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Information for Contributors appears on pages 93–94 of the 5 January 1996 issue. Editorial correspondence, including requests for permission to reprint and reprint orders, should be sent to 1333 H Street, NW, Washington, DC 20005.

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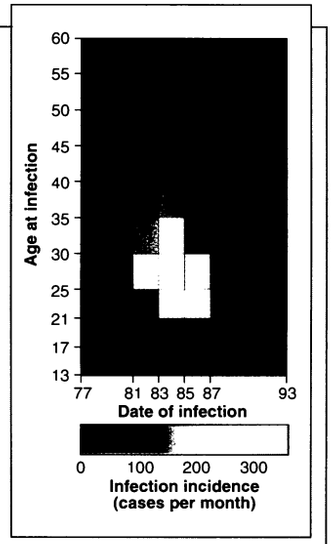
LETTERS

Political points

Sharply different approaches to U.S. science policy and funding are highlighted: Is the White House emphasizing "politics over science"? Three researchers discuss how looking at patients by ethnic group can sometimes be helpful (if one is learning about AIDS awareness and prevention) or harmful (if one mistakenly infers causation from the categorization): Should such comparisons be made? [At right, estimated number of AIDS infections per month among white males in the United States, 1977–1993 (P. S. Rosenberg *et al.*, Reports, 24 Nov., p. 1372)]. The validity of a global warming trend is further debated: How much of the related climate research has been peer-reviewed? Another writer alleges that NASA is "continually reducing support" for technical infrastructure at U.S. universities: Will this lead to the disappearance of "hands-on training" for young scientists and engineers?



Rohrabacher re Gore

I read with interest the coverage of "Heavy hitter," U.S. Vice President Al Gore's speech to the recent AAAS annual meeting (News & Comment, 16 Feb., p. 903). On reflection, I must admit that Al Gore was right about one thing: There are indeed major differences between the White House and the Republican-led Congress on science and technology issues.

While Gore may maintain that weakening our patent system to the lower standards of Japan helps American inventors, more than 200 members of the House of Representatives who have co-sponsored the Patent Term Restoration Act do not.

While Gore may maintain that it is more important to pay Fortune 100 corporations to develop product-related technology than it is to fund basic science, Republicans in Congress do not.

While Gore may maintain that it is more important to subsidize foreign-owned corporations in selling solar technology to the Third World, we in Congress would rather allocate every single solar energy research and development dollar to real research.

And while Gore may choose to ravage NASA's space science budget to fund the engineering-driven Mission to Planet Earth and to push corporate welfare "conservation programs" over scientific research at the Department of Energy, those choices clearly belie his exhortation to support "knowledge . . . for knowledge's own sake."

In the context of balancing the federal budget, Republicans in Congress have largely protected basic, fundamental research as a wellspring of progress and freedom. It has been Gore's White House that has emphasized politics over science, to the detriment of both.

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AIDS and Ethnicity

Philip S. Rosenberg (Reports, 24 Nov., p. 1372) finds human immunodeficiency virus (HIV) more prevalent in younger and nonwhite groups. His figures indicate that AIDS is largely a disease of poverty, not of age or ethnicity.

U.S. black women are 15 times, and Hispanic women 7 times, more likely to be HIV-infected than are white non-Hispanic women. Black males are 4.7 times and Hispanic men are 3 times more likely to be infected than are white males. This HIV pattern corresponds to the rates of blacks (33%), Hispanics (29%), and whites (9%) living on incomes below poverty guidelines in 1992. Within each race, younger people (15 to 24 years old) are twice as likely to be impoverished (21%) than are older people (25 to 59 years old) (11%) (*1*).

When the surplus or deficit of HIV acquisition (assuming it is 10 years earlier than age at AIDS diagnosis) for each age and ethnic group is standardized according to each group's surplus or deficit of poverty, the discrepancy between young and old age groups disappears for both sexes (*2*). The racial discrepancy for men also disappears. An HIV surplus among black and Hispanic women compared with white women remains. This argues that poverty places women at a higher net risk, perhaps rendering them vulnerable at young ages to sexual violence, prostitution, and sexual contact with older men. The HIV surplus among males is not explained by poverty.

There is virtually no morbidity from AIDS among white low-risk youth, while in lower income, primarily nonwhite, high-risk youth, morbidity ranges up to 17% (*3*). These factors indicate that critical issues in AIDS prevention are not demographic or behavioral, but relate to reversing the United States' consignment of a uniquely high and rising proportion of its youth and nonwhites to poverty.

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References

1. *Poverty in the United States—1992* (Bureau of the Census, Washington, DC, 1993), pp. 60–185 and table 17.
2. M. Males, data not shown.
3. P. Sweeney *et al.*, *Arch. Pediatr. Adolesc. Med.* **149**, 521 (1995).

I do not believe it is appropriate to present data on HIV infection rates or AIDS incidence according to ethnic groupings unless we can show that such disease incidence rates are causally related to ethnicity, for example, in the way that sickle cell anemia is related. To the best of my knowledge, we cannot at this time assert such causality.

Rosenberg suggests that as many as 1 in 50 black males in the United States may be infected with HIV. Does this figure apply to both middle-class professional black males and inner city socio-economically deprived black males? Probably not, in which case we should correlate HIV infection rates with a putatively causal attribute—socio-economic status—rather than ethnicity. A similar argument can be asserted with respect to incidence data reported by race for phenomena such as infant mortality (*1*) or homicide. Are we ready to say that infant mortality rates are *causally* associated with race simply because a full explanation of the difference in race-specific rates may continue to elude us (*2*)?

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1. Centers for Disease Control and Prevention, *Morbid. Mortal. Wkly. Rep.* **44**, 922 (1995).
2. ———, *ibid.* **43**, 288 (1994).

Response: McMillan and Males are concerned about the possible misuse and misinterpretation of HIV prevalence estimates for groups defined by race and ethnicity. I agree with both correspondents: Race and ethnicity are not causally associated with increased risk of infection, but are simply markers for social factors such as low socio-economic status (SES) that are the root causes of the high prevalence rates seen in minorities.

Unfortunately, it is not possible to derive SES- or income-specific estimates from national AIDS surveillance because such data are not collected. Even if it were, inadequacies in available measures of SES and other social factors might preclude a complete explanation of the large excess of AIDS cases among minorities. For example, in a nationally representative health survey,

African Americans had significantly higher rates of infection with hepatitis B virus than did whites even after SES was controlled for with the best available measures (*1*). In Philadelphia, income level explained some, but not all, of the favorable reductions in the rate of increase of AIDS in white residents compared with that in nonwhite residents (*2*). Thus, even when good data on SES exist, SES as a risk factor may be an imperfect surrogate in a complex web of underlying causes that include cultural variations in behavior that are distinct from SES.

In practice, people want to know how HIV affects persons of their own race and ethnicity and respond positively to public health messages that are culturally appropriate and targeted to individuals with whom they can identify. It is difficult enough for social scientists to adequately measure SES; imagine the general public trying to decode an AIDS awareness poster featuring a person of low SES.

Improving the health of racial and ethnic minorities in the United States is a priority of the Public Health Service (*3*). Data on race and ethnicity can help target interventions and resources to those communities that are most in need (*4*). Minority communities are particularly hard hit by the AIDS epidemic, regardless of the underlying causes. It is hoped that greater recognition of this problem will help prevent further spread of HIV.

Philip S. Rosenberg
National Cancer Institute, 6130 Executive Boulevard, Rockville, MD 20852, USA

References and Notes

1. G. M. McQuillan *et al.*, *Am. J. Med.* **87** (Suppl. 3A), 55 (1989).
2. D. Fife and C. Mode, *J. AIDS* **5**, 1105 (1992).
3. Public Health Service, *Healthy People 2000: National Health Promotion and Disease Prevention Objectives* (Publ. [PHS] 91-50212, U.S. Department of Health and Human Services, Washington, DC, 1991).
4. Centers for Disease Control and Prevention, *Morbid. Mortal. Wkly. Rep.* **42** (No. RR-10) (1993).
5. I thank J. Karon, G. McQuillan, S. Melnick, and R. Hahn for helpful discussions.

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Climate Change Report

S. Fred Singer (Letters, 2 Feb., p. 581) refers to the "summary for policy-makers" (SPM) ratified by the Intergovernmental Panel on Climate Change (IPCC) at its meeting in Madrid in November 1995. We are responding as a subset of the lead authors of the full IPCC report and participants at the Madrid meeting.

Singer writes that there has been no global warming trend "in the last 50 years." This is not supported by the data; the trend from 1946 to 1995 is 0.3°C. As shown in chapter 8 of the full report (figure 8.4), there is no inconsistency between the observed temperature record and model simulations.

Singer notes differences between satellite estimates of lower tropospheric temperatures (derived from microwave sounding unit data) and surface temperatures from 1979 to the present. As explained in chapters 3 and 8 of the full report, there are good physical reasons to expect differences between these two climate indicators. They differ, but they are not inconsistent.

Singer writes that climate models lack validation. Chapter 5 in the 1995 report deals with the validation issue. Current general circulation models (GCMs) have well-known and well-documented weaknesses, but they still perform remarkably well in simulating important features of current climate conditions.

In noting that global mean temperature projections made in the latest assessment are lower than those given in 1992, Singer says that this fact and the reasons for it are not mentioned in the SPM. This is incor-