BOOKS: EVOLUTIONARY BIOLOGY

An Evolutionary Dead End?

H. Allen Orr

Good science demands two things: that you ask the right questions and that you get the right answers. Although science education focuses almost exclusively on the second task, a good case can be made that the first is both the harder and the more important. Getting Mendel's laws from Mendel's data may not be easy, but surely the hardest part is daring to ask Mendel's question: Despite all appearances to the contrary, might heredity obey simple laws?

Phenotypic Evolution's strengths and weaknesses map neatly onto this distinction between right questions and right answers. The book's chief accomplishment is that it forcefully reminds evolutionists of a set of important but neglected questions. Do novel characters arise by mutations of large or small effect? Is long-term evolution constrained by the genetic variation found at any one time or by the range of morphologies that is developmentally possible? At their best, Schlichting and Pigliucci's discussions force biologists to face a fact whose magnitude has been obscured by a good deal of wishful thinking: Our understanding of phenotypic evolution remains appallingly weak.

Phenotypic Evolution
A Reaction Norm Perspective
by Carl D. Schlichting and Massimo Pigliucci
Sinauer Associates, Sunderland, MA, 1998. 400 pp. Paper, $38.95. ISBN 0-87893-799-4.

The cause of our situation is clear. Over the last few decades, evolutionary geneticists have been obsessed by evolution at the level of molecules. This focus has had two consequences. The first is success—no sane person could deny the spectacular achievements of molecular evolution. But the second is neglect. Evolutionary biology, a notoriously faddish field, seems constitutionally incapable of holding two problems in its head simultaneously and, consequently, a whole range of fundamental questions has been sidestepped. Recently a number of us have made much noise about the neglect of the phenotype—especially of the genetics of adaptation—and Schlichting and Pigliucci's book represents the loudest outburst yet.

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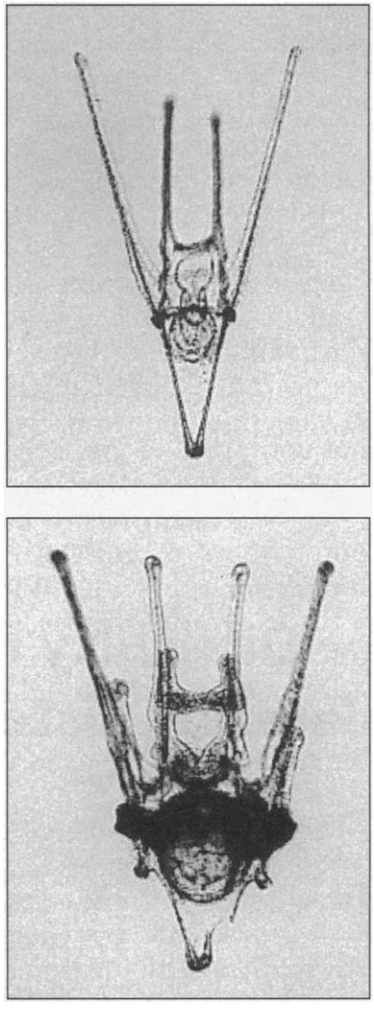
Unfortunately, their attempt to answer these neglected questions is less satisfactory. Schlichting and Pigliucci believe that subtle interactions between genotype and environment play a key, but overlooked, role in evolution. They are particularly interested in adaptive plasticity, in which a genotype gives rise to different but appropriate characters in differing environments. For instance, genetically identical caterpillars raised on different colored twigs might each assume the appropriate cryptic color. Schlichting and Pigliucci bring plasticity together with development and allometry to form their central idea, which they call the "developmental reaction norm," that range of developmental paths a genotype can take when exposed to a variety of environments. Evolution, in their view, means selection on these norms. To survive, organisms must weather environmental noise as well as throw the right switches when confronted with a given environment.



Form follows feeding. Phenotypic plasticity in the morphology of 9-day-old larvae of the sea urchin *Paracentrus lividus*. With reduced food (top), larvae exhibit increased allocation to food-catching structures; with enhanced food (bottom), they invest more in juvenile structures.

Although there is some truth to all this, big questions remain: Why give such primacy to plasticity and the developmental reaction norm? Why build a whole new perspective on evolution from the DRN point of view? Schlichting and Pigliucci give three reasons for doing so, but none seems terribly convincing.

First, they suggest that the developmental reaction norm is the true target of selection, not individual characters. But this is at best confused. While it is sensible to suggest that the developmental reaction norm is as real a character as any other, there is no reason to think it is more real. As Alan Robertson emphasized, the only character that can claim to be the literal target of selection is fitness itself, with all other characters changing as correlated responses. Squabbles over whether selection really "sees" viability or body size or, now, the developmental reaction norm, are misguided.

Second, the authors argue that much evolution involves not simple gene substitution but rather genetic assimilation, in which a new character first appears as a plastic response to environmental shock and only later gets genetically codified. But most evolutionists view assimilation as a curiosity of unclear relevance. So what new experiment or theory has convinced Schlichting and Pigliucci that assimilation is the modus operandi of evolution? Remarkably, the answer is none. Instead they rattle off a few less-than-convincing reasons for doubting the traditional view. But this won't do. Evolution has been directly observed in a good number of cases—including industrial melanism, and resistance to insecticides or antibiotics—and, as far as I can tell, none involves a hint of assimilation. Unless and until there are hard data demonstrating the frequent occurrence of assimilation, evolutionists will rightly refuse to ground theories of adaptation on such a baroque hypothesis.

Finally, Schlichting and Pigliucci suggest the developmental reaction norm may allow a unified view of phenotypic evolution. But their own book undermines this claim. Entire chapters (that on allometry, for example) seem detached from the rest of the book, and the summary chapter reads like a random walk—we are treated to the developmental genetics of butterfly spots, fitness sets, metabolic theory, duplicate

genes, the evolution of pleiotropy, the accumulation of modifier mutations, and so on. The authors themselves brand the chapter "conceptual meanderings." But it is one thing to meander, another to synthesize. This lack of coherence lends the book a diffuse, unrigorous quality and is, in the end, its most serious problem. Now and then the clash of ideas gets so bad that the text slips into unintelligibility, as when we are told, "The innate complexity of genetic systems necessarily leads to emergent properties arising from epigenetic processes that integrate the output components of myriad local genetic programs into a functional global phenotype." Such talk seems an unlikely first step to clearer understanding.

Phenotypic Evolution is representative of a popular trend—some pet theory (take your pick: plasticity, development, complexity theory, or chaos) gets elevated to its rightful place as "the" way to think about evolution. But this longing to dress up biology in unusual new perspectives has, so far, yielded more book deals than results. Although new ways of thinking will surely be required in the attempt to unravel the genetical evolution of the phenotype, considerable care and taste are needed in their selection. Schlichting and Pigliucci's confused admixture is not the perspective we have been waiting for.

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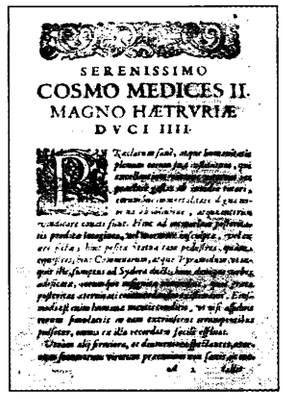

Galileo Galilei's *Sidereus Nuncius* (1610) has been called the most earthshaking book published in the history of astronomy. In this work, Galileo defined a modern view of the universe and our place in it. His most important observation—that Jupiter was at the center of what appeared to be a mini–planetary system—simultaneously demonstrated that the sun and humankind were not foci of the heavens. Benjamin Franklin's *Experiments and Observations on Electricity* (1751) is one of the most important treatises of the 18th century because of its systematic approach to a physical phenomenon. The rarity and fragility of these original documents have for centuries put them out of reach for all but a very select, privileged few. Thanks to digitizing technology and Adobe's Acrobat Reader, however, we now can scroll page-by-page through carefully scanned original versions of these fascinating works. Other significant historic scientific books also available on CD-ROMs (and as downloadable Acrobat files) from Octavo Corporation (Oakland, CA; www.octavo.com) include Nicholas Copernicus' *De Revolutionibus Orbium Coelestium* (1543), Robert Hooke's *Micrographia* (1664), William Harvey's *Exercitatio Anatomica de Motu Cordis et Sanguinis in Animalibus* (1628), and Isaac Newton's *Opticks* (1704).

The detail and beauty of these documents in full color is breathtaking. One can see the texture of the pages, down to the actual indentations made by the metal type on the original paper and the inevitable marks and colorings of age. Users can "thumb through" the books as many times as desired without worrying about damaging fragile pages. Complete English translations are provided where appropriate, and computer search tools allow the reader to easily locate text of interest. Best of all, the modest price of the products ($25 to $75) enables virtually anyone with access to a computer to view these classic volumes in all of their original glory. —KEVIN AHERN

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PERSPECTIVES: CELL CYCLE

The Difficulty in Separating Sisters

Terry L. Orr-Weaver

One of the defining events during cell division (mitosis) is the separation of sister chromatids that are attached to each other and to the mitotic spindle, a process called cohesion release. But we are only now beginning to understand how the powerful cohesive forces that hold the sister chromatids together are overcome. In the budding yeast, *Saccharomyces cerevisiae*, the chief player seems to be the Espl protein—a so-called separin—which is also found in human cells, raising the possibility that the mechanism of cohesion release is highly conserved (*1*). On page 418 of this issue, Zou *et al.* add another piece to the puzzle with their report that an oncogene, *PTTG* (pituitary tumor-transforming gene), acts as a regulator of Espl. This suggests that defective regulation of cohesion may contribute to cancer by promoting chromosome instability (*2*). Like a classic murder mystery plot, the prime suspect for "doing in" sister-chromatid cohesion, the anaphase-promoting complex (APC), now turns out to be a mere accomplice, with the less colorful Espl character actually responsible for the deed.

Physical association of the sister chromatids is crucial for their stable attachment to microtubules from opposite spindle poles and for their proper segregation at the transition from metaphase to anaphase. Cohesin, a conserved multiprotein complex, is essential for the tight association of the sister chromatids, which is established as the DNA is replicated during S phase (*3*). The cohesin complex is localized along the length of the sister chromatids and, as shown by a report in last week's *Science*, is also bound to the centromere (the constricted area of the chromosome to which spindle microtubules bind) (*4*). In budding yeast, two cohesin subunits, Scc1p (also called Mcd1p) and Scc3p, dissociate from the chromatids at the onset of anaphase, coinciding with release of cohesion.

Ubiquitin-mediated proteolysis is necessary to activate the transition from metaphase to anaphase. The APC tags mitotic cyclins with a ubiquitin marker, targeting them for degradation; the cells are then able to exit mitosis. The APC also has other substrates that must be degraded to ensure sister-chromatid separation (*5*). The initial theory that the APC directly releases cohesion by degrading cohesin was disproved by the finding that cohesin subunits persisted into telophase, the final step of cell division (*6, 7*). Rather, it turned out that APC triggers the degradation of a group of proteins called securins that inhibit sister-chromatid separation. Securins include Pds1p in *S. cerevisiae* and a different protein, encoded by the *cut2*

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