the mycobacterium stopped 20 years ago" (News & Comment, 28 Feb., p. 1064), I owe an apology to many, particularly to those who have developed the means for the present-day sequencing of the genome of *Mycobacterium tuberculosis*; the cloning, sequencing, and provision of many of its key antigens; the development of recombinant live Bacille Calmette-Guérin (BCG) and other vaccines expressing M. *tuberculosis* antigens; the cloning of DNA responsible for the synthesis of complex secondary gene products; the characterization of repeated insertion sequences and their application to the diagnosis and epidemiology of tuberculosis; and so on. And, of course, there has been considerable activity in the chemical definition of the cell wall of M. *tuberculosis* and the chemistry and physiological function of many of its proteins.

The point I made was that research on the molecular mode of action of the frontline tuberculosis drugs stopped in the mid-1970s, and work on the genetic basis of resistance to these drugs was never initiated at that time. For instance, Winder and Collins (*1*) showed in 1970 that isoniazid had an instantaneous and profound effect on mycolic acid synthesis, and Takayama *et al.* (*2*) in 1975 were to show that fatty acyl chain elongation and insertion of a Δ-5 double bond were probably primarily affected by isoniazid. However, subsequent work on the mechanism of isoniazid action and mycolic acid synthesis virtually stopped at that time. I also made the point that elucidation of the biosynthetic pathways leading to the complex D-arabino–D-galactan–peptidoglycan of M. *tuberculosis*, a molecule that governs many of the pathogenic features of the tubercle bacillus and is the site of action of several drugs, had not been pursued, unlike analogous structures in gram-positive and gram-negative bacteria, and that now there was a dearth of biochemists and molecular geneticists in these specialized areas, areas crucial to our understanding of drug action on M. *tuberculosis* and the development of new drugs.

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REFERENCES

1. F. G. Winder and P. B. Collins, *J. Gen. Microbiol.* **63**, 41 (1970).
2. K. Takayama, H. K. Schnoes, E. L. Armstrong, R. W. Boyle, *J. Lipid Res.* **16**, 308 (1975).

Provocative Crystallography

I am quoted in a boldface italic display as saying (Research News, 13 Mar., p. 1355) that "I wrote [my 24 February *Physical Review Letters*] article to be downright provocative and almost insulting." I was explaining to your reporter that I had adopted a polemical tone in the hope of enticing a response out of the users of the crystallographic scheme I was trying to improve upon. In the context of our conversation it was clear that my intent was to provoke, not insult. My respect for the designers of the superspace scheme is considerable, and I am pained by the prominent display of a fragment of an informal conversation so that it appears otherwise.

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Neem Seed Oil Toxic to Crustaceans

Before getting too enthusiastic about the potential of neem seed oil, which appears to be a promising biological control agent (Research News, 28 Feb., p. 1070), one should note that the mechanisms of action of this agent (blocking ecdysone and inhibiting chitin formation) are ones that are likely to cause problems in most arthropods, including crustaceans. Other control agents with similar modes of action, such as Dimilin, have been found to be extremely toxic to crustaceans and therefore risky for use in areas where they could enter aquatic systems and damage organisms that are important in food webs.

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Corrections and Clarifications

The author notes for the report "Multiple intestinal neoplasia caused by a mutation in the murine homolog of the APC gene" by L.-K. Su *et al.* (1 May, p. 668) should have indicated that there were two authors to whom correspondence should be addressed: Amy Rapaich Moser, McArdle Laboratory for Cancer Research, University of Wisconsin, Madison, WI 53706; and Kenneth W. Kinzler, Molecular Genetics Laboratory, Johns Hopkins University School of Medicine, Baltimore, MD 21231.

An item in ScienceScope (14 Feb., p. 787) should have stated that a supercapacitor could extend battery life in an electric vehicle from 400 to 800 cycles, thereby effectively doubling vehicle range.