topics are chosen, the cosponsors of the act in Congress have maintained that funds allocated for basic or applied research or development should not be shifted out of those categories. Neither the Senate nor House version requires agencies to reallocate funds traditionally set aside for university and medical school basic research or funds going to current demonstration projects. The NSF's estimates of the fiscal year 1982 science budget show that each agency could fund even a purely applied program if it so chose.

The act is sensitive to the importance of ensuring quality R & D. Both the Senate and House versions emphasize the importance of peer review, and, unlike most other federal programs, require applicants to successfully complete a feasibility study before they can compete for major funding. In addition, the Office of Science and Technology Policy is granted a major role in implementing the act.

As scientists and as citizens, we should remember what the growth of small firms in California, Massachusetts, and elsewhere has meant for job opportunities; scientific, technological, and economic development; and funding university-based research institutes and academic departments. Throughout the debate over this legislation, no one has denied that research consistently indicates that small R & D firms are (i) the primary source of major innovations in our economy; (ii) have one of the fastest U.S. rates of growth in net employment, sales, exports, productivity, revenue, and tax dollars (and we should also remember that federal support for science requires tax dollars); (iii) are among the most cost-efficient performers of R & D; (iv) find government awards a major stimulus for their formation and growth; and (v) rapidly diversify into private sector work after receiving government work. The Small Business Innovation Research Act of 1981 will simultaneously serve the interests of industrial and academic scientists and our fellow citizens.

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Radiation Effects

In "Genetic effects of the atomic bombs: a reappraisal" (11 Sept., p. 1220) W. J. Schull *et al.* provide interesting data and analyses. Their major conclusions could be presented more fairly, however, if they reported standard errors along with statistical estimates.

For example, from data in their table 5 they estimate the increase per rem in sex chromosome aneuploids at 4.65 per million and zygotic doubling dose at 504 rems, but they provide no explicit assessment of the variability of these estimates. According to their table, 12 of 5058 children of distally exposed parents and 16 of 5762 children of proximally exposed parents exhibited sex chromosome aneuploids. They estimate the average gonadal dose at 87 rems and compute the increase per rem as

$$\frac{1}{87}\left(\frac{16}{5762} - \frac{12}{5058}\right) = 4.65 \times 10^{-6}$$

and the zygotic doubling dose as

$$\frac{12/5058}{4.65 \times 10^{-6}} \doteq 504 \text{ rems}$$

The standard error of the difference in two binomials produces a standard error for the increase per rem of

$$\frac{1}{87}\left[\frac{(16)\,(5746)}{(5762)^3} + \frac{(12)\,(5046)}{(5058)^3}\right]^{1/2} = 11.2 \times 10^{-6}$$

Applying the empirical logit (*1*) produces a one-sided 95 percent lower confidence bound for the zygotic doubling dose of 75.8. The upper bound is infinity, since the confidence interval for the increase per rem includes zero.

These computations incorporate only binomial sampling error and not the uncertainty associated with the average dose of 87 or with nonsampling errors. They show that, although the estimates may be the best possible, they should not be memorialized.

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References

1. D. R. Cox, *The Analysis of Binary Data* (Methuen, London, 1970), chap. 3.

Schull *et al.* report that they find little in the way of phenotypic evidence of heritable effects in the progeny of persons exposed to irradiation from the atomic bombs dropped on Hiroshima and Nagasaki, this in spite of the somatic mutational events (chromosome anomalies and neoplasms) observed earlier in the same population of exposed individuals. Implied by this disparity is a potential differential sensitivity of somatic and germ cells to radiation-induced mutagenicity.

Evidence being gathered in studies of chemical mutagenesis points in a similar direction. In a recent examination of available information about the cytogenetic effects of chemicals in somatic and germ cells in vivo I found 76 chemicals to have been tested in both cell types. Of the 45 that elicited positive responses in somatic cells, 19 were negative in germ cell assessments. More important, no compound was found to produce a positive effect in germ cells but not in somatic cells. In other words, the germ cell models detected only about 60 percent of mutagenically active substances.

This, as well as the conclusions reached by Schull *et al.* about human effects of radiation exposure, implies a relative insensitivity of either the germ cells themselves or of the assays used for detecting germ cell mutations. The inability to detect significant effects in the radiation study by means of several highly sensitive indicators of genetic damage suggests the former to be the case, perhaps because of DNA packaging, repair mechanisms, meiotic "sieve" and so on. It is not clear, however, whether the germ cells of animals are similarly unsusceptible or whether the assays used to detect such effects are relatively insensitive.

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Inasmuch as we regarded our estimate of the doubling dose as preliminary, subject to many sources of error in addition to the traditional sampling one (particularly with respect to the subject of dose), we have been reluctant to place errors on our estimates. We hope to do so later. We certainly share with Louis the concern that these values "not be memorialized," for data continue to accumulate and doubtless will do so for some time; also, new technological developments make alternative methods of estimation possible.

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