Toward a Modern Revival of Darwin's Theory of Evolutionary Novelty

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Darwin proposed that evolutionary novelties are environmentally induced in organisms "constitutionally" sensitive to environmental change, with selection effective owing to the inheritance of constitutional responses. A molecular theory of inheritance, *pangenesis*, explained the cross-generational transmission of environmentally induced traits, as required for evolution by natural selection. The twentieth-century evolutionary synthesis featured mutation as the source of novelty, neglecting the role of environmental induction. But current knowledge of environmentally sensitive gene expression, combined with the idea of genetic accommodation of mutationally and environmentally induced change, supports a revival of Darwin's original theory that is consistent with modern molecular and population genetics.

1. Introduction. An evolutionary novelty can be defined as *a discrete phenotypic trait that is new in composition or context of expression relative to established ancestral traits* (see, e.g., West-Eberhard 2003; Hall 2005; Kaplan 2007). In this essay, I will consider novelties that are somewhat complex—the kind of phenotypic traits that Darwin attempted to explain as products of selection and gradual change. And I will include the establishment of novelties in populations or lineages as regularly occurring traits, not just their initial appearance. The initial appearance of a novelty is an event that depends on preexisting developmental capacities used in other aspects of development, or on "latent capacities" that become evident (reawakened or newly assembled) when a novelty is initially evoked (Hall 2005). I will consider only the evolution of adaptive novelties—that

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is, Darwinian evolution by natural selection. I will not consider pre-Darwinian or non-Darwinian explanations.

2. Darwin's Theory as a Causal Chain to Explain Adaptive Evolution. A satisfactory theory of novelty has to have a series of explanatory elements lined up in what might be called a "causal chain," with a satisfactory mechanism for each step. As outlined by Darwin, the evolution of an adaptive novelty under natural selection is a two-step process: first variation, then spread. Beginning with an ancestral population, such a theory has to describe two aspects of variation: its inception—the initiation of change (the new input that gets it started—an upstream, stimulatory, or regulatory event); and the origin of new form—where the phenotypic end product comes from. Then, to account for spread in Darwinian terms, there has to be an explanation for how the novel variant affects selection (differential reproductive success), which causes an increase in frequency of the novelty within a population. But selection alone is not enough to explain evolution, because it does not explain cumulative phenotype-frequency change that continues across many generations. Such cross-generational change requires inheritance of the trait, which, together with selection, produces a change in frequency over time (adaptive evolution). Differential reproduction without inheritance of the fitness-affecting trait variation, as could occur between differently nourished members of a clone, produces only short-term change in phenotype frequencies.

Darwin's theory of novelty is a marvelously complete and ingenious analysis of this causal chain. In describing it, I will translate partially into modern terms for the sake of clarity and brevity. According to Darwin, the initiation of change is often associated with changed conditions ([1859] 1872, 15–16). The new conditions cause increased variability, including small discrete novel variants, which Darwin called "individual differences" or "pecularities." These, he observed, can be small, medium, or large—even "monstrous"—in size ([1859] 1872, 38–39). The variation can be due to direct effects of the new conditions on the preexisting phenotypes (as in the use and disuse of limbs). But Darwin considered newly induced variations to be influenced significantly by the nature of the organism itself, or what Darwin called "constitutional" properties: "Organic beings, when subjected during several generations to any change whatever in their conditions, tend to vary; the kind of variation that ensues depending in most cases in a far higher degree on the nature or constitution of the being, than on the nature of the changed conditions" ([1868] 1972, 237). In other words, Darwin observed that variation generated in response to conditions depends on what we would now call the genetically influenced makeup of the individual phenotype.

Given an array of newly induced variations, individuals showing dif-

ferent degrees of development of a novel trait will exhibit different degrees of survival or success in reproduction (natural or sexual selection; Darwin [1859] 1872). But the problem of identifying a mechanism for inheritance remained to be solved. If a novelty is originally environmentally induced, how can natural selection increase that novelty's predominance in a population over time? Even if the conditional responses are seen as being to some degree inherited, or "constitutional," from their initiation, the response is still dependent upon conditions. How can there be a lasting effect on the constitutional properties of individuals to explain the observed increase in the frequency of environmentally induced traits?

Once the material basis for inheritance was known to be the gene, the spread of genetically influenced conditional phenotypes could be identified with the spread of genes. This would have followed easily from Darwin's observation (discussed above) that *variation* in the response to conditions depends significantly on variation in constitution. But Darwin could not make the connection between conditional expression and genetic inheritance the way we can make it today nor could he have solved the problems of inheritance he observed had he known about Mendel's work,[1] which did not explain the inheritance of condition-sensitive or of quantitatively variable traits.

Darwin reasoned clearly that an increase in the commonness of an environmentally induced trait would have to depend on inheritance, not on the environmental factor itself: "A fancier who wished to decrease the size of his bantams or tumbler-pigeons would never think of starving them, but would select the smallest individuals which spontaneously appeared. . . . Although every variation is either directly or indirectly caused by some change in the surrounding conditions, we must never forget that the nature of the organization which is acted on, is by far the more important factor in the result" ([1868] 1972, 415).

To solve the problem of the inheritance of environmentally induced novelties under positive selection, Darwin invented a molecular theory of the gene—his theory of *pangenesis* (see esp. [1868] 1972). He hypothesized the existence of tiny particles, which he called "gemmules," that accumulate over generations of positive selection in the region of the body where a novelty is expressed. These particles could then be transmitted between generations. Darwin realized—and this is something that is often forgotten, for it was a feature of genetics ignored by the twentieth-century synthesis—that inheritance involves both the *transmission* and the *expression* of traits; the molecular gemmules, like DNA, were the material

1. Despite Darwin's considerable research and correspondence as a plant breeder (see esp. [1868] 1972), including extensive work on peas, there is now virtually no doubt that he was not aware of Mendel's work (Sclater 2003).

basis for both. Only now, when the age of Mendelian transmission genetics has given way to the age of gene expression, are we beginning to adequately appreciate this twofold role of the genome in both the transmission and the expression of phenotypic traits.

In sum, Darwin explained the increased inheritance of acquired traits with a kind of "genetic assimilation," erroneously ascribed to phenotypically local effects on the particles of inheritance under natural selection. Darwin had to imagine a purely hypothetical mechanism to account for the observed and reported facts because in the late 1800s he had no other choice. He was completely aware of the limitations of his speculations in these respects: he termed his hypothesis of pangenesis "provisional" and acknowledged that it was subject to "a certain portion of incompleteness, and even of error" ([1868] 1972, 349–350). And he repeatedly mentioned the inadequacy of contemporary understanding of the causes of variation: "At the present time there is hardly any question in biology of more importance than this of the nature and causes of variability" (1882, vi).

The claim by some (e.g., Pinker 1998, 522) that "Darwin's . . . embrace of Larmarckism . . . was perhaps his biggest scientific blunder" seems unfair when one considers Darwin's caution regarding his speculations on the mechanism of inheritance, the tendency in subsequent discussions (including those taking place during the twentieth-century synthesis) to brush aside observations that Darwin considered of major importance (the eventual inheritance of environmentally altered phenotypes), and Darwin's seeming ambivalence regarding the inheritance of acquired traits. On the one hand, his highly tentative theory of pangenesis was Lamarckian (and erroneous) in hypothesizing that use could alter the material of inheritance. On the other hand, his discussion of inheritance was sometimes explicitly dismissive of Larmarck: upon noting that social insect workers (which do not reproduce) have traits passed on via the reproduction of queens (which do not work), Darwin concluded, "I am surprised that no one has hitherto advanced this demonstrative case of neuter insects, against the well-known doctrine of inherited habit, as advanced by Lamarck" ([1859] 1872, 207). Much of Darwin's discussion ([1859] 1872, [1868] 1972) of "direct and definite action of the external conditions of life" and on "use and disuse" refers not to Lamarckian inheritance but to what we would now call "phenotypic plasticity."

Finally, Darwin placed enormous emphasis on the importance of cumulative gradualism to explain the origin of complexity. In fact, he staked his whole argument on the principle of gradual change and asserted that the whole theory of evolution by natural selection could be falsified if any exception could be found. He wrote: "If it could be demonstrated that any complex organ existed, which could not possibly have been formed by numerous, successive, slight modifications, my theory would

absolutely break down" ([1859] 1872, 135). It is worth noting that with this statement, and others like it, Darwin's theory meets the scientific requirement of falsifiability.

In that passage of his writing, Darwin places great emphasis on gradualism, when at the same time he stated that novel variants could be large and even "monstrous" in size. Isn't this a contradiction? It would seem that the existence of monstrous selectable variants would make his theory absolutely break down. Although Darwin recognized the existence of large variants, he consistently downplayed their importance. He did so in order to emphasize the central importance of selection. If he had given too much credit to variation, that would have detracted from the power of selection and would, in turn, have opened the door to the possibility of saltatory developmental origins, or even divine creation, of complex novel design. But Darwin had a clever way to remove this contradiction between gradualism and the occurrence of large developmental variants, which I call *retrospective gradualism*: he argued that some saltatory phenomena such as heterochrony and one-step reversions were simply the altered expressions of traits that had evolved gradually in the past. In other words, complex reorganizational variants are not really novelties—they are just old products of gradual evolution whose timing of expression has changed.

3. Mendelian Genetics: A Mutation-Based Incomplete Causal Chain. People are fond of saying that Mendelian genetics solved the problem of inheritance that Darwin had failed to understand. But Mendelian genetics did not account for all of the facts of inheritance that Darwin had observed. The version of evolutionary theory inspired by Mendelian genetics—the "synthesis" of mid-twentieth-century evolutionary biology—was in some respects oversimplified and incomplete. The mutation-based causal chain for the origin of novelty was depicted as follows (see Huxley 1942, 51): a genetic mutation leads (by an undescribed mechanism) to the production of a small phenotypic change in a single individual. Then, owing to a fitness increase associated with the mutant genetic allele, the mutant gene and the associated phenotype increase in the population over subsequent generations. A complex novelty requires a series of mutations. The problem of inheritance is solved by the cross-generational transmission of genetic alleles that have a one-to-one correspondence to the selected phenotype. This scheme was an incomplete account of the phenomena to be explained:

- There is no role for environmental induction—even though the environmental induction of developmental variants is an obvious and undeniable fact, as extensively documented by Darwin and many studies since.

- The mechanism for an effect of mutation on trait development is a black box.
- And any hint that large developmental variants could play a role in evolution is strongly rejected (by, e.g., Fisher 1930; Charlesworth, Lande, and Slatkin 1982), even though such variants occur and evidently can become established during evolution (see West-Eberhard 2003, Chapters 9–19, on evolution by developmental reorganization of the phenotype, sometimes gradual, sometimes by large steps). No hopeful monsters or macromutations are allowed. This synthesis is a strict version of gradualism, in some respects more uncompromising than Darwin's. It followed Darwin in emphasizing the importance of selection, and it went further than Darwin in the corresponding de-emphasis of developmental causes of variation.

In some respects, this is an evolutionary theory in denial, conceptually blind to the environmental sensitivity of phenotypes and to the fact that selection, which acts on phenotypes, must necessarily act on environmentally induced variation as well as on mutationally induced change.

This is a caricature, of course, and there were exceptions. C. H. Waddington was one of them—others were Baldwin (1896, 1902) and Schmalhausen ([1949] 1986), but their ideas were less explicitly related to genetics than were Waddington's. Waddington did figure out a way, through his concept of "genetic assimilation" (1953), to incorporate environmental induction into the genetic theory. Genetic assimilation treated environmentally induced traits as polygenic threshold traits, which could spread if positive selection lowered the threshold for their expression.

Even if this idea had been incorporated into the twentieth-century synthesis, which it was not (for various interesting reasons; see West-Eberhard 2003, 415–416), Waddington's genetic assimilation did not really complete the synthesis as an explanation of novelties. In Section 4, I outline an expanded, remedial proposal.

4. The Origin of Novelty: A Modern Darwinian Causal Chain.

4.1. Phases in the Origin and Evolution of a Novel Trait. I propose that we consider a causal chain that builds on Waddington's insights, as well as on those of the twentieth-century synthesis, but is more complete—more like Darwin's but taking advantage of what we now know about the molecular basis of inheritance and trait expression.

As in Darwin's theory, innovation begins with a population of constitutionally (genetically and phenotypically) variable environmentally sensitive organisms. Then a novel input or initiator impinges on some individuals. The novel input can be either a mutation or an environmental change, such as the uptake of a new building block from outside the

organism, or a new environmental stimulus whose effect is mutation-like in that it induces some novel developmental response. The initiator interacts with the existing, genetically variable individuals, which then react to it with purely phenotypic adjustments (phenotypic accommodation). Some are more responsive than others, and they respond in somewhat different ways to produce a population of individuals possessing the novel phenotype. From the beginning, the novel phenotype would be variable both in its form and in the threshold for its production, due in part to the preexisting genetic variation in the responding population. Then positive or negative selection can produce the *genetic* accommodation of the trait. Genetic accommodation (West-Eberhard 2003) is just gene frequency change due to selection on the genetically variable regulation and form of a novel trait, or of a trait undergoing selection in altered (new) circumstances. Genetic accommodation does not require mutation. It depends on the existence of a standing crop of genetic variation in the population under selection. That such genetic variation exists for virtually all traits is supported by research on genetic polymorphisms and selection experiments using populations of wild and domesticated organisms (Schmalhausen [1949] 1986; reviews in West-Eberhard 2003; Kaplan 2007).

Note that in this scheme much phenotypic change can take place before there is any appreciable gene-frequency change, due to phenotypic accommodation (West-Eberhard 2003), which can be an adaptively appropriate phenotypic adjustment and can involve extensive remodeling of development. Even if a novelty begins with a mutation, most of the genetic change affecting its spread would usually come later, in the form of genetic accommodation.

4.2. Genetic Accommodation and Genetic Assimilation Compared. Genetic accommodation allows for a more complete description of genetic change during the evolution of novelty than that suggested by the idea of genetic assimilation, which depicts all change as moving toward increased frequency of expression and increased genetic control and which considers only environmentally induced traits. Genetic accommodation, by contrast, applies to both mutationally and environmentally induced novelties and can either raise or lower the frequency of expression of a trait by lowering or raising the threshold for its expression or by effects on the liability to produce a response. Moreover, it includes genetic modification of trait form, in addition to regulation. It also can result in more or *less* genetic control of the phenotype: when environmental sensitivity is advantageous, genetic accommodation can act to favor it; and if some environmental factor is essential for the development of the trait, genetic accommodation would favor such traits as habitat selection and other

kinds of "niche construction" (Odling-Smee, Laland, and Feldman 2003) that increase the probability of access to those factors. Genetic accommodation, then, can explain how conditional alternative behavioral phenotypes and polyphenisms can originate, beginning with an environmentally induced novelty that evolves, under genetic accommodation, to be maintained as an adaptive alternative to an established trait. Genetic assimilation, by contrasts, envisions only evolution toward fixation (increased frequency and genetic control).

Genetic accommodation is a universally applicable concept, not an exceptional one applicable only to special cases in the evolution of novelty. It describes the genetic response that is expected whenever a polygenic novel or established trait comes under a new regime of natural selection. In this sense, it is nothing new: genetic accommodation is simply the quantitative genetic response to selection on polygenic thresholds and the coexpressed sets of genes of the "downstream" traits whose expression the threshold-affecting gene modulates or controls. This is a thoroughly documented and formally well-characterized process (see, e.g., Falconer and Mackay 1996). Given the modular nature of discrete phenotypic novelties, genetic accommodation is the necessary substitute for a single-locus model of change that implies an erroneous one-gene, one-phenotype view of the genetic architecture of evolving traits.

4.3. The Nature and Origin of Selectable Variation. In this proposal, much of the *phenotypic* change in the origin of novelty is reorganizational, a product of reexpression of preexisting sets of genes. Reorganizational phenotypic change, or developmental recombination (West-Eberhard 2003), is based on modular dissociability—it is like moving the furniture. Modular organization of the phenotype and adaptive phenotypic accommodation (West-Eberhard 2003, 2005) permits what Kirschner and Gerhart (2005) call "facilitated variation," variation that capitalizes on already functional subunits for the assembly of new structures.

One might well ask if there is any real novelty, given the prominence of reorganizational change, which characterizes even genomic "mutations," which in turn often involve duplication and reorganization of DNA subunits (see diverse examples in West-Eberhard 2003, Chapter 17). But there is one kind of evolutionary change whose true novelty cannot be denied, and that is the kind that occurs when a lineage incorporates some new element from the external environment. The heme group of the hemoglobin molecule is a good example. This is a true novelty. No amount of reorganization can explain the iron atom in hemoglobin, and genes cannot make it by themselves. True novelties also arose with the advent of calcified shells during the Cambrian explosion, when calcium became newly common in the oceans; with the novel incorporation of essential

vitamins or microorganisms that aid digestion in cows, termites, and aphids; and with the incorporation of luminescent bacteria into the light organs of some marine invertebrates. No amount of reorganization could account for the origin of novel form that depends on new elements from outside the organism for its innovative nature.

5. Conclusion. Whether or not the scheme for the origin of novel traits that I have outlined above is completely convincing, we are in an exciting era for biologists and philosophers who aspire to formulate a complete causal theory of evolutionary novelties. We no longer have to guess at the mechanisms of inheritance and development. But modern molecular genomics and evolutionary developmental biology, important sources of the new information, still pay too little attention to the developmental role of the environment. I find it remarkable that we have yet to achieve the comprehensiveness of Darwin's original theory of novelty, and we will not match it until there is a wider appreciation of the role of the environment in the production of the selectable variation that creates novel form.

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