetic variation. However, the relationship between canalization and plasticity is more complex, in fact, rather than being mutually exclusive, I argue below that in many cases one enables the other, and that plasticity oftentimes amplifies development's capacity to act as a reservoir for genetic variation. Phenotypic plasticity has many manifestations, and considerable debate exists as to whether it is productive to lump them into the same category.[83] On the simplest level, plastic responses may

arise solely from the interplay between gene products, developmental processes and the biophysical and biochemical environment in which they function, a category that Schlichting and Pigliucci refer to as *allelic sensitivity*.[83] For example, changes in temperature or pH often have profound consequences for the rate and outcome of biochemical reactions, including those underlying growth and reproduction. Developmental plasticity arising from allelic sensitivity is commonplace and, as such, probably constitutes as close an opposite to canalized development as is possible. Matters are more complicated with the other major category of plastic responses, often referred to as *regulatory plasticity*, which involves the sensing of changes in environmental conditions followed by coordinated responses in phenotype expression.[83] Regulatory plasticity, too, is ubiquitous and often involves the expression and adjustment of complex phenotypes, ranging from the physiological responses to seasonal change to the production of environment-induced alternative phenotypes and the flexible allocation of resources during organismal growth and differentiation.[4] More generally, regulatory plasticity generates a particular response by a given genotype to a particular environmental gradient, or a *norm of reaction*,[59] a term originally coined by Woltereck[84] and later elaborated upon by Goldschmidt[85] and especially Schmalhausen,[86] whose understanding of the nature and consequences of the interplay between development, environment and adaptive evolution anticipated many of the debates now occurring half a century after the publication of his major work. Paradoxically, even though regulatory plasticity requires environmental sensitivity, it also typically involves a great deal of canalization, as organisms evolved mechanisms that allow them to be responsive to only certain environmental fluctuations while being able to buffer against others at the same time. Canalization also allows plastic organisms to respond only in the expression of selected aspects of their phenotype, permitting others to be expressed unaltered. Lastly, and possibly most profoundly, canalization permits *threshold responses*, i.e. adjustments in phenotype expression may occur only once changes in the environment have passed a certain threshold value. Consequently, regulatory plasticity does not necessarily limit development's ability to act as a capacitor for genetic variation; in fact, it may even amplify it under certain conditions.[87] Such amplification may be particularly obvious whenever regulatory plasticity involves threshold responses, be it to temperature (e.g. diapause), nutritional (e.g. social insect castes), or social (e.g. alternative reproductive morphs in insects) gradients.[4] The existence of thresholds in regulatory plasticity is pervasive, and often involves endocrine processes that mediate between the environmental changes experienced by an individual and the physiological and developmental adjustments initiated in response to such changes.[4] For example, juvenile hormone titers above a certain threshold induce queen development in honey bees, soldier development in ants, horn development in horned beetles, solitary morph development in plague locusts etc, whereas titers below the threshold initiate the development of the alternate phenotype.[82,88] Importantly, while response thresholds such as these can generate precise transition points between very different alternative phenotypes, they permit the accumulation of variants below and above the threshold value. For example, in case of endocrine thresholds, genotypes that differ in precisely how much their hormone titers can fall below or be above a given threshold value in response to a certain environmental gradient will still produce the same alternative phenotypes, *and the same norm of reaction*, as long as they converge on the same threshold. This genetic variation would remain cryptic until external or internal perturbations expose it to selection.[87] This was beautifully illustrated by a recent artificial selection experiment on the endocrine regulation of caterpillar cuticle coloration by Suzuki and Nijhout.[89] Here, a combination of a mutation of large effect and heat shock released cryptic genetic variation for sensitivity to juvenile hormone underlying cuticle coloration. This variation then permitted, over the course of just 13 generations, the selection of both a *monophenic* line, in which cuticle color became insensitive to rearing temperature, and a *polyphenic* line, in which cuticle color changed from black to green in response to increases in rearing temperature in a threshold-like manner. These and other studies therefore suggest that the canalizing nature of development, including the processes underlying developmental plasticity, can act as a natural capacitor for cryptic genetic variation of one kind or another (see also Refs 90–94). Developmental capacitance therefore allows for the origin, accumulation and ultimately release of cryptic genetic variation in response to genetic and environmental perturbations. If the release of this cryptic genetic variation coincides with the appearance of adaptive phenotypes, these could then be stabilized in a population via *genetic accommodation*, a termed coined by West-Eberhard[4] to describe how environmental alterations of development could fuel adaptive evolution, including the origin of novel traits.

Genetic accommodation can be defined as a mechanism by which environmentally induced phenotypic changes that provide a selective advantage are genetically stabilized, or accommodated, through the subsequent selection of genetic modifiers available in a population.[4,95] As a consequence, genetic accommodation may result in altered sensitivity to the originally inducing environment, as in the caterpillar cuticle example above, including genetic assimilation via complete loss of sensitivity in extreme cases, as in the *bithorax* and *Hsp90* examples discussed earlier. Developmental capacitance relates to genetic accommodation, because it provides a plausible mechanism that would permit the accumulation of cryptic genetic variation alongside *cryptic phenotypes*, i.e. phenotypes that development is capable of

producing yet generally does not unless particular environmental perturbations occur. Two important aspects are worth pointing out. First, there is no mechanism available that would allow development to respond to environmental perturbations by generating phenotypes that will subsequently prove adaptive. Just as the vast majority of mutations is prone to be either neutral or detrimental, the vast majority of environment-induced new phenotypes will probably be non-adaptive. However, if *by chance* a certain environmental perturbation alters development such that it produces an adaptive phenotype, and *by chance* releases previously cryptic genetic variation, selection on which could stabilize the newly adaptive phenotype, then evolution by genetic accommodation may occur, and may permit environmentally induced phenotypic variation to become heritable. At the same time, the presumably low probability of environment-induced alteration of development resulting in both the origin of a novel adaptive phenotype, *and* the release of selectable cryptic genetic variation makes the process of evolution by genetic accommodation seem unlikely to occur very often. This, however, may be juxtaposed by the temporal and geographic scale of environmental perturbations, which can operate immediately on the level of populations rather than individuals. By operating on a large number of individuals at once, environmental perturbations can generate phenotypic changes in a large number of individuals simultaneously and increase the amount of previously cryptic genetic variation available to selection.[4] As pointed out earlier, whether induced phenotypes ultimately prove both adaptive and selectable is a chance event but, by virtue of operating on the level of populations rather than individuals, the probability of such an event occurring may be much increased. Furthermore, as environmental perturbations persist over generations, environmental induction of adaptive new phenotypes and the appearance of genetic modifiers suitable for their subsequent accommodation no longer have to co-occur in the same individual or even generation but can, at least initially, be temporally dissociated. Lastly, if environment-induced phenotypes happen to be selectively favorable *and* suitable genetic modifiers happen to exist in a population, such modifiers may well surface in many more than just a single individual, further increasing the chances of new phenotypic variants persisting and even spreading within a population. Evolution by genetic accommodation thus appears clearly feasible, and several elegant studies on a variety of organisms have demonstrated that genetic accommodation can occur in laboratory studies and in the context of artificial selection experiments.[77,89–91] What remains an open question is how frequently it does indeed occur in nature, and whether, by itself, it has the capacity to generate the kinds of major novel traits whose origins we seek to explain. These questions remain to be answered yet one might argue that now more than ever, interesting opportunities exist to examine if and how large-scale environmental perturbations of major ontogenetic consequences, from global climate change to endocrine disruptors, alter magnitude and mode of adaptive evolution in natural populations.[71]

Fake novelties?

I will close by briefly discussing two categories of evolutionary innovation that highlight a central theme of this essay: that to understand where innovation begins we need to search for where, exactly, the already existing ends. We may be surprised by how much new is possible through modifications of the familiar.

The first category starts with the now already familiar notion that homologous traits need not be homologous in their underlying developmental and genetic underpinnings. One avenue that may lead to such an at first perplexing situation may lie in the ubiquity of canalization: many genetic variants often give rise to the same phenotypes, allowing equivalent genotypes to drift within populations, including the possibility that different genotypes may drift to fixation in different populations and species, resulting in genetic and developmental differentiation without leaving a signature in the corresponding phenotypes. Another avenue not yet mentioned involves the notion of developmental redundancy. Often, multiple, redundant pathways ensure the same phenotypic output during development. Differential loss of redundant pathways in different taxa may not yield any phenotypic consequences, but now causes the same phenotype to appear under the control of different developmental and genetic mechanisms. Developmental systems drift is obvious in cases such as the divergent vulva development in different nematode genera,[42] appendage formation in different insect orders,[40] or sex determination in different fly families mentioned earlier.[43,44] We do not need to think twice to establish homology within each of these examples. Things become more difficult, however, if we realize that developmental systems drift is likely to have been omnipresent during the history of life on earth and thus had the opportunity to shape development for a long time. At the same time, developmental systems drift does not prevent homologous structure from also experiencing divergent selection. If developmental drifting alternates with, or is followed by, extensive periods of divergent selection, we may quickly lose any signature of obvious homology between two structures, including their underlying development. In such cases, we may be inclined to postulate more evolutionary innovation than might actually have been necessary.

A possible example for such a scenario is the origin of holometabolous development in insects. Holometabolous development, as seen in butterflies, moths, beetles or bees, involves development from an embryo to an immature larva, which represents the primary feeding stage of most holometabolous insects. Larvae undergo several larval-to-larval molts before molting into a pupa, and ultimately an adult. The pupal stage thus effectively decouples the larval from the adult

stage, and is credited with having allowed larval holometabolous insects to utilize feeding niches otherwise unavailable to other insects, culminating in the dramatic divergence in form, physiology and behavior between larval and adult stages of extant holometabolous insects.[21] This is in contrast to hemimetabolous development as observed e.g. in grasshoppers or cockroaches, in which embryos molt via a brief pronymph stage into a nymphal stage, which in many ways resembles a miniature and incomplete version of the final adult. Through a series of nymphal-to-nymphal molts animals grow in size, eventually add wing buds whose size increases through subsequent molts, culminating in a final nymphal-to-adult molt by which the animal acquires fully functional wings and genitalia. The most widely held hypothesis regarding the origin of holometabolous development postulates that holometabolous larvae are homologous to hemimetabolous nymphs, and that the origin of holometabolous metamorphosis was made possible through the invention of the pupal stage which, consequently, lacks a homologous counterpart among the Hemimetabola.[96–99] However, exactly how an entire new life stage such as the pupa could have been intercalated between larva and adult is unclear.

In an important study, Truman and Riddiford[100] challenged this view and argued in favor of a hypothesis put forward originally nearly a century ago by Berlese.[101] This alternative hypothesis proposes that the holometabolous pupa arose from a compacting of the nymphal stages into a single life stage, making pupal and nymphal stages homologous. The holometabolous larvae in turn arose as an elaboration of the hemimetabolous *pro*nymph stage. The pronymphal stage of hemimetabolous insects is a distinct stage directly following the embryo, but it is so brief and ephemeral that it is spent entirely to largely while the animal is still inside the egg. The pronymphal cuticle is shed either during (as in bugs and lice), or a few minutes to hours after, hatching from the egg (as in grasshoppers[102]). Consequently, the pronymph stage has generally received less attention than other life stages. Truman and Riddiford,[100] however, present compelling evidence consistent with the notion that the holometabolous larva may have arisen through a ''de-embryonization'' of the pronymph stage, converting a largely embryonic stage into a free living larva. The hemimetabolous nymphal stages, in turn, collapsed into what we now recognize as the holometabolous pupa. Consequently, a three-part life cycle already existed prior to the origin of holometabolous development, which instead arose via heterochronic changes in the endocrine regulation of growth and molting. If correct, this hypothesis does not require the invention of a new life stage, just the elaboration and modification of already existing ones. Whether developmental systems drift could have helped initiate such a transition is unknown, but also not really important. What is important, is the realization that homology may at times be very subtle, and if obscured may cause us to postulate origins where modifications may do. Larval evolution in the Holometabola yielded without doubt much amazing innovation and diversification, but if Truman and Riddiford[100] are correct, the origins of the holometabolous pupa itself may be easier to explain than traditionally assumed.

The second category concerns the loss and recurrence of complex traits. Traditionally assumed to be impossible, the recurrence of complex traits is now at least under discussion, and several prominent examples such as the postulated recurrence of wings and flight in stick insects,[103] coiling of shells[104,105] or reversal of digit loss in lizards[106] have reopened the discussion as to whether complex traits, once no longer expressed in a lineage, are necessarily doomed to be lost forever.[107–109] Recurrence of complex traits, and thus homology to a preexisting ancestral trait, is obvious in cases in which recurring traits have retained their ancestral identity. Stick insect lineages that may have ''re-evolved'' wings and flight have done so by re-expressing wings clearly homologous to those of lineages that never lost them in the first place. Recurrence may be less obvious, however, in cases in which more time has passed between loss and return, and in which mechanisms such as developmental systems drift may have set the stage for rapid modification of a recurrent trait once it becomes visible again to selection. Such a scenario is presently entirely speculative, but should at least be considered as an alternative before postulating the de-novo innovation of structures. Possible examples include the paired abdominal claspers of male sepsid flies mentioned at the beginning of this essay. Ancestral arthropods expressed paired appendages from every single segment, including those that have since evolved into clasper-bearing segments of present-day sepsids.[20] And even though ancestral insects subsequently lost abdominal appendages long before claspers appeared, their origin may at least in part be more of a recurrence rather than an invention from scratch. This is supported by the observation that many other groups of extant insects still grow abdominal appendages at least during immature development.[21] Moreover, extant adult insects, including flies, also appear to have retained the ability to grow abdominal appendages, but have made it the responsibility of the abdominal Hox genes *Ultrabithorax* (*Ubx*), *abdominal-A* (*abd-A*), and *abdominal-B* (*abd-B*) to inhibit appendage formation in the abdomen during normal development.[110] If this inhibition is lifted experimentally, each abdominal segment produces a pair of appendages.[52,111] suggesting that all that it may have taken to initiate the origin of sepsid claspers was a developmental failure to inhibit an already existing developmental program. Clearly, much diversification occurred during the evolution of sepsid claspers, but the origin of the appendages themselves may be simpler to explain than we may initially consider.

Conclusions

The origin of novel features continues to be a fascinating and challenging topic in evolutionary biology. Here I have argued that definitions of novelty are significant in helping us capture important components of innovation in evolution, but generally fail to provide obvious starting points for exploring the mechanisms by which novel features arise. Instead, I argue that, in order to understand exactly where, and under what conditions, evolutionary innovation occurs, we need to search for exactly where preexisting variation ends. We may be surprised how much novelty and innovation may arise out of the already familiar, and may grow well within the confines of strict homology. To do so, developmental processes and how they link environmental variation to ontogenetic properties of individuals, and both to the genetic properties of populations, deserve particular attention. Specifically, the canalizing nature of development, the resulting accumulation of cryptic genetic variation free to drift within populations until freed by above-threshold environmental or genetic perturbations, and the process of evolution by genetic accommodation provide a set of interrelated mechanisms of much unexplored potential to explain adaptive evolution, including the origin of major novel traits.

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