the result of infection by HIV-1 B. The latter has been linked primarily to receptive rectal intercourse among homosexual men and injection drug use. We postulated in our report that prevailing strains of HIV-1 B in the United States and Europe might have reduced capacity for transmission by vaginal intercourse.

Most researchers would agree that LCs represent the most logical target for cervico-vaginal entry and exit of HIV-1. We postulated that most isolates of HIV-1 subtypes such as E and C, which have been linked to the major heterosexual epidemics, might grow more efficiently in LCs. The data we published support this hypothesis, which has at times been misconstrued to mean that no subtype B could ever be adapted to heterosexual transmission.

Pope *et al.* cite two recent papers that they interpret as not supporting our hypothesis. The data in the paper by Dittmar *et al.* (*1*) seem compatible with our hypothesis. We believe that the other paper cited (*2*) has several internal inconsistencies and is not relevant to our hypothesis. These two papers, cited by Pope *et al.* to make their point, are in major disagreement with each other. One shows that direct replication of HIV-1 occurs in LCs (*1*); the other states that such replication cannot occur (*2*).

A third paper, not cited by Pope *et al.*, defines two separate pathways by which LCs may be infected (*3*). The first, described earlier by Pope *et al.* (*4*), requires fusion between LCs and T4 cells (LC/T conjugates) for productive HIV infection to occur. Entry for this first pathway is based on the capture of the virus and is independent of the known receptors and co-receptors (*3*). Without evidence for LC/T conjugates in vaginal mucosa, it is unclear how this capture pathway could be involved in the selective production of HIV-1 for heterosexual transmission, as has been described (*5*).

The second pathway for infection of LCs is productive infection involving LCs alone (*3*). This is more relevant to our results and to our hypothesis. This pathway appears to be dependent on cytokines and to be mediated through CD4 and chemokine receptors (*3*). It is this second pathway that best fits the results of Dittmar *et al.* (*1*), which describe productive replication in LC in the absence of LC/T conjugate formation (*1*). Others have also described this productive infection pathway (*6*).

Dittmar *et al.* examined 26 HIV-1s from six subtypes, HIV-1 A to F. On the basis of their data (*1*, table 1), we believe there is a statistical difference (*7*) in that the 17 HIV-1 non-Bs grew better in LCs than did the nine HIV-1 Bs. Also, of the six HIVs found to grow better in LCs than in peripheral blood mononuclear cells, five were of non-B subtypes. Is this result not compatible with our data and our hypothesis?

Pope *et al.* also state that the selective transmission of NSI viruses cannot be attributed to selective expression of certain co-receptors on LCs. However, recent observations of Blauvelt *et al.* show that tissue LCs, unlike cells in culture, show selective surface expression of CD4 and CCR5, but not CXCR4 (*8*). Whether this can fully explain the selective release of NSI viruses remains to be determined. However, the selective release of NSI viruses cannot be readily explained by the T cell conjugate capture pathway presented by Pope *et al.* (*2*, *4*). Also, differences in receptor binding affinity need not be the only explanation for differences in cell tropism. Different cell types express different amounts of DNA binding proteins that interact with different enhancers and promoters in the long terminal repeat region of HIV-1. We recently showed that both the regulatory gene sequences and the transactivator genes of HIV-1s are conserved within HIV-1 B subtypes, but that there is significant independent evolution in the case of HIV-1 Es and Cs (*9*).

Recent results from South Africa also suggest that the long-term presence of HIV-1 B did not result in a heterosexual epidemic, while the more recent introduction of HIV-1 C is causing a serious heterosexual epidemic (*10*). Others have recently found that HIV-1 As, associated with heterosexual epidemics in other regions of Africa, also grow better in LCs (*11*).

In summary, independent observations by several groups support our hypothesis that LC tropism may help explain the association between certain HIV subtypes and the major heterosexual epidemics. It is important to recognize that receptor-mediated productive infection of LCs and the ability of LCs to capture virus are mediated through separate pathways. We propose that the "productive pathway" is more relevant for heterosexual transmission.

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Color Vision, Genetics, and Computers?

In their thought-provoking report "Visual pigment gene structure and the severity of color vision deficits" (1 Nov. 1996, p. 801), J. Neitz *et al.* used genetics to study the relative preservation of "trichromatic" vision in deuteranomalous individuals—color-blind men who lack the gene for the middle-wavelength cone pigment.

Color vision in some deuteranomalous individuals can approach that of normal people. Neitz *et al.* found that the degree of preservation of trichromatic vision was inversely correlated with the similarity of the individual's long-wavelength pigments. In other words, the brains of deuteranomalous individuals with relatively preserved trichromatic vision presumably use the short-wavelength pigment and two slightly split long-wavelength pigments to produce color vision—a task that is accomplished in normal people with the use of short-, middle-, and long-wavelength pigments.

Software could be developed for computers (or other monitors) that would take advantage of the brain function of deuteranomalous individuals to give them a fuller range and vividness of color vision. Furthermore, even for non-color-blind individuals (especially women, who have twice as many pigment genes as do most men), one can imagine that genetic and psychophysical testing might be useful in conjunction with such software to individualize color monitor displays.

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LKLF and FasL Expression: Correction and Clarification

In the report "LKLF: A transcriptional regulator of single-positive T cell quiescence and

survival" (26 Sept., p. 1986) (*1*), we found increased Fas ligand (FasL) expression on lung Kruppel-like factor (LKLF)–deficient T cells. This result was based on flow cytometric data obtained with two different FasL antibodies, NOK-1 and K10. Only the NOK-1 data were shown in figure 4C of the report (p. 1988). Our subsequent experiments have demonstrated high-affinity nonspecific binding of the NOK-1 antibody to Fc receptors on the surface of T cells. Therefore, the data shown in figure 4C did not accurately reflect FasL expression on these cells. However, parallel experiments in which the K10 antibody (which specifically detects mouse FasL) was used (*2*) demonstrated significantly increased FasL on the surface of both splenic and lymph node single positive LKLF-deficient T cells (Fig. 1). These experiments validated our conclusion that increased FasL expression on LKLF-deficient single positive T cells correlates with increased apoptosis of these cells in vivo. We apologize for any confusion caused by the inclusion of the NOK-1 experiments in our report (*1*).

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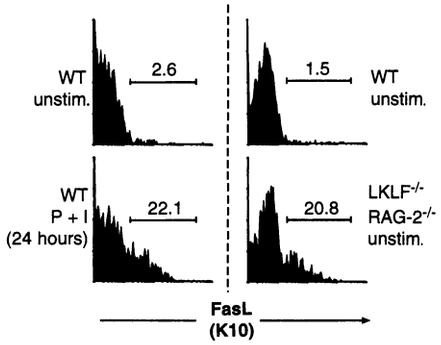

Fig. 1. Fas ligand expression on LKLF-deficient T cells. Unstimulated (unstim.), wild-type (WT), or LKLF-deficient (LKLF$^{-/-}$ RAG-2$^{-/-}$) lymphocytes isolated from lymph nodes were stained with monoclonal antibodies (mAbs) against CD4 and CD8 as well as with the K10 mAb against mouse Fas ligand (FasL). As a positive control, WT lymph node T cells were also stimulated for 24 hours with PMA (5 nanograms/milliliter) + ionomycin (0.25 micrograms/milliliter), (P + I). Profiles show FasL expression on CD8$^+$ cells; *y* axis is in arbitrary units. All experiments were performed in the presence of an Fc receptor blocking agent (Fc Block, PharMingen, San Diego, CA). Fraction (%) of FasL$^+$ cells is shown above each profile.

Corrections and Clarifications

In the News & Comment article "Environment institute lays plans for gene hunt" by Jocelyn Kaiser (24 Oct., p. 569), one version of paraoxonase should have been described as converting sarin "more slowly," not "more quickly," than a more common version of the enzyme.

The Research News article "Slicing an electron's charge into three," by David Ehrenstein (19 Sept., p. 1766) should have mentioned an earlier measurement of fractional charge by V. J. Goldman and B. Su [*Science* 267, 1010 (1995)].

Letters to the Editor

Letters may be submitted by e-mail (at science_letters@aaas.org), fax (202-789-4669), or regular mail (*Science*, 1200 New York Avenue, NW, Washington, DC 20005, USA). Letters are not routinely acknowledged. Full addresses, signatures, and daytime phone numbers should be included. Letters should be brief (300 words or less) and may be edited for reasons of clarity or space. They may appear in print and/or on the World Wide Web. Letter writers are not consulted before publication.