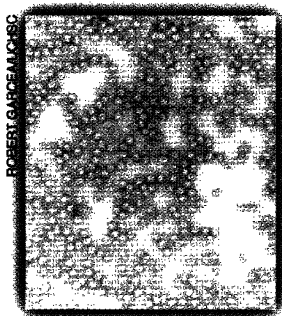


LETTERS



Seeking a pattern

An author questions whether "the standards and procedures" used in "advocacy research" are "minimally sufficient to support the conclusions reported." A reason why simian virus 40 (left), which was a contaminant in some batches of polio vaccine in the 1950s, could play a role in human cancer is presented. And a debate continues about the nature of patterns that some call "punctuated equilibria" in the evolution of bacteria in vitro and organisms in the fossil record.

Standards for Advocacy Research

As one of the four Brooklyn College scientists who drafted the petition citing the New York Public Interest Research Group (NYPIRG) for research misconduct, I would like to clarify several points in the article "Charges fly over advocacy research" by Jock Friedly (News & Comment, 7 Mar., p. 1411). The issue is not whether advocacy research should be held to the same standards as those used for peer-reviewed science (our petition did not address that question), but whether the standards and procedures used are minimally sufficient to support the conclusions reported. Our petition states that NYPIRG research does not "meet accepted minimum standards."

Contrary to NYPIRG's statement, its study of the Audi 500 car (1) contains original data [as does its landfill study (2)], which as NYPIRG acknowledges in print (1, p. 95), were gathered in a survey it prepared. In any case, the premise that studies without original data should be judged by lower standards than studies with such data is fallacious. Any data, original or not, can be manipulated to support a desired, albeit invalid, result.

The implication of political scientist Michael Kahan that our petition was issued in lieu of publishing a rebuttal to NYPIRG's studies is incorrect: three of the five NYPIRG studies at issue were critically examined in scholarly journals (3). The evidence developed in those publications is unassailable. For example, the analytical procedure used in NYPIRG's recycling report (4) provides a result which, while favorable to their position, is physically impossible (its proposed recycling plants yield 3600 more tons of material daily than they take in).

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SV40 and Human Cancer

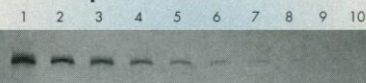
The principal reason for concern that simian virus 40 (SV40) might play a role in human cancer is not mentioned in the article "Monkey virus DNA found in rare human cancers" by Elizabeth Pennisi (News & Comment, 7 Feb., p. 748). The reason is that SV40 has been shown to be capable of transforming normal human cells into cancer cells in vitro (1). Enormous efforts were made after the discovery of SV40 in polio vaccines in the 1960s to have vaccine manufacturers switch from monkey kidney cells as the substrate for virus replication to normal human cells, for the very reasons (2) described in Pennisi's article.

Unlike the case with primary monkey kidney cells, no extraneous viruses have been found in normal human diploid cell

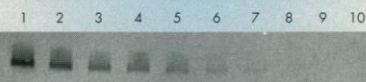
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populations such as the well-characterized strain WI-38. Yet it took 10 years for the production of polio vaccine in WI-38 to be accepted by the Division of Biologics Standards (now the Center for Biologics Evaluation and Research of the U.S. Food and Drug Administration), even as the risk of SV40 exposure continued.

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On Punctuated Equilibria

In their technical comment of 6 December (p. 1748) on a report of punctuated patterns of evolutionary change in a laboratory cul-

ture of *Escherichia coli*, Jerry A. Coyne and Brian Charlesworth perpetuate several incorrect perceptions of the original notion, and subsequent discussions, of punctuated equilibria (1).

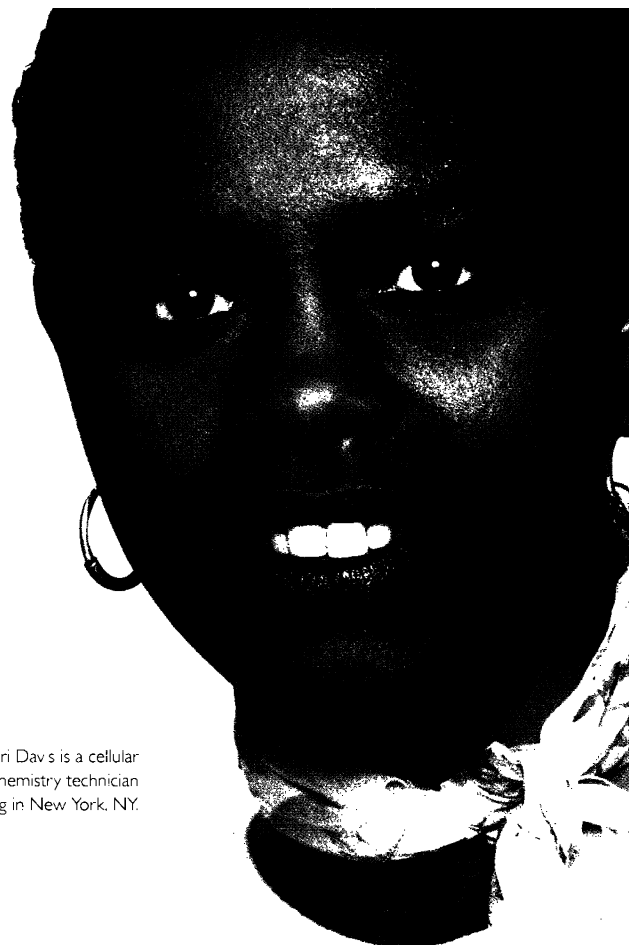
They are correct that punctuated equilibria apply to sexually reproducing organisms and that morphological evolutionary change is regarded as largely (if not exclusively) correlated with speciation events. However, they err in suggesting that we attribute stasis strictly to "developmental constraints," which represent only one of a set of possible mechanisms that we have suggested for the causes of stasis. Others include habitat tracking and the internal structure of species themselves [for example, (2)].

Habitat tracking [as when the distributions of Pleistocene species oscillate on a north-south gradient in concert with global temperature fluctuations (3)] demonstrates the stability of species morphologies in the face of environmental change as long as suitable habitat can be found and occupied. Although habitat tracking does not rule out developmental homeostasis, it suggests that stabilizing selection, rather than directional selection, will be the rule as long as species can continue to "recognize" and occupy suitable habitat elsewhere under a regime of environmental change.

Moreover, the very structure of species, broken up into semi-isolated populations each integrated into different ecosystems, should for the most part preclude the possibility of any widespread species evolving as an entirety in any one particular direction (4). Genetic theory should have explicitly predicted stasis in numerically rich species. B. S. Lieberman *et al.* (5) present empirical data suggesting that stasis in two species lineages of Devonian brachiopods was governed more by such constraints of species organizational structure than by habitat tracking.

Coyne and Charlesworth further state that "the punctuated changes in the fossil record are said to occur via 'species selection,' in which descendant species rapidly supplant their ancestors"—a statement they attribute to Santiago F. Elena *et al.* (Reports, 21 June 1996, p. 1802). Species selection is one of a set of models proposed to explain evolutionary trends in the fossil record—given the absence of strong evidence that long-term trends are merely the accumulated outcome of within-species gradual directional evolution (1, 2, 6). Replacements of ancestral species by presumed descendants (which usually appear "punctuational" in the fossil record) are generally interpreted as ecological or biogeographic

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