Introduction

ATSDR produced a draft Public Health Assessment (May 24, 2002) on the impact of tritium released from LLNL in 1965 and 1970. The report includes discussions of environmental pathways, community health concerns, community exposures, and public health implications. ATSDR’s estimate of the public health impact of tritium released from LLNL is flawed by a biased review of the literature on biological effects of low-level radiation, misinterpretation of epidemiologic principles, omission of information from studies of humans exposed to tritium, and a lack of consideration of important determinants of susceptibility to radiation exposures.

The form of dose response relationships for biological effects of ionizing radiation

Although the ATSDR report states that ionizing radiation from tritium can have health impacts including cancer and genetic effects, it interprets animal and human studies of gamma radiation and x-rays as showing there is a threshold dose for health effects of tritium. This interpretation is at odds with biological theory and recent evaluations of evidence from in vitro, animal and human studies (1-3). The energy in a single beta particle produced by radioactive decay of tritium is sufficient to cause ionization in a cellular environment and thereby directly or indirectly cause damage to genetic material. This means that, on theoretical grounds, there is no threshold for biological damage. The capacity of low level radiation doses from tritium to produce health effects is dependent on the population dose and the susceptibility of the individuals exposed, which may be related to their developmental stage, the effectiveness of genetic repair mechanisms, other carcinogenic exposures, reproductive behavior, and other factors. The National Academy of Science’s BEIR V committee concluded that it is reasonable to assume there is no threshold dose below which health effects of ionizing radiation do not occur (2).

Reliance on animal studies

ATSDR relies on studies of laboratory animals to provide assurance that tritium exposures from LLNL releases did not produce health effects. ATSDR fails to note that laboratory animals have life spans that are shorter than the latency periods for many human cancers. There can be large
inter- and intra-species variation in susceptibility to carcinogens. Although animal studies are important, their relevance to effects of radiation on heterogeneous human populations in complex environments that include exposures to other initiating and promoting agents should not be over-stated.

Reliance on studies of the Japanese atomic bomb survivors

It is increasingly clear that there are fundamental flaws in studies of the survivors of the atomic attacks on Hiroshima and Nagasaki (4-11). High mortality in the aftermath of the bombings, selective survival of persons with low sensitivity to radiation effects, and uncertainties in estimating doses from the bombs, including a lack of data on doses from radioactive fallout, raise serious questions about the validity of risk estimates from this population. The ATSDR report relies on risk estimates from studies of the A-bomb survivors without acknowledging that there are several reasons why these studies should be expected to underestimate radiation health effects. Furthermore, the report misinterprets analyses of risk in arbitrary dose groups, fails to point out that standard dose response analyses which avoid arbitrary dose categorization suggest no threshold for radiation induced cancer (2), and fails to cite recent reports which support a linear no-threshold model for radiation health effects (12).

Studies of occupational exposure to radiation

Beginning during the Manhattan Project to develop atomic weapons, workers exposed to chronic, low level ionizing radiation have been monitored using dosimeters incorporated into security badges. Dosimeters have been used primarily to identify excessive exposures, however, beginning in the 1960s, badge readings were assembled for studies of radiation risks. Some early studies emphasized low death rates of nuclear workers compared to national statistics (13), however, low mortality among workers can be misleading because death rates are typically low among employees of large corporations, which conduct health screening and offer benefits of employment, compared to the general public, which includes people who are too sick to work. Other research has examined cancer death rates of workers according to the levels of radiation recorded on their badges. A growing number of studies that carefully evaluate radiation measurements and other worker characteristics have shown that cancer rates rise with increasing radiation exposure, even at dose levels permissible under current standards (10, 14-29), adding to other literature that suggests there is no threshold for carcinogenic effects of ionizing radiation.

Although most of the whole body radiation exposures of nuclear workers comes from external penetrating radiation, some workers have been exposed to tritium, which distributes throughout the body. Where nuclear facilities have monitored tritium exposures by urinalysis, tritium doses can be combined with external radiation doses to yield a total whole body dose. Several epidemiologic studies have demonstrated relationships between these combined whole body doses and mortality from leukemia, multiple myeloma, and other cancers (29, 30).

The availability of individual radiation measurements for thousands of workers whose cancer mortality has been monitored over decades is a unique epidemiologic resource that does not exist for any other occupational exposure (10). Although workers are generally healthy, they are more similar to the average members of the public than are subjects of some other types of radiation
epidemiology studies, such as patients under treatment or survivors of atomic attack. These studies are therefore especially important to consider in evaluating public concern about radiation releases. ATSDR does not discuss occupational radiation studies in its draft report.

**Studies of in utero exposures**

In the early 1950s, Alice M. Stewart began the Oxford Survey of Childhood Cancers. British children who died of cancer were compared to healthy controls in an attempt to identify exposures that might be causes of childhood cancer. In 1956, the first of many reports from the OSCC, which became the largest study of childhood cancer ever to be conducted, showed that the only factor that differed systematically between cancer cases and controls was a history of maternal pelvic x-ray during pregnancy (31). Because of the widespread assumption in the medical community that low level radiation posed no risk, in part due to the absence of an effect in the study of in utero exposed A-bomb survivors, the initial OSCC findings were treated with great skepticism. However, the medical x-ray link to childhood cancer held up as additional cases and controls were added to the OSCC (32, 33), and subsequent studies in other countries confirmed the British findings (34). Exposures of pregnant women to diagnostic x-rays have been greatly curtailed even though the doses from diagnostic x-rays are well below the levels that ATSDR cites as posing no risk.

Although the issue of in utero radiation exposure is raised in the ATSDR report, there is misinterpretation of the literature, perhaps due to reliance on a health physics text rather than a review of the literature on the carcinogenic effects of obstetric x-rays. Extensive evidence now shows that childhood cancers in general (not just leukemia, as cited in the ATSDR report) are related to fetal exposure to diagnostic x-rays. Mean whole body fetal dose estimates in these studies are approximately 0.6 rad, well below the 10-50 rad figure cited by ATSDR. (35, 36)

**Susceptible sub-populations**

A growing literature suggests that older adults are more sensitive than younger adults to carcinogenic effects of ionizing radiation. This may be due to the accumulation of genetic “hits” from previous exposures, functional declines DNA repair processes, and lowered immune function. The potential for tritium releases to disproportionately impact older adults, as well as populations with other inherited or acquired forms of heightened susceptibility, is an important public health issue that is not addressed in the ATSDR report.

**Epidemiologic concepts**

Basic epidemiologic concepts are poorly applied in the ATSDR report. First, as noted above in discussion of the A-bomb survivors, the ability of epidemiologic studies to detect low-level effects is often compromised by problems of poor measurement, confounding, and inadequate sample size (10). No evidence of an effect is not the same as evidence of no effect. Many studies cannot detect effects because of inadequate dose information, reliance on cause of death information rather than incidence, selective exposure of individuals with lower susceptibility, and lack of long-term follow-up of large populations. The studies cited in the report section entitled “Health outcome data evaluation” (pp26-28) suffer from these problems and should not be
interpreted as providing evidence of no effect. Furthermore, ATSDR’s assumption that “tritium doses from the accidental LLNL tritium releases are below levels expected to produce adverse health outcomes” (p 26) suggests that any positive findings from occupational and community studies would not be interpreted by ATSDR as evidence of health effects due to their a priori assumption that doses and dose-response relationships are too small for effects to occur.

Another basic epidemiologic concept is that small relative risks can have a large impact if many people are exposed. Small doses to large numbers of people further away from LLNL could produce a larger health impact than higher doses to small numbers of people near the site.

Conclusion

ATSDR has conducted a biased review of evidence for health effects of ionizing radiation in general and tritium in particular. Important scientific and public health considerations are omitted. The report should be revised to reflect the substantial uncertainties in the scientific basis for understanding the full range of biological effects of low level ionizing radiation and the existence of a growing body of evidence that radiation health effects have been underestimated in the past.

References: