Letters

Monitoring the U.S. AIDS Epidemic

The report by E. O. Laumann *et al.* entitled "Monitoring the AIDS epidemic in the United States: A network approach" (9 June, p. 1186) is a creative attempt to estimate the relative prevalence of acquired immunodeficiency syndrome (AIDS) in various groups and geographic locations. The authors suggest that national AIDS surveillance data underestimate the prevalence of AIDS in the white population relative to that in minority populations and underestimate the prevalence of AIDS in the Midwest relative to that in the East. These conclusions are suspect because the methodology employed invokes several questionable assumptions, the term "AIDS" may be subject to broad interpretation, the sample size is small, and the results contradict other independent efforts to evaluate AIDS and human immunodeficiency virus (HIV) surveillance efforts.

First, the implication by Laumann *et al.* that the results on homicide support their methodology may be questioned; an "underestimate" for homicide victims among minorities and a higher estimate for persons from the Midwest were found similar to those differences in estimates found for persons with AIDS. Second, the authors' results depend on the key assumption that the structure of personal networks for persons with AIDS are not systematically different from those for the entire population, but they do not evaluate this assumption. The social networks of white homosexual men may well differ in size, social composition, geographic mobility, and other important characteristics from the networks of minority intravenous drug users with AIDS and those of homicide victims.

More important, the assumption that a person with AIDS could be assigned to the geographic location of the respondent is not a reasonable one, since a large proportion of early reported cases of AIDS, particularly in homosexual men, were reported among men who had migrated to New York and California from other states. In a case-control study conducted in 1981, 23% of homosexual men with AIDS residing in New York, Los Angeles, San Francisco, and Atlanta had been born in the Midwest; 26% had lived in their cities of residence less than 5 years, and another 23% had lived there 5 to 9 years (*2*). Also, the first cases in many southern and midwestern states were among such men who had returned home after diagnosis in a coastal city (*2*).

The design of the survey of Laumann *et al.* assumes equal representation of all persons at risk for AIDS. However, populations with a high incidence of AIDS other than homosexual men (for example, intravenous drug users, who may be homeless and of lower socioeconomic status, especially minorities) would be less likely to be "captured" using the household-based design of the General Social Survey. The effect of this bias would be to underestimate AIDS cases among minority populations and women, which appears to have occurred in this study. While the authors state that they found the same gender imbalance as the surveillance data of the Centers for Disease Control, they in fact estimated only 4% of persons with AIDS were women as compared to 8% in CDC surveillance reports.

Also, respondents may differ in what they consider to be "AIDS." Homicide is clear-cut; what is meant by "AIDS," however, may be subject to wide interpretation. Is the person with hemophilia who carries the "AIDS" virus considered by his neighbor to have AIDS? Does the homosexual man living down the street who appears to be losing weight have AIDS? Furthermore, the authors do not provide data on how many respondents knowing persons with AIDS were incidental acquaintances as opposed to persons with more personal ties.

In discussing the national surveillance system for AIDS, Laumann *et al.* state that many private physicians may be reluctant to report their patients with AIDS to the health department. Most state health departments, however, work directly with hospitals to identify AIDS cases; and most persons with AIDS have become sufficiently ill in the course of the disease to require hospitalization. Thus, these independent reporting networks have decreased the impact of individual physicians who do not report cases of AIDS.

Certainly, AIDS cases are underreported; additionally, reported AIDS cases do not represent the full spectrum of illness associated with HIV infection (*3–6*). However, underestimates are probably greater for women and minorities than for white homosexual men, on the basis of unpublished and recently published data (*4, 5*). Stoneburner *et al.* have documented an increasing mortality in intravenous drug users in New York City, which may represent a spectrum of serious HIV-related diseases that have not been identified through AIDS surveillance and has probably resulted in an underestimation of the impact of HIV infection on intravenous drug users, blacks, and Hispanics (*4*). In addition, HIV seroprevalence surveys conducted in various populations, including military recruit applicants, childbearing women, blood donors, homosexual men, and intravenous drug users, have demonstrated a considerably lower prevalence of HIV infection in the Midwest, supporting the relative distribution of AIDS cases found through surveillance reports (*6*). For example, the percent of childbearing women positive for HIV in Michigan and Illinois are 0.06% and 0.09%, respectively, compared with 0.66% and 0.49% for New York and New Jersey, respectively (*2*).

An alternative interpretation of the results would be that the higher "proportion" of whites from the Midwest observed in this study may result from incomplete ascertainment by the survey of minority cases (including women) due to inadequate sampling of intravenous drug users, the homeless, and those with less geographic mobility. Regardless of interpretation, the survey sample size is small and no confidence limits are provided, making it unclear whether observed differences are statistically significant.

HIV infection and AIDS remain a national and worldwide problem, affecting every community and most individuals either directly or indirectly. We need creative approaches to monitoring the HIV epidemic; a study such as this one may be useful to measure attitudes and behaviors, but the methodology is not adequate to replace or to validate more directly measured surveys of HIV morbidity or mortality.

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Response: Berkelman *et al.* make several important points. The most important issue is the concern that "populations with a high incidence of AIDS . . . would be less likely to be 'captured' using the household-based design." The network sampling procedure assumes that members of populations with a high incidence of AIDS have family, friends, and acquaintances who will fall into the household sample and thus will be reported. Suppose there are in fact two distinct populations, one of household members with one distribution of AIDS cases and the other of

nonhousehold members, perhaps drug users, with a high prevalence of AIDS and a distinct distribution of AIDS cases. If the two populations are wholly isolated from each other, with no one in the first group knowing anyone in the second group, our procedure will miss all those AIDS cases in the second population and thus misspecify the overall distribution of AIDS cases. But if, instead, the two populations fully comingle, our procedure can accurately capture the overall distribution of AIDS cases.

The truth surely lies somewhere between these two extremes. For the case in hand we believe, for two reasons, that we have probably captured most but not all of the distribution of AIDS cases among those nonhousehold populations. First, there is no evidence that the social networks of persons with AIDS are so unlike persons without AIDS that they will differentially appear in the network reports from a general population sample. While persons in the crisis stages of drug use or a fatal disease may not be able to sustain extensive social ties, this does not mean that in relatively recent periods they were atypical members of society in terms of their social bonds to family, friends, neighbors, or workplace acquaintances. The advantage of the network approach is that it does not depend on the immediate accessibility of an individual to a scientific observer for his or her behavior to be reported. Second, the General Social Survey (GSS) is estimated to include 95% of the population resident in the United States in its targeted universe.

Berkelman *et al.* report that there is substantial evidence of undercounting of AIDS and HIV-related morbidity among intravenous drug users (IVDUs), minority women, and children. Indeed the recent report of the Government Accounting Office (*1*) suggests more generally that the undercount of AIDS cases may approach 40%. This is an estimate based on those populations in urban centers where there are the most cases and the greatest effort devoted to their ascertainment. Given the size of the undercount in those places where there are many AIDS patients and a sensitized reporting system, surely one can expect that many cases will slip through the net in those places outside the epicenters of the epidemic. It is this imperfection of the decentralized monitoring system which may be the source of the undercounting of cases which the network procedure finds in the Midwest.

The other comments by Berkelman *et al.* surely have merit, but their import rests on the interpretation of the glass being half empty or half full. We see the similarity of official statistics and our estimates on homicide as reassuring, but as Berkelman *et al.* rightly say, they are not exactly identical. That can raise doubt, as it does for them, just as the overall similarity raises confidence, as it does for us.

In short, we would not argue, and indeed do not suggest, that our procedure should replace the CDC surveillance system; but we do suggest that it—like the important evidence cited by Berkelman *et al.* about incidence of HIV infection in blood samples—offers a means for validating or raising concerns about the details of the CDC statistics on this very important topic. One way to do this would be to collect similar data using large samples with more attention given to issues of network size and composition. We are attempting to do this by including network questions in the GSS for 1989 to 1992.

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NRDC on Alar

Since its release in February, the report of the Natural Resources Defense Council (NRDC) *Intolerable Risk: Pesticides in our Children's Food* (*1*) has generated substantial discussion, including two articles by Leslie Roberts (News & Comment, 10 Mar., p. 1280; 17 Mar., p. 1430) and an editorial by Daniel E. Koshland, Jr. (7 Apr., p. 9), in *Science*. The report estimated the potential health risks to children ages 1 to 5 from dietary exposures to 23 pesticides resulting from consumption of 27 fruits and vegetables. We would like to respond to the following specific questions that have been raised by the Environmental Protection Agency (EPA) (*2*), by Koshland, and by others and to present new information.

1) *Was the study based on a worst-case estimate of exposure*? Because of the limited data available, the NRDC report actually underestimated total pesticide exposure. NRDC estimated exposures to only 7% (23/300+) of the pesticides currently registered for use on food, including 12% (8/66) of the food use pesticides known or suspected to be carcinogenic (*3*). In addition, NRDC used *average* residue values, derived from government monitoring programs, in its exposure assessments. Tolerances, or legal limits, were not used because pesticide residues in food are generally considerably lower than the tolerance levels.

Because the report identified a high cancer risk resulting from exposure to the daminozide breakdown product UDMH, critics have questioned the exposure estimates used in the calculations. Average daminozide and UDMH residues were derived from a 1985–1986 market basket survey (*4*). Daminozide levels in apples averaged 1 part per million or 1/20 of the existing EPA tolerance for daminozide, while UDMH levels averaged 2 to 23 parts per billion (*5*). NRDC did not factor into its exposure estimates metabolic conversion of daminozide into UDMH, which EPA now estimates to be 1% (*6*). Had a 1% metabolic conversion been included, NRDC's exposure estimates for UDMH would have been increased by 36%.

Consumption data used in the report were derived from the 1985–1986 nationwide survey of daily food intakes of 489 children ages 1 to 5 (*7*). Exposure estimates for UDMH were calculated on the basis of an average consumption of approximately 3 ounces of apple products daily (*8*).

EPA's current estimate for UDMH exposure, based on a larger 1977–1978 U.S. Department of Agriculture dietary survey, differs only slightly from NRDC's. For commodities covered by the NRDC report, EPA currently estimates that the average daily exposure to UDMH for children ages 1 to 6 is 0.066 μg/kg/day (*9*) or 80% of the NRDC estimate for children ages 1 to 5 (*10*). EPA's current estimate of children's (1 to 6) total exposure to UDMH (*9*) is approximately twice NRDC's.

The NRDC's risk assessment has been challenged as overestimating risk because of the belief that daminozide use has decreased since 1986 and that currently "only 5% of apples are treated with Alar." However, 1988–1989 federal, state, and independent surveys found that 22% to 55% of apples tested had been treated with daminozide (E. Groth, III, Letters, 19 May, p. 755), indicating that the 5% figure may significantly underestimate the amount of daminozide used during the last growing season. Although EPA estimated earlier this year that 5% of the apples were treated with daminozide, the agency has recently revised that estimate upward to 5 to 15% (*6*).

2) *Was a valid cancer potency estimate used by the NRDC study for UDMH?* NRDC used a UDMH carcinogenic potency factor (q_1*) calculated by EPA in 1984 and listed in 1987 by the Office of Pesticide Programs (OPP) and the Carcinogen Assessment Group (CAG) (*11*). At the time that NRDC conducted its risk assessment, this was the only available estimate of carcinogenic potency for UDMH.

NRDC has been criticized (*2*) for using this q_1* because the Science Advisory Panel (SAP), an advisory panel to EPA's Office of Pesticide Programs, gave the opinion in 1985 that the existing bioassays on daminozide and UDMH were not adequate as the basis for quantitative risk assessment (*12*). The SAP opinion, however, was not consistent with other scientific analyses by EPA and other expert groups. EPA's CAG concluded that the existing evidence was more than adequate to classify UDMH as a "probable human carcinogen" (*13*) and was sufficient to serve as the basis for calculating a carcinogenic potency factor for this compound (*14*). Two independent EPA audits of the study that served as the basis for the CAG potency estimate agreed that, despite limitations, the bioassay clearly demonstrated that administration of UDMH led to a significantly increased incidence of multiple types of tumors at multiple sites in both sexes of test animals (*15*).

Similarly, 1 month after the SAP review, EPA's Environmental Criteria and Assessment Office concluded that the existing UDMH studies "provide sufficient quantitative evidence that 1,1-dimethylhydrazine represents a potential carcinogen" and that criticisms raised do not "constitute a basis for altering the fundamental conclusions of EPA's risk assessment for UDMH" (*16*). In addition to the EPA reviews, both the International Agency for Research on Cancer (IARC) and the National Toxicological Program (NTP) concluded that there was sufficient evidence of carcinogenicity (*17*).

Just before the release of the NRDC study, EPA published an updated assessment of the carcinogenic risk resulting from daminozide use based on interim (12-month) results of a new UDMH bioassay being conducted by Uniroyal. Two revised cancer potency factors were calculated that were lower than the previous agency q_1* used in the NRDC computations by factors of 10 (based on hemangiosarcomas) and 4 (based on benign lung tumors) (*18*).

Cancer is a disease with a long latency, and interim results may lead to underestimates of potency. EPA attempted to allow for latency by multiplying the cumulative incidence at 1 year by a factor of 8 to obtain an estimate of the cumulative 2-year "lifetime" incidence (*6*). This factor of 8 appears to be inappropriately small, however. The age-specific incidence of hemangiosarcomas in the Uniroyal bioassay (male and female UDMH exposed groups combined) is approximately proportional to the fourth power of time from first exposure (*19*). If this proportionality is maintained in the second year, the cumulative 2-year incidence will be about 30 times the number of cancers present at the end of 1 year (the sum of effects of a t^4 incidence function is proportional to t^5, so doubling the duration of the experiment would increase the cumulative incidence by 2^5, rather than 2^3, as proposed by EPA). Using the multiplier of t^5 would bring the interim q_1* based on hemangiosarcomas in the current experiment to about 4 mg/kg/day^{-1}, four times higher than the revised EPA q_1* discussed above and approximately half the q_1* used in the NRDC computations.

EPA anticipates that the final q_1*s based on the completed Uniroyal bioassay may be considerably higher than the q_1*s based on the interim data (*20*). On the basis of consideration of subsequent findings at lower doses, the agency predicts that hemangiosarcomas, which are currently significantly in-