one transferases, and so forth, which may make low doses of mutagens protective in some circumstances. Even radiation—the classical DNA-damaging agent and carcinogen—may be protective in small doses against DNA damage at higher doses, as shown by recent work in human cells (*19*). Also, recent radiation experiments in mice show a dose threshold for the latency of tumor appearance (*20*). Thus, low doses of carcinogens appear to be both much more common and less hazardous than is generally thought. These scientific questions about mechanisms of carcinogenesis and the preventable causes of human cancer, in any case, are being resolved by the scientific community as quickly as resources allow.

Regulation of low-dose exposures to chemicals based on animal cancer tests may not result in significant reduction of human cancer, because we are exposed to millions of different chemicals—almost all natural—and it is not feasible to test all of them. Most exposures, with the exception of some occupational, medical, or natural pesticide exposures, are at low doses. The selection of chemicals to test, a critical issue, should reflect human exposures that are at high doses relative to their toxic doses and the numbers of people exposed. Epidemiology has been reasonably successful in identifying risk factors for human cancer, such as smoking, hormonal and dietary imbalances, asbestos, and several occupational chemicals; the data suggest that pesticide residues are unlikely to be a significant risk factor (*6, 21*). Epidemiology, with molecular approaches, is becoming more sophisticated and will continue to be our main tool in analyzing causes of cancer. In order to minimize cancer and the other degenerative diseases of aging [which are associated with our constantly increasing life expectancy (*6, 7*)], we need to obtain the knowledge that will come from further basic scientific research.

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Erratum: Table 3 of the report "Seroprevalence and epidemiological correlates of HTLV-I infection in U.S. blood donors" by Alan E. Williams *et al.* (29 Apr. 1988, p. 643) contained errors. The correct table is printed below.

Variable	Cases	Controls	Odds ratio	95% Confidence interval
History of IV drug use or sex with IV drug user	4/7†	0/35	20.0	NA§
Black race	6/7	4/35	14.0	(3.2–62.0)
History of more than two STDs	1/7‡	0/35	14.0	NA
HAV seropositive	5/7	3/35	8.9	(3.5–17.1)
Unmarried	6/7	17/35	7.5	(1.2–47.6)
No education past grade 12	4/7	5/35	6.0	(1.4–25.9)
History of transfusion	2/7	2/35	5.0	(0.9–29.2)
History of skin rash	2/7	3/35	4.5	(0.6–33.9)
One or more STDs	2/7	3/35	4.5	(0.6–33.9)
Sexual contact in Orient	1/7‡	2/35	NA	NA
HSV seropositive	6/7	26/35	2.3	(0.2–32.2)
Travel to HTLV-1 endemic areas	1/7	14/35	1.5	(0.1–48.0)
Family history of cancer	2/7	9/35	1.2	(0.2–8.5)
Exposure to swine	1/7‡	5/35	1.0	NA
Breastfed as infant*	4/5	13/17	0.3	(0.2–3.2)
CMV seropositive	3/7	18/35	0.7	(0.1–3.9)
Birth outside of the United States	0/7	0/35	0.0	0.0
Gay, lesbian, or bisexual	0/7	0/35	0.0	0.0
Exposure to cattle	0/7	2/35	0.0	0.0
Numbness or weakness, difficulty walking, or poor health	0/7	0/35	0.0	0.0
Neurologic disease in family	0/7	0/35	0.0	0.0

*Two seropositive cases and eight controls did not know whether they were breastfed as infants. Only controls for whom case data were available were used. †Significantly different from controls: $\chi^2 = 14.5$, $P < 0.001$. ‡Not significantly different from controls. §Not applicable.