## **AIDS Biosafety**

At least two cases of accidental, laboratory-acquired human immunodeficiency virus (HIV) infections have been documented (1). Laboratory workers who routinely handle HIV and HIV-infected materials risk accidental infection by the virus that causes AIDS. Use of attenuated HIV strains for commercial production and routine laboratory procedures could prevent or minimize disease in the event of such laboratoryacquired infections. Recently, Kestler et al. have presented strong evidence for the importance of the nef gene for the development of AIDS (2). We propose the use of engineered strains of HIV that have deletions in the nef gene as a means for decreasing risk in laboratory workers.

HIV and simian immunodeficiency virus (SIV) nef can be deleted without abrogating the ability of virus to replicate in cell culture. HIVs and SIVs deleted in nef replicate in a variety of cultured cells with similar kinetics and to similar extents when compared to their nef-open counterparts. In the experiments of Kestler et al., rhesus monkeys infected with SIV with nef deleted did not develop AIDS-related complex (ARC) or AIDS, remained healthy, had extremely low virus loads, and maintained normal CD4 lymphocyte concentrations. Rhesus monkeys infected with a counterpart SIV having an open nef gene developed ARC and AIDS, and 50% have died to date. The extensive similarity between SIV and HIV suggests that results in the SIV system will likely be applicable to HIV in humans (3). Although it is not yet clear whether the presence of nef is an absolute requirement for the development of AIDS, the results indicate that HIV with nef deleted should, at the very least, be much less pathogenic than nondeleted counterparts.

The use of HIV with a deletion in nef will likely yield comparable results in a variety of HIV-based clinical and research tests. These include whole virus ELISA, Western immunoblotting, and virus neutralization tests. Commercial manufacturers and research laboratories should thus consider using cloned virus strains with a deletion in nef.

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## REFERENCES

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- 2. H. W. Kestler et al., Cell 65, 651 (1991).
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## **Monsanto Dioxin Studies**

Leslie Roberts' article about dioxins (News & Comment, 8 Feb., p. 626) included a box (p. 626) with the title "Monsanto studies under fire." Since reading this commentary on studies performed a decade ago by epidemiologists and physicians from Monsanto and the University of Cincinnati-and about alleged fraudulent manipulation of data and criminal investigation-I have been doing the proverbial "slow burn."

Even though Science provided a Monsanto spokesman with the opportunity to firmly deny all of these allegations and so reported in the article, I was disappointed that a publication dedicated to science would stoop to dignifying hearsay and allegations stemming from lawsuits. Such hype may raise unwarranted suspicion in the minds of some readers, despite our denials and despite the fact that Monsanto invited an audit by the Environmental Protection Agency and the National Institute of Occupational Safety and Health of the studies to resolve the matters.

Especially repugnant to me was a sentence in the last paragraph which read, "Everyone Science spoke with who is familiar with the Monsanto studies agrees that they are flawed, but probably not as the result of criminal intent." Who are these unidentified "experts" who, without a detailed review of the actual study, its analysis, and interpretations, can render judgment on flaws (let alone gratuitously comment on criminal intent)? Is the reader told that no experteither epidemiologist or trained public health physician-has ever come forward to actually criticize the adequacy of these studies? Or that a morbidity study at the same Monsanto plant-commissioned and paid for by the union representing workers at that plant-was conducted by Irving Selikoff, Marion Moses, and their colleagues (1), who arrived at essentially the same conclusion as first reached by the University of Cincinnati: that chloracne was the only evident finding and "that it is unlikely that permanent, severe, and debilitating toxicological sequelae are inevitable after exposure to TCDD sufficient to produce chloracne"?

In my opinion, a publication with the stature of Science has better things to do than involve itself in the sort of mess described in Roberts' article.

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## **Dendrimer Research**

Joe Alper's Research News article, "Rising chemical 'stars' could play many roles" (29 Mar., p. 1562) was appreciated. However, several inaccuracies need to be addressed.

First, I am conducting Starburst dendrimers research at the Michigan Molecular Institute (MMI), not the Midland Molecular Institute. Second, MMI is a member of the Michigan Polymer Consortium. It is in this organization that MMI, Michigan State University, and Michigan Technological University (not "Institute") are affiliated, together with Central Michigan University, Eastern Michigan University, and Wayne State University. Third, I have never alleged that Starburst dendrimers are micelles. I have called them "unimolecular assemblages reminiscent of spheroidal micelles" (1) and "unimolecular mimics of micelles" (2), but never micelles.

Finally, I did not leave Dow Chemical with an adversarial opinion of their support for Starburst dendrimer research. I would like to emphasize that the enlightened Dow management approach resulted in the Starburst dendrimer technology package being transferred to MMI and my being allowed to pursue this research at MMI.

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