tween well-below replacement fertility and well-above) and nearly 50% of births in California (4). (The year 2000 census will likely not measure this source of population growth because there is no question asking for the birthplace of parents.) These data portend a substantial increase in childbearing-age population by 2010. By choosing to maximize rather than optimize population, our political leaders have sealed our ecological fate.

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Defining "Microbiology"

Should the term "microbiology" and its derivatives be limited to situations when all microorganisms (bacteria, viruses, and mycoplasmas) are implied? I propose that, when bacteria alone are meant, the term "bacteriology" should be used. The profound differences between bacteria and viruses have been recognized for at least 100 years. While bacteria are of a cellular nature, viruses are acellular; they replicate only as parasites of host cells, and they are metabolically inert outside host cells. "Microbiology" may have been synonymous with "bacteriology" until the turn of the century, but virology is now an important branch of microbiology. The scientific output of virologists in the last decades has undoubtedly equaled if not exceeded that of bacteriologists. Why is it then that "microbiology" is still used when bacteria are exclusively discussed (E. J. Strauss and S. Falkow, "Microbial pathogenesis: Genomics and beyond," Articles, Frontiers in microbial biology, 2 May, p. 707)?

The same holds true when advertisements are placed for employment opportunities, pharmaceutical products, and so forth. An opening for a microbiologist might be advertised, when, in fact, a bacteriologist is sought. This results in a waste of time and money while inquiries about the exact nature of the position are made. Is it unreasonable to ask that we recognize that bacteria are not the only form of microorganisms?

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Response: We agree with Cepica, for the most part, and apologize for any confusion our title may have caused. Indeed, viruses and bacteria are distinct entities and should be treated as such. Along the same lines, language associated with them should reflect the differences. Of course, there is probably some blurring of the biology at the edges; for example, many bacteria (for example, species of Rickettsia and Chlamydia), as well as many bacterial endosymbionts, are obligately intracellular, replicating "only as parasites of host cells, and . . . are metabolically inert outside host cells."

Occasionally it makes sense to take some liberties with language to avoid excessively clumsy prose. Everyone probably has his or her own sense of where these lines should be drawn.

With regard to our title, we did carefully consider these issues and opted for "Microbial pathogenesis" as opposed to "Bacterial pathogenesis" because we in-

cluded discussion of a virus (vaccinia). Although we understand Cepica's (and presumably others') disappointment about the relative lack of "viral" subject matter in our article, we did not want to be inaccurate and lump vaccinia in with a bunch of bacteria.

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Timing of Permian-Triassic Anoxia

Yukio Isozaki outlines thresholds in deepening oceanic anoxia and subsequent oxygenation across the Permian-Triassic boundary (Reports, 11 Apr., p. 235). He implies that mass extinction at this time was a product of oceanic stagnation many millions of years in the making. There is, however, continuing uncertainty about the position of the Permian-Triassic boundary in the Japanese section studied. That section lacks critical ammonites and conodonts, or even geochemical markers, such as the change in carbon-13 isotopic lightening, that characterize the boundary elsewhere (*1*, *2*). The Permian-Triassic boundary could have equally preceded or coincided with the onset of "climax superanoxia," rather than postdated it, as proposed in Isozaki's stratigraphic section. If "climax superanoxia" postdated the Permian-Triassic boundary, as in the best documented sections elsewhere (*1*), then there are alternative hypotheses to long-term descent into "superanoxia" and recovery. Instead, Late Permian anoxia could have been related to an extinction event during the Late Permian (end-Guadalupian extinction) (*3*), with "climax superanoxia" and delayed recovery forced by the greatest of all mass extinctions at the end of the Permian (end-Changxingian extinction) (*1*). Oceanic anoxia could then have been a consequence of death, decay, and boom-or-bust population cycles (*4*) forced by other agencies (*5*), such as voluminous volcanic eruptions (*6*), impact of an unusually large bolide (*7*), or both (*8*).

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Response: Concerning the age assignment of the Permian-Triassic boundary section in Japan, microfossil age constraints have recently been enhanced: (i) conodonts (*Neogondolella chanxingensis* and *N. subcarinata*) of the Changxingian stage were found in lower gray cherts below the claystones; (ii) three radiolarian zones were newly defined for the interval of the late Wuchapingian and Changxingian in the uppermost part of the lower gray chert; (iii) Paleozoic-type radiolaria *Follicucullus* species were found (although few) from the black "boundary" claystone without any Triassic