factors, for example, active hepatitis, cytomegalovirus, Epstein-Barr virus; nonintravenous drug use; autoimmune conditions; malnutrition; infections requiring antibiotic use; and so forth (*1*, *2*)] and (ii) free of coinfection with suspected AIDS cofactors such as mycoplasmas, viruses, or human leukemia cells that are universally present with HIV in laboratory situations (*2*, *4*).

Indeed, Cohen's articles do not mention idiopathic CD4–T cell lymphopenia (ICL)—cases that match the clinical description of AIDS but lack all sign of HIV infection. If three cases of "risk-free" HIV infection "prove" causation, then the more than 100 ICL cases "disprove" it. Logically, of course, neither conclusion follows. What we must focus on are the more than 500,000 AIDS cases in North America and Europe in which HIV is accompanied by exposure to cofactors (and the universality of such cofactors in sub-Saharan Africa and Asia). There lies the clue to understanding AIDS, and through intervention with these cofactors, an alternative mode of preventing and treating AIDS.

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Corrections and Clarifications

In the report "Activation and regeneration of rhodopsin in the insect visual cycle" by A. Kiselev and S. Subramaniam (25 Nov., p. 1369), the second and third sentences of the last paragraph on page 1371 were incorrectly printed. They should have read, "When rhodopsin was re-excited immediately after it was regenerated, most of the metarhodopsin formed was thermally stable (that is, M^b was formed predominantly; Fig. 4A). However, when rhodopsin was re-examined after waiting for various lengths of time (in the dark), an exponential increase was observed in the amount of thermally unstable metarhodopsin produced, implying heterogeneity in the rhodopsin population."

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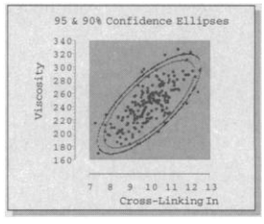

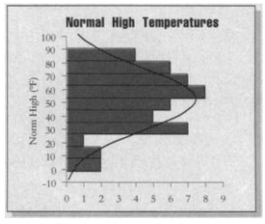

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