

tor, radiation, is the only cause can yield meaningless results. A cautionary note on the last page of the article stated, "Other factors studied, such as birth cohort and pay code, showed much stronger relationships to mortality than radiation." The focus on the much weaker association with radiation was justified by the "public health impact of these radiation exposures and the industry that produces them."

The study of small health data sets must deal with an almost undefinable level of noise; controlling for superficial demographic factors is little more than an act of faith. The conclusions drawn are often an artifact of both method and predilection. The Oak Ridge National Laboratory leukemia mortality data may represent the impact of an unknown set of risk factors for the varied life histories of the 28 patients. But the article only seems to offer evidence of an answer in search of a question.

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1. Wing S, Shy CM, Wood JL, et al. Mortality among workers at Oak Ridge National Laboratory: evidence of radiation effects in follow-up through 1984. *JAMA*. 1991;265:1397-1402.
2. Ichimaru M, Ohkita T, Ichimaru T. Leukemia, multiple myeloma, and malignant lymphoma. In: *GANN Monograph on Cancer Research* 32. 1986:113-127.
3. Kato H. Cancer Mortality. In: *GANN Monograph on Cancer Research* 32. 1986:53-74.
4. United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR) 1988. *Sources, Effects and Risks of Ionizing Radiation*. New York, NY: United Nations; 1988.
5. National Academy of Science, National Research Council. *Health Effects of Exposure to Low Levels of Ionizing Radiation*. The Committee on the Biological Effects of Ionizing Radiation (BEIR V). Washington, DC: National Academy Press; 1990:242-248.
6. Surveillance Epidemiology and Results (SEER). *Incidence and Mortality Data: 1973-77*. National Cancer Institute (NCI) Monograph 57. Bethesda, Md: National Institutes of Health; 1981. NIH publication 82-2330.

To the Editor.—The recent article by Wing et al¹ concluded that there is an increased incidence of leukemia due to occupational radiation exposure at fairly low levels. I believe that there are several major reasons why this conclusion is incorrect and, in fact, unsupported by the data in the article.

First, there were only 28 cases of leukemia, and the distribution of cases may have been due in large measure to the problem of small numbers. Second, the conclusion is inconsistent with virtually all other data in the field, some of which involve many more cases. Leukemias have supposedly been produced at far lower exposure rates than in other exposed populations, and the latent periods here are inconsistent with those generally seen for leukemia (minimum, 2 years; peak, 5 years) and for solid tumors (10 to 40+ years).

This radiation exposure is comparable to background levels over a slightly longer period of time. If this conclusion

were correct, we should be seeing increased cancer rates in areas of the world with higher background radiation levels, such as Brazil, India, and China. Cancer rates in Colorado should be higher than those in New York. Instead, they are the same (Brazil, India) or lower (China, Colorado).

The most significant problem with this conclusion is that there is no dose-response effect. As shown in Table 5 of the article, all but two of the cases occurred at exposures of 39 mSv or less, and seven occurred in workers with zero exposure. Two cases were in workers with greater than 120 mSv cumulative exposure, and there were no cases in workers with cumulative exposures between 40 and 120 mSv. If there really is an increase in leukemia due to occupational radiation exposure, we should be seeing an increase in the leukemia rate with increasing exposure. This is clearly not the case. It is also interesting to note that there are fewer lung cancers in particular and fewer of all cancers than would be expected, but this finding is essentially ignored. Other potential causes, such as toxic chemicals, are briefly mentioned and dismissed. If there are increased rates of leukemia in workers at Oak Ridge National Laboratory, they are almost certainly not due to occupational radiation exposure.

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1. Wing S, Shy CM, Wood JL, et al. Mortality among workers at Oak Ridge National Laboratory: evidence of radiation effects in follow-up through 1984. *JAMA*. 1991;265:1397-1402.

To the Editor.—We read with interest the article by Wing et al,¹ citing an increase in cancer risk of 5% per 10 mSv of cumulative radiation exposure in workers at Oak Ridge National Laboratory. We are unable to translate this into practical terms, however. For example, how many extra deaths would this represent per 100 000 population exposed to this total dose (10 mSv) over their lifetimes? How can we relate this to other, self-inflicted risks; eg, diagnostic radiology,² smoking one cigarette per day, riding a bicycle, traveling in a jet plane, or visiting New York City? Can the authors express these risks as shortening of the average life span? In view of the benefits that nuclear workers may derive from their employment (better than normal health care, for example), what is the outcome of a risk-benefit analysis in these workers compared with similar workers in non-nuclear-related industries?

Since inhabitants of Colorado have similar, increased, cumulative, environmental exposures of 10 mSv every 10

years (due to increased terrestrial and cosmic radiation³), are there data to show that they, too, have the increased cancer risk detected in Wing's study? For example, if the increased risk is 5% per 10 mSv as the article suggests, then a lifetime in Colorado should be associated with a 30% higher cancer rate compared with areas of less intense background radiation. If such a geographic cancer risk cannot be detected, then unmeasured factors other than radiation dose must have caused the increased cancer risk observed in the Oak Ridge study.

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1. Wing S, Shy CM, Wood JL, et al. Mortality among workers at Oak Ridge National Laboratory: evidence of radiation effects in follow-up through 1984. *JAMA*. 1991;265:1397-1402.
2. Baker DG. Medical radiation exposure and genetic risks. *South Med J*. 1980;73:1247-1250.
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In Reply.—Our study showed that the previously reported elevation of the leukemia SMR at Oak Ridge National Laboratory¹ continued with longer follow-up, was confined to the time period after 1965 among workers hired before 1960, and was larger in a subgroup of workers monitored for internal radionuclide contamination than in the cohort as a whole. We nowhere concluded that the leukemia excess was due to occupational radiation; however, these results are not inconsistent with an occupational effect.

Relationships between mortality and radiation exposure were studied among the workers; the general population was not used as "unexposed" controls. Dose-response estimates for leukemia were highly unstable, reflecting in part the small number of leukemia deaths and the generally low occupational exposures. There were no leukemia deaths between 40 mSv and 120 mSv and 1.9 expected; all the observed and expected values in Table 5 are summarized by the dose coefficient of 9.14% increase in leukemia per 10 mSv (Table 4). As reported, when analyses were repeated excluding CLL, we found similar results. Brown's SMR calculation fails to account for the age and calendar time differences between death rates for CLL and other leukemias.

A practical interpretation of the estimate of 5% per 10-mSv increase in risk of cancer death can be given by considering a hypothetical worker with characteristics studied in our analysis: age 55 years, born between 1905 and 1914, paid monthly, and no longer working at the lab. The estimated risk of cancer death