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ENVIRONMENT AND HEALTH MANAGEMENT DIVISION

January 21, 1982

Mr. Ralph G. Page, Chief
Uranium Fuel Licensing Branch,
Division of Fuel Cycle and Materials Safety,
Office of Nuclear Materials Safety and Safeguards,
U. S. Nuclear Regulatory Commission
Washington, D.C. 20555

Dear Mr. Page:

Attached are Kerr McGee comments on the "Disposal or Onsite Storage of Thorium or Uranium Wastes from Past Operations" published in the Federal Register October 23, 1981. As you can see from the attachment, we have many reservations as to the technical and legal positions stated in this Branch Technical Position.

We appreciate the extension of time for submitting these comments granted by your office.

Sincerely,

W. J. Shelley, Vice-President
Nuclear Licensing & Reg. Environmental &
Health Management Div.

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BEFORE THE NUCLEAR REGULATORY COMMISSION

Comments on Branch Technical Position
on Disposal or Onsite Storage of Thorium or
Uranium Wastes from Past Operations
46 Federal Register 52601 (Oct. 23, 1981)

Kerr-McGee Corporation
Kerr-McGee Nuclear Corporation
Kerr-McGee Chemical Corporation

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These comments, submitted on behalf of Kerr-McGee Corporation, Kerr-McGee Nuclear Corporation, and Kerr-McGee Chemical Corporation (Kerr-McGee), are in response to the Branch Technical Position on Disposal or Onsite Storage of Thorium or Uranium Wastes from Past Operations. The Branch Technical Position is described in a notice published at 46 Fed. Reg. 52061 (Oct. 23, 1981). The Branch Technical Position establishes five options for disposal of thorium and uranium wastes.

The first four options are in the form of numerical limits on concentrations of radionuclides which may be disposed under certain conditions. The limits as applied to uranium wastes are purportedly based on an EPA 5 pCi/gm radium-226 standard. The limits as applied to thorium wastes are represented to be based on certain EPA exposure standards for the lung and bone and on an external gamma standard. Under the fifth option, NRC proposes a position that uranium and thorium wastes may not be disposed except in accordance with options one through four or at a licensed disposal facility. Option five envisions indefinite interim storage of uranium and thorium

wastes not qualified for disposal under options one through four if a licensed repository is unavailable.

The Branch Technical Position ostensibly is directed at sites formerly used for processing thorium and uranium and, in many cases, the quantities are large but the activity concentrations low. Since some of these sites are still licened, while others are not, different legal requirements apply. Specifically, currently licensed former mill sites and their mill tailings are subject to Title II of the Uranium Mill Tailings Radiation Control Act of 1978, as amended (UMTRC Act) and, accordingly, are not subject to the Board Technical Positions. These comments do not address whether the NRC has, or could assert, jurisdiction over former processing sites which are not currently licensed.

The Branch Technical Position should be withdrawn. All the standards purportedly embodied in Options One through Four are beyond the agency's authority, premature, and unsupported. Option Five is flatly contrary to the Uranium Mill Tailings Radiation Control Act, 10 C.F.R. § 20.302, and the public interest.

Interest of Commenter

Kerr-McGee Nuclear owns and operates a uranium mill located at Ambrosia Lake, New Mexico. The Company also owns and operates a uranium conversion facility at Sequoyah, Oklahoma. NRC may seek to apply the Branch Technical Position to uranium wastes at these facilities. Kerr-McGee Chemical owns

a thorium mill located at West Chicago, Illinois. NRC staff in fact already has sought to apply a portion of the Branch Technical Position to thorium wastes allegedly associated with that facility. The Branch Technical Position may also impact other facilities owned or planned by Kerr-McGee.

Summary

NRC lacks authority under the Atomic Energy Act, as amended by the Uranium Mill Tailings Radiation Control Act, to establish standards such as those embodied in Options One through Four in the absence of valid standards issued by EPA. EPA has not yet promulgated any applicable standards on which NRC may base generic standards such as those contained in the Branch Technical Position. NRC's Options One through Four are thus beyond the agency's authority and are premature.

Options One through Four are also unsupported. Although the underlying documents are unclear, the four options are evidently based upon an assumption known as the linear non-threshold model, which purports to project the effects of low-level radiation. The linear non-threshold model is contrary to much of the evidence and is unduly stringent. The various "standards" on which Options One through Four purport to be based are inapposite to the situations to which these four options are applicable. Moreover, NRC's calculations which purport to link the underlying standards to the various concentration limits are based upon a variety of unrealistic

assumptions which render the whole exercise invalid. Correction of these various deficiencies will lead to a substantial increase in the maximum concentrations allowed under each disposal option.

NRC's Option Five envisions interim storage of licensed concentrations of uranium and thorium on-site pending the availability of a licensed disposal site. This option is contrary to the Uranium Mill Tailings Radiation Control Act which envisions on-site disposal. The option also contravenes 10 C.F.R. § 20.302 and the agency's past practice pursuant to it. Furthermore, temporary or interim storage is contrary to the public policy favoring prompt decommissioning of facilities and permanent disposal of associated waste. As a general policy, the NRC should treat onsite disposal as the preferred option for uranium or thorium mill tailings. On-site disposal may be so designed as to satisfy all reasonable health or safety concerns. It is fully justified by cost-benefit analyses.

Kerr-McGee is particularly troubled with the suggestion, embodied in a memorandum from Mr. Dircks to the Commission, that Option Five may apply to the Company's West Chicago Facility. That facility is a thorium mill and is clearly regulated under the UMTRC Act. The UMTRC Act provides for permanent on-site disposal of thorium mill wastes. Upon conclusion of the proposed on-site stabilization plan, the disposal site, in accordance with the Act, will be transferred to the

Department of Energy (DOE) or to the State government. The Facility does not, and cannot, fall under Option Five.

The Branch Technical Position falls within the definition of "rule" for purposes of the Administrative Procedure Act (APA) and the Atomic Energy Act. It must be proposed and ultimately issued as a rule should the Commission decide to go forward with it. When promulgated in final form, it will be subject to judicial review. The technical justification supplied for the Branch Technical Position contains serious flaws and gaps. It is insufficient to support the proposed rule under applicable standards of judicial review.

Consistent with these comments, NRC should:

(1) eliminate sites subject to UMTRC Act from its proposed Branch Technical Position

(2) defer republication of a revised Branch Technical Position applicable to non-UMTRC Act sites until EPA has issued governing standards

(3) repropose the revised Branch Technical Position only following publication of final EPA standards and in a manner consistent with the APA; and

(4) provide sufficient data to disclose the NRC's calculations and rationale.

COMMENTS

I. NRC Lacks Authority to Issue Standards

The Commission lacks authority to establish the minimum standards and requirements specified in the Branch

Technical Position. Reorganization Plan No. 3 of 1970[1] transferred to EPA all authority under the Atomic Energy Act to issue generally applicable standards for the protection of public health and the environment. NRC's authority was confined to enforcing and to implementing standards adopted by EPA.[2] NRC thus lacks authority to issue standards such as those on which the Branch Technical Position is grounded. The Branch Technical Position is thus invalid as beyond the agency's authority.

NRC's lack of authority is especially clear with respect to "byproduct material" as defined by Section 11(e).2 of the Act, 42 U.S.C. § 2014(e)(2). This type of byproduct material includes uranium or thorium waste resulting from "extraction or concentration of uranium or thorium from any ore processed primarily for its source material content." It thus specifically covers uranium and thorium wastes generated at Kerr-McGee's Ambrosia Lake and West Chicago facilities,

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[1] EPA was established by Reorganization Plan No. 3 of 1970, 35 Fed. Reg. 15623, 84 Stat. 2086, 42 U.S.C.A. § 4321 note. Section 2(a)(6) of the Plan transferred to EPA all authority to set generally applicable standards for the protection of public health and the environment under the Atomic Energy Act. The uranium milling industry is challenging the validity of the pertinent provisions of Reorganization Plan No. 3 in a separate proceeding. See Kerr-McGee Nuclear Corp., et al. v. EPA, No. 81-1630 (10th Cir.). This portion of Kerr-McGee's comments assume arguendo the validity of the Reorganization Plan.

[2] This arrangement has been confirmed in a Memorandum of Understanding between the AEC (NRC's predecessor) and EPA. 38 Fed. Reg. 24936 (September 14, 1973).

and, we believe, the Sequoyah facility as well. With respect to these wastes, Section 206(b)(1) of the Uranium Mill Tailings Radiation Control Act, 42 U.S.C. 2022(b)(1), specifically provides that the Environmental Protection Agency (EPA), not NRC, is to exercise the standard-setting function. That provision specifically requires EPA to issue standards for

> "hazards associated with the processing and with the possession, transfer, and disposal of [the uranium and thorium wastes in question]. . . ."

The legislative history confirms that Congress intended EPA, not NRC, to set the pertinent standards. Indeed, NRC specifically requested Congress to preserve the split in authority established by Reorganization Plan No. 3 in enacting the UMTRC Act. NRC Chairman Hendrie actually testified in opposition to legislation assigning the "standard-setting task to the NRC with EPA in the consulting role," characterizing this as an "undesirable reversal of the present regulatory roles of the two agencies."[3] Congress in fact evinced no interest in reversing the roles of the two agencies. Section 206 of the UMTRC Act was adopted to clarify that intent.[4]

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[3] Hearings on H.R. 13382, 12938, 12535 and 13049, before the Subcomm. on Energy and Environment of the House Comm. on Interior and Insular Affairs, 95th Cong., 2d Sess. 78 (1978); Hearings on S. 3008, 3078 and 3257 before the Subcomm. on Energy Production and Supply of the Senate Comm. on Energy and Natural Resources, 95th Cong., 2d Sess. 48 (1978) (same).

[4] See, e.g., Hearings on H.R. 11698, 12229 12938, 12535, 13049 and 13650 before the Subcomm. on Energy and Power of the House Comm. on Interstate and Foreign Commerce, 95th Cong., 2d Sess. 343, 366, 296 (1978); H.R. Rep. No. 1480, pt. 1, 95th Cong., 2d Sess. 25 (letter, Administrator Costle to Committee on Interior and Insular Affairs).

The legislative history also specifically demonstrates that Congress did not intend NRC to interlope in EPA's standard-setting authority. Senator McClure admonished against "duplicative regulatory procedures. We do not want to see NRC out there doing exactly what EPA is required to do and is doing."[5] Other legislators similarly indicated that industry was not to be whipsawed by first one set and then another set of regulations. Congressman Dingell pointedly charged EPA to "come up with one set of standards . . . so that the rules won't change on operators."[6]

This construction of the UMTRC Act has recently been confirmed by Congress. On July 24, 1981, the House of Representatives adopted an amendment to the Energy and Water Appropriations Bill (H.R. 4144) precluding NRC from expending funds to implement its new Uranium Mill Licensing requirements, promulgated at 45 Fed. Reg. 65521 (Oct 3, 1980). The House amendment specifically provides that

> "no funds appropriated to the Nuclear Regulatory Commission in this Act may be used to implement or enforce any portion of the Uranium Mill Licensing Requirements published as final rules at 45 Federal Register 65521 to 65538 on October 3, 1980."

Congressman Stratton, the sponsor of this language, explained that the amendment was offered because the NRC regulations in

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[5] 124 Cong. Rec. S. 15324 (daily ed. Sept. 18, 1978) (emphasis added).

[6] See Hearings on H.R. 11698, 12229, 12938, 1235, 13049 and 13650, supra, at 393.

question were unlawfully issued in advance of EPA standards. The Uranium Mill Tailings Radiation Control Act ("UMTRC Act"), the Congressman explained,

> "required the Nuclear Regulatory Commission to promulgate rules which would implement and enforce the EPA final standards. There are no EPA final standards; and there should not be NRC rules." 127 Cong. Rec. H 4873 (daily ed. July 24, 1981).

The Senate Appropriations Committee adopted similar language in the Senate mark-up of the relevant bill.[7] The

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[7] The language adopted by the Senate Appropriations Committee provided that:

> "no funds appropriated to the Nuclear Regulatory Commission in this Act may be used to implement or enforce any portion of the Uranium Mill Licensing Requirements published as final rules at 45 Federal Register 65521 to 65538 on October 3, 1980, or to require any State to adopt such requirements in order for the State to continue to exercise authority under State law for uranium mill and mill tailings licensing, or to reinstate NRC regulatory authority for uranium mill and mill tailings licensing in any State that has acted to exercise such authority under State law; provided, however, that the Commission may use such funds to continue to regulate byproduct material, as defined in section 11 e.(2) of the Atomic Energy Act of 1954, as amended, in the manner and to the extent permitted prior to October 3, 1980."

The additional language was added to deal with concerns raised by NRC with respect to the effect of the suspension and to clarify the intent of Congress that Agreement States not be compelled to adopt NRC's regulations in contravention of local conditions. See 127 Cong. Rec. S 12982 (daily ed. Nov. 5, 1981).

Committee's Report makes clear that the Committee agreed that NRC acted in violation of section 275 in adopting its regulations in advance of EPA standards. "The Committee agrees with the House that the Commission should not have adopted these technical requirements for uranium mill tailings prior to EPA's promulgation of final environmental standards." S. Rep. No. 97-256, at 154 (Oct. 22, 1981).

The full Senate adopted the suspension language on November 5. Senator Simpson, Chairman of the Nuclear Regulatory Subcommittee, cogently explained that NRC acted unlawfully and improperly in adopting its regulations prior to action by EPA. "The issuance of these regulations," the Senator said,

> "violates the careful division of regulatory responsibilities for mill tailings in the [A]ct and creates the potential for future shifts and conflicts in regulatory requirements affecting present and new uranium milling operations." 127 Cong. Rec. S 12982 (daily ed. Nov. 5, 1981).

Similarly, Senator Domenici, a sponsor of the UMTRC Act which added section 275 to the Atomic Energy Act, explained that NRC's action in advance of EPA standards violated the Act. Senator Domenici incisively declared that:

> "Under section 275 of the Atomic Energy Act, EPA must issue the standards, not NRC. EPA has not yet issued standards. NRC erred in issuing standards and more detailed requirements in advance of EPA's standards. NRC's action is not only contrary to the statute but also subjects [A]greement States and regulated industry to shifting and conflicting regulatory requirements." 127 Cong. Rec. S 12984 (daily ed. Nov. 5, 1981).

The Conference Committee adopted the Senate language. The Energy and Water Development Appropriation Act, which contains the pertinent suspension amendment, was signed into law by the President on December 4.

It is well established that "[s]ubsequent legislation declaring the intent of an earlier statute is entitled to great weight in statutory construction." Red Lion Broadcasting Co. v. FCC, 395 U.S. 367, 380-81 (1969). See also Seatrain Shipbuilding Corp. v. Shell Oil Co., 444 U.S. 646, (1980) (subsequent legislation entitled to "significant weight"); NLRB v. Bell Aerospace Co., 416 U.S. 267, 275 (1974) (same). The suspension language adopted by both Houses and signed into law by the President (and the legislative history supporting it) concisely and undeniably declares Congress' earlier intent and compels NRC obedience to it. This action by Congress fully confirms that NRC may not issue standards, as it purports to do in the Branch Technical Position, in lieu of EPA under section 275 of the Act.[8] The standards proposed in the Branch Technical Position are accordingly invalid.

II. NRC, Through its Purported Reliance on Proposed EPA Standards, Has Overestimated the Risk from Exposure to Radiation from Natural Uranium and Natural Thorium

The supporting documents for the Branch Technical Position do not reveal the basis for NRC's apparent belief

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[8] Pertinent legislative history is collected in Attachment A.

that natural uranium and natural thorium pose a significant health hazard on disposal. Judging from the documents, it appears that NRC utterly failed to analyze the health risks against which its Branch Technical Position is presumably directed. The only health risk analysis associated with the Branch Technical Position is that which was undertaken by EPA in proposing the various standards on which NRC purportedly relies. The various EPA analyses, insofar as they exist at all, are not part of the record for the Branch Technical Position. The Position is thus manifestly deficient in terms of supporting rationale. Even if health risks were analyzed, however, the Position would be deficient for lack of consideration of other relevant factors, such as costs of compliance, effect on energy supplies, and so forth. Given the lack of any express risk analysis on the part of NRC, Kerr-McGee's remarks on that subject must necessarily be directed at the generic approach to risk assessment ordinarily employed by NRC and EPA in standard setting. As shown below, the approach employed by EPA in issuing its proposed standards (on which NRC purportedly relies) and the approach used elsewhere by NRC in setting standards are fatally deficient in several respects.

A. NRC's Analysis of Risk with Respect to Uranium Wastes Is Overly Conservative and Otherwise Defective

There is no data establishing that exposure to low-level radiation is harmful. In the absence of data, NRC

and EPA assume that some health effects may occur on the basis of the "linear non-threshold model." This model was in fact employed by EPA in devising the proposed standards on which NRC purports to rely. The linear non-threshold model has been aptly criticized by numerous authorities. For example, the model is based on the assumption that tumor induction is a straightforward "one-hit" process in which any unit of exposure will result in some carcinogenic activity. But "the bulk of the evidence" argues against the hypothesis that neoplastic transformation is a linear function of dose.[9] Additionally, many prominent health physicists and other experts believe that the body is capable of repairing damage caused by low-level radiation.[10] Moreover, other authorities have pointedly observed that the linear non-threshold model is contrary to recent evidence that "ecologically realistic, low-level radiation is biologically stimulatory, and presumably beneficial."[11] This effect, known as "radiation hormesis," is a

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[9] Anderson, Pathology 347 (7th ed. 1977).

[10] See, e.g., Robbins & Cotran, Pathologic Basis of Disease 552 (2d ed. 1979); Transcript of New Mexico EIB hearing at 495-96 (testimony of Dr. Evans) reprinted in Uranium Ore Residues: Potential Hazards and Disposition, Hearings before the Procurement and Military Nuclear Systems Subcomm of the House Armed Services Comm., 97th Cong., 1st Sess (June, 1981); In the Matter of Duke Power Co. (Perkins Nuclear Station, Units 1, 2 and 3), 8 NRC 87, [1975-78 Transfer Binder] Nuclear Reg. Rep. (CCH) ¶ 30,312 at p. 28,669 (1978).

[11] See, e.g., Hickey, Letter: Cancer and Concensus, Chemical & Engineering News 65 & 75 (Sept. 14, 1981) (Attachment B).

restatement of the Arndt-Schulz law that small doses of substances are stimulatory (i.e., beneficial) even if large doses are harmful. As one biochemist recently explained,

> "[r]adiation hormesis denies the validity of straight line extrapolation from known harmful doses to zero. The argument that low doses give harmful effects in proportion to dosage is invalid."[12]

Finally, pertinent epidemiological studies are consistent with the proposition that low-level radiation is not harmful. Indeed, those studies indicate that people in high background radiation areas enjoy better health than people in low background radiation areas.[13] For example, people living on the

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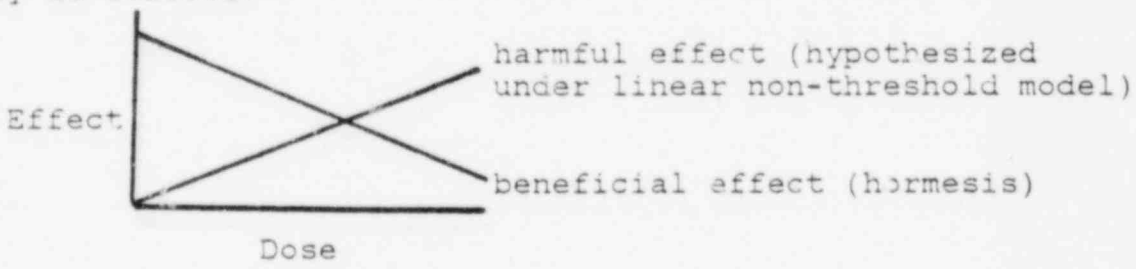
[12] T. Luckey, Radiation Hormesis 162 (CRC 1980). See also Luckey, Letter: Hormesis, Nuclear News 48 (Dec. 1981) (Attachment C); Hickey, Letter: Hormesis, Nuclear News, at p. 54 (Dec. 1981) (Attachment D). In any event, even if carcinogenesis is linearly related to dose, there may also be a second beneficial effect at low doses, and these benefits may outweigh the risks. Such a relationship would appear graphically as follows:



See Sagan, Some Thoughts on Dose-Response, Hormesis and All That, Nuclear News, at p. 82 (Oct. 1981).

[13] See, e.g., Hickey, et al, Low Level Ionizing Radiation and Human Mortality: Multi-Regional Epidemiological Studies, 40 Health Physics 625 (1981) (Attachment E); Frigerio, et al, The Argonne Radiological Impact Program (AGIP)-1. Carcinogenic Hazard from Low-level, Low-rate Radiation (Argonne Nat'l Lab. Report ANL/ES-26, Part 1) (1973) (Attachment F); High Background Radiation Research Group (China), Health Survey

(footnote cont'd)

Colorado plateau in the United States have significantly lower death rates from cancer and chronic disease that people in the eastern coastal plains where natural ionizing radiation is much lower. Another striking example involves the people of Kerala, India.

> "The people of Kerala are reported to have the highest literary rate and the best health status in India; yet their expenditure on health care is not appreciably above average, and they have the lowest food intake and the least adequate diets of people of all states of India. A partial explanation for this paradox is that radiation from the unusually high radium and thorium in coastal and river rocks exposes many residents to 10 times more terrestrial radiation than the U.S. average.[14]

NRC admits that the linear nonthreshold model may be erroneous.[15] In fact, as one statistician has charged, the purported general regulatory "acceptance" of the model is "in large part [a] misuse of statistics."[16] Exclusive or even primary reliance on the linear non-threshold model is an over-simplication and may result in an over-investment in

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(footnote cont'd)

in High Background Radiation Areas in China, 209 Science 877 (1980) (Attachment G); Gopal-Ayengar, et al, Evaluation of Long-Term Effects of High Background Radiation on Selected Population Groups on the Kerala Coast in Peaceful Uses of Atomic Energy, Vol. II, Proc. 45th Int. Conf. Peaceful Uses of Atomic Energy, pp. 31-51 (1971) (Attachment H); Cullen, et al., Dosimetric and Cytogenetic Studies in Brazilian Areas of High Natural Activity, 19 Health Physics 165 (1970) (Attachment I).

[14] Luckey, Letter: Hormesis, Nuclear News 52 & 54 (Dec. 1981) (emphasis added).

[15] GEIS U-4.

[16] Hickey, supra note 15.

controls to avert hypothetical risks.[17] This translates into an unwarranted burden on atomic energy activities and a waste of society's resources.

In view of these considerations, EPA and NRC should re-evaluate and modulate their reliance on the linear non-threshold model for purposes of assessing the risk from exposure to low-level radiation. In particular, the agencies

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[17] In the words of Professor David Okrent of UCLA, "Resources for the reduction of risks to the public are not infinite. At some point, a greater improvement in health and safety is to be expected from a more stable and viable economy than from a reduction in pollution or the rate of accidents." Okrent, Comment on Societal Risk, 208 Science 372, 374 (1980). NRC should also recognize that stringent controls aimed at reducing exposure to radiation from atomic energy activities can easily have a reverse affect. For example, new and costly NRC controls will result in increased energy costs to consumers. This will induce increased reliance on insulation. This means fewer air changes in residences and businesses. This in turn leads to increased exposure to naturally occuring indoor radon. EPA estimates (under the linear non-threshold model) that the risk posed by indoor radon is about 1 in 300 lifetime (approximately 1 in 21,000 per year). EPA, Draft EIS for Remedial Action Standards for Inactive Uranium Processing Sites at pp. 4-20 & 21. This means that, under government estimates, about 10,000 people perish per year from natural indoor radon exposure in this country alone. This is far more than the maximum number of deaths (6) projected by NRC from radon emanating from three times the amount of uranium mill tailings now in existence. Increased insulation, prompted by rising energy costs, already is leading to about 1,000 additional hypothetical deaths from indoor radon per year and will eventually lead to about 10,000 additional hypothetical deaths. Cohen, Health Effects of Radon from Insulation of Buildings, 39 Health Physics 937, 940 (1980). Less costly uranium fuel cycle regulatory requirements can make a major contribution in decreasing hypothetical radiation fatalities in this country because less costly regulation will result in less expensive electricity. This in turn will lead to less reliance on conservation measures which are 100 to 1000 times more hazardous, radiologically speaking, than radon releases from the uranium fuel cycle.

should give greater weight to the possibility that ionizing radiation, particularly from naturally occurring concentrations of radionuclides such as are involved here, is not harmful and may in fact be beneficial.

B. NRC's Analysis of Risk with Respect to Thorium Wastes Is Deficient.

NRC's risk analysis for low-level gamma radiation, the primary hazard the agency identifies with respect to thorium, is even more suspect than NRC's risk estimates for exposure to uranium and its decay products. NRC's estimates (through EPA) are again traceable to exclusive reliance on a linear non-threshold model. All the criticisms already made with respect to the linear non-threshold model apply with equal or greater force. Indeed, the objections are even more telling, because the epidemiological studies in India, Brazil and China on high-background radiation were in thorium-rich areas. None of these epidemiological studies detected adverse effects. They are instead consistent with the view that higher exposure to natural radiation may be beneficial.[18] Moreover, various advisory groups have acknowledged the lower likelihood of gamma radiation to result in harm. The ICRP has indicated that gamma and beta radiation (termed "low-LET") is an order of magnitude less effective in doing biological damage than high-LET radiation.[19] The BEIR-III Committee have

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[18] See text and notes at pages 14-15 supra.

[19] International Commission on Radiological Protection, Report of Committee II on Permissible Dose for Internal Radiation (1959).

also admitted that the linear nonthreshold model "probably leads to overestimates of the risk of most cancer . . . for exposure to low-LET radiation at low doses."[20] The BEIR-III report in fact recommends the use of a range of estimates for the risk of such radiation, and notes that the risk may be zero.[21] NRC and EPA have improperly relied exclusively on a linear nonthreshold model for projecting the risk from gamma radiation from thorium wastes.

III. NRC Has Employed Additional Unduly Conservative Assumptions in Calculating the Limits Specified in Options One through Four

The staff's de facto total reliance on the linear non-threshold model results in overly stringent limits with respect to each of the first four disposal options described in the Branch Technical Position. The limits are also unduly conservative due to methodological deficiencies employed in their calculation. Some of these deficiencies are explored below.

Option 1. Option one permits unrestricted disposal of natural uranium and natural thorium, with daughters in equilibrium, so long as the concentrations are less than 10

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[20] See National Academy of Science, The Effects on Population of Exposure to Low Levels of Ionizing Radiation 4 (1980) (BIER III Report).

[21] BEIR-III Report at p. 226.

pCi/gm.[22] This section of Kerr-McGee's comments will first analyze deficiencies in the calculations with respect to uranium wastes. It will then discuss deficiencies with respect to thorium wastes.

The Position makes only a single argument for the 10 pCi/gm figure as applied to natural uranium and its daughters.[23] Specifically, it indicates that the 10 pCi/gm figure for uranium is based on the 5 pCi/gm radium-226 limit contained in EPA's proposed inactive uranium processing site regulations. Even assuming the EPA proposal were final, it is designed for, and should be applicable only to, inactive uranium processing sites. NRC's extension of the standard is totally unwarranted. It overlooks the myriad of other factors, including costs and impact on the policies and purposes of the Atomic Energy Act, which must be considered before a standard may be extended into a new area.

The NRC standard is also unreasonably low for a variety of reasons. EPA suggests that the 5 pCi/gm limit will result in no more than 80 mrem exposure per year.[24] This amount is considerably less than natural background radiation,

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[22] See 46 Fed. Reg. 52062 (Oct. 23, 1981). Kerr-McGee understands that NRC intends to permit disposal if the concentrations average less than 10 pCi/gm.

[23] See 46 Fed. Reg. 52062.

[24] EPA, Draft EIS for Remedial Action Standards for Uranium Processing Sites at 4-33.

particularly in western mining areas.[25] It is unduly stringent in terms of the exposure limit which it prescribes. However, even assuming that 80 mrem exposure is a sound standard, it clearly warrants much higher radium-226 concentrations than 5 pCi/gm. EPA's calculations linking 5 pCi/gm ra-226 to 80 mrem are based on numerous improper assumptions which overstate exposure. For example, EPA made a 100% occupancy assumption. This is totally unrealistic. A person working outdoors could be expected to occupy a site no more than 20-30% of the time -- not 100%. EPA also did not take into account shielding for a person residing at the site. A concrete house slab would reduce the dose rate by at least a factor of two to three. The 5 pCi/gm radium-226 standard will result in a dose of about 5 mrem assuming realistic outdoor occupancy, or 15 mrem per year based on continuous outdoor occupancy. The radium-226 standard may accordingly be raised by at least a factor of 5 and still not result in exposures exceeding 80 mrem per year.

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[25] Average natural background radiation (cosmic, terrestrial and internal body radiation) varies from State to State between approximately 90 mrem and 180 mrem exposure per year. Variation is caused primarily by different altitudes above sea level and by natural rock formations. Living near a granite rock formation, for example, may result in 25 to 100 mrem additional exposure per year. See Low-Level Ionizing Radiation, Hearings before the Subcomms. on Energy Research and Production and Natural Resources and Environment of the H. Comm. on Science and Technology, 96th Cong., 1st Sess. at 8-9 (1979); Biological Effects of Radiation, 15 Encyclopedia Britannica 382 (1979). Epidemiological studies indicate that background radiation has not resulted in any statistically detectible harm and may in fact be significantly beneficial.

DOE has specifically criticized the 5 pCi/gm radium-226 standard in comments filed with EPA.[26] The Department, among other things, has aptly explained that the standard is in fact so low that it will be difficult, if not impossible, to achieve as an engineering matter.

The Position purports to apply two different standards to exposure to radiation from natural thorium and its daughters. The first standard provides that no person may receive a 50 year dose commitment in excess of 1 mrad/yr to the lung or 3 mrad/yr to the bone from inhalation or ingestion under any foreseeable use from such disposal. The Position suggests that these limits were proposed by EPA as dose limits resulting from unplanned contamination.[27] The two limits are deficient and inappropriate for several reasons.

First, they were proposed only in the form of guidance, not as binding standards. Moreover, they related only to transuranics, not naturally occurring radionuclides such as thorium and its decay products. Significantly, the proposed transuranic guidance embodying the standards was rejected by President Carter in October 1979 because of numerous objections lodged by other agencies. It is totally inappropriate to generalize standards designed for a particular class of radionuclides to another class of radionuclides in a totally

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[26] See, Letter, Mr. Greenleigh (DOE) to Ms. Selander (EPA) (July 15, 1981) (Exhibit J).

[27] Id., citing 42 Fed. Reg. 60956-59.

different context. The factors pertinent to standard-setting -- costs, population at risk, chemistry, and so forth -- may be, and in this case are, significantly different.

Second, the proposed guidance on transuranics was designed for the protection of workers who were assumed to be chronically exposed to transuranics. The guidance limits are accordingly in the form of dose commitments. The dose commitment approach is far too stringent as applied to the general population. "Radiation dose commitment" means, for a radionuclide with a very long half life such as thorium-232, that an actual physical dose of less than 0.02 mrad per year is "counted" for regulatory purposes as a dose of 1 mrad. As a result, a standard specifying a 1 mrad dose commitment is extremely conservative, and rather deceptive, for the doses which it permits are in fact far less.

The dose-commitment approach is reasonable only if one assumes that all exposed people will be exposed every year for a lifetime to the same level of dose. This obviously is not the case, particularly in our highly mobile society. Moreover, even if the dose is received every year, people over age 25 will never receive part of the dose which the "dose-commitment" assumes. Additionally, assuming a 10 year latency period for most common cancer, people over 13 years old will never suffer some of the risks which the dose-commitment approach assigns to them.

Third, the EPA limits were derived under a wholly different set of assumptions than those employed by NRC. For example, EPA used a quality factor of 10 for alpha radiation, NRC employs a quality factor of 20. EPA employed a seventy year dose commitment; NRC employs a fifty. Given the significant differences in assumptions underlying the calculations, the whole exercise must be redone.

NRC next argues that no person will receive an external exposure rate in excess of 10 μR per hour above background under these standards. Staff asserts that this is compatible with EPA's proposed clean-up standards for inactive uranium processing sites. It is not. NRC employs the 10 μR/hr standard as an unshielded outdoor rate. However, the EPA proposed standard is an indoor exposure rate of 20 μR/hr. The comparable outdoor value is 40 μR/hr (assuming a 0.5 shielding factor for calculation of the indoor dose).

The underlying documentation for the Branch Technical Position indicates several additional deficiencies in the calculations purportedly supporting the 10 pCi/gm natural thorium limit specified in Option One. For example, NRC appears to assume that 100% of the Bi-212 in the thorium decay chain decays to Tl-208. In actuality, decay of Bi-212 is branched with only 35.93% decaying to Tl-208 and the remainder to Po-212. This results in an important overestimate of external exposure since Tl-208 is the most important gamma emitter contributing to external dose in the thorium chain.

To take another example, NRC appears to assume that all radiation is from the top 1 cm of soil. Calculations should instead be based on a volume radiation source.[28]

Kerr-McGee has an additional objection to Option One. The option was evidently developed and proposed by NRC in the Branch Technical Position in order to govern certain disposals. However, Kerr-McGee now understands that NRC intends to apply it as a clean-up and decontamination standard. The 10 pCi/gm standard is totally inconsistent with NRC's previous approach on cleam up and decontamination. NRC previously employed the exposure standards for unrestricted areas contained in 10 CFR Part 20 for these purposes. These exposure standards would permit concentrations at least an order of magnitude higher than those set forth in Option One even where people were actually being exposed. The 10 pCi/gm standard is also inconsistent with NRC's various decontamination guidelines. For example, the November 1976 Guidelines for Decontamination of Facilities and Equipment Prior to Release for Unrestricted Use or Termination of Licenses for Byproduct, Source, or Special Nuclear Material indicates that 1,000 dpm/100cm^2 of thorium-natural is "acceptible" upon decontamination. This is equivalent to over 400 pCi/gm of thorium-natural. NRC's radical departure from its prior standards and requirements

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is totally unexplained. NRC has utterly failed to establish that a disposal standard may so readily be transfigured into a standard governing clean-up and decontamination. Moreover, many of the factors relevant to standard-setting such as costs, are entirely different, and much greater, in the clean-up or decontamination context than in the disposal context. This suggests that clean-up standards should be different, and higher. Finally, clean-up concerns are extremely site-specific. For example, the hazard posed by radionuclides in stream sediment may be much less than radionuclides in fill used under houses. The clean-up costs may also vary enormously. NRC (and EPA) accordingly should not issue generic clean-up standards but instead devise requirements appropriate for each site on its own merits. Finally, DOE has specifically criticized the purported foundation of Option One -- the proposed 5 pCi/gm Ra-226 standard -- as an undesirable clean-up standard in comments filed with EPA. See Exhibit J. NRC obviously should not employ Option One as a clean-up standard in the face of these criticisms by DOE.

Option 2. Option Two permits disposal of natural thorium with daughters in amounts up to 50 pCi/gm by burial under at least four feet of earth.[29] The Position states that that the NRC calculates that such a disposal will not result in exposures greater than in Option One. Option Two, which is

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[29] 46 Fed. Reg. 52062.

based on an extrapolation from Option One, is defective in that the Option One limits on which it is based are too low. The limit in Option Two also purports to be set such that no member of the public will likely receive doses in excess of 170 mrem to any organ on account of intrusion. This approach is far too conservative. 10 C.F.R. Part 20 calls for a 170 mrem limitation only with respect to whole body exposure of the general population, not with respect to exposure to any given organ of a maximally exposed individual. In any event, the underlying documentation available for our review was not adequate to evaluate whether NRC's assumptions with respect to this calculation are supportable. However, Kerr-McGee surmises that NRC made its intruder calculation on the basis of occupancy, shielding, and other assumptions similar to those in Option One and that the agency's exposure estimates are therefore overly conservative by a substantial factor.

Option 3. Option Three permits disposal of natural uranium, with daughters in equilibrium, in amounts up to 40 pCi/gm by burial under at least four feet of earth in areas zoned for industrial use, provided that deed covenants also restrict the disposal area to non-residential use.[10] The limit specified in Option Three is derived from EPA's proposed 5 pCi/gm radium-226 standard on which Option One is based. It is accordingly deficient for the reasons noted in the discussion of Option One. In addition, NRC made numerous assumptions

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[10] 46 Fed. Reg. 52062.

which further invalidate the 40 pCi/gm limit. For example, NRC assumed a 50% occupancy factor in an industrial building atop the site. The occupancy factor is too high (30% is conservative). NRC also assumed a dirt floor. Buildings are no longer constructed with dirt floors. NRC further assumed one air change per hour. This assumption is unduly conservative; dirt floored industrial buildings would likely require three to four air changes per hour under OSHA regulations. NRC should either assume a dirt floor and four air changes, or a slab floor and one air change, but not the worst case (and probably unlawful) combination of the two. Correction of NRC's assumptions will lead to much lower radon concentrations in the building. This will translate into a far less stringent limit on the natural uranium concentration which may be disposed under the specified conditions. In any event, even if NRC's assumptions were adopted, the resulting radon daughter concentrations would amount to only 0.04 WL. This is far less than naturally occurring radon concentrations in dwellings.[31] NRC also may be resting Option 3 on a 170 mrem exposure limit for intruders. NRC's proposed 10 C.F.R. Part 61 ("Licensing Requirements for Land Disposal of Radioactive Waste") employs an inadvertent intruder limit of 500 mrem whole body.[32] NRC offers no rationale for its different approach here.

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[31] Indeed, a recent study indicated that indoor radon concentrations may commonly be in the range of 12 to 33 pCi/l (0.13 WL to 1 WL using the conversion factor set forth in 10 C.F.R. Part 20, App. B, footnote 3). See Rundo, et al., Observation of High Concentrations of Radon in Certain Homes, 36 Health Physics 729 (1979).

[32] 46 Fed. Reg. 38095 (July 24, 1981) proposed § 61.42.

Option 4 Option Four permits disposal of up to 500 pCi/gm natural thorium (and daughters) and up to 200 pCi/gm natural uranium (and daughters) by burial in areas zoned for industrial use provided (1) that title documents specify that the land contains radioactive material and (2) that the documents are conditioned by a covenant. The covenant must provide that the land (1) may not be excavated below stated depths unless excavation is cleared by appropriate health authorities; (2) may not be used for residential or industrial structures; and (3) may not be used for agricultural purposes.[33]

The limits specified in Option Four are derived from the limits specified in Option One and are defective for similar reasons. However, the limits specified in Option Four are unduly conservative for an additional reason. Under the conditions specified in Option Four, there can be no buildings erected or agricultural activities conducted at the burial site. Under these conditions, the only significant radiological concern would be radon-222 emanating through the soil. However, significant exposure to radon would be impossible.

Radon-222 emanation from uranium wastes does not pose a significant risk to persons living outside the near-vicinity of the wastes in question. The amount of radon likely to result from the wastes, particularly in the low concentrations specified by the Branch Technical Position, is

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[33] 46 Fed. Reg. 52062.

miniscule compared to the amount of radon released naturally from soils in the United States and from such accepted economic activities as agriculture.[34] Moreover, increased radon from uranium wastes cannot be detected more than a short distance from such wastes, even in a downwind direction.[35] The effect of the wastes is vastly overborne by natural discharges.

Stringent controls for radon emanation similarly cannot be justified on the basis of risk to nearby residents. NRC acknowledges that there are no discernible adverse health effects from exposure to radon from uranium wastes.[36] NRC assumes that health effects will occur on the basis of the linear non-threshold model. However, this model is subject to the criticisms described earlier in these Comments.

Even assuming that the linear non-threshold model is appropriate, the risk which it projects in the situations involved here is insubstantial. NRC calculates that the

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[34] See, e.g., Transcript of Hearing before the New Mexico EIB at 461-62 & 470 (testimony of Dr. Evans) reprinted in Uranium Ore Residues, Hearings, supra, at 453.

[35] See, e.g., Shearer & Sill, Evaluation of Atmospheric Radiation in the Vicinity of Uranium Mill Tailings, 17 Health Physics 77 (1969) (Attachment K); Letter, Greenleigh (DOE) to Selander (EPA), dated July 15, 1981, at 2 (DOE comments on EPA inactive site standards); Uranium Ore Residues Hearings, supra, at 457; 127 Cong. Rec. S 12984 (daily ed. Nov. 5, 1981).

[36] NRC, Generic EIS on Uranium Milling (GEIS) at A-35 ("We [NRC] know of no data or studies which indicate definitively that health effects do or do not occur at the low levels of exposure that are anticipated to result from operation of uranium mills").

maximum risk posed by radon-222 from even totally unregulated mills is 1 in 70,000,000 for three times the number of mills now in existence.[37] This maximum risk, which is only hypothesized to exist anyway, is insignificant and de minimis. NRC has admitted that even if it actually eventuates, it is "about equal" only to the risks posed by "a few puffs on a cigarette, a few sips of wine, driving the family car about 6 blocks, flying about 2 miles, canoeing for 3 seconds, or being a man aged 60 for 11 seconds."[38] Indeed, the risk perceived by NRC is far less than many risks commonly and ordinarily accepted in our society.[39]

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[37] See, e.g., GEIS at 19.

[38] 46 Fed. Reg. 15167 (March 4, 1981). See also 127 Cong. Rec. S 12984 (daily ed. Nov. 5, 1981).

[39] The following table sets forth many commonly and ordinarily accepted risks in our society.

Cause	Individual risk/year
smoker	1/300
agricultural employment	1/1,700
motor vehicle - Total (1975)	1/4,500
air pollution - sulphates	1/6,700
government employment	1/9,100
truck driving employment	1/10,000
falls	1/13,000
alcohol	1/20,000
living for one year downstream from a dam	1/20,000
motor vehicle - pedestrian (1975)	1/25,000
drowning (from recreational activities)	1/53,000
inhalation and ingestion of objects	1/71,000
home accidents (1975)	1/83,000
bicycling	1/100,000
person in room with smoker	1/100,000
one pint of milk per day (aflatoxin)	1/100,000
accidental poisoning - solids and liquids	1/170,000

(footnote cont'd)

Kerr-McGee understands that NRC staff may take the position that this de minimis risk is not dispositive because the risk to people living next to, or on top of, uranium wastes, is greater. To Kerr-McGee's knowledge, NRC has not provided a detailed quantification of this alleged risk for public review. However, the risk -- which is purely hypothetical to begin with -- cannot be large in comparison to many customarily accepted risks. This view is strongly supported by the Commission's admission that radon exposures at the edge of uranium wastes stabilized in accordance with the agency's now suspended Uranium Mill Licensing Requirements will result in exposures which are only "a small fraction of any reasonable health protection limit."[40] Moreover, purported fears for persons living atop tailings are groundless in view of the requirement in the Uranium Mill Tailings Radiation Control Act that ownership of the disposal site generally be transferred

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(footnote cont'd)

Cause	Individual risk/year
electrocution	1/200,000
vaccination for small pox (per occasion)	1/330,000
air travel - one transcontinental flight/yr	1/330,000

Source: OSHA Testimony of Professor Richard Wilson (Exhibit L) reprinted in Hutt, Unresolved Issues in Conflict Between Individual Freedom and Government Control of Food Safety, 33 FDC L.J. 558, 564-66 and 568 (1978).

[40] GEIS at p. 12-15 explains that "[e]xposures as close in as a fencepost near the edge of the pile would be about 1.1 x 10-4 WL above background levels. . ., which is a small fraction of any reasonable individual health protection limit (1% of the Surgeon General's guidelines)."

to the government for actual tailings.[41] Obviously, Congress presumed that the government can and will keep people from occupying the disposal area. NRC's deed restrictions and covenants will accomplish the same purpose here.

NRC's risk estimates under the linear non-threshold model, which already predict insubstantial hazards, are in fact excessive because the agency has relied on erroneously high risk estimators. The latest study by a panel of eminent scientists from EPA, the Department of Energy (DOE), Germany England and Canada indicates that the maximum hypothetical risk from radon-222 can be no greater than 1/3 that employed by NRC per unit exposure and may in fact be zero.[42] Moreover Professor Cohen, after analysis of risk estimates for radon-induced lung cancer employed in the BEIR-III report, concluded that the risk estimators advocated in that report overstated the risk from low-level exposure by a factor of twenty to forty.[43] To the extent that NRC purports to rely on a risk of exposure through an ingestion pathway, that risk estimate is similarly excessive. Professor Evans has specifically

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[41] 42 U.S.C. § 2113(b).

[42] Evans, et al., Estimate of Risk from Environmental Exposure to Radon-222 and its Decay Products, 390 Nature 98 (March 12, 1981) (Attachment M). Significantly, DOE has expressly endorsed reliance on the study by Evans, et al., for standard-setting purposes. Uranium Ore Residues Hearings, supra, at 176.

[43] See also Cohen, Failures and Critique of the BEIR-III Lung Cancer Risk Estimate, ____ Health Physics ____ (in publication) (Attachment N).

criticized risk estimates for ingestion of low-levels of radium as unduly conservative and as unsupported by the evidence.[**]

Occupancy of an Option Four disposal site would be of limited duration (certainly no more than the 10% assumed by NRC). The radon directly over the site would be "fresh," containing few radon daughters. Since the radon daughters, not the radon, pose the purported health risk, the site itself would accordingly not be hazardous. There would be no significant off-site exposure because radon from a volume source disperses readily and the amount of radon released is de minimis compared to natural releases. In any event, burial under even a nominal amount of soil will attenuate radon emanation by a substantial amount. In sum, under the specified restrictions, the site could be used for disposal of ore-grade material without danger of posing a health hazard. This conclusion is amply supported by prominent health physics experts. See, e.g., Testimony of Dr. Evans before the N.M. EIB reprinted in Uranium Ore Residues: Potential Hazards and Disposition, Hearings before the Procurement and Military Nuclear Systems Subcomm. of the House Comm. on Armed Services, 97th Cong., 1st Sess. at 461 (June 1981).

[**] R. Evans, et al., in Radiobiology of Plutonium (Stover & Jee, eds. 1972) at pp. 431-68 (practical threshold for humans); Raube, et al., Bone Cancer from Radium: Canine Dose Response Explains Data for Mice and Humans, 208 Science 61 (1980).

A similar conclusion holds with respect to thorium wastes. Thorium wastes do not pose a significant radon problem. The radon decay product (known as thoron) in the thorium decay chain has a much shorter half life (only 55.6 seconds) in comparison to radon-222 and accordingly cannot be expected to diffuse in significant amounts through even nominal cover. It furthermore cannot be expected to travel any significant distance off-site. Moreover, the decay products of thoron, with one exception, are short-lived in comparison to decay products of radon-222.[45] Since an Option Four site cannot be used even for agricultural purposes, there obviously will be no hazard posed by ingestion of thorium or thorium daughters. Thus, disposal of ore-grade thorium-bearing material should also be permitted in the context of Option Four.

IV. Option Five Is Inconsistent with the Public Interest in Prompt Decommissioning, Is Contrary to Applicable Statutes and Regulations, and Should Not be Applied to Kerr-McGee's West Chicago Facility

Option Five authorizes storage of licensed concentrations of uranium and thorium wastes on-site pending the availability of a licensed disposal site.[46] As drafted, it

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[45] The properties of the thorium decay series are described in Rundo, The Radioactive Properties and Biological Behavior of ^{224}Ra (Th X) and its Daughters, 35 Health Physics 13 (1978).

[46] 46 Fed. Reg. 52063.

suggests that such wastes may be stored on-site for an indefinite period of time on the ground that a licensed disposal site is unavailable.[47] This option is thus directly contrary to the public interest which requires prompt decommissioning of nuclear facilities after completion of useful activities.[48]

Option Five is also contrary to applicable statutes and regulations. The UMTRC Act specifically contemplates on-site disposal of byproduct material as defined in section 11(e).2 of the Atomic Energy Act, 42 U.S.C. § 2114(e)(2). Indeed, NRC personnel have repeatedly represented that on-site disposed is the norm contemplated under the UMTRC Act.[49] Option

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[47] As NRC is aware, space at the three current low-level waste disposal facilities is limited. Moreover, all three facilities are one to two thousand miles from the Chicago area. Transporting substantial volumes of uranium or thorium residues to these sites raises risks of traffic accidents and fatalities, is extremely costly, and will unduly strain the capacity of these sites. The Low-Level Radioactive Waste Policy Act, which calls on states to locate sites for the establishment of new low-level repositories, does not apply to byproduct material as defined in section 11(e).2 of the Atomic Energy Act. Moreover, the availability of a low-level radioactive waste repository in the Midwest is at this time totally speculative even for material which is not covered by section 11(e).2.

[48] See, e.g., NRC, Draft Generic EIS on Decommissioning of Nuclear Facilities at p. v & vii (Jan. 1981).

[49] For example, NRC recently explained to Congress that

> "Moving an entire talings pile is an extreme worst case in that all other options would have to have been evaluated and found to be unsatisfactory. A great deal of flexibility exists in terms of options to stabilize the tailings pile in place. Our licensing experience indicates that through recontouring and

(footnote cont'd)

Five is inconsistent with the UMTRC Act and these representations insofar as it projects indefinite interim storage of byproduct material pending availability of an off-site licensed repository.

Option Five also contravenes 10 C.F.R. § 20.302. Under that regulation, the Commission has long authorized disposal of uranium and thorium wastes in concentrations much higher than that in Option Five by on-site burial. Option Five appears to preclude such disposal unless it can be accomplished in accordance with Options One through Four. This prevents disposal of higher concentration uranium or thorium wastes except in licensed disposal facilities. This marked change in NRC's construction of section 20.302 is without foundation. It is a significant curtailment of that regulation and imposes substantial additional costs upon the regulated public without

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(footnote cont'd)

> covering and hardening the tailings pile in place, the necessary level of assurance concerning long term stability can be achieved at most, if not all, existing sites."

Uranium Ore Residue Hearings, supra, at 543 (letter, Mr. Kammerer (NRC) to Chairman Stratton). Mr. Martin of NRC also testified that "frankly . . . we [NRC] have some concerns that perhaps the Department [of Energy] is contemplating excessive movement of those piles. We share EPA's view that they should not be moved except as a last resort, and even then they shouldn't be moved very far . . . ." Uranium Ore Residue Hearings, supra, at 155-56. See also 47 Fed. Reg. 1820, 1824 (Jan. 13, 1982) (EPA statement that "we believe perhaps only one [inactive uranium mill tailings] pile might have to be moved once all pertinent site-specific factors are fully analyzed.").-

commensurate benefits. It is extremely prejudicial and must be deleted entirely from the regulation.

Mr. Dircks' Memorandum to the Commission dated October 30, 1981, which conveyed the Branch Technical Position to the Commissioners, suggests that NRC staff may seek to apply Option 5 to Kerr-McGee's West Chicago Facility. This implies that staff contemplates possible indefinite interim storage of waste at the West Chicago Facility. Kerr-McGee objects strongly to any such indefinite interim storage. The West Chicago Facility is regulated under the UMTRC Act. The wastes at that Facility are byproduct material as defined in section 11(e).2 of the Atomic Energy Act. The UMTRC Act envisions prompt, permanent on-site disposal of the wastes in question. Indefinite interim disposal is patently contrary to the Act.

Kerr-McGee understands that the Office of the Attorney General (OAG) of the State of Illinois has already filed comments objecting to interim storage of uranium and thorium residues at Kerr-McGee Chemical's West Chicago facility.[50] Kerr-McGee joins in this aspect of OAG's comments. Interim storage of the material in question contravenes not only the public interest in prompt decommissioning but also the UMTRC Act. It additionally is uneconomic and wasteful of society's reserves. OAG also observes that the residues in question are

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[50] Letter, Ms. Rapkin (OAG) to Mr. Page (NRC) (Dec. 23, 1981).

regulated under the UMTRC Act, another point in which Kerr-McGee concurs.

VI. Legal Status of Branch Technical Positions

The Administrative Procedure Act (APA) specifically defines "rules" to include any "agency statement of general . . . applicability and future effect designed to implement, interpret or prescribe law or policy . . . ." 5 U.S.C. § 551(4). The APA is applicable to actions taken by the NRC.[51] Its definition of rule encompasses the Branch Technical Position issued here. In particular, it is clear that the Branch Technical Position is intended to be an agency statement of general applicability and future effect. The cover memorandum from Mr. Dircks to the Commission dated October 30, 1981 indicates that the standards are intended to be prescriptive. Indeed, NRC has already sought to apply Option One to Kerr-McGee in West Chicago as a clean-up standard. NRC must accordingly conform to the APA in issuing the Branch Technical Position in final form. Moreover, the Branch Technical Position, when so issued, will be subject to judicial review.

Kerr-McGee has noted a number of objections to the the Branch Technical Position including NRC's lack of authority to issue it, its prematurity, its inconsistency with applicable statutes and regulations and its lack of basis. Kerr-McGee

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[51] 42 U.S.C. § 2231; Siegel v. Atomic Energy Commission, 400 F.2d 778, 785 (D.C. Cir. 1968).

also notes that NRC made insufficient information available to permit the Company to review all of the agency's rationale for some of the options. The APA specifically guarantees interested parties "an opportunity to participate in the rule-making." 5 U.S.C. § 553(c). As a corollary of this right, agencies must "disclose in detail . . . the data upon which [a] rule is based."[52] This is particularly true with respect to mathematical, statistical, and scientific analyses such as those which must underlie calculation of standards for disposal of radionuclides.[53] Assuming that NRC wishes to go forward with the Branch Technical Position, the agency as a minimum should

(1) eliminate sites subject to UMTRC Act from its proposed Branch Technical Position

(2) defer republication of a revised Branch Technical Position applicable to non-UMTRC Act sites until EPA has issued governing standards

(3) repropose the revised Branch Technical Position only following publication of final EPA standards and in a manner consistent with the APA; and

(4) provide sufficient data to disclose the NRC's calculations and rationale.

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[52] Home Box Office, Inc. v. FCC, 567 F.2d 9, 35 (D.C. Cir.), cert. denied, 434 U.S. 829 (1977). See also Portland Cement Ass'n v. Ruckelshaus, 486 F.2d 375, 393 n.67 (D.C. Cir. 1973), cert. denied, 417 U.S. 921 (1974).

[53] See United States v. Nova Scotia Food Products Corp., 568 F.2d 240, 252 (2d Cir. 1977).

Conclusion

For the reasons stated above, the Branch Technical Position must be withdrawn.

Kerr-McGee Corporation
Kerr-McGee Nuclear Corporation
Kerr-McGee Chemical Corporation

sent that the remainder of title IV be considered as read and open to amendment at any point.

The CHAIRMAN. Is there objection to the request of the gentleman from Alabama?

There was no objection.

The CHAIRMAN. Are there any points of order against the provisions of title IV?

AMENDMENT OFFERED BY MR. STRATTON

Mr. STRATTON. Mr. Chairman, I offer an amendment.

(The portion of the bill to which the amendment relates is as follows:)

NUCLEAR REGULATORY COMMISSION

SALARIES AND EXPENSES

For necessary expenses of the Commission in carrying out the purposes of the Energy Reorganization Act of 1974, as amended, and the Atomic Energy Act, as amended, including the employment of aliens; services authorized by 5 U.S.C. 3109; publication and dissemination of atomic information; purchase, repair, and cleaning of uniforms; reimbursements to the General Services Administration for security guard services; hire of passenger motor vehicles and aircraft; $477,534,000, of which not to exceed $1,455,000 shall be available for the Office of the Commissioners, and of which $80,610,000 shall be available for the Office of Nuclear Reactor Regulation, and of which $62,667,000 shall be available for the Office of Inspection and Enforcement: *Provided*, That from this appropriation, transfer of sums may be made to other agencies of the Government for the performance of the work for which this appropriation is made, and in such cases the sums so transferred may be merged with the appropriation to which transferred: *Provided further*, That moneys received by the Commission for the cooperative nuclear safety research programs may be retained and used for salaries and expenses associated with those programs, notwithstanding the provisions of 31 U.S.C. 484, and shall remain available until September 30, 1983: *Provided further*, That funds available for nuclear reactor research shall remain available until September 30, 1983: *Provided further*, That transfers between accounts may be made only with the approval of the Committees on Appropriations of the House of Representatives and the Senate: *Provided further*, That no part of the funds appropriated in this Act may be used to implement section 110 of Public Law 96-295.

The Clerk read as follows:

Amendment offered by Mr. STRATTON: Page 30, line 3, strike out the period and insert in lieu thereof the following: ": *Provided further*, That no funds appropriated to the Nuclear Regulatory Commission in this Act may be used to implement or enforce any portion of the Uranium Mill Licensing Requirements published as final rules at 45 Federal Register 65521 to 65538 on October 3, 1980.".

Mr. STRATTON (during the reading). Mr. Chairman, I ask unanimous consent that the amendment be considered as read and printed in the RECORD.

The CHAIRMAN. Is there objection to the request of the gentleman from New York?

There was no objection.

Mr. STRATTON. Mr. Chairman, my Subcommittee on Procurement and Military Nuclear Systems of the Armed Services Committee held 2 days of hearings on the remedial action programs for the decontamination and decommissioning of sites formerly associated with nuclear defense programs and for sites which contain uranium mill tailings or other uranium ore residues. These remedial action programs are being developed to meet the requirements of Public Law 95-604, the Uranium Mill Tailings Radiation Control Act of 1978.

The act assumes that uranium mill tailings pose a potential and significant radiation health hazard to the public. Without any evidence that uranium mill tailings are a hazard, Public Law 95-604 requires EPA and NRC to develop standards and regulations that have been interpreted, for instance, to require dirt piled 10 to 30 feet on the piles because our future Government might not continue for another 1,000 years. Without any evidence of hazard, EPA proposed the promulgation of standards that could require cleanups at certain sites that would place those sites at or below the background levels for radon and radium that occur naturally in neighboring areas.

The act directed EPA to issue final cleanup and disposal standards by November 8, 1979. This deadline was not met, but interim standards were proposed on April 22, 1980, and January 19, 1981. A comment period was established which ended on July 15, 1981. The Deputy Administrator of EPA testified at hearings on June 24, 1981, that EPA would take 2 to 3 years to establish final standards because they wished to consider very carefully all of the public comments available to this agency.

Public Law 95-604 required the Nuclear Regulatory Commission to promulgate rules which would implement and enforce the EPA final standards. There are no EPA final standards; and there should not be NRC rules.

The cost to the private sector and to the U.S. Government to follow the NRC final rules could be in the billions of dollars without providing any real improvements in the health and safety of the American public. Until such time as epidemiological studies are done, until such time as EPA publishes its standards and until we know what hazards actually exist, the Nuclear Regulatory Commission should be prohibited from enforcing the arbitrary, capricious and expensive-to-apply rules they have promulgated.

Mr. BEVILL. Mr. Chairman, will the gentleman yield?

Mr. STRATTON. I yield to the gentlman from Alabama.

Mr. BEVILL. Mr. Chairman, I agree with the gentleman from New York that in this era of budget stringency there should be careful controls on the expenditure of funds which provide marginal benefits to the public. I thank the gentleman for bringing this serious deficiency to our attention. We accept this amendment on this side.

Mr. MYERS. Mr. Chairman, will the gentleman yield?

Mr. STRATTON. I yield to the gentleman from Indiana.

Mr. MYERS. Mr. Chairman, we certainly do not want to get into a situation where we would needlessly spend hundreds of millions or billions of dollars to modify areas that pose little, if any, hazard to the public. We accept the amendment on this side.

The CHAIRMAN. The question is on the amendment offered by the gentleman from New York (Mr. STRATTON).

The amendment was agreed to.

□ 1430

Mr. DECKARD. Mr. Chairman, I move to strike the last word.

(Mr. DECKARD asked and was given permission to revise and extend his remarks.)

Mr. DECKARD. Mr. Chairman, it was at this point that I intended to introduce an amendment affecting the Columbia Dam. For technical reasons, I am unable to do so.

Last December, the Government Operations Committee, of which I am a member, unanimously approved a report criticizing the Columbia Dam project in Tennessee as a waste of money. Completion of the project would cost taxpayers an additional $100 million, simply to create the eighth recreational reservoir within a short radius. To that end, TVA proposes to destroy a river, condemn 440 farms, and force 1,500 people from their homes.

The TVA appropriation in this bill is $15.8 million—the amount of money for the Columbia Dam project for fiscal year 1982. These funds were deferred in the fiscal 1981 supplemental appropriation and rescission bill which we adopted a short time ago, pending acquisition by TVA of the necessary permits. These have now been obtained, and only by cutting these funds can the project be halted. A vote to do so would save $15.8 million immediately and more than $100 million in downstream costs.

The committee found that—

First, this project would not produce a single benefit that could not be achieved by a less costly alternative. Indeed, the Columbia Dam is the highest cost alternative for addressing the problems noted by project sponsors.

Second, the primary justification of the project—fully 60 percent of claimed benefits—is for flat water recreation in an area which already has seven reservoirs within a short drive of the project site.

Third, 440 farms would be destroyed and 1,500 people would be forced from their homes to make way for this recreational reservoir.

Fourth, over $100 million remains to be spent on this project, but all the claimed benefits—aside from those associated with a speedboating lake—could be realized by spending between

Calendar No. 352

97TH CONGRESS } SENATE { REPORT
1st Session } { No. 97-256

ENERGY AND WATER DEVELOPMENT APPROPRIATION BILL, 1982

OCTOBER 28 (legislative day, OCTOBER 14), 1981.—Ordered to be printed

Mr. HATFIELD, from the Committee on Appropriations, submitted the following

REPORT

[To accompany H.R. 4144]

The Committee on Appropriations, to which was referred the bill (H.R. 4144) making appropriations for energy and water development for the fiscal year ending September 30, 1982, and for other purposes, reports the same to the Senate with various amendments and presents herewith information relative to the changes recommended:

AMOUNT IN NEW BUDGET (OBLIGATIONAL) AUTHORITY, FISCAL YEAR 1982

Amount of bill as passed by House	$13,189,674,000
Budget estimates considered by House	13,410,287,000
Budget estimates considered by Senate	12,096,547,000
Amount of bill as reported to Senate	12,418,611,000

COMMITTEE ALLOCATION

Amount allocated from 1st Budget Resolution	13,500,000,000

dation proposes a $35,000,000 general reduction from the March budget request. However, the recommended appropriation is still $10,100,000 over the revised request for 1982, and more than $23,000,000 over the funding level for 1981.

Nuclear data link.—The Committee recommendation restores the House reduction and includes the requested funding for the nuclear data link system. This prototype system is designed to improve emergency response and enhance the quality and quantity of technical data available to NRC headquarters emergency response personnel.

The Committee believes that the Commission should proceed with a prototype system to further determine feasibility and cost effectiveness of this project. Although the full budget request is approved, the Commission is directed to spend no more than $1,000,000 until such time as it has submitted a report to the Congress and has received the approval of the Committees on Appropriations.

Licensing recovery.—As noted in the House report, the Commission has shown improvement in the handling of operating license requests and construction permits. The Commission has initiated a program to recover from the delays in issuing operating licenses that resulted from the accident at Three Mile Island. This effort to reduce licensing delays was accelerated by the reprograming of $4,300,000 in fiscal year 1981 to support licensing activities.

During fiscal year 1982, the Commission should pay particular attention to plants scheduled for completion and licensing in 1983. The Commission should identify early those plants that are likely to be strongly contested and allow sufficient staff and hearing time for those contested plants to be licensed without unnecessary delay.

Loss-of-fluid test program.—Within the funds appropriated to the Nuclear Regulatory Commission, the Committee notes that $44,000,000 is recommended to be allocated to the loss-of-fluid test (LOFT) program at Idaho National Engineering Laboratory. The Committee underlines its support for this program which is designed to conduct experiments to determine the results of the loss of reactor coolant water.

The Committee notes further that the LOFT Special Review Group (LSRG) was established to review the LOFT program and report its findings to the Nuclear Regulatory Commission. The primary purpose of the group was to consider whether LOFT should be decommissioned in fiscal year 1983, as recommended by the Advisory Committee for Reactor Safeguards. On February 11, 1981, the NRC Commissioners met to hear the report of the LSRG and the response of the Office of Nuclear Regulatory Research (RES).

RES developed a testing scheme which drew from tests suggested by LSRG. The RES testing scheme proposed a particular sequence which would provide, in their view, an optimum feedback and scientific return. The views presented by RES were consistent with those presented by the LSRG. The proposed RES testing matrix and schedule estimates would put the final LOFT test in January 1983. The Committee specifies that the $44,000,000 are appropriated for the conduct of those tests identified in the proposed RES testing matrix.

Uranium mill licensing requirements.—The House of Representatives adopted a provision that would prohibit the use of appropriated funds

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by the NRC to implement or enforce any portion of the uranium mill licensing regulations promulgated by the Commission on October 3, 1980.

The Commission has advised the Committee that the House provision would have several detrimental effects on the uranium mill licensing programs of the NRC and those Agreement States that have elected to regulate uranium mills under State law. In particular, the NRC has expressed the concern that the House provision would prevent the Commission from imposing on a case-by-case basis license conditions on uranium milling needed to protect the public health and safety, as it did prior to the promulgation of the NRC uranium mill licensing regulations. The Commission has also expressed the concern that the House provision would force the Commission to terminate all Agreement State uranium mill licensing programs after November 8, 1981—the present statutory deadline for Agreement State compliance with certain requirements of the Uranium Mill Tailings Radiation Control Act of 1978 (UMTRCA).

The Committee agrees with the House that the Commission should not have adopted these technical requirements for uranium mill tailings prior to EPA's promulgation of final environmental standards. Nevertheless, the Committee believes that the House provision is broader than necessary to carry out the purpose of preventing the implementation and enforcement of the NRC technical requirements. In addition, the Committee believes that the detrimental effects identified by the NRC, as described above, should be eliminated. For these reasons, the Committee has modified the House provision.

As modified by the Committee, the provision would prevent the Commission from implementing or enforcing the technical requirements in uranium mill licensing regulations promulgated by the NRC on October 3, 1980, or from adopting by regulation similar technical requirements during fiscal year 1982 until EPA promulgates its final environmental standards for uranium mill tailings at active uranium mill sites. However, the NRC would retain its authority to regulate uranium mill tailings on a case-by-case basis in the manner and to the extent permitted prior to the promulgation of its uranium mill licensing regulations. Thus, the Commission would retain the authority to impose through license conditions those requirements for the management of uranium mill tailings that the Commission can demonstrate in each case are necessary to protect the public health, safety, and the environment. This preserves the situation in effect prior to the promulgation of the NRC uranium mill licensing regulations. The provision, as modified by the Committee, is intended to permit the Commission also to retain its authority under UMTRCA to promulgate by regulation technical requirements for uranium mill tailings after EPA promulgates its final environmental standards for uranium mill tailings at active sites.

The House provision has also been modified by the Committee to prevent the Commission from terminating any Agreement State program for uranium mill licensing, and from reinstating NRC regulatory authority in such a State, if the State fails to adopt technical requirements that are comparable to the NRC technical requirements suspended by the provision. Under the provision as modified by the

Committee, the NRC would retain the authority to enter into an amended State agreement if the revised State uranium mill licensing program meets all other requirements of UMTRCA. However, during the period in which NRC is prohibited from enforcing the technical requirements in its regulations, it would lack the authority to require any State to adopt comparable requirements in order for the State to continue to exercise authority under State law for uranium mill licensing. The provision would not preclude, however, any Agreement State from adopting technical requirements for uranium mill tailings as permitted under State law.

In its comments on the House provision, the Commission noted that there is the potential for renewed litigation on the issue of the fuel cycle health effects of radon in some individual reactors license proceedings if the House provision is enacted. The Committee expects the Commission and its Atomic Safety and Licensing and Appeal Boards to review these ongoing reactor licensing proceedings on the health effects of radon and to take appropriate action to minimize the potential for disruption from this provision.

The following table shows the allocation of the funds recommended by the Committee.

[In thousands of dollars]

		1982		
	Appropriation 1981[1]	Budget estimate	House allowance	Committee recommendation
Nuclear reactor regulation	$73,590	$75,610	$80,610	$75,610
Standards development	16,650	17,350	17,350	17,350
Inspection and enforcement	57,500	67,680	62,667	67,680
Nuclear material safety and safeguards	38,090	46,700	46,700	46,700
Nuclear regulatory research	215,750	231,940	222,340	231,940
Program technical support	18,990	19,140	19,137	19,140
Program direction and administration	38,946	41,680	41,280	41,680
Total	459,516	500,700	490,684	500,700
Travel			-1,392	
Personnel costs			-3,077	
Consultants			-500	
SLUC charges			-432	
Consolidation			-500	
General reduction		-45,100	-5,000	-35,000
Other			-2,249	
Total	459,516	455,600	477,534	465,700

[1] Includes $11,996,000 in prior year unobligated balances.

particular interest or to demean the importance of the questions they are raising here, but from a fiscal point of view, we are resisting all amendments, as I have just tried to resist the Tower amendment and failed. We are resisting all amendments to this bill because we are $350 million above the President's level. Now, with the Tower amendment, we are going to move close to the $700 million over the President's requested level. That puts us in real jeopardy of getting the bill signed. Nowhere does this remark reflect at all on the project itself.

Mr. GLENN. Mr. President, I appreciate very much the remarks of the distinguished floor manager of the bill. As he has pointed out, the language in the committee report says:

> The $90,000,000 general reduction proposed for uranium supply and enrichment activities should be prudently allocated so that, to the extent possible, there is minimal impact on production capability.

So we can spell this out, Mr. President, is it the view of the appropriations committee that "minimal impact on production capability" could be achieved by allocating the general reduction in such a way that planned increases in the efficiency of production are allowed to move forward on presently projected time frames?

Mr. HATFIELD. Yes, Mr. President, future increases in efficiency as a means of maintaining or increasing production capability would be highly desirable, and should be taken into account by DOE in allocating any general reduction.

Mr. METZENBAUM. Mr. President, I inquire of the manager of the bill, the distinguished Senator from Oregon, is it the view of the Appropriations Committee that, in drafting the passage referred to by the senior Senator from Ohio, the committee did not wish to suggest that DOE should make a decision in this area that could cause delays in bringing enrichment capacity on line that would be needed for competing for new contracts for enriched nuclear fuel?

Mr. HATFIELD. It is definitely the intent of the committee that DOE should avoid any delays at all.

Mr. GLENN. Mr. President, I appreciate that statement by the distinguished chairman. I want further to ask, does the chairman of the Committee on Appropriations agree with the assessment that the gas centrifuge enrichment project will be an important tool for easing U.S. balance-of-payments problems, for promoting electric energy efficiency while increasing our nuclear fuel production capability, and for aiding the U.S. nuclear nonproliferation effort by making the United States a reliable supplier of low-cost nuclear fuel to foreign as well as domestic utilities?

Mr. HATFIELD. Absolutely; I believe GCEP can help very definitely in these areas, as well as all other areas where it has already proven its worth.

Mr. METZENBAUM. Mr. President, does the chairman of the Appropriations Committee agree that the allocation of the general reduction by DOE should not create a situation in which costs to the Government for enhancing uranium enrichment capacity in the future will rise by an amount greater than the general reduction?

Mr. HATFIELD. I agree that the allocation of any general reduction by DOE should be performed in a way that is cost effective and every effort should be made by DOE to avoid significantly greater costs for DOE programs in the future.

Mr. METZENBAUM. I have one last question, Mr. President. We are aware of the fact that we are $90 million below the House bill in this area. I also respect and I know that my colleague from Ohio equally well respects the position of the chairman of the Appropriations Committee in resisting any effort to go beyond the excesses that are in the bill, excesses over the amount. My question is, Can we who have a major concern about this subject be reasonably well-assured that, when this matter is considered by the conference committee, the Senator from Oregon, the chairman of the Committee on Appropriations (Mr. HATFIELD), will give every possible consideration to bringing the $90 million back into it? Can he, hopefully, find ways to do that, as is provided in the House bill and, rather than make the issue on the floor of the Senate, can we hope that the matter can be resolved at the conference committee level?

Mr. HATFIELD. I shall make sure this Senator will put a double asterisk on this item in conference to remind us to be very sympathetic to the request of both Senators from the State of Ohio.

Mr. METZENBAUM. Both of the Senators from Ohio will be doubly appreciative of the double asterisk and be even more grateful if they put the $90 million back in.

I yield to the senior Senator from Ohio.

Mr. GLENN. Mr. President, I thank the distinguished floor manager of the bill for his consideration in this matter. I think this colloquy very well spells out what we hope will take place. It is an important project. It is one of the very few facilities in the world like this; in fact, it is the only one like this exactly. It is energy efficient, as I pointed out in my earlier statement. If we cut back now, it will have some very serious implications in international markets as well as domestic.

I point out once again this facility is a moneymaker for the country. It is not a drain on the budget. We are particularly grateful for the support indicated by the floor manager of the bill.

Mr. METZENBAUM. Mr. President, in view of the double asterisk assurances given by the chairman of the Committee on Appropriations, on behalf of the senior Senator from Ohio and myself, I now withdraw the amendment.

The amendment (UP No. 591) was withdrawn.

Mr. SIMPSON. Mr. President, I want to express my support for the limitation now in this bill on NRC's implementation of its uranium mill licensing regulations. I believe that the limitation is an appropriate and necessary step in restoring a rational and workable regulatory program for uranium mills and mill tailings.

As chairman of the Nuclear Regulation Subcommittee, I have directed considerable attention to the implementation of the Uranium Mill Tailings Radiation Control Act of 1978 (UMTRCA). On June 16, 1981, the Nuclear Regulation Subcommittee held a hearing to review certain problems that have developed in the implementation of UMTRCA. I believe that the hearing record discloses a number of serious problems in the implementation of the act. These include the following:

First, the Environmental Protection Agency has failed to adopt final environmental standards for uranium mill tailings at active sites, even though the act imposed a statutory deadline of May 8, 1980, for such standards. These environmental standards were an essential element in the integrated regulatory program involving EPA and NRC established by the act, and were to serve as the basis for NRC's subsequent development of specific technical requirements for mill tailings disposal at active mill sites.

Second, NRC has proceeded to adopt its final technical requirements for uranium mill licensing without the benefit of the EPA standards. These NRC regulations, which became effective on August 5, 1981, include general environmental standards such as specified reductions in radon releases from mill tailings disposal sites, and technical requirements for earth and rock cover to achieve these standards. The issuance of these NRC regulations violates the careful division of regulatory responsibilities for mill tailings in the act and creates the potential for future shifts and conflicts in regulatory requirements affecting present and new uranium milling operations.

Third, there appears to be substantial disagreement within the scientific community regarding the basis for certain of the technical requirements adopted by NRC. In particular, there appears to be considerable disagreement over whether the NRC's stringent radon release limits, and the cover requirements to achieve these limits, are actually needed to protect the public health, safety and the environment. This is one of the questions the EPA environmental standards were intended to resolve.

Fourth, certain of the technical requirements adopted by NRC—most notably, the radon release limits, and the cover requirements to achieve these limits—are cast as absolute requirements, and NRC has provided no flexibility in applying these requirements at existing sites where large quantities of mill tailings already exist. In adopting these absolute requirements, particularly for existing sites, NRC does not appear to have given sufficient attention to considerations of practicability such as the economic and environmental cost of meeting the requirements.

Finally, from the date of enactment of the act until the hearing held by the Nuclear Regulation Subcommittee on June 16, the NRC appears to have insisted that all agreement States must adopt the NRC regulations as an absolute minimum without regard to differing local or regional conditions, if the State is to retain its authority for uranium mill licensing. This position, which was modi-

fied in testimony before the subcommittee, stands in marked contrast to the words of the act, which provide that a State must adopt standards that are "equivalent, to the extent practicable, or more stringent than," the NRC standards. This position may also have denied some agreement States the opportunity until recently to develop and propose alternatives to those NRC requirements that a State believes are not practicable.

Mr. President, it is clear to me that the technical requirements contained in NRC's uranium mill licensing regulations are a significant contributor to many of the problems experienced in the implementation of the act. Now that the NRC technical requirements are in effect and the deadline for amended agreement State programs is approaching, the adverse impact of these technical requirements is likely to increase substantially. For these reasons, I believe that a prompt suspension of NRC's implementation and enforcement of these technical requirements is an essential first step in correcting the problems described above. Enactment of the provision in this bill would acomplish this objective and would provide the time needed to accomplish more detailed changes to the regulatory program through subsequent legislation and administrative action.

Mr. President, the Commission has recently advised me that enactment of the House provision might have several detrimental effects. In particular, the Commission notes that the House provision would prevent the Commission from imposing on a case-by-case basis license conditions on uranium milling needed to protect the public health and safety, as it did prior to adopting its uranium mill licensing regulations. In addition, the Commission notes that the House provision would force the Commission to terminate all agreement State uranium mill licensing programs after November 8, 1981—the present deadline for amended agreements.

Mr. President, I believe that the changes made to the House provision fully address the NRC concerns. Specifically, the provision in the bill makes it clear that NRC retains the authority to regulate mill tailings in the manner and to the extent permitted prior to the adoption of the NRC's technical regulations, and that agreement States may continue to regulate uranium mills during the period in which NRC's technical requirements are suspended.

The Commission has also indicated that the House provision might lead to renewed litigation on the question of the fuel cycle health effects of radon in some individual reactor license proceedings. On further study, however, it appears that the NRC's Atomic Safety and Licensing Appeal Board has also calculated radon source terms assuming that the NRC technical requirements do not take effect. Including these radon source terms in the ongoing NRC proceedings to determine the health effects of radon should minimize the potential for future disruption of these reactor license proceedings.

Mr. President, the provision in the bill would simply preserve the situation in effect prior to the effective date of the NRC regulations, thereby allowing time to develop the more detailed legislative and administrative changes needed to correct the problems in implementing the Mill Tailings Act. During this 1-year period, NRC would be prohibited from implementing or enforcing their uranium mill licensing regulations; those States that have acted to exercise uranium mill and mill tailings regulatory authority under State law would not be required to adopt requirements comparable to the suspended NRC regulations; and NRC would be prohibited from exercising any regulatory authority for uranium mills and mill tailings in any State that has acted to exercise regulatory authority for uranium mills and mill tailings under State law. I strongly support the provision in the bill as an essential first step in restoring a rational and workable regulatory program for uranium mills and mill tailings.

● Mr. HART. Mr. President, the Energy and Water Appropriations Act (H.R. 4144), under consideration by the Senate today, contains a provision that would drastically restrict the authority of the Nuclear Regulatory Commission to carry out its responsibilities under the Uranium Mill Tailings Radiation Control Act of 1978 (P.L. 95-604). This provision prohibits the NRC from spending appropriated funds to implement and enforce the final regulations, which it promulgated 1 year ago, for managing and cleaning up piles of uranium mill tailings scattered around the country.

I understand that the chairman of the Senate Appropriations Committee accepted this provision on the advice that these restrictions enjoyed the support of the Environment and Public Works Committee, on which I sit. Although I will not press today for a vote opposing this provision, I want the record to show that I do not support efforts to delay implementation of the Mill Tailings Act.

The Mill Tailings Act has two purposes. First, it establishes a remedial action program to clean up over 25 million tons of tailings at two dozen abandoned uranium mills in several Western States and Pennsylvania. The affected States and the Federal Government will share the cost of the cleanup program, currently estimated at around $405 million.

Second, and just as important, the act provides a new Federal program for regulating management and disposal of uranium tailings to protect public health and safety from the radiological and nonradiological hazards of these tailings. As part of this regulatory scheme, the NRC has promulgated health and safety standards. The four States that have received authority, delegated from the NRC through the agreement States program, to regulate uranium millings with their boundaries must adopt, by November 8, 1981, standards "which are equivalent, to the extent practicable, or more stringent than" the NRC's standards. The NRC will continue to regulate directly uranium mill tailings in those States that do not participate in the agreement States program.

The Mill Tailings Act, in essence, established a uniform system for protecting the public from the dangers of uranium milling and the resulting tailings.

Several arguments have been made for suspending NRC's authority under the Mill Tailings Act. I do not find any of these persuasive.

First, it has been argued that Agreement States cannot meet the November 8 deadline for conforming their standards to the NRC regulations. This apparently is the primary stated reason for suspending NRC's regulatory authority. Yet, three of the four agreement States—Texas, Washington, and Colorado—will come very close to meeting the November 8, deadline. In fact, only New Mexico has appealed to the Environment and Public Works Committee for an extension of the deadline—for up to 2 years. Thus, the provisions in the Energy and Water Appropriations Act constitute a private bill for New Mexico. In granting New Mexico relief, however, this provision disrupts efforts in three other States to comply with Mill Tailings Act. Indeed, the suspension of the NRC's regulatory authority could cause all the staff resources and funds spent by the other agreement States in establishing adequate programs for regulating uranium milling to go for naught.

Second, those supporting a suspension of the NRC's regulatory authority maintain that because the Mill Tailings Act assumes the EPA would issue its mill tailings standards prior to promulgation of the NRC's final regulations, the NRC regulations should not go into effect before the EPA has acted. Admittedly, the EPA has missed by almost 2 years the deadline in the Mill Tailings Act for issuing its standards. I have repeatedly criticized the EPA's chronic inability to meet deadlines for issuing its various radiation standards, including those for mill tailings. After working closely with EPA staff to anticipate the EPA standards, the NRC properly decided to go ahead and promulgate its final regulations. None of the testimony taken by the EPA standards, when finally issued, would significantly alter the NRC regulations. If EPA is still unable to issue its standards, the proper solution is not to disrupt the entire Federal regulatory program by suspending the NRC's authority under the Mill Tailings Act, but rather to discharge the EPA from further responsibility under the act. Indeed, given the administration's apparent intent to dismantle the agency through unprecedented budget cuts over the next few years, it seems highly unlikely that EPA will issue its standards soon.

Finally, it has been argued that the NRC has not given agreement States the flexibility provided under the Mill Tailings Act to adopt standards "equivalent, to the extent practicable" to the NRC's final regulations. If indeed this is the case, then it is more appropriate for authorizing legislation to make clear that the NRC should give agreement States the flexibility provided under the Mill Tailings Act to adopt standards that deviate from the NRC's final regulations. It is highly inappropriate, however, to use an appropriations bill to suspend the entire NRC mill tailings regulation program, particularly for those agreement States that have already adopted their own standards based on the NRC regulations.

Mr. President, I strongly object to this apparent attempt at end-running the authorization process to disrupt the uranium mill tailings regulation program. Uranium mill tailings pose a significant hazard to public health and safety. They contain 85 percent of the original radioactivity of the uranium ore. Moreover, according to the U.S. Geological Survey:

Windblown tailings from inactive, unstabilized piles in the Western States are responsible for (radiation) dose rates greater than 25 millirems per year at distances up to one mile from the pile.

This dose rate equals the rate EPA permits per person each year for the entire nuclear fuel cycle.

Attempts to disrupt the uranium mill tailings regulation program, or to relax the restrictions on managing and disposing of mill tailings, places the public health and safety at continued risk.

We should realize that it is not the additional cost of properly controlling mill tailings that has created the financial problems the uranium industry now faces. Rather, these financial difficulties result from pure economic—from the decreased demand for uranium to fuel commercial power reactors, which in turn stems from a decreased demand for nuclear-generated electricity.

Mr. President, this provision does not foreclose further debate on the Federal program for regulating uranium mill tailings. I hope the full Senate will carefully review this issue, when it comes up again in the next few weeks as part of Senate consideration of the NRC Authorization Act for fiscal years 1982 and 1983 (S. 1207). That is the only proper forum for debating an issue that could seriously affect the health and safety of people living in many States throughout the Nation.●

Mr. DOMENICI. Mr. President, NRC's uranium mill licensing requirements have been roundly criticized by Government agencies and by responsible representatives of the uranium industry. The Department of Energy has specifically declined to support the 2 pico curie per meter squared per second radon emanation standard which is a key element of NRC's regulations. DOE has stated:

Because radon dispersion from unstabilized piles does not produce measurable effects even at short distances from their boundaries, we cannot support the proposed 2pCi/m2-sec radon flux standard, which is very close to or lower than background in many parts of the nation.

The New Mexico Environmental Improvement Division has explained that NRC's radon limit is "based on highly uncertain and speculative estimates of health effects and on unfounded assumptions that adverse health effects might occur in certain hypothetical situations."

Similarly, the New Mexico Environmental Improvement Board, after taking extensive testimony and public comments, found that NRC's "standards are unsupported, unreasonable, and impracticable."

NRC's uranium mill licensing requirements are troublesome for an independent reason: They were issued in advance of EPA standards. Under section 275 of the Atomic Energy Act, EPA must issue the standards, not NRC. EPA has not yet issued standards. NRC erred in issuing standards and more detailed requirements in advance of EPA's standards. NRC's action is not only contrary to the statute but also subjects agreement States and regulated industry to shifting and conflicting regulatory requirements.

NRC's uranium mill licensing requirements will be extremely expensive. NRC estimates that they will cost the domestic industry $1 billion or more. Industry believes that the cost is far greater. Moreover, NRC has indicated to DOE that it intends to apply its regulations to inactive sites for which DOE is responsible. DOE estimates that comparable requirements imposed on inactive sites will cost the taxpayer almost $500,000,000, even before inflation. The burden to the taxpayer will be far more if the standards are applied to other sites for which DOE is responsible or to that portion of the tailings at active sites which was produced under Government contract.

This is a tremendous cost for taxpayers and domestic industry to bear. This is especially true in view of the fact that NRC admits that there is no evidence that establishes that exposure to radon at low levels associated with tailings is hazardous and the fact that NRC's estimate for the average hypothetical risk under the linear nonthreshold model from tailings is only 1 in 70,000,000.

Incidentally, this risk, even if it occurs, is equivalent only to the risk from a few puffs on a cigarette, a few drinks of wine, or driving the family car a few blocks. And NRC represents that this remote risk is for three times the numbers of mills now in existence even if the tailings from all those mills are totally unregulated. It is difficult to explain to unemployed uranium miners why this remote and hypothetical risk warrants these tremendous costs. And there are a lot of unemployed uranium miners. Half the uranium miners in Wyoming, Colorado, and New Mexico are already out of work. New closings and cutbacks are being announced on an almost weekly basis.

The provision adopted by the Appropriations Committee is responsive to these concerns. It has the effect of suspending the NRC regulations until EPA issues standards. It does not interfere with NRC's ability to regulate uranium mills as it did prior to the adoption of the regulations. In fact, all problems raised by NRC with respect to the amendment have been carefully considered and addressed by the Senate Appropriations Committee, as indicated in the detailed discussion of the committee's version of the amendment in the committee's report.

Some may ask why the suspension should be included in this bill. The fact is that NRC has indicated that it intends to treat all State regulatory programs pertaining to uranium mill tailings as automatically terminated on November 8, 1981, unless the States adopt regulations patterned on NRC's standards and requirements before that date.

It now appears that most States—including Colorado, Texas, and New Mexico—will be unable to meet this date. Termination of these State programs will cause totally unnecessary regulatory confusion and will be an unwarranted blow to Federal-State cooperation in the regulation of atomic energy as provided in section 274 of the Atomic Energy Act. The suspension language, as modified by the Appropriations Committee, makes clear that Congress did not intend any automatic termination of agreement State programs on November 8. Because of this imminent date, it is essential that we act immediately to suspend NRC's uranium mill licensing requirements. I therefore support the Appropriations bill as reported by the committee on this subject.

UP AMENDMENT NO. 581

Mr. PRYOR. Will the Senator from Oregon yield to me?

Mr. HATFIELD. Does the Senator wish to offer an amendment?

Mr. PRYOR. I say to the distinguished floor manager, Mr. President, I should like to ask unanimous consent to make a correction to an amendment that Senator BUMPERS and I introduced yesterday and the Senator from Oregon and the Senator from Louisiana so graciously accepted. It is a one-word correction, not an amendment.

Mr. HATFIELD. Mr. President, we have discussed this matter. I yield to the Senator to describe it. It is perfectly satisfactory to the managers of the bill.

Mr. PRYOR. Mr. President, this amendment basically relates to the Ouachita River navigation project. UP amendment 581, accepted by the Senate yesterday afternoon. I ask unanimous consent that one word be changed from "authorization" to "appropriations" in the amendment. I understand both sides of the aisle are in agreement with the amendment.

The PRESIDING OFFICER. Is there objection? Without objection, it is so ordered.

So the amendment (UP No. 581) reads as follows:

UP AMENDMENT 581

(Purpose: To provide that no funds may be used to construct channel realignment work on the Ouachita and Black Rivers navigation project until a restudy is made)

On page 8 following line 23 add the following new section:

Section 107—No funds appropriated in this Act may be used to construct channel realignment work on the Ouachita and Black Rivers navigation project in Arkansas and Louisiana until such time as the Chief of Engineers has completed a restudy of the various options for navigation above Crossett, Arkansas, including the two barge abreast configuration, with a view toward reducing the number of cutoffs and bend-widenings required. The results of this restudy should be reported to the respective appropriations committees of both houses of the Congress for review, and should accurately reflect the economic and environmental tradeoffs of providing greater than two-barge navigation.

UP AMENDMENT NO. 582

Mr. DOMENICI. Mr. President, I have a technical amendment with reference to the Nuclear Regulatory Commission language in this bill. It changes two

words in the amendment which are improper. I have checked with the floor manager and the minority manager, and they are willing to accept it. I send the amendment to the desk.

The PRESIDING OFFICER. Do the managers of the bill set aside the committee amendment for the purpose of considering the amendment of the Senator from New Mexico?

Mr. JOHNSTON. I ask unanimous consent that that be done.

The PRESIDING OFFICER. Without objection, it is so ordered.

The amendment will be stated.

The bill clerk read as follows:

The Senator from New Mexico (Mr. DOMENICI) proposes an unprinted amendment numbered 592:

On page 34, lines 11 and 12, strike "reinstate NRC" and insert in lieu thereof "exercise any".

Mr. DOMENICI. Mr. President, this amendment would make a technical correction to the provision now in the bill to reflect that any action by NRC to assert regulatory authority over uranium mills and milltailings in a State that has acted to exercise regulatory authority over these activities under State law would be a new exercise of authority rather than a reinstatement of any previous authority exercised by the Commission. Thus, the amendment simply provides the correct terminology to make it clear that NRC will not assert any regulatory authority over uranium mills and milltailings in a State that has elected to exercise regulatory authority over uranium mills and milltailings during the period in which the NRC regulations are suspended. During this period, the State's authority under State law to regulate these activities will remain in effect and the State will not be required to adopt requirements comparable to the suspended NRC regulations in order to continue to exercise this authority.

Mr. HATFIELD. Mr. President, this is a technical amendment. It has been checked by the staff, and we have no objection to it.

The PRESIDING OFFICER. The question is on agreeing to the amendment.

The amendment (UP No. 592) was agreed to.

Mr. DOMENICI. Mr. President, I move to reconsider the vote by which the amendment was agreed to.

Mr. HATFIELD. I move to lay that motion on the table.

The motion to lay on the table was agreed to.

Mr. DOMENICI. I thank the distinguished managers of the bill.

Mr. HATFIELD. Mr. President, I believe we have one more amendment, to be offered by the Senator from Oklahoma.

The PRESIDING OFFICER. Does the Senator ask unanimous consent to set aside the pending amendment?

Mr. JOHNSTON. I ask unanimous consent.

The PRESIDING OFFICER. Without objection, it is so ordered.

UP AMENDMENT NO. 593

Mr. BOREN. Mr. President, I send an amendment to the desk.

The PRESIDING OFFICER. The amendment will be stated.

The bill clerk read as follows:

The Senator from Oklahoma (Mr. BOREN) proposes an unprinted amendment numbered 593.

Mr. BOREN. Mr. President, I ask unanimous consent that reading of the amendment be dispensed with.

The PRESIDING OFFICER. Without objection, it is so ordered.

The amendment is as follows:

On page 17, line 20, beginning with "Appropriations" strike all that follows through "Project" on page 18, line 7, and insert in lieu thereof the following:

"Appropriations in this title shall be available for acquisition of land for the McGee Creek Project, Oklahoma; provided that land required for the dam, dike, and any other authorized permanent features shall be acquired in fee title (surface and minerals); provided further, that mineral and subsurface interests shall be acquired by subordination in the conservation pool area of the reservoir, natural scenic recreation area and the wildlife management area in such a manner as to allow the present mineral owners, their successors and assignees the right to explore for and extract minerals under restrictions required to protect the project; provided further, that only the surface estate be acquired for any other lands required for the McGee Creek Project."

Mr. BOREN. Mr. President, this is a noncontroversial technical amendment which I understand has been cleared with the majority and the minority.

The amendment simply clarifies language in the committee amendment to make it clear that the mineral development will proceed in a manner that will also protect the wildlife management area as well as the conservation pool and the wilderness area. The measure would subordinate the mineral interests to those purposes. It would allow the mineral ownership to continue, but it subordinates use and development of those minerals to methods that would be in keeping with the preservation of the purposes of those areas.

Mr. HATFIELD. Mr. President, the Senator has stated the situation correctly. We have checked this, and it is a local matter relating to the State of Oklahoma. The approach the Senator has made in this amendment is satisfactory.

Mr. JOHNSTON. Mr. President, we are willing to accept the amendment.

The PRESIDING OFFICER. The question is on agreeing to the amendment.

The amendment (UP No. 593) was agreed to.

Mr. HATFIELD. Mr. President, I move to reconsider the vote by which the amendment was agreed to.

Mr. BOREN. I move to lay that motion on the table.

The motion to lay on the table was agreed to.

USER-COUPLED CONFIRMATION DRILLING PROGRAM

● Mr. CANNON. Mr. President, I wonder if the distinguished floor manager of the bill would yield for a question or two.

Mr. HATFIELD. I am happy to yield to my good friend, the senior Senator from Nevada.

Mr. CANNON. I thank the Senator.

It is my understanding from reading the committee's report on the pending legislation that $2.7 million was included in the committee amendment for the two remaining geothermal energy projects under the Department of Energy's user-coupled confirmation drilling program.

Mr. HATFIELD. The Senator is correct. Some $2.7 million was included in our amendment for reservoir confirmation drilling work for the final two user-coupled projects—a space heating and cooling project in Reno, Nev., and an alcohol fuel plant at Vale Hot Springs in Oregon.

Mr. CANNON. Could the Senator explain, for the benefit of our colleagues, how the user-coupled program works?

Mr. HATFIELD. I will be happy to do that.

The user-coupled confirmation drilling program is an innovative public-private sector cooperative effort. It was started by the DOE to encourage geothermal developers to sink wells into promising geothermal areas in order to verify the presence of economically recoverable geothermal resources for nonelectrical applications.

To obtain funding, a geothermal developer must conduct appropriate exploration activities and pay for drilling the confirmation wells. Once completed and tested, if the wells are as successful as anticipated, DOE will reimburse the developer for 20 percent of the drilling and associated costs.

If the wells are less than a complete success, the Federal commitment increases on a sliding scale. The maximum Federal contribution for a totally unsuccessful project has been set at 90 percent in the past.

Mr. CANNON. The Senator said that "in the past" the maximum Federal commitment was 90 percent of the costs associated with a completely unsuccessful project. Does the present legislation change that upper limit?

Mr. HATFIELD. It would under certain circumstances. Because of other pressing budgetary priorities, the total Federal contribution for the two remaining projects is limited to $2.7 million. That means that the maximum available for either project, in the unlikely situation where both fail, would be approximately 78 percent. In other situations, the upper bound would still be set at 90 percent.

I should emphasize, however, that the Energy Department has carefully limited participation in the program to experienced companies with promising geologic sites. The final Federal expenditure for these two projects should be a lot less than the $2.7 million amount.

Mr. CANNON. I believe that the user-coupled program is a good way to stimulate the direct-heat utilization of geothermal resources with a minimal expenditure of Federal funds.

Mr. HATFIELD. I agree with the Senator. I look forward to seeing the administration move ahead promptly with the remaining two projects.●

Mr. McCLURE. Mr. President, I was pleased to note the inclusion on page 70

dent, will the Senator from Oregon yield?

Mr. HATFIELD. I yield.

Mr. HARRY F. BYRD, JR. As I understand it, the bill as it now stands is approximately $700 million——

Mr. HATFIELD. Mr. President, may we have order, so that I can hear the Senator?

The PRESIDING OFFICER. The Senate will be in order.

Mr. HARRY F. BYRD, JR. As I understand it, the bill as it now stands is approximately $700 million over the President's request.

Mr. HATFIELD. The precise figure is $632 million over the President's request; $309 million of that is the amendment adopted by the Senate, by a vote. The other was that adopted by the committee.

Mr. HARRY F. BYRD, JR. So that the total exceeds $600 million.

Mr. HATFIELD. It is $632 million.

Mr. HARRY F. BYRD, JR. I thank the Senator.

Mr. DOLE. Mr. President, I rise today in support of the committee's recommendations for energy and water appropriations in fiscal year 1982. This bill does an excellent job of allocating our scarce Federal resources and I applaud the members of the committee for the many difficult decisions they made to reduce Federal spending.

Mr. President, it is not my intent to attempt to alter the numbers in the Senate bill. I do, however, want to express my concern that an unintentional injustice may occur as a result of efforts by both Houses to reduce spending and appropriate money in a responsible manner. Specifically, I am referring to the $50,000 appropriated in the House-passed version of H.R. 4144 for study at the El Dorado Reservoir in El Dorado, Kans., an appropriation which is not included in the Senate bill.

I do not believe that it was anyone's intention to eliminate funding for this badly needed study of alternative access routes to the reservoir. The appropriation included in the House bill was achieved by reducing the construction account for the El Dorado Reservoir by $50,000 and transferring those funds to a general investigation account for study at the El Dorado Reservoir. In so doing, the House did not exceed the President's budget request for the El Dorado Reservoir as a whole. The transfer of funds was, in my mind, a very responsible action on the part of the other body, satisfying the concerns of the area's residents and the administration.

Unfortunately, the Appropriations Committee in the Senate correctly noted that the $50,000 appropriation for study was not requested by the administration and consequently deleted it from the bill. In almost all instances, I would support that action. However, in this case, the deleted appropriation was achieved by an earlier reduction in a companion account by the House.

By accepting the administration's zero appropriation we have in this bill eliminated a very valuable study. A study that the residents of the area, the Corps of Engineers and the House of Representatives felt was more worthwhile than an additional $50,000 for construction as requested by the administration. A study which does not do violence to the original budget request.

Mr. President, I make these points because I want to encourage the Senate conferees to accept the House figure in conference. The conferees will face many, more difficult, and much more costly issues when they reach the conference with the House. I would hope that they will see their way clear to accept this relatively minor item and restore a badly needed study that was consciously funded, in a responsible manner, by the House.

AUTOMATIC TERMINATION

● Mr. SCHMITT. Mr. President, this bill contains language which I sponsored barring NRC from implementing its uranium mill licensing requirements until EPA issues pertinent standards. This language is well-explained in the conference report. One point, however, merits some amplification. Because of the necessary delays between final passage of this bill, the House-Senate conference and signature by the President, it is unlikely that the November 8 deadline will be met.

Advocates of needless and mindless regulation should not take advantage of the necessary delay to thwart the will of Congress. Amended agreements will not have been entered by New Mexico, Colorado, Texas, and several other uranium-producing States by November 8.

The Senate does not wish the NRC to reassert jurisdiction on November 9. This not only will disrupt State regulatory programs, hamper the scheme of Federal-State cooperation envisioned under section 274 of the Atomic Energy Act, and cause unwarranted regulatory confusion, but also is contrary to the intent of Congress.

Neither the Mill Tailings Act nor its transitional provisions confer authority on NRC to automatically terminate all or any part of a discontinuance agreement. A discontinuance agreement may only be terminated as provided under section 274(j) of the Atomic Energy Act.

That section provides that NRC may terminate an agreement only upon request of the Governor of the State or after reasonable notice and an opportunity for a hearing.

The language adopted by the Appropriations Committee and now the full Senate and the comparable House prohibition reaffirms this intent and thus prevents NRC from unnecessarily terminating valid and successful State regulatory programs. ●

Mr. HATFIELD. Mr. President, I know of no other amendments. Therefore, I request third reading.

Mr. DOMENICI. Mr. President, I have no amendments, but will the Senator yield 30 seconds?

Mr. HATFIELD. I yield.

The PRESIDING OFFICER. The question is on the engrossment of the amendments and the third reading of the bill.

The amendments were ordered to be engrossed and the bill to be read a third time.

The bill was read the third time.

Mr. HATFIELD. Mr. President, I ask for the yeas and nays.

The PRESIDING OFFICER. Is there a sufficient second? There is a sufficient second.

The yeas and nays were ordered.

The PRESIDING OFFICER. The bill having been read the third time, the question is, Shall it pass? On this question the yeas and nays have been ordered, and the clerk will call the roll.

The assistant legislative clerk called the roll.

Mr. BAKER. I announce that the Senator from New York (Mr. D'AMATO), the Senator from Arizona (Mr. GOLDWATER) and the Senator from Utah (Mr. HATCH) are necessarily absent.

I also announce that the Senator from Alaska (Mr. STEVENS) is absent due to death in the family.

I further announce that, if present and voting, the Senator from New York (Mr. D'AMATO) would vote "yea."

Mr. CRANSTON. I announce that the Senator from Nevada (Mr. CANNON), the Senator from New York (Mr. MOYNIHAN), the Senator from New Jersey (Mr. BRADLEY) are necessarily absent.

I further announce that, if present and voting, the Senator from Nevada (Mr. CANNON) would vote "yea."

The PRESIDING OFFICER (Mr. SPECTER). Are there any other Senators in the Chamber wishing to vote?

The result was announced—yeas 71, nays 22, as follows:

[Rollcall Vote No. 360 Leg.]

YEAS—71

Abdnor	Glenn	Murkowski
Andrews	Gorton	Nunn
Baker	Hart	Packwood
Bentsen	Hatfield	Pressler
Boren	Hawkins	Pryor
Boschwitz	Hayakawa	Quayle
Burdick	Hecht	Randolph
Byrd, Robert C.	Heinz	Rudman
Chafee	Hollings	Sasser
Chiles	Huddleston	Schmitt
Cochran	Inouye	Simpson
Cohen	Jackson	Specter
Cranston	Jepsen	Stafford
Danforth	Johnston	Stennis
DeConcini	Kassebaum	Symms
Denton	Kasten	Thurmond
Dodd	Laxalt	Tower
Dole	Levin	Tsongas
Domenici	Long	Wallop
Durenberger	Lugar	Warner
East	Matsunaga	Weicker
Exon	Mattingly	Williams
Ford	McClure	Zorinsky
Garn	Melcher	

NAYS—22

Armstrong	Grassley	Nickles
Baucus	Helms	Pell
Biden	Humphrey	Percy
Bumpers	Kennedy	Proxmire
Byrd, Harry F., Jr.	Leahy	Riegle
	Mathias	Roth
Dixon	Metzenbaum	Sarbanes
Eagleton	Mitchell	

NOT VOTING—7

Bradley	Goldwater	Stevens
Cannon	Hatch	
D'Amato	Moynihan	

So the bill (H.R. 4144), as amended, was passed.

Mr. HATFIELD. Mr. President, I move to reconsider the vote by which this bill was passed.

Mr. HAYAKAWA. I move to lay that motion on the table.

The motion to lay on the table was agreed to.

Mr. HATFIELD. Mr. President, I move that the Senate insist on its amendments and request a conference with the House

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roast beef or chicken breast) is $12.50 per person, including tax and gratuities. Tickets and further information are available from Eileen Reilley at ACS headquarters, 1155—16th St., N.W., Washington, D.C. 20036. Make checks payable to ACS.

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Letters

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U.S. Interagency Toxic Substances Data Committee (ITSDC), and at meetings of its CSIN and Public Liaison Subcommittees. To scope out the complexity of tasks associated with identifying the quality of data, Network Administration has invited experts from the Office of Standard Reference Data of the National Bureau of Standards and the Federation of American Societies for Experimental Biology to discuss problems associated with establishing the quality of chemical and biological data with the CSIN Subcommittee. A similar discussion is planned for the next ITSDC meeting in October.

Even these preliminary efforts have made it apparent that identifying the quality of data is a time-consuming, costly, and complex operation. Through its involvement on issues concerning data quality, Network Administration is encouraging the use of existing "mechanisms of review," as well as identifying the need for new or complementary review activities and, as appropriate, seeking the advice of owners of information resources both in the public and private sector. The problem is best addressed through joint or cooperative efforts of many communities.

It is also appropriate to mention that while the Office of Network Administration presently resides in the Environmental Protection Agency, CSIN is a project that requires and does solicit involvement from interested communities in industry, academia, and government (federal and state).

Sidney Siegel
Administrator, Chemical Substances Information Network, Office of Toxics Integration, EPA

Cancer and consensus

CR: Some observations by John Higginson of the International Agency for Research on Cancer (IARC) ("Cancer Research Priorities," C&EN, June 29, page 2) need clarification.

He distinguishes between (1) "causal factors that have been identified with considerable certainty," and (2) whether "environmental etiology could only be inferred as the most rational" epidemiological interpretation. Epidemiological studies usually are based on statistical associations. Does Higginson imply that any "most rational" functional interpretation of an association is factual, unbiased, and scientifically valid? If so, it is questionable, since it is a basic principle of statistics that functional causality *cannot* be inferred from statistical association alone. Properly used, statistics can *reject* a hypothesis to the extent that it is incompatible with observed data; but statistics can *never* establish that a hypothesis is certainly true [Fisher, R. A., "Statistiscal Tests," *Nature*, 136, 474 (1935); see also Hickey, R. J., and colleagues, "Chemicals and Cancer," C&EN, June 22, page 4.]

A confirmed statistical association in an epidemiological study requires setting forth testable alternative functional (biological, biochemical) hypotheses for experimental confirmation or rejection. Some popular causal beliefs have been based on statistical associations and subjective judgment, with allegations that the association has been adequately "explained."

An example of an epidemiological association for which functional causality has been demonstrated involved human occupational exposure to certain chloromethyl ethers, particularly bis(chloromethyl) ether [Figueroa, W. G., Razkowski, R., Weiss, W., "Lung Cancer in Chloromethyl Ether Workers," *New Engl. J. Med.*, 288, 1096 (1973)]. Subsequently, animal studies [Kuschner, M., Laskin, S., Drew, R. T., Cappiello, V., Nelson, N., "Inhalation Carcinogenicity of Alpha Halo Ethers. III. Lifetime and Limited Period Inhalation Studies with Bis(Chloromethyl) Ether at 0.1 PPM," *Arch. Environ. Health*, 30, 73 (1975)] demonstrated that bis(chloromethyl) ether is in fact a rapidly acting carcinogen that can induce lung cancer. In this case, regulatory action apparently was warranted. However, there are associations of unclear, obscure, and unproven etiologies.

It is curious that Higginson asserts that "there is no evidence that the vast majority of tumors [cancers?] are related to diffuse chemical pollution in the ambient environment. . . ." This is the same error of fact encountered by John A. Todhunter in his letter (C&EN, Feb. 23, page 4), to which we responded (see above, C&EN, June 22, page 4). Other studies also have reported statistically significant, multiregional epidemiological results involving "diffuse," multichemical data [(a) Hickey, R. J., "Air Pollution," Chapter 9, page 189, in "Environment: Resources, Pollution & Society," W. W. Murdoch, editor, Sinauer Associates Inc., Stamford, Conn., 1971], and also involving low-level, ambient, ionizing radiation in addition to air pollutant chemicals [(b) Hickey, R. J., Bowers, E. J., Spence, D. E., Zamel, B. S., Cleiland, A. B., Cleiland, R. C., "Low-Level Ionizing Radiation and Human Mortality: Multi-Regional Epidemiological Studies. A Preliminary Report," *Health Phys.*, 40, 525 (1981)].

The finding of significant negative associations between ecologically realistic, ambient radiation levels and mortality rates for several classifications of cancer, including cancer of the respiratory organs, is contrary to many conventional causal claims and beliefs. However, claims that any and all levels of ionizing radition are carcinogenic or otherwise hazardous are based largely on linear or other monotonic extrapolation of dose-response data obtained at ecologically unrealistic, very high doses, or dose rates, downward toward zero exposure, through the ecologically realistic, low-level exposure

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zone for which effects rarely are measured reliably. From such extrapolation, widespread beliefs exist that all levels of ionizing radiation is hazardous to health, and that there is no threshold level below which radiation is harmless [Radford, E. P., "Health Effects of Low Doses of Ionizing Radiation: The BEIR III Controversy," *Radiation Res.*, 84, 369 (1980)].

To the contrary, ecologically realistic, low-level radiation is biologically stimulatory, and presumably beneficial [Luckey, T. D., "Hormesis with Ionizing Radiation," CRC Press, Boca Raton, Fla., 1980]. But extrapolation is subjective and unreliable, as some textbooks of statistics warn, and extrapolated "data" may be false. Consider extrapolation of high-level exposure dose-response data for copper downward toward zero exposure, through the nutritionally essential range!

Higginson, referring to radiation, cigarette smoking, and other presumed hazards, without considering exposure level, or hormesis, or behavioral physiology, appears to rely on "reasonable consensus regarding public health strategy," such as for "control of cigarettes." "Reasonable consensus" implies majority opinion and belief, possibly biased, in claiming "facts" that may be unproven. Facts or truth are not reliably determined by that "many doctors believe." As noted in (a), the Surgeon General's report of 1964 states (page 182): "The causal significance of an association is a matter of judgment which goes beyond any statement of statistical probability." This is opinion, not evidence or science. It is an example of abuse of science by a federal agency.

The "consensus" fallacy exists today in large part through misuse of statistics, such as through inference of causality from statistical association, through acceptance of a linear or other extrapolation model as a scientifically acceptable method for deducing effects "data," and by use of nonrandom samples with the presumption that they are not biased. Consider that sample populations of high-blood-glucose, low-insulin individuals are biased (diabetics). Samples of habitual smokers and of habitual nonsmokers are nonrandom and very likely biased, considering the reality of behavior-genetic heterogeneity of human populations [e.g., Hirsch, J., "Behavior-Genetic Analysis," McGraw-Hill Book Co., New York, 1967]. An assumption that populations are genetically homogeneous is an example of the fallacy of typology.

Regarding Higginson's reference to "diffuse chemical pollution," analyses involving data for specific chemicals have been performed through the use of methods of multivariate statistical analysis [see (a) and (b)]. Regarding this complex problem, Th. Dobzhansky ["Proceedings of the 5th Berkeley Symposium on Mathematical Statistics and Probability," Vol. IV, L. M. LeCam and J. Neyman, editors, University of California Press, Berkeley, page 303, 1967] has advised: "The abandonment of the assumption of uniform environments makes . . . mathematical models uncomfortably complex. Dealing with this complexity, however, is a compelling necessity if we are to understand the biological reality."

Richard J. Hickey
Department of Statistics, Wharton School, University of Pennsylvania, Philadelphia

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Hormosis

Unlike the rainbow, which can be viewed from only one side, ionizing radiation is harmful in large doses, but appears to be beneficial when administered in minute doses. If radiation were found to be essential, the arch of the rainbow would resemble the complete dose-response curve for ionizing radiation, as it does for other essential elements of our environment: light, heat, oxygen, water, or nutrient. Too much is harmful; too little leads to a deficiency; only the optimum amount allows full development and health.

Sagan (*NN*, Oct. 1981, pp. 80–82) apparently prefers either the threshhold or the no-threshhold concept of the dose response curve to represent the effects of low doses of ionizing radiation extrapolated from high doses (Fig. 1A) and chooses to ignore the paradoxically opposite data taken at low doses (Fig. 1B). The following paragraph summarizes Chapter 2 of my work, *Hormesis with Ionizing Radiation* (CRC Press, Boca Raton, 1980), which documents information that allowed the prediction that results from ionizing radiation would both conform to and confirm the thesis of hormesis.

Hormesis, a term coined by Southam and Ehrlich in 1943, is the stimulation of any system by subharmful amounts of any agent. This was expressed as the 1888 Arndt-Schultz law and dropped from U.S. pharmacology texts in the late 1930s. My perception of hormesis began in 1945 with the dietary antibiotic stimulation of animal growth; the general thesis of hormesis was proposed in 1956 using the name *hormoligosos* from the Greek *hormo*, I excite, and *oligo*, small amounts. A 1969 literature review is available in "Antibiotics in Nutrition," pp. 179–321, in H. S. Goldberg, ed., *Antibiotics, Their Chemistry and Non-Medical Uses* (D. Van Nostrand Co., Inc., Princeton). In order to test the validity of the thesis, the results of a series of experiments was predicted. I.

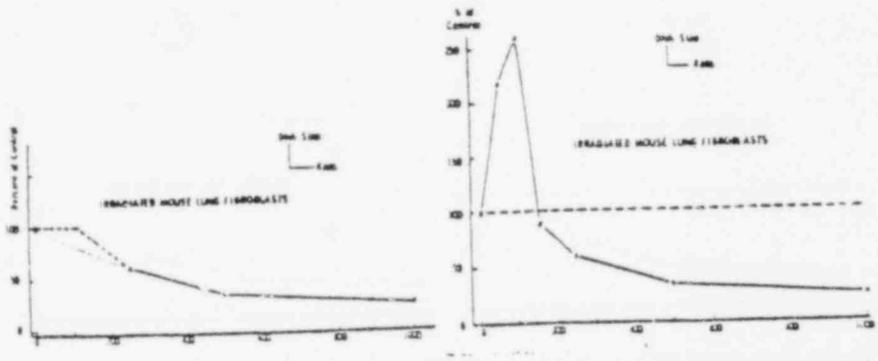

Fig. 1A (left). Viscoelastic analysis of the molecular weight of DNA taken from control and irradiated mice (Uhlenhop, E. L., *Biophysics Journal*, 15: 233–237, 1975). The data fit either a threshold or a no-threshold concept. Fig. 1B (right). More complete examination of the dose-response curve of Fig. 1A showed a distinct reversal of the effect when high and low doses were used.

Vinegar eels grew faster ($p = 0.001$*) in the presence of each of four antibiotics. 2. Five different antibiotics greatly improved the survival of vinegar eels subjected to severe heat stress ($p = 0.01$–0.001), sometimes 100 percent of the treated eels survived while only 60 percent of the controls survived. This experiment was performed with a species never before used experimentally and involved a parameter, heat stress, which had never before been studied with antibiotics. 3. Antibiotics in bacterial medium caused superinduction of enzyme synthesis. 4. Increased growth of crickets was noted when 12 insecticides were fed individually at different concentrations; the data reached statistical significance ($p = 0.001$) with 10 of these. 5. Chick growth was increased ($p = 0.01$) when either germanium oxide or rutin was fed at appropriate concentrations. 6. Bacterial vegetation was increased by relatively low doses of ionizing radiation. 7. The growth increment noted in mice exposed to minute daily doses of x rays was confirmed ($p = 0.01$). The success of these predictions, combined with the extensive literature, was convincing evidence that the hormesis thesis was valid generally.

Hormesis with Ionizing Radiation provides much evidence that low-level ionizing radiation conforms to the thesis. Experiments with plants were most numerous, about 800 references, with many showing statistical significance and excellent reproducibility—i.e., experiments with cloned strawberry plants by the group at Hannover. The animal data are less extensive, about 200 references were summarized. Few investigators knew of the work of others; there was little systematic work; and fragmented or little support was available. The frequency of the observed beneficial action of ionizing radiation for any physiologic parameter measured in a great diversity of phyla must be considered when interest centers upon a single species or function for which there is little or no objective data—e.g., man. Sagan's question about the effects of low doses of ionizing radiation should be thoroughly examined with funds from both public and private sources.

Results from published animal experiments indicate that low-level ionizing radiation is beneficial for growth and development, fecundity, resistance to subsequent harmful doses of radiation, resistance to infection, resistance to cancer induction, and increased average lifespan for a variety of species. Without using unusual numbers of animals, one should program several doses in the range of 10 to 1000 times ambient radiation. Realistic experimental design might include the effect

*Probability is 999/1000 that the differences in the results between experimental and control groups were significant.

of ionizing radiation upon animals subsequently challenged with carcinogens or bacteria. Different types of experiments suggest the value of low doses of ionizing radiation in delaying and/or decreasing cancer induction. In 1915, Murphy and Morton (*Journal of Experimental Medicine*, 22:800) excised spontaneous tumors from mice and irradiated animals of one group before inserting each tumor into its original host. Tumor resorption occurred in 50 percent of the irradiated animals and 3.4 percent of the controls. Subsequently, fourfold fewer new spontaneous tumors were palpated in the experimental animals than were found in the unirradiated controls. Several reports indicate that methylcholanthrene-induced tumors were delayed in appearance and/or were fewer in number in irradiated mice than in controls. These and epidemiologic studies on humans are included in my review titled "Physiologic Benefits from Ionizing Radiation" (*Health Physics*, in press).

A number of recent epidemiologic studies suggest a negative correlation between human cancer and cancer deaths with ambient levels of ionizing radiation. The investigators could not find valid correlations to other environmental factors or social or economic status. Compatible with the correlations noted in both epidemiologic and experimental studies is the suggestion that higher levels of ambient radiation contribute positively to health.

Background ionizing radiation levels could help to explain enigmas and paradoxes in epidemiology. For example, the high death rates in the eastern U.S. coastal plains correlate well with chronic diseases but no factors have been identified to explain "the enigma of the Southeast." The average terrestrial ionizing radiation of the Southeast coastal area is 23 mrem/yr, an amount which is about one-half of the average for the United States and only about one-fourth of that received in the Colorado plateau, where cancer deaths and death rates from chronic diseases are lower than the average for the country. This negative correlation is particularly valid for areas of the low, average, and high terrestrial radiation compared with the death rates for cardiovascular diseases for white males, 35–74 years of age. Ionizing radiation levels could help one to understand this enigma.

Another example is taken from the opposite end of the dosage spectrum, the "paradox of Kerala." The people of Kerala are reported to have the highest literacy rate and the best health status in India; yet their expenditure on health care is not appreciably above average, and they have the lowest food intake and the least adequate diets of people of all states of India. A partial

explanation for this paradox is that radiation from the unusually high radium and thorium in coastal and river rocks exposes many residents of Kerala to 10 times more terrestrial radiation than the U.S. average. Similarly, the health spas of Europe and Brazil are found to be, and openly advertise, high radioactive areas.

The real meaning of data showing that ionizing radiation stimulates physiologic functions or correlates with certain parameters of health can become evident only when the sub-ambient component of the dose-response curve is defined. Might a dramatic deficiency develop in environments that were substantially lower in ionizing radiation than we presently enjoy? We need to know. Viability was poor in "radiation deficient" Artemia barley and algae (*Aerospace Medicine* 35:524, 1964). Pioneering experiments of Planel and his group over the past decade (IAEA Report #134/17, Vienna, 1979) suggest that lowering radiation below ambient levels reduced the rate of growth (reproduction) in protozoa. Our laboratory has verified this result. Rigorous experiments must be performed with human cell cultures, insect colonies, and a mammalian species through several generations maintained in an environment where both endogenous and exogenous ionizing radiation is substantially reduced.

If the results from such experiments indicated that a radiation deficiency is produced, then radiation should be considered to be essential for life. The data reviewed as radiation hormesis would then provide evidence that most ambient levels of ionizing radiation are sub-optimal. If ionizing radiation were essential and if our environments were deficient, as indicated by present information, then appropriate supplementation would allow mankind to reach a new plateau of health.

T. D. Luckey
Professor of Biochemistry
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Columbia, Mo.

•My letter, "Hormesis," published in NUCLEAR NEWS, Nov., 1981, p. 18, is perhaps rather terse for so complex and controversial a topic. Also, another publication has appeared that pertains to the problem of estimation of health effects of ecologically realistic low-levels of ionizing radiation.

The following report has been published: *Uranium Ore Residues: Potential Hazards and Disposition*, Hearings before the Procurement and Military Nuclear Systems Subcommittee of the Committee on Armed Services, U.S. House of Representatives, 97th Congress, 1st Session, June 24 and 25, 1981, Washington, D.C. (H.A.S.C. No. 97-14), 550 pages.

Involved are legal actions or lawsuits. These pertain to requests for relief from what some consider over-regulation or unjustified regulation and restriction regarding the safety and handling of uranium ore residues. The American Mining Congress (AMC) seeks relief from certain actions of the U.S. Environmental Protection Agency (EPA) (pp. 254–288) and of the U.S. Nuclear Regulatory Commission (NRC) (e.g., pp. 527–545). On p. 527, an NRC letter refers to a ". . . lawsuit filed by the American Mining Congress."

AMC is a consortium of about 40 commercial organizations that includes United States Steel Corp., Bethlehem Steel Corp., International Minerals and Chemical Corp., Westmoreland Coal Co., Ingersoll-Rand Co., Union Carbide Corp., Cities Service Co., The Anaconda Co., Conoco, Inc., Homestake Mining Co., and others, identified on p. 289 of the Hearings.

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That very high levels of ionizing radiation in single or continued expo-

sures are hazardous to health is not in question. The problem and controversy pertains to biological effects of *low-level* radiation, such as ecologically realistic background radiation which is, and presumably has been, ubiquitous over the millenia of biological evolution. Natural low-level radiation arises mostly from radioactive isotopes, or radionuclides, of geological origin and from cosmic rays. Some additional deposits have originated from human activities such as military test explosions. There is also diagnostic X irradiation. Also, traces of radionuclides are absorbed from natural food and drink, and contribute to total radiation exposure. However, DNA-repair and other repair mechanisms exist and therefore, evidently, evolved.

Dr. T. D. Luckey, long a student of chemical and radiation hormesis, has defined hormesis as beneficial stimulation of biological organisms by subharmful quantities or levels of any agent to any biological system (*Hormesis with Ionizing Radiation*, CRC Press, Boca Raton, Fla., 1980). Activation of natural physiological homeostatic mechanisms is likely partly involved. Since life forms evolved in the presence of environmental chemicals and other potential hazards, as in seawater, the life forms presumably adapted to ecologically realistic levels of the hazards. The scientific problem, encountered by some, is the assumption or belief that because some variable, such as ionizing radiation, is hazardous at high levels, it must be hazardous at all levels. This is the extrapolation error or fallacy. High levels of insulin and copper are toxic but low levels are essential to optimum health and to survival.

Regarding the controversial question of "essentiality" of radiation, Luckey and colleagues [see (Lu78), cited in my letter, *NN*, Nov. 1981, p. 18] investigated effects of subambient radiation on survival and multiplication of *paramecium bursaria*. They found that populations of cells/ml were much lower under lead-shielded, subambient radiation conditions than under normal background control conditions. Comparable experimental evidence regarding health and survival of small animals, such as mice, raised for their lifetimes under subambient radiation conditions, as compared with normal controls, would be of great value toward resolving the controversy of beneficial effects of low-level ionizing radiation. Such studies, which Luckey and colleagues wish to perform, had not led to necessary funding as of last April (*Nucleonics Week*, April 30, 1981, p. 9).

While high-level radiation data are generally positively correlated with detrimental effects, comparable low-level data for populations have been reported to be *negatively* correlated with detrimental effects, such as cancer mortality rates (*NN*, Nov. 1981, p. 18). Further evidence is given by B. S. Sanders, "Low-Level Radiation and Cancer Deaths," *Health Phys.* 34: 521-538 (1978). Negative correlations were reported. Additional information may be found in: High Background Radiation Research Group, China, "Aspects of Environmental Radiation and Dosimetry Concerning the High Background Radiation Area in China," *J. Rad. Res.* 22: 88-100 (1981). Some negative associations are reported. Such findings, considering the various reports showing negative associations, necessitate a U-shaped dose-response model.

Regarding those who claim that because radiation is damaging at high exposure levels it is damaging at all levels (a version of the extrapolation position), it seems necessary that they justify and validate extrapolation as an acceptable and valid scientific method. This is a statistical problem concerning which caveats exist. Extrapolation to yield "data" estimates might be designated as a method of "creative statistics."

Richard J. Hickey

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LOW LEVEL IONIZING RADIATION AND HUMAN MORTALITY: MULTI-REGIONAL EPIDEMIOLOGICAL STUDIES

A PRELIMINARY REPORT

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Abstract—Analyses of relationships involving environmental chemicals, background radiation, and mortality rates for diseases of the heart and several cancer categories are presented. Bivariate correlation coefficients between radiation and mortality rate were significantly negative for cancer of the lung and respiratory organs, cancer of the buccal cavity and pharynx, cancer of the digestive organs and peritoneum, total cancer and diseases of the heart. Only the correlation coefficient between background radiation and leukemia–aleukemia was positive, and that not significantly positive. Initial multivariate statistical studies support the bivariate results in that all significant regression terms that represent background radiation have been negative. Background radiation level was not a significant predictor for leukemia–aleukemia mortality rate when examined without regard to age. This preliminary work suggests that any health effects of background radiation on the diseases studied do not exceed in magnitude those of environmental chemicals. It also suggests that models implying important long-term deleterious effects of low levels of ionizing radiation on humans may be invalid.

INTRODUCTION

A MAJOR controversy exists regarding the health effects of low levels of ionizing radiation. At least two kinds of explanatory models have been proposed. One class of models assumes that all levels of ionizing radiation, no matter how low, are hazardous to health and that the detrimental effects tend to be an increasing function of dose or exposure (Ad79; Ba73; BEIR72; BEIR77; BEIR79; Bow80, Ga80; Gof72; Ke79; Mo78; Na79; Ster72). The other class of models proposes that human populations have been, and continue to be, subjected to low levels of environmental ionizing radiation so that adaptive mechanisms have evolved which cope with ambient radiation. It proposes further that, although ionizing radiation is damaging at elevated levels and rates of exposure, there is a level of radiation exposure below which health effects at the level of the organism are negligible or beneficial (Car57; Car59; Cas68; Cr50; Cl70; Fri73; Jan65; Jac76; Lor50; Lu80; Va68).

These two classes of models have very different implications for public health policy. Particularly important in influencing policy in this area have been reports produced by committees of the National Research Council of the U.S. National Academy of Sciences.

These committees on the "Biological Effects of Ionizing Radiations" (BEIR) have published reports BEIR-I (BEIR72) and BEIR-II (BEIR77). The report of a third committee (BEIR79) is in the process of being revised for publication (Bow80). BEIR-III was to have been published in 1979, but serious factional conflict has resulted in delay of issuance. A press report (Bow80) regarding BEIR-III described NAS President Dr. Philip Handler as being surprised to learn that "... there were two groups so bitterly opposed to one another on the committee". Handler also observed that "when the voting comes out so close, it tells me that the evidence being considered doesn't compel any conclusion" (Bow80, p. 3B).

Epidemiological investigations on human populations from a number of localities in different ecological regions, such as a number of metropolitan areas of the U.S., should be useful in distinguishing between these classes of models. If ionizing radiation contributes to mortality from chronic disease as an increasing function of dosage, then background levels of ionizing radiation should show positive statistical associations with mortality rates for cancers and perhaps cardiovascular diseases in these populations.

For a number of years we have been conducting studies that have examined the relationships of mortality from a variety of chronic diseases to the prevalence of various chemical pollutants in the atmosphere. A measure of water hardness and several socioeconomic predictors have been included in some of the analyses. Data were collected and analyzed for 38 Standard Metropolitan Statistical Areas (SMSAs) as is described elsewhere (Hic70a; Hic70b; Hic71a; Hic71b; Hic76). Among urban air pollutant chemicals whose concentrations were found to be significantly positively associated with mortality rates for several categories of cancer and cardiovascular disease were sulfur dioxide (SO_2), nitrogen dioxide (NO_2), and particulate sulfate ($SO_4^=$), while copper (Cu) was negatively associated.

Regarding radiation effects, Jacobson *et al.* (Jac76) have cautioned against failure to consider other environmental variables that could mask the epidemiology of radiation leukemogenesis, e.g. socioeconomic factors, mobility and ethnic variations in susceptibility. This position was reinforced by Neyman (Ney77; Ney79) who cautioned that air pollutant chemicals are among the multiplicity of environmental variables of epidemiological significance that cannot be ignored.

Of critical importance in the elucidation of information from epidemiological studies aimed at etiological understanding is the problem of competitive mortality. This phenomenon was pointed out clearly by Neyman (Ney77; Ney79). He (Ney77) advised that if, for example, one wishes to identify the causes of an "... increase in the frequency of cancer, [and if] one omits from an observational study a really important agent, then the effects of this agent will be ascribed to some other agent, possibly quite innocent" (pp. 754, 755). Numerous epidemiological studies, however, have concentrated on a single environmental pollutant, such as radioactivity (Gof72; Ste72), or sulfur dioxide (A172), or carbon monoxide (Lon77), while neglecting the synergistic interactions of, for example, atmospheric chemicals, or socioeconomic factors, or both.

Recently we have expanded our data base for the period 1957–64 by obtaining data for 43 urban regions rather than 38 and by inclusion of the additional air pollutant chemical, nitrate. Following Neyman's advice, background radiation data were located (Oa72) and incorporated. The present report is a preliminary description of early findings in the analysis of relationships among background radiation levels, environmental chemicals, and chronic disease mortality rates. These results contain information of potential relevance to current discussions of the health effects of low levels of ionizing radiations.

METHODS AND MATERIAL

The basic analytical method used here is an optimal subset selection modification of multiple regression analysis as described by Boyce *et al.* (Boy74). This computer-based procedure employs an algorithm that selects that linear function of the set of predictors

numbering $N = 1, 2, 3, \ldots, n$ that maximizes R^2, i.e. that makes the greatest joint contribution to the proportion of explained variance in mortality rate. On the first search, the computer program selects the single predictor that makes the greatest contribution to R^2; on the second search, it selects the two predictors that make the greatest combined contribution to R^2, and so on for three or more predictors. Thus, at any stage a predictor found in one set need not be included in a subsequent set. The computer used currently is the IBM S/370 model 168 MP. We have reported results of application of this regression method previously (Hic70a; Hic70b; Hic71a; Hic71b; Hic76; Hic80).

The analyses presented here examine data for 39 Standard Metropolitan Statistical Areas (SMSAs) and 4 Standard Economic Areas (SEAs) of the U.S. The predictor variables include natural logarithms (ln) of the arithmetic means of the annual concentrations of 13 air pollutant chemicals in $\mu g/m^3$ of air found between 1957 and 1964 as available (see Hic71a). It is assumed on biological grounds that the relationships between mortality rates and concentrations of environmental chemicals are non-linear (Hic76). Our empirical work suggests that the natural logarithm function gives a reasonable fit. The air pollution chemicals in question are Cd, Cr, Cu, Fe, Pb, Mn, Ni, Ti, V, NO_2, SO_2, $SO_4^=$ and NO_3^-. Also included are ln water hardness (WH) in parts per million of calcium carbonate equivalent (Du64; Loh54a; Loh54b), and background radiation levels (RA) in mrem/yr (Oa72). The predicted variables are arithmetic means of observed mortality rates (Pu60; Bur67) per hundred thousand population [Census of the U.S. 1960] for 1961–64 in the following disease categories [Seventh Revision of the International Statistical Classification of Diseases and Causes of Death (ICD) (Wo55)]:

- M_{re}: Cancer of the respiratory organs (ICD Nos. 160–164);
- M_{bu}: Cancer of the buccal cavity and pharynx (ICD Nos. 140–148);
- M_{di}: Cancer of the digestive organs and peritoneum (ICD Nos. 150–156A, 157–159);
- M_{leu}: Leukemia and aleukemia (ICD No. 204);
- M_{tc}: Total cancer (ICD Nos. 140–205);
- M_{dh}: Diseases of the heart (ICD Nos. 400–402, 410–443).

Mortality rates for these causes of death are presented in Table 1.

Observed rates are used as is appropriate in examining associations related to the effects of environmental differences on the autecology of populations of *Homo sapiens* (Hil39; Hic80). However, in later studies we expect to investigate these relationships in more detail using age- and sex-cohort analyses. Early results suggest that age adjustment does not alter the basic negative relationship between low-level radiation and respiratory tract cancer.

It has sometimes been suggested that socioeconomic variables would overwhelm any effects of differences in biological environments upon chronic disease mortality among various areas in the U.S. This has not proved to be the case in our analyses (Hic71a; Hic76). Socioeconomic and environmental variables have both contributed to explained variance in many cases. Examination of socioeconomic data is, however, beyond the scope of this preliminary report. Migration is one variable that might have a confounding effect (Hic71a; Hic76; Ki73). However, migration would have to be extensive and consist of individuals who are biologically different from non-migrants to overwhelm the effects of other variables. Another variable having possible confounding effects in studies of malignant and cardiovascular diseases is smoking. We have been unable to locate reliable data thus far on level of smoking by SMSA. We are skeptical of the estimation of urban cigarette consumption from modified state tax data (Li78) since the logic of the modifications is unclear.

BIVARIATE STATISTICAL ANALYSES

Table 2 presents correlation coefficients between mortality rates for several causes of death and observed background levels of ionizing radiation for the 43 urban populations (39 SMSAs and 4 SEAs) given in

Table 1. Mortality rates per 100,000 persons for chronic disease categories, 1961–64, and background levels of ionizing radiation, for 43 urban populations of the U.S.

Observation No.	State	SMSA or BEA[†]	Cancer of Respiratory Organs (ICD Nos. 160-164)	Cancer of Buccal Cavity and Pharynx (ICD Nos. 140-148)	Cancer of Digestive Organs and Peritonium (ICD Nos. 150-156A, 157-159)	Leukemia-Aleukemia (ICD No. 204)	Total Cancer (ICD Nos. 140-205)	Diseases of the Heart (ICD Nos. 400-402, 410-443)	Radiation* mrem/yr
1	Ala.	Birmingham	24.01	3.75	39.58	5.83	141.05	306.01	88.4
2	Ariz.	Phoenix	24.57	2.69	40.99	7.35	136.92	289.53	90.4
3	Calif.	Los Angeles	28.94	3.26	47.64	6.78	154.20	304.73	73.6
4	"	San Francisco	29.24	4.93	55.11	7.58	167.60	291.97	68.7
5	Colo.	Denver	19.15	2.30	40.00	7.61	128.74	298.38	164.6
6	Conn.	Hartford[‡]	26.73	4.85	59.09	7.50	173.12	363.08	82.0
7	"	New Haven[‡]	26.37	5.27	63.30	8.41	183.67	427.95	91.3
8	Del.	Wilmington	28.74	2.66	54.55	7.37	161.27	384.01	77.5
9	D.C.	Washington	25.66	4.92	48.55	6.07	141.98	298.63	76.7
10	Ga.	Atlanta	22.05	3.22	35.47	6.96	131.24	284.65	101.9
11	Iowa	Des Moines	27.88	3.10	47.88	7.60	156.30	369.13	89.2
12	Ill.	Chicago	29.51	4.40	61.18	6.97	173.37	454.84	81.6
13	Ind.	Indianapolis	29.53	4.09	52.04	6.99	165.81	362.69	90.8
14	La.	New Orleans	33.91	5.67	47.53	6.42	156.74	377.01	83.7
15	Mass.	Boston[‡]	29.51	5.19	65.14	7.39	183.27	434.81	86.6
16	Md.	Baltimore	31.24	4.07	52.66	6.06	166.59	411.86	68.3

Table I. *(Contd)*

Observation No.	State	SMSA or SEA[†]	Cancer of Respiratory Organs (ICD Nos. 160-163)	Cancer of Buccal Cavity and Pharynx (ICD Nos. 140-148)	Cancer of Digestive Organs and Peritonium (ICD Nos. 150-154A, 157-159)	Leukemia-Aleukemia (ICD No. 204)	Total Cancer (ICD Nos. 140-205)	Diseases of the Heart (ICD Nos. 400-402, 410-443)	Radiation[‡] mrem/yr
17	Mich.	Detroit	27.58	4.07	52.11	6.30	354.85	329.32	88.4
18	Minn.	Minneapolis	19.72	2.65	50.05	8.59	350.50	330.69	74.8
19	Mo.	Kansas City	25.04	3.75	46.69	6.90	333.81	371.30	89.9
20	"	St. Louis	31.00	4.79	55.86	7.18	373.52	407.49	87.6
21	Nebr.	Omaha	28.88	4.15	53.85	7.59	354.47	362.87	90.2
22	N.J.	Newark	28.93	4.34	64.69	7.71	388.76	460.59	86.6
23	N.Y.	Buffalo	25.80	4.42	57.98	7.54	370.33	429.24	88.4
24	N.Y.	New York City	33.46	4.68	72.95	7.85	301.89	477.01	86.6
25	N.C.	Charlotte	19.75	3.40	37.93	6.76	307.31	313.03	88.9
26	Ohio	Cincinnati	33.01	5.30	64.31	7.51	390.38	405.67	72.8
27	"	Cleveland	31.46	4.40	61.87	7.38	381.46	399.06	88.3
28	"	Columbus	26.36	3.37	50.01	6.83	352.20	329.41	85.4
29	"	Youngstown	26.87	3.59	60.66	7.51	366.04	374.50	84.6
30	Pa.	Allentown	27.48	3.30	61.39	7.47	377.63	491.60	87.3
31	"	Philadelphia	31.76	4.14	62.98	7.16	381.38	396.91	83.5
32	"	Pittsburgh	30.03	4.05	62.78	7.29	378.54	437.69	95.4

Table 1. (Contd)

Observation No.	State	SMSA or REA[a]	Cancer of Respiratory Organs (ICD Nos. 160-164)	Cancer of Buccal Cavity and Pharynx (ICD Nos. 140-148)	Cancer of Digestive Organs and Peritonium (ICD Nos. 150-156A, 157-159)	Leukemia-Aleukemia (ICD No. 204)	Total Cancer (ICD Nos. 140-205)	Diseases of the Heart (ICD Nos. 400-402, 410-443)	Radiation[b] mrem/yr
33	Pa.	Scranton	31.55	5.12	92.72	7.46	212.55	610.79	88.9
34	R.I.	Providence[a]	31.19	5.04	66.20	7.82	200.87	502.02	83.0
35	Tenn.	Chattanooga	27.27	4.41	37.08	5.30	139.23	301.53	88.7
36	"	Nashville	26.70	3.88	35.21	5.54	137.15	350.60	87.9
37	Texas	El Paso	12.97	3.39	26.83	6.25	87.72	147.58	106.8
38	"	Houston	28.02	3.76	34.65	6.50	123.84	270.26	80.7
39	Utah	Salt Lake City	15.40	3.70	31.39	6.53	111.68	240.91	110.9
40	Wash.	Seattle	24.63	3.87	51.46	9.01	159.75	362.44	86.8
41	"	Tacoma	22.98	2.98	49.36	7.63	153.15	379.21	87.3
42	W. Va.	Charleston	27.48	2.97	38.85	7.04	133.24	316.13	88.4
43	Wis.	Milwaukee	24.01	4.37	57.02	7.26	164.30	354.02	88.5

[a] All mortality rates are for Standard Metropolitan Statistical Areas (SMSAs), except for areas Nos. 8, 7, 15, and 34, i.e., the Hartford, New Haven, Boston, and Providence areas. These rates are for Standard Economic Areas (SEAs).

[b] Background radiation data are those reported by Oakley (Oa72).

Table 2. Coefficients of correlation between background levels of ionizing radiation and chronic disease mortality rates for 43 urban populations of the U.S., 1960 period

Cause of Death	Correlation Coefficient, r, Versus Radiation*	Level of Statistical Significance
Cancer of Respiratory Organs (ICD Nos. 160–164)	−0.514	$p < 0.001$
Cancer of Buccal Cavity and Pharynx (ICD Nos. 140–148)	−0.438	$p < 0.01$
Cancer of Digestive Organs and Peritoneum (ICD Nos. 150–156 A, 157–159)	−0.235	$p < 0.10$**
Leukemia–Aleukemia (ICD No. 204)	+0.023	not sig.
Total Cancer (ICD Nos. 140–205)	−0.300	$p = 0.05$
Diseases of the Heart (ICD Nos. 400–402, 410–443)	−0.257	$p < 0.10$**

*Correlations are based on data of Table 1.

**Correlation coefficients for x_{20} and x_{21}, and x_{19} and x_{21}, barely fall short of significance at the 0.05 level. The critical value given in Table A10b,' page 569, of Gibson and Massey (1958), is $r = \pm 0.244$.

Table 1. Also shown are the levels of significance of these coefficients using a null hypothesis of zero correlation. With the exception of leukemia–aleukemia, all are negative. The correlation of background radiation level with mortality rate from cancer of the respiratory organs is significant at the 0.001 level; with cancer of the buccal cavity and pharynx at the 0.01 level; with total cancer at the 0.05 level; and with cancer of the digestive organs and peritoneum at the 0.10 level. Background radiation level is also significantly correlated with mortality rate for diseases of the heart at the 0.10 level. Leukemia–aleukemia mortality rate is not significantly correlated with background radiation level.

MULTIVARIATE STATISTICAL ANALYSES

In this preliminary communication we report only that we have conducted multivariate statistical analyses in which the associations between levels of several environmental chemicals and of background radiation on the one hand, and certain chronic disease mortality rates, on the other, have been analyzed by multiple regression. When radiation was selected by the algorithm as a significant predictor variable, the regression coefficient was negative. This occurred in these preliminary analyses for the two respiratory tract cancer categories.

Table 3 reports six regression equations (Nos. 2, 4–8) with the largest sets of predictors for which R^2 is significant ($\alpha = 0.05$) by the usual F-test, and for which all regression coefficients are also significant ($\alpha = 0.05$) by the usual t-test. In *none* of these six maximum predictor equations is the radiation (RA) variable included. The radiation variable was selected, however, in the optimal selection sequence for two mortality categories where N was less than maximum, i.e. equation (1) for cancer of the respiratory organs

Table 3. Multiple regression equations associating disease with natural logarithms of concentrations of also with background level of ionizing radiation

Equation No.	Mortality rate for:	n[1]	Constant	Regression Coefficients and t-Statistics							
				ln (NO_3^-)	ln (SO_4^{2-})	ln (H^+)	ln (NO_3^-)	ln (Cs)	ln (Cd)	ln (Cr)	ln (Tl)
1	Cancer of the Respiratory Organs (ICD Nos. 160-164)	4	-0.342	+4.912 (1.92)*	-	-	+3.816 (2.07)*	-2.477 (3.01)*	-	-	-
2	"	7	-18.728	+4.759 (1.62)	-	-	+3.755 (2.28)	-1.846 (2.13)	-0.918 (2.63)	+1.256 (2.58)	+4.994 (4.47)
3	Cancer of the Buccal Cavity and Pharynx (ICD Nos. 140-148)	1	+6.261	-	-	-	-	-	-	-	-
4	"	4	-0.125	-	+0.503 (2.56)	-	-	-	-0.328 (3.61)	+0.616 (2.80)	-0.858 (4.06)
5	Leukemia and Aleukemia (ICD No. 204)	4	+11.262	-1.133 (3.08)	+0.364 (3.13)	-	-	-	-	-	-
6	Cancer of the Digestive Organs and Peritoneum (ICD Nos. 150-156A, 157-159)	5	-11.613	-	+6.199 (5.70)	-	-	-	-2.956 (3.07)	-	+7.302 (2.29)
7	Total Cancer (ICD Nos. 140-205)	5	-146.822	-	+20.038 (4.64)	-	+26.844 (1.79)	-	-6.119 (4.47)	-	-14.298 (2.18)
8	Diseases of the Heart (ICD Nos. 400-402, 410-443)	5	+165.509	-	+61.718 (6.67)	+52.843 (2.06)	-	-	+27.422 (4.33)	+26.135 (3.17)	-

* Parenthetical values are t-statistics for the significance of the regression coefficients.

**Radiation and mortality rate data are given in Table 1.

† n = number of predictors in the equation.

mortality rates for several categories of chronic 13 air pollutant chemicals and water hardness, and for 43 urban populations of the U.S., 1957–64.

La (Fe)	La (MS)	La (Mn)	La (NM)	HA**	χ^2	Degrees of Freedom	F-Statistics	Significance of χ^2	Significance of Hardness t-Statistic
-	-	-	-	−0.093 (1.03)*	0.592	4,38	13.8	<0.01	<0.05
+1.833 (1.84)	-	-	-	-	0.693	7,35	11.3	<0.01	<0.05
-	-	-	-	−0.228 (3.12)	0.172	1,41	8.5	<0.01	<0.01
-	-	-	-	-	0.528	4,38	10.6	<0.01	<0.01
-	+0.648 (3.89)	−0.575 (3.31)	-	-	0.224	4,38	4.5	<0.01	<0.05
+7.996 (2.14)	+6.005 (2.31)	-	-	-	0.648	5,37	14.9	<0.01	<0.05
+16.367 (2.13)	-	-	-	-	0.686	5,37	14.1	<0.01	<0.01
-	-	-	−28.491 (2.64)	-	0.636	5,37	12.9	<0.01	<0.05

for $N = 4$, and equation (3) for cancer of the buccal cavity and pharynx for $N = 1$. For larger values of N for these mortality categories, radiation was not among the optimal combinations of predictors selected for maximum R^2. Thus in some combinations radiation was selected among the optimal R^2 predictors, and, when selected, it was negatively associated.

For cancer of the respiratory organs, background radiation level appears as a statistically significant predictor in the presence of environmental chemical variables. Radiation was initally selected when $N = 2$, with a negative regression coefficient. Negatively associated radiation level was also selected for $N = 3$ and 4. This last relationship is shown in equation (1) of Table 3; the predictors account for approx. 59% of the variance in the criterion. The algorithm continued to produce significant sets of predictors through $N = 7$, as shown in equation (2), but radiation was not included. The chemical predictors selected have appeared in previous analyses with coefficients of the same signs (Hic70a; Hic70b; Hic71a; Hic71b).

In the analysis of mortality rates for cancer of the buccal cavity and pharynx, radiation level was the first variable selected by the algorithm, as shown in equation (3) for $N = 1$. Radiation accounted for approx. 17% of the variance in this criterion, and the association is negative. Radiation was not selected subsequently, although significant sets occurred through $N = 4$ (equation 4). This equation explains about 53% of the variance in this criterion, based on chemical predictors discussed previously (Hic70a; Hic70b; Hic71a; Hic71b).

In the analysis of mortality rate for leukemia–aleukemia, ionizing radiation level was not selected as a predictor in any significant set. It was selected with a negative coefficient both for $N = 8$ and 9. However, neither the t-statistics nor the F-statistics were significant.

The equation for $N = 4$ yields the maximum R^2 with significant t- and F-statistics, as shown in equation (5). This group of chemical predictors includes negatively correlated lead. Lead was not selected previously in this analysis, and the predictors explain only about 32% of the variance in leukemia–aleukemia mortality rate. Accordingly, we consider this a weak result.

Clinically, leukemia is a symptomatic diagnosis, like pneumonia, not an etiological diagnosis, like tuberculosis. The age distribution of leukemia incidence is bimodal, and the prognosis usually differs with age. For younger ages, leukemia tends to be acute, and survival is often relatively brief. At older ages, leukemia is frequently chronic. The literature suggests that the etiologies of acute and chronic leukemia may be different (Fre78; Nec79). Should this be the case, the explanatory power of our analyses could be reduced. To investigate this possibility, we plan to separate the mortality data into juvenile and adult leukemia, compute rates, and repeat the analysis.

In the regression equations for cancer of the digestive organs and peritoneum, the radiation variable was not selected for any set of predictors chosen by the algorithm. In equation (6), for which $N = 5$, about 67% of the variance in the criterion was explained by the environmental predictors selected. Again, these are familiar chemical predictors (Hic70a; Hic70b; Hic71a; Hic71b).

Also for total cancer, radiation level was not selected by the algorithm. For this criterion the maximum percentage of variance explained is about 66, as shown in equation (7). The five predictors are, again, familiar (Hic70a; Hic70b; Hic71a; Hic71b).

The fact that the radiation variable does not appear in the equations for cancer of the digestive organs and peritoneum or total cancer does not imply that radiation is without effect. It simply indicates that, statistically, in this analysis, the associations of chemicals with these criteria were stronger than those for radiation level. As will be reported subsequently, inclusion of socioeconomic variables among the predictors alters the combinations of chemicals and radiation selected by the algorithm. Furthermore, it must be understood that in a regression equation, when a set of environmental predictors explains about 66% of the variance in mortality rate of a cancer cate-

gory, this does *not* necessarily imply functional causality. It may reflect the effects of additional variables, or even represent a spurious correlation.

Since our studies are largely concerned with diseases of aging, we also included in our analyses diseases of the heart. Cardiovascular diseases are the major cause of death in mature individuals in Western cultures. Radiation level was not selected as a statistical predictor for diseases of the heart (see equation 8). In this equation, 63.6% of the variance in M_{ch} was explained by five chemical predictors. The selection of $c(NO_3^-)$ as a positively correlated predictor for M_{ch} makes equation (8) differ from both the results of previous analyses (Hic71a; Hic71b; Hic76) conducted without the $c(NO_3^-)$ variable, and from the cancer analyses reported here.

In summary, our preliminary results appear to be of two kinds. First, based on the signs of the bivariate correlation coefficients shown in Table 2, the only significant statistical associations are *negative*. That is, mortality rates for the indicated chronic diseases tend to be consistently low where background radiation level is high, and high where radiation level is low. The positive association for leukemia is very weak and not statistically significant. These preliminary observations are not compatible with models that assert that all levels of radiation, no matter how low, are damaging. Because the age distribution of leukemia incidence is bimodal, it appears necessary to conduct analyses on juvenile and adult leukemia mortality rates separately.

Second, the occurrence of only negative coefficients for the background radiation predictor variable, whenever it appeared as a significant term in multiple regression equations, casts further doubt upon the validity of the no-threshold model. The absence of the radiation variable for some causes of death, such as cancer of the gastrointestinal organs and peritoneum, suggests that, compared with the environmental chemicals, the contribution of background radiation level to explained variance of mortality rates of several major chronic diseases is rather small.

It appears that the statistical effects of the environmental chemicals in these systems are not overwhelmed by inclusion of background radiation level among the available predictors. While radiation does appear to have a detectable effect, it is not of a greater order of magnitude than chemical effects, and the consequences of sustained exposure to low level radiation require further examination.

DISCUSSION

This study utilizes vital statistical data on populations to examine problems of radiation as was suggested by the U.N. Seminar on the Use of Vital and Health Statistics for Genetic and Radiation Studies of some years ago (UN62). We employ statistical methodologies to relate variations in the compositions of ecosystems to the health of the human populations inhabiting them. This study appears to have implications for the on-going discussion of public policy with respect to the health consequences of exposure to various levels of ionizing radiation.

For purposes of discussion, the alternative hypotheses that purport to describe human health effects of ecologically realistic low levels of ionizing radiation require amplification. They can be stated as:

(1) *The continuous increase—no threshold hypothesis.* The biological effects of ionizing radiation, including low levels of natural radiations that result from terrestrial geological chemicals, from cosmic rays, and from the internal body burden of radionuclides acquired from food and drink, are hazardous at all levels, with the degree of hazard increasing with the dose and dose-rate (Ad79; Ba73; BEIR72; BEIR77; BEIR79; Bow80; Jac76; Ke79; Mo77; Mo78; Na79).

(2) *The threshold hypothesis.* Ionizing radiation is damaging at elevated levels and dosages, the damage increasing with exposure, but there is a threshold exposure level below which there is no health hazard to human populations (Ba73; Fri73; Jac76).

(3) *The beneficial effects hypothesis.* Ecologically realistic low levels of ionizing radiations, such as environmental background radiations, that have existed over the millenia of biological evolution, are beneficial to human populations, resulting in extended life

span, reduced risk of chronic disease, and reproductive advantage in intergenerational functions (Car57; Car59; Cas68; Cl70; Cr50; Fre73; Jan65; Jac76; Lor50; Lu78; Lu80; Va68; WHO55; WHO57).

The negative correlation coefficients of Table 2 certainly do not suggest that, at low levels, increases in ionizing radiation will lead to increased mortality from chronic disease. Neither does the fact that in the multiple regression equations computed thus far, radiation level usually occurs with a negative coefficient and vanishes as other significant variables are selected. Apparently the statistical associations of various environmental chemicals with the mortality rates examined are stronger than those involving radiation. So in a very preliminary way, the evidence reported here is incompatible with hypothesis (1), and is consistent with either hypothesis (2) or (3). This is a surprising result. It led us to examine the literature to ascertain what data exist that might support each of these three hypotheses.

We found that much of the support for hypothesis (1) comes from scholars who believe that the result of studies using high exposure levels of ionizing radiation on experimental animals can be extrapolated to low levels—usually using a linear model (Ad79; BEIR72; BEIR77; BEIR79; Bow80; Br77; Ke79; Mo77; Mo78; vo76). However, both toxicological and statistical results have shown that such extrapolation is frequently incorrect (Am74; Ke67; Sn76). This is consistent with the view of Frigerio *et al.* (Fr73), of the Argonne Radiological Impact Program (ARIP). In Part I of their study on the "Carcinogenic Hazard from Low-level, Low-rate Radiation", they stated:

> "Various models and predictions of carcinogenic hazard are examined and compared with actual experience in U.S. and foreign populations. All of the models predict a significant increment in malignant mortality with increasing background. Observation of the actual populations at risk shows not only no increment but an actual decrement, so that these predictions are left quite without observational support. It is concluded that extrapolation of high-rate and usually high dose level studies to low rates and low levels is probably invalid, and that radiation at such levels and rates does not constitute an environmental carcinogen of significance" (p. 5).

This ARIP warning (Fri73) has been widely ignored, though it was cited, for example, by Wolfe in 1976 (Wo76) and 1977 (Wo77). Wolfe (Wo76) observed that at low radiation levels "... it is not even plain that a beneficial effect is precluded" (p. 479). Objections to Wolfe's views were expressed by von Hippel (vo76), Morgan (Mo77) and Brown (Br77) who favored the linear extrapolation hypothesis, or some variant of it. Brown (Br77) asserted, for example, that "... the dose-response curve ... must be linear", and that "... since much of our knowledge of carcinogenesis by radiation comes from doses of around 100 rad, this means that, for all practical purposes, linear extrapolation obtained from these doses will be a good way of estimating effects at very low doses" (p. 349). This, however, is not evidence, and Wolfe (Wo77) observed in response that "... no meaningful evidence of the validity of the linear theory is presented" (p. 1387).

On the same point, Jacobson *et al.* (Jac76) commented that "... assessment of radiation risk below this range [20–50 rem] requires the guesswork of extrapolation" (p. 32). They also pointed out that radiation levels "... substantially above the U.S. background dose-rate such as those in Kerala, India, where the average dose-rate is about 1300 mrem/yr, have not produced statistically significant effects in the human population" (p. 36).

Support for models of the first sort seems to stem from the work of Muller (Mu57; WHO57) based largely on the genetics of male *Drosophila* and dependent upon very strong assumptions. Muller also pointed out the difficulties of testing linearity below the 25-R level; however, Bonnier and Lüning report increases in visible mutations in *Drosophila* in response to radiation below this level (Bon49). Bustad and colleagues also reported results that support linearity at low doses (Bus65). Russell's early work (Ru52; Ru56) has also been cited in support of linearity at low

radiation levels; however in recent years he has deviated from the linear model (Ru72; Be72).

For carcinogenic effects, Morgan (Mo78) denies the existence of a threshold, citing Stewart's and Kneale's analysis of pelvimetry exposures (Stew70; Stew71) and Sternglass' analysis of mortality in the vicinity of nuclear power plants (Ster63; Ster71). Serious questions have been raised, however, concerning the validity of Sternglass' results (La75; Un79). Smith (Sm78) and Warren and Gates (Wa71) also support hypothesis (1), but inbred laboratory animal strains were used in some of their studies. Such strains tend to withstand environmental hazards less well than wild strains found in the natural world.

Much of the actual data from well-designed studies tends to favor either hypothesis (2) or (3) or both. The work of Gopal–Ayengar and colleagues on populations from the Kerala coast of India (Gop72), and that of the Chinese High Background Radiation Research Group (Hig80) on populations from Guangdong Province, China, are consistent with hypotheses (2) or (3), but not with hypothesis (1). Moreover, a number of experimental animal studies have been conducted on populations of animals exposed for all or most of their lifetimes to various levels of ionizing radiation. At exposure levels and rates of 300–2000 mrem/yr, average survival times were longer and risks of chronic diseases were lower than for controls exposed to about 60–165 mrem/yr, the range found in various regions of the U.S. (Car57; Car59; Cas68; Lor50; Oa72; Va68). A warning issued by Jankowski (Jan65) was not incompatible with hypothesis (3), and Van Cleave (Va68) discussed the "negative life shortening" effects of ionizing radiation. Similarly, Casarett (Cas68) noted that in "... certain lifespan experiments, when the radiation dose has been quite low, the irradiated animals live longer, on the average, than do the control animals". He also stated that "... animals in some of the irradiated groups do not have as high an incidence of certain diseases as is found in control groups" (pp. 273, 274).

Furthermore, in an experimental study of *Paramecium bursaria*, Luckey and colleagues found that lead shielding of the organisms from ambient radiation resulted in much poorer growth and reproduction than observed for the controls (Lu78). In a forthcoming report on hormesis in ionizing radiation, Luckey (Lu80) states that, based on his analysis of the evidence, "... radiation hormesis is not predictable with data from high doses... The argument that low doses give harmful effects in proportion to dosage is invalid". Hormesis is a general phenomenon in which exposure of biological organisms to traces or low levels of hazardous substances or agents stimulates natural physiological defense mechanisms in a manner that benefits health and survival (Lu80). Radiation hormesis tends to resemble biological responses to low levels of hazardous substances such as toxins and antigens.

Natural defense mechanisms are stimulated by toxoids, viruses, vaccines and bacteria. Immunological mechanisms are, of course, well known. Some individuals exist whose defense mechanisms are genetically deficient. However, the existence of a number of different DNA repair mechanisms has become known in recent years (Ar78; Ay80; Ha78; Ro79; Se78). The hypothesis may be considered that under ecologically realistic conditions of exposure to low levels of ionizing radiations, DNA repair (and other defense mechanisms) are stimulated to such an extent that efficiency is elevated above the level necessary to repair low-level radiation damage. The result could be that the activated defense mechanisms improve health and survival of the exposed individuals compared with similar individuals exposed to lower levels of ionizing radiation. Such reactive mechanisms could explain the experimental observations of extended survival of irradiated animals observed by such investigators as Lorenz (Lo50), and Carlson *et al.* (Car57; Car59), as well as the related observations of Cronkite *et al.* (Cr50) and of Clarke *et al.* (Cl70). Biochemical investigations should provide enlightenment on this general question.

CONCLUSIONS

This preliminary report can make no firm

causal statements. It can only call attention to certain relationships and suggest further study. With these caveats applying, we can conclude with three statements:

(A) There is continuing need for multivariate epidemiological studies aimed at elucidating the health effects of low-level ionizing radiation, particularly in the presence of other environmental variables.

(B) Extrapolation from high exposure levels to low exposure levels is methodologically unsound. Reassessments of any hazards involved with exposure to low-level ionizing radiation should be based on ecologically realistic data acquired at the levels of concern without extrapolation.

(C) Our preliminary results suggest that adopting the no-threshold hypothesis in the absence of strongly supportive observational data at the actual exposure levels and levels of biological organization of concern is a dubious procedure.

We plan to report further on our studies after we have investigated the multivariate aspects of the problem more thoroughly and have examined the influences of age and socioeconomic variation upon the observed relationships.

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Addendum. Since completing this manuscript we have obtained a copy of the 1980 version of BEIR-III. It does not contemplate a beneficial effects hypothesis. Moreover, the very recent report by a BEIR-III committee member, Charles E. Land ["Estimating Cancer Risks from Low Doses of Ionizing Radiation", *Science* **209**, 1197–1203 (1980)] also does not examine this alternative hypothesis.

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THE ARGONNE RADIOLOGICAL IMPACT PROGRAM (ARIP)

Part I. Carcinogenic Hazard from Low-level, Low-rate Radiation

by

Norman A. Frigerio, Keith F. Eckerman,
and Ralph S. Stowe

Environmental Statement Project

September 1973

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THE ARGONNE RADIOLOGICAL IMPACT PROGRAM (ARIP)
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ABSTRACT

The entire Argonne Radiological Impact Program is briefly outlined, and part of the program dealing with radiation hazards from nuclear power plants is discussed in detail. Various models and predictions of carcinogenic hazard are examined and compared with actual experience in U. S. and foreign populations. All of the models predict a significant increment in malignant mortality with increasing background. Observation of the actual populations at risk shows not only no increment, but an actual decrement, so that these predictions are left quite without observational support. It is concluded that extrapolation of high-rate and usually high dose-level studies to low rates and low levels is probably invalid, and that radiation at such levels and rates does not constitute an environmental carcinogen of significance.

INTRODUCTION

In late 1971 the Environmental Statement Project was formed at Argonne National Laboratory to aid the U. S. Atomic Energy Commission in the preparation of environmental statements for the nuclear facilities being licensed by the Commission. Since methodology for assessing the impact of such facilities was in its infancy, it was necessary for us to develop methods and programs adequate to the task. For questions of radiological impact this eventually took the form of a series of studies, reports, recommendations and computer programs which, taken together, formed the Argonne Radiological Impact Program (ARIP). This report, ARIP I, concerns itself with evaluation of the carcinogenic hazard that might be associated with the radiation and radioactivity from nuclear facilities. Future ARIP reports will address other parts of the program, e.g.: the determination of optimum sites for nuclear facilites (II); evaluation of the doses received by man and other biota from the releases of nuclear facilities (III); storage and access of available data on radiation and radioactivity levels in the U. S. (IV); etc.

A recent report has summarized what is known of the genetic and somatic hazards of radiation.[32] The principal somatic hazard was found to be carcinogenesis, and genetic and somatic hazards were found to be of about the same severity per unit dose. Our evaluations of nuclear power reactors, present and proposed, revealed that the major fraction of the dose received by man and other biota was somatic rather than genetic. The spectrum of

radionuclides emitted by a power plant, in conjunction with the pathways by which they reached man and other biota almost invariably resulted in critical organ doses about an order of magnitude higher than the genetically significant doses received from the same source. The inexorable arithmatic of carcinogenesis renders the critical organ dose truly critical to the survival of the individual irradiated. While the probability of cancer in any organ is proportional only to the dose to that organ, the survival of the individual is largely independent of which organ becomes cancerous. At the present state of the art in cancer therapy, the survival of the individual is seriously threatened by cancer in any organ. Thus, radiation hazard from nuclear plants, small as it may be, is defined by the critical organ doses received. Thus such concepts as "whole body" dose are germane to the question of hazard only at a secondary level relative to critical organ doses.

So far as is known the radiation and radioactivity from a nuclear power plant is not uniquely hazardous and that, so long as doses are expressed in common units (i.e. rem), the hazard is independent of source.[32] In terms of both dose rate and dose level, radiation hazard from nuclear plants most closely resembles background radiation. Thus, we opined that a study of carcinogenesis occasioned by the radiation background would constitute the most pertinent test of the potential carcinogenicity radiation and radioactivity from nuclear power plants.

THE PROBLEM

In recent years, the hypothesis has been advanced that a significant fraction of human cancer mortality may be due to the human radiation background.[1-3,13,31-33] For a normalized irradiation of 170 millirem/yr, these authors have estimated U. S. cancer mortality excesses of about 3,000 to 100,000 per year, i.e., about 1% to 30% of current experience. Since the identification of so important an etiologic factor would be an event of major significance in the field of cancer epidemiology, we addressed ourselves to the examination[24] of the degree to which these hypotheses could be justified from current vital statistics and from the known variations in the radiation background.

This examination occupied a fair span of time, during which the mists of our comprehension cleared only slowly.[55] We began with vital statistics, recognizing that they form only a small part of the epidemiologic method, but intending to go on when they wore thin. Much to our surprise they continued to lead us on until, at the end, we had hardly applied anything else. We beg the reader's indulgency, therefore, if what we present is less in logical than in chronological order. We hope thus to indicate how it was possible for us to begin with the presumption that background radiation must be carcinogenic only to be forced, after something very much like the classic Drunkard's Walk,[19] to conclude that it is not.

LINEAR MODELS — THE GIVENS

The estimates given above all depend on linear extrapolation of data obtained at high dose-rates, and generally high doses, to the low-rate, low-dose condition of the natural background. The linear models involved have taken two forms; the *additive*, or absolute-risk, model, and the *multiplicative*, or relative-risk, model. Formally, these may be written as:[22]

$$r = r' + kD \qquad \text{additive,}$$
$$r = r' + r'(D/DD) \qquad \text{multiplicative.}$$

Here r is the observed rate of age-adjusted mortality, r' the "spontaneous" or radiation-independent rate, and D the dose. k is the absolute risk per unit dose, and DD the so-called "doubling dose," a measure of relative risk.

These two models differ in the role assigned radiation as a carcinogen. The additive model treats radiation as a complete carcinogen, sufficient unto itself, and with no need for co-carcinogens. The multiplicative model treats radiation as a pure co-carcinogen, amplifying whatever malignancy already exists, but not necessarily adding any on its own.

The estimates cited have all made use of five "generalizations," although these have been made most explicit only in the most recent reports.[2,32] Following Gofman and Tamplin,[2] we have termed these "generalizations" rather than "assumptions" since they are based on an impressive assemblage of human and animal data as well as an extensive collection of theoretical discussions. Properly speaking, the "assumption" at issue was whether this data, involving small, selected groups at high doses and/or high dose-rates, could be linearly extrapolated to the U. S. population as a whole.

In the estimates presented to date, the following generalizations have been employed:[1-3,13,31-33]

(1) Radiation is a *pan-* to *polycarcinogen*, i.e. all/many sites are subject to radiation carcinogenesis, and in about the same measure;

(2) Radiation sensitivity is independent of dose rate, but drops sharply during the first three decades of life, so that only the background dose received during the first 30 years is considered in estimating population risks over all ages. For a normalized background irradiation of 170 mrem/yr, this amounts to a total dose, D, of about 5.1 rem;

(3) Estimates of total risk can be made assuming an age-averaged sensitivity over the entire population. For the additive model, estimates of k have ranged from 0.3 to 9.0 deaths per year per 100,000 population per rem. For the multiplicative model, estimates of doubling dose have ranged from about 50 to 500 rem. These ranges correspond, roughly, to from 1% to 30% of the current U. S. malignant mortality of about 320,000/yr. While many workers have used only a single value for sensitivity, others have utilized specific functions for generalization (2), and integrated these over

age to provide the most explicit estimates;[2,32]

(4) Aside from age, populations have been treated as equivalent — regardless of "race, creed or color," so to speak. While this generalization has seldom been made explicit, it has always been implicit, as witness the extrapolation of data from Japanese bomb victims, uranium miners, spondylitics, infants radiographed *in utero* malignancy-bearing patients and the like, to the U. S. population as a whole; and

(5) Latent periods for the initial appearance of radiogenic malignant mortality range from a few years for infants to as much as 30 years for adults. The bulk of the values used have been ≤5 years for infants and ≤20 years for adults.

In testing the two forms of the linear model, we utilized these generalizations as they stood. Natural background for each state were taken from Minx, Schleien, *et al.*[6], and applied equally to both sexes and all races within the state. A man-made background (medical, fallout, nuclear devices, etc.) of 40 millirem/yr was added to the natural background. While this is admittedly lower than the 95 millirem given for 1972[6] or the 45-61 given for the 1960's,[12] it was chosen as a reasonable, conservative average for the period 1950-1968.[32] It makes some allowances for the presence in the population of groups whose medical exposure is probably low, and adds nicely to the U. S. average natural background[6] of 130 mrem/yr to give the 170 mrem/yr which has so often been used as the basis of estimates. For some tests we needed a distribution function for radiogenic malignant mortality. We could find nothing explicit on this in the presentations. However, from the Poisson nature of radiation itself and the linear form of the model, we inferred Poisson statistics for radiogenic components of malignant mortality.

The major disagreements among the various authors seem to have centered on (1.) and (3.), as well as on the length of the plateau to be anticipated once the latent period had been exceeded. In order to arrange tests of all the variations, therefore, we added the following operational provisos:

(1a.) We would begin with the assumption of pancarcinogenesis. But we would also arrange the tests so that, if this were not the case, sites would be eliminated until only those corresponding to polycarcinogenesis would be left.

(3a.) We would begin with a high sensitivity and apportion it equally among the various sites. But we would also decrease this value progressively, toward zero, so as to encompass predictions made on the basis of various sensitivities and plateaus.

THE ADDITIVE MODEL

From generalizations (1) and (4) and the form of the additive equation, no general population may show a rate, r, of zero. Even if r' = 0, r will be equal to kD. Of course, an observation of r = 0 may be made if the statistics of sampling are such that kD cannot be distinguished from zero. Similarly, if other carcinogens are present,[9] or the population shows a heterogeneity of sensitivity,[34] r may be greater than kD. In such cases there is simply no test. But r should never be lower than kD in this model.[9]

Following the methodology of Higginson,[9] we attempted to determine if there were any subpopulations in the U. S. that violated this stricture, i.e., for which r was less than kD.

We were fortunate to have before us Burbank's[5] outstanding recent compadium of U. S. cancer mortality for the 18-year period 1950-1967. Our analyses were performed on his Static Geographic Tables, using his ICD types, age-adjusted death rates, population bases, etc. These group the U. S. population into 200 groups for each ICD (International Classification of Death)[5] type, i.e., 50 states, two sexes, and two races (white and non-white). ICD types 171-179 concern sites specific to only one sex, and were thus represented by 100 groups each. For details not available in Burbank, we utilized the available U. S. Statistics for 1950-1968,[27,28] but the same methodology.[5]

These groups are listed by 55 specific ICD malignancy (Mn) types, plus one "all other" category, — Burbank No. 65. The latter includes ICD malignancy types Mn 156, 165, 195, 198, and 199.

From generalizations (1, 1a) there must be roughly equal distribution of radiogenic malignant mortalities among the 56 Mn types. At k = 3.2 and D = 5.1, this would lead to an excess national mortality of about 33,600/yr, i.e., about 10% of current experience. Spread over 56 Mn types, this corresponds to r = 0.3 per Mn type, i.e., an expectation of about 54 radiogenic deaths per million population per Mn type for the 18-year period of observation. In order to run the gamut of proposed expectations, we first identified populations presenting at rates below 0.3 and 0.03, discriminated by sex, race, Mn type, and state of residence. The hope was that we would thus bracket some r at which the linear additive model would be easily tenable.

Observed mortalities were derived from Burbank[5] or obtained from the U. S. Vital Statistics.[27,28] "t" values were taken (expectation-observation)/(standard error) in the usual fashion.[11,18] Radiogenic expectation was taken as:

$$\text{expt.} = \sum_i (18)(k/56)(30)\, B_i\, P_i,$$

where each population subgroup, P_i, had been exposed to radiation at its particular background rate, B_i, for the first 30 years of life and observed over 18 years for the designated malignancy at a sensitivity of (k/56) per Mn type.

These expectations are given in the first column of each r group. Although, in cancer epidemiology, one would not usually consider expectations less than 5 or so,[11] much less express them as decimals, this *has* been the practice in radiation carcinogenesis studies.[13,32] Thus, we have allowed this practice at r = 0.3 or 0.03. At r = 0.003 we have used only a plus sign (+) to indicate that the value for t is mathematically real but less than 1.

Population of each cohort presenting at $\leq r$ is also given (in thousands) as well as the weighted average dose level characterizing the cohort. At the bottom of the table we have summarized the number of malignancy types at various t levels, signaling those of observations less than expectation by the device of a "positive" t, and the converse by a "negative" t. The latter have also been signaled by dashes in the body of the table. In each case, the total of types is 56.

With so many Mn sites violating the requirements of the model, even as judged simply by the normal "t" test, we had to admit that it was extraordinarily improbable, at least at these levels. So, we continued dropping our search value for r until, at 0.006, *all* of the observations went to zero except for those three stalwarts, ICD 151, 153 and 171. Since we thought we might have something here, we did our estimations on the basis of r = 0.003, the mid-value of the interval, rather than 0.006, its upper bound.

In this range level, the normal "t" test becomes awkward.[11,18] Thus, we resorted to the much more powerful, albeit lengthy and expensive, Monte Carlo method.[19] Briefly the population of the U. S. was subjected to a random "rain" of radiocarcinogenci deaths at r = 0.003 for 100 18-year periods, and the results of each period analyzed as above. Where the r = 0.003 column of Table 1 shows zero observed deaths for 53 tumor types we found a range of 11-33. By the 35th trial, a mean of 20 ± 2 was reached, and this held to the end of trials. Ergo, not only is the null hypothesis (r = 0.003) improbable, but the Monte Carlo results suggest that a level of roughly 0.003/20 would be needed to reach even a 63% confidence level. This corresponds to about 16 deaths/yr per 200,000,000 population, or about 0.005% of current U. S. mortality. Since the same pattern of zero observations had been found at all levels of r below 0.006, where the Monte Carlo expectation was about 41, a null hypothesis (r = 0.006) was comensurately less probable.

Now, the observation that "large," geographically contiguous populations exist in which malignant mortality rates are quite low is hardly novel.[9,26,43] But, by making this sort of observation explicit and more-or-less quantitative, it is more readily seen how this phenomenon places upper bounds on the involvement of background radiation in carcinogenesis. If there is a surprise, it is that the upper bound is as low as it is, at least in this otherwise persuasive model.

In any case the model certainly seemed untenable at any level much greater than $r = 0.003/20 = 1.5 \times 10^{-4}$, at least as its authors originally presented it. In theory, we thought it might be saved by abandoning generalization (1) and confining higher expectations to 10 of the 56 types, even though these 10 corresponded poorly to those for which human radiocarcinogenesis had been shown.[32] In practice, though, even this turned out

Table 1 Observations of U. S. Cancer Mortalities vs. Radiation Exposition at Three Exposition Levels

Malignancy type, ICD No.	r = 0.5 expt.	obs.	%	pop., thous.	Dose, mrem/yr	r = 0.05 expt.	obs.	%	pop., thous.	Dose, mrem/yr	r = 5.025 expt.	obs.	%	Pop., thous.	Dose, mrem/yr
140	6714	1930	58.3	189,859	173	550.8	842	5.8	66,150	173	8.5	0	1.5	4404	132
141	1438	1257	4.8	26,813	179	1.4	0	1.2	219	207	0.14	0	+	818	207
142	4933	5679	17.3	93,255	172	3.4	0	1.8	586	165	0.34	0	+	536	183
143	6531	8514	30.6	125,[illegible]	175	83.8	12	8.4	4,175	156	1	0	1.0	1536	196
144	5405	8456	16.6	63,850	174	2.0	0	1.4	324	204	0.20	0	-	324	204
145	5548	8059	46.8	104,251	174	14.5	3	3.0	2,394	198	1	0	1.0	1525	198
146	5637	8558	39.7	106,579	173	1.9	0	1.4	313	193	0.19	0	+	313	193
147	7095	8392	33.8	134,277	173	128.4	85	5.5	22,099	184	8	0	1.4	5173	191
148	3477	8443	12.3	65,090	175	2.9	0	1.7	501	191	0.29	0	+	501	191
150	35	81	8.1	668	220	0.3	0	0.5	50	213	0.05	0	+	50	213
151	--	--	-	--	--	-	--	-	--	--	-	-	-	--	--
152	1103	943	4.8	21,056	171	1.6	0	1.3	249	207	0.16	0	+	249	207
153	--	--	-	--	--	-	--	-	--	--	-	-	-	--	--
154	1	0	1.0	11	185	0.1	0	0.3	11	185	0.01	0	+	11	135
155	0	0	0.0	4	170	0	0	0.0	4	170	0.00	0	0	4	170
157	0	0	0.0	7	170	0	0	0.0	7	170	0.00	0	0	7	170
158	535	437	4.2	9,709	169	1.8	0	1.3	335	150	0.18	0	+	335	150
159	768	567	7.3	15,[illegible]2	184	0.7	0	0.8	151	132	0.07	0	+	151	182
160	4436	5490	14.9	85,627	171	1.5	0	1.2	265	180	0.15	0	+	865	180
161	3753	8506	15.5	71,496	172	1.2	0	1.1	203	194	0.12	0	+	808	194
162	1	0	1.0	15	222	0.1	0	0.3	15	222	0.01	0	+	15	222
163	0	0	0.0	8	170	0	0	0.0	8	170	0.00	0	0	8	170
164	8273	4576	4.1	157,352	172	14.1	4	2.7	2683	185	0.78	0	+	1473	173
170	3116	8604	9.2	59,719	171	1.3	0	1.1	258	179	0.13	0	+	259	179
171	--	--	-	--	--	-	--	-	--	--	-	-	-	--	--
172	2	0	1.4	39	209	0.2	0	0.4	39	209	0.02	0	+	39	209
173	4765	1254	50.9	90,819	172	14.8	7	8.0	2763	175	0.40	0	+	735	177
174	0	0	0.0	5	272	0	0	0.0	5	272	0 00	0	0	5	272
175	0	0	0.0	8	170	0	0	0.0	8	170	0.00	0	0	8	170
176	6	0	8.5	92	214	0.6	0	0.8	92	214	0.06	0	+	92	214
177	0	0	0.0	1	170	0	0	0.0	1	170	0.00	0	0	1	170
179	893	832	5.6	5,601	171	0.6	0	0.8	105	189	0.06	0	+	105	189
179	1433	1357	5.3	28,817	169	1.0	0	1.0	170	193	0.10	0	+	170	193
180	1	0	1.0	15	208	0.1	0	0.3	18	208	0.01	0	+	18	208
181	1	0	1.0	12	213	0.1	0	0.3	12	213	0.01	0	+	12	213
190	454	585	5.2	8,836	168	2.5	0	1.6	414	189	0.25	0	+	414	193
191	87	10	3.3	595	174	1.1	0	1.0	189	184	0.11	0	+	189	184
192	9094	6143	30.9	173,537	171	9.3	2	2.4	1702	178	0.59	0	+	1054	183
193	5	0	2.2	79	219	0.5	0	0.7	79	219	0.05	0	+	79	219
194	550	503	8.0	10,432	172	0.9	0	0.9	161	168	0.09	0	+	161	192
195	19	10	2.1	304	162	0.4	0	0.6	75	195	0.04	0	+	75	195
197	47	85	3.2	875	175	0.8	0	0.9	145	191	0.08	0	+	145	191
200.0	337	811	5.9	5,704	176	1.2	0	1.1	183	207	0.12	0	+	183	207
200.1	7	1	2.3	110	209	0.4	0	0.6	72	191	0.04	0	+	72	191
200.2	543	595	6.5	9,313	181	2.0	0	1.4	320	203	0.20	0	+	320	233
201	9	3	2.0	150	205	0.2	0	0.4	27	220	0.02	0	+	27	220
202.0	9411	8791	73.5	179,503	172	158.5	85	5.3	29,934	173	4	0	2.0	7925	175
202.1	8297	8071	4.7	45,538	173	0.9	0	0.9	152	196	0.09	0	+	152	196
202.2	9407	1501	83.6	179,254	172	372.0	808	5.4	70,945	171	2	0	1.4	3485	181
205	7	0	2.6	134	181	0.7	0	0.8	134	181	0.07	0	+	134	181
206.0	18	10	1.9	320	174	0.5	0	0.7	87	199	0.05	0	+	87	199
206.1	3	0	2.2	66	224	0.5	0	0.7	66	224	0.05	0	+	66	224
206.2	1820	1350	11.0	34,122	174	5.2	0	1.8	541	191	0.32	0	+	541	191
206.3	1	0	1.0	14	219	0.1	0	0.3	14	219	0.01	0	+	14	219
206.4	65	50	1.8	1,201	165	1.3	0	1.1	202	210	0.13	0	+	202	210
All Other	0	0	0.0	1	170	0	0	0.0	1	170	0	0	-	1	170

Types at t ≥ 3	22	5	0	
" " 3 > t ≥ 2	9	4	1	
" " 2 > t ≥ 1	11	17	5	
" " 1 > t > 0	7	26	46	
" " t = 0	5	4	4	

to be improbable when we examined this unholy decade by criteria other than r < kD *(vide infra)*.

As an aside, we might note that this sort of epidemiological approach possesses some peculiar advantages over less direct studies of radiation effects. For one thing it addresses itself directly to the population of interest, in this case that of the U. S. rather than to small, select populations of the war-torn (e.g., Hiroshima and Nagasaki), of the ill (e.g., those irradiated for spondylitis, tuberculosis, thyroiditis, malignancies, thymic disorders, etc.), of the young (e.g., irradiation for tinea), or of the occupationally stressed (e.g., uranium miners). It may be noted, for example, that even the smallest population groups in Table 1 are as large and usually much larger than these select irradiated groups.

Then, too, the time span of observation is large. Although we have dealt only with an 18-year span here,[36] the data can quite properly be regarded as an 18-year sampling of a continuing procession of cohorts which span the full biblical "three score years and ten". And, of course, the radiation is being delivered over this entire time span, at the very rates of interest, and compounded, so to speak, for effects *in utero*, on the young, on the general population, and on the aged. Admittedly, epidemiological data are susceptible to problems of over- and under-reporting, but these are hardly likely to be more severe than the parallel problems in small-group studies. Finally, the sheer mass of data permits far better cross-checking and, in many instances, provides insight into phenomena too minor to be observed in small-group studies but which may spark an idea or hypothesis in the mind of the investigator.[47] Quite possibly it is this aspect which makes addicts of the adherents of epidemiology.

With this in mind, several curious observations appear in Table 1. While each malignancy type at r = 0.003 is within its respective statistical expectation, the consistent string of zero observed deaths, over populations that range from tens of thousands to nearly ten million, is a bit unsettling. Admittedly very low incidences of certain malignancies are characteristic of ethnic and geographic groups throughout the world.[9,26] But one is not usually prepared for such variations in a population supposedly so culturally uniform as that of the U. S. It appears that the U. S. population comprises groups whose range of cancer mortality is nearly as wide as that of the world at large, substantiating previously puzzling observations of wide disparities between major U. S. cities.[8,26,30]

Despite this heterogeneity, some socioeconomic bias exists in the U. S. data. Many of the 10 apocalyptic horsemen of Table 1, who refused to show significant groups below r = 0.3, appear less formidable when viewed in the light of worldwide malignancy mortalities. In the first part of Table 2, we have presented the lowest national rates that we were able to find on inspection of the more readily available literature. Considering the usual urban-rural, male-female, ethnic-racial and socioeconomic diversities that one invariably finds in such national collections, one can anticipate finding sizeable groups at rates of zero or near-zero on closer study, even for these remaining Mn sites. Indeed, there appear to be large homogeneous groups below r = 0.3 for *all* malignancies.[9,26,41,42]

Several features of Table 2 may be worth noting. First, the "secondary,

Table 2. Minimum National Rates for Ten Malignancy Types

	All Ages		U. S., white, male		U. S., white, female		Early Adulthood		
Mn Type	r	Ref.	r	dr/dt	r	dr/dt	R	Age ś	Ref.
151	0.6	43	15.6	- 0.69	8.0	- 0.39	0.0	35	27, 37
153	0.0	43	16.1	+ 0.09	16.3	- 0.09	0.0	45	27, 37
155	0.0	43	3.0	+ 0.03	3.7	- 0.05	0.0	40	27, 37
157	0.2	39	9.3	+ 0.16	5.7	+ 0.06	0.0	50	27, 37
163	0.0	37	19.1	+ 0.96	3.6	+ 0.12	0.0	All	37
171	0.6	8	-	-	8.0	- 0.21	0.0	40	27, 38
174	0.5	37	-	-	4.8	- 0.26	0.0	50	37
175	0.6	39	-	-	8.5	+ 0.04	0.0	45	27
177	0.0	43	17.6	- 0.09	-	-	0.0	45	27, 37
"65"	0.1 - 4.0	8, 37	11.6	+ 0.01	9.7	- 0.17	0.0	40	27, 37

all other, and unspecified" categories 163, 174, and Burbank 65 characteristically constitute one of the major banes of the epidemiologist's existence (e.g.[8]). One hardly anticipates data of much analytical value in such catchall categories. Even so, the fact that cancer is a reportable disease in the Scandanavian countries,[37] with their remarkably complete registries, eliminates 163 entirely and produces some very low rates for 174 and for some of the ICD types included in 65. It seems quite likely that more data of this quality would eliminate them completely.[41,43] After all, they must, from their very nature, vanish to zero in the limiting case of perfect diagnoses.

Secondly, the very low rates observed help to dispel the enticing, if mildly parochial, notion that the remaining 7 types somehow constitute "common" malignancies, while the 46 eliminated via Table 1 are "rare". While these 7 do account for perhaps a third of total U. S. malignant mortality,[5] they hardly constitute important malignancies in other lands. Indeed, the only one of these to coincide with the BEIR list[32] of important radiogenic malignancies, ICD 151, is dropping so linearly and rapidly in the U. S. that it bids fair to reach zero within the comi. ; two decades.[5,8] That something of the sort might transpire for several of the others is suggested by the data given in the second part of Table 2, i.e., rates and values of dr/dt, the yearly change in age-adjusted mortality rate.[5]

Some further test of the stature of these ten malignancy types may be made by considering generalizations (2) and (5). If these are indeed valid, a goodly fraction of the total radiogenic insult must have been received by age 10 and a significant number of radiogenic mortalities should have appeared by age 30. However, this does not seem to be the case, as shown in the third part of Table 2, where R is a specific age group rate. Here we have isolated those national rates for which we had age-specific data, and for which R = 0 up to age 30 or beyond. Again, if these ten horsemen were truly riding to the beat of a radiogenic drum, they were certainly riding more slowly than predicted by the linear additive models so far proposed.

All in all, then, it appears that even the abandonment of polycarcinogenesis would do little for the additive model, especially in the long run, and this model will probably have to be abandoned *in toto*.

ADDITIVE MODEL IN INFANCY

Infants irradiated *in utero* may well represent a special case. The heroic, case-by-case studies of Stewart[20,47] have shown not only a very marked effect of diagnostic radiography at very low doses, but a broad spectrum of Mn types as well, so that her results have often been used to buttress the generalization of pancarcinogenesis.[2,3,13,31-33] Stewart has reported a value of 572 malignant mortalities per million live births per rem of irradiation *in utero*, essentially all of them occurring before age 10.[20,47] Stewart also showed that sensitivity during the first trimester *in utero* was about thrice that during the last trimester. Following Sternglass[21] we budgeted the background by trimester at relative sensitivities of 3:2:1. This yielded an age-specific rate, R = 1.11, for *in utero* irradiation by the 130 millirem/yr U. S. natural background. We then added 40 millirem as a probable average *in utero* X-irradiation[4,20] at a relative sensitivity of 1. The sum, R = 1.34, was distributed among the 56 tumor types to give R = 0.024 per type as an upper working level for test of hypothesis.

We screened U. S. experience as before, but with R = 0.024 and for the 0-9 year groups only. All states were accumulated so that only the two sexes and two race groups were discriminated, in addition to the available age discrimination of 0-4 and 5-9. Results are presented in Table 3, with populations given in millions. With only 19 of the 56 Mn types at t less than 2, a hypothesis of pancarcinogenesis was untenable at the 0.024 level of sensitivity. As before, we progressively lowered R until, at R $\leq$ 0.0018, the pattern stabilized at the values given in the second column of Table 2. Here, at an implied sensitivity about 8% of that found by Stewart for other malignancies, we were left with 34 Mn sites at r = 0.

We turned again to the Monte Carlo method, and, for the mid-value R = 0.0009, obtained an expectation range of 11-42, with a mean of 27, where only zero had been observed. Thus, the null hypothesis (R = 0.0009) was also untenable. The results, rather, suggested that the actual sensitivity of the infant to background was of the order of $\leq$ 0.15% of the sensitivity observed for diagnostic radiography, at least for these 34 types.

The remaining 22 Mn sites are in excellent agreement with the spectrum found by Stewart for diagnostic radiation. Since we had no way of removing the radiographic component from these populations, these 22 remaining sites may well include radiogenic deaths due to radiography, precisely as per Stewart. For the natural background, they simply represent cases of "no test".

The results are not consistent with a more selective model of additive carcinogenesis applicable only to these 22 sites. However, the cancers of early childhood form a rather special case of human malignancy.[7,8,10,20,25,44,47] They seem to be chiefly embryogenic in origin, have rates which are relatively constant over quite varied populations, and decrease in rate with increasing age (the converse of the situation above age 10 or so).[5,7,8,10,20,25,26,44] And, the 22 above only represent the most common malignancies in this age group. Thus, unlike the adult 10 horsemen above, it is improbable that zero rate groups will be found for more than a few of the 22, and the question of applicability of a partial additive model in this area is not likely to be answered by closer examination of worldwide experience along the lines used

Table 5. Cancer Mortality and Expectations, 0-9 Year Group

ICD Mn	R = 0.024				R = 0.0018			
	Exp.	obs.	t	pop., Mil.	Exp.	obs.	t	pop., Mil.
140	58	0	7.6	40	4.4	0	2.1	40
141	58	1	7.5	40	4.2	0	2.0	39
142	58	17	5.4	40	0.5	0	0.7	4.6
143	58	6	6.8	40	0.7	0	0.8	6.2
144	58	7	6.7	40	1.4	0	1.2	13
145	58	4	7.1	40	1.6	0	1.3	15
146	46	18	4.1	32	0.5	0	0.7	4.7
147	58	0	7.6	40	4.4	0	2.1	40
148	58	9	6.4	40	0.5	0	0.7	4.6
150	58	1	7.5	40	3.4	0	1.9	32
151	58	13	5.9	40	0.3	0	0.6	3.0
152	58	3	7.2	40	1.6	0	1.3	15
153	56	27	3.8	39	0	0	-	0
154	58	11	6.2	40	0.5	0	0.7	4.6
155	4.3	4	0.1	3	0	0	-	0
157	58	8	6.6	40	1.6	0	1.3	15
158	16	11	1.3	11	0	0	-	0
159	58	7	6.7	40	1.4	0	1.2	13
160	58	20	5.0	40	0.5	0	0.7	4.7
161	58	2	7.4	40	3.3	0	1.8	31
162	58	20	5.0	40	0.7	0	0.8	6.2
163	41	22	2.9	28	0	0	-	0
164	54	23	4.2	37	0.2	0	0.4	1.5
170	56	4	6.9	39	2.2	0	1.5	21
171	28	10	3.4	20	0.2	0	0.4	1.5
172	28	0	5.3	20	2.1	0	1.5	
173	28	1	5.1	20	2.0	0	1.4	18
174	28	8	3.8	20	0.2	0	0.4	1.5
175	0	0	-	0	0	0	-	0
176	16	2	3.5	11	0.3	0	0.6	3.1
177	17	6	2.7	12	0.2	0	0.4	1.5
178	15	13	0.5	10	0	0	-	0
179	30	7	4.2	21	0.3	0	0.6	3.1
180	0	0	-	0	0	0	-	0
181	43	23	3.1	30	0.2	0	0.4	1.6
190	58	17	5.4	40	0.2	0	0.4	1.5
191	58	21	4.9	40	0.2	0	0.4	1.5
192	0	0	-	0	0	0	-	0
193	0	0	-	0	0	0	-	0
194	58	9	6.4	40	0.5	0	0.7	4.6

Table 3. (Contd.)

ICD Mn	R = 0.024 Exp.	obs.	t	pop., Mil.	R = 0.0018 Exp.	obs.	t	pop., Mil.
196	0	0	-	0	0	0	-	0
197	0	0	-	0	0	0	-	0
200.0	2.3	2	0.2	1.6	0	0	-	0
200.1	0	0	-	0	0	0	-	0
200.2	2.1	2	0.1	1.5	0	0	-	0
201	29	17	2.2	20	0	0	-	0
202.0	58	0	7.6	40	4.4	0	2.1	40
202.1	0	0	-	0	0	0	-	0
205	58	0	7.6	40	4.4	0	2.1	40
203	58	5	7.0	40	2.3	0	1.5	22
204.0	0	0	-	0	0	0	-	0
204.1	2.1	2	0.1	1.5	0	0	-	0
204.2	0	0	-	0	0	0	-	0
204.3	0	0	-	0	0	0	-	0
204.4	0	0	-	0	0	0	-	0
All other	0	0	-	0	0	0	-	0
Sums	1880	385	-	-	51	0	-	-

THE MULTIPLICATIVE MODEL

Now, none of the foregoing bears on the multiplicative model except, possibly, to strengthen its position by removing a competitor. Indeed, the multiplicative model tends to predict much of what we saw. Relatively rare malignancies might be expected to show $r' = 0$, hence $r = 0$ as well. To be sure, it was a bit distressing that the anticipated mortalities below age 30 did not show up even though r' was clearly < 0 over the remaining age range. But then, this could be repaired by some changes in the values proposed for generalizations (2.), (3.) and (5.). Admittedly, radiation at low dose rates does seem to be remarkably ineffective as a complete pancarcinogen, or even as a complete carcinogen of any sort. But it could well be a pan-co-carcinogen, precisely as envisioned by the multiplicative model.

If this were the case, one would predict a fair increase of malignant mortality with increasing background, and this prediction has been made quite explicit by the model's authors,[2,3] e. g., from 1% to 30% increase at 170 mrem/yr, depending on various assumptions of latency, plateau, and doubling dose.[2,3,32]

With this in mind it was intriguing to note, in Table 1, the resolute insistence on dwelling in regions of high background that seemed to characterize the low mortality groups. At $r = 0.03$ and 0.003 only six groups were at the 170 mrem/yr national average, none were below the average, and at least 40 were above 180 mrem/yr. At first we thought this might only be a secondary association with the well-known urban trend of U. S. cancer mortality.[14,26] Tests failed to substantiate this, however.[24] A white female resident of Dallas, for example (140 mrem/yr), simply seems to be about twice as likely to contract leukemia as her counterpart in Denver (290 mrem/yr). Since we doubted that anyone was prepared to ascribe oncolytic properties to the radiation background, we felt obliged to search for some other association. Surely there must be *some* sort of mortality increase with increasing background.[2,3,32]

However, plots of U. S. rates for white, malignant mortality[5] against natural background for the 50 states showed, if anything, the reverse[54] — e.g., Figure 1. Now, were it not for the insistence of the hypothesis[2,3] that there *must* be a correlation between malignant mortality and background, we would be inclined to dismiss Figure 1 as an example of simple noncorrelation.[46] However, of the 14 states above 140 mrem/yr, 12 were very significantly ($P < 0.01$) below the U. S. average, one insignificantly lower, and only one slightly, but significantly, higher. The probability of this occurring by pure chance proved to be < 0.001. Similar results were obtained with an independent estimate of natural backgrounds.[35]

Several features of Figure 1 might be worth noting. First of all, some states at common background had rates identical to the third significant figure, so that some of the single points actually represent pairs.

Secondly, no error bars are shown because the standard errors are less than the size of the points. The data base is, literally, enormous. Each point represents an average of about 10^5 deaths, and a coefficient of variability, V, of about 0.3%. Even the smallest states are represented

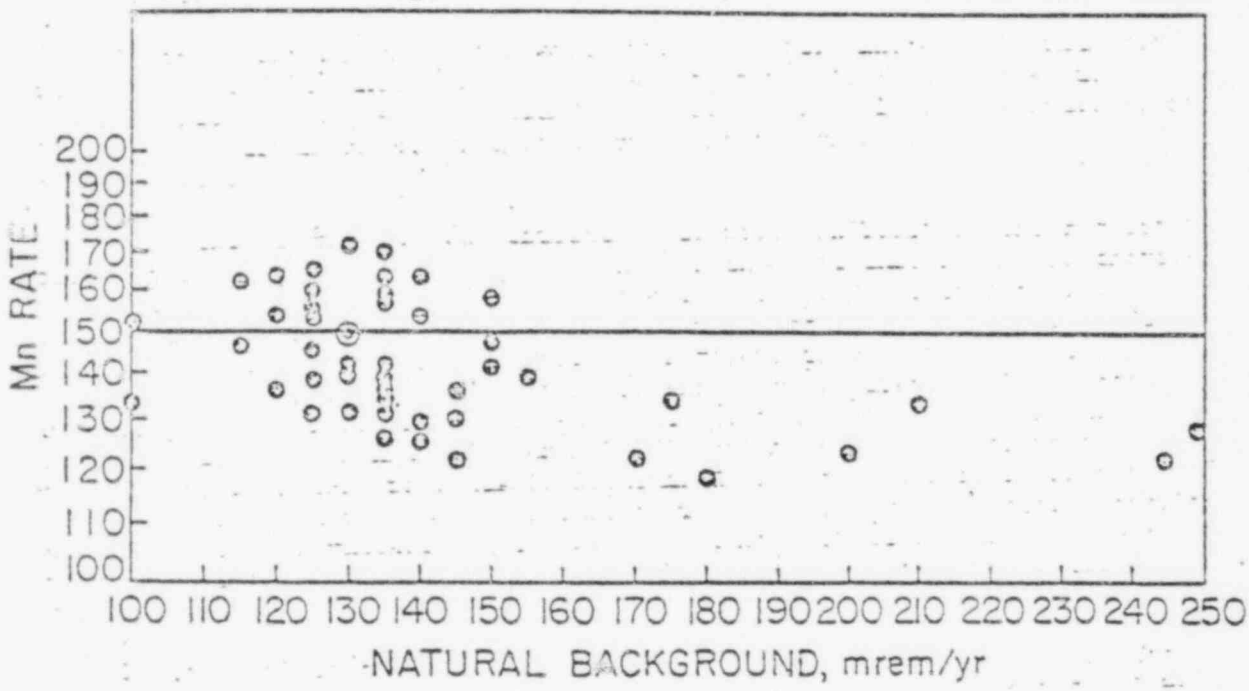

Fig. 1. Malignant Mortality Rates for the U. S. White Population, 1950-1967, by State and Natural Background. The horizontal line and open circle indicate the rate and background for the U. S. as a whole.

by about 10^4 deaths, with V = 1%. With this sort of precision, it is evident that the vertical dispersion displayed is not "scatter", at least not in the usual sense. Rather, it reflects the operation of the genetic, cultural, socioeconomic and other environmental factors so well known in the epidemiology of malignancy.[7-10,14,20,25,26,39,41,43,44,46,47]

Finally, in addition to the seeming negative correlation of rate with background,[49] the ten lowest states in the U. S. all lay at backgrounds of ≥ 135 mrem/yr. Thus, there seemed to be some real, if hidden, association between high backgrounds and low malignant mortalities. Although a similar and even more dramatic effect was noted in the non-white population, we confined ourselves to the white population because of its greater homogeneity, better statistics, the better availability of socioeconomic data, etc.[27,28]

For purposes of further comparison, we discriminated three groups: A, the seven states of natural background above 165 mrem/yr; B, the fourteen states of natural background above 140 mrem/yr; C, the fourteen states with the lowest backgrounds. These were compared with all 50 U. S. states,[24,27,28] and some of the more pertinent results are summarized in Table 4.

We first analyzed the 50 states for each of the 56 (Mn) types to see if the low mortalities of groups A and B could be due to particularly low rates for a few types. These two groups, however, proved to be lower in all categories than the U. S. average, and this premise had to be discarded. A summary is presented in lines 3-7 of Table 3. The rates for all categories, in fact, tended to decrease with increasing background.

Regressions against background and rate were then run for some 30 geographic, climatological and socioeconomic factors in an attempt to find some secondary association.[24] Some of these are summarized in Table 4. No associations were found beyond the obvious ones, i.e., high backgrounds[6,35] in the U. S. tend to be associated with higher altitudes[23] because of the increased cosmic ray component. In the U. S., high altitude states are often located in the more arid regions and can be somewhat less urbanized than plains, or coastal states, e.g., group B. However, none of these associations was of the sort or strength that would be expected to lead to some marked difference in malignant mortality.[7,8,9,14,15,24,25,26,47] In fact, the majority of the associations were in a direction *inverse* to that expected. Thus, the high background groups A and B had a generally lower or equal socioeconomic status (lines 10, 11, 12, 13, 15, 16, 17) to the U. S. or group C, rather than the higher status that usually accompanies lower malignancy rates.[7,8,25,26] The sole exception in this area was the median years of schooling (line 14). Further, A and B had lower Mn rates than U. S. or group C despite a generally higher level of chemical and radioactive pollution (lines 21-24) and also despite a slightly longer life expectancy, which would be expected to *increase* the mortality fraction due to malignancies.[8,26] The larger fraction of militarily acceptable males (line 18) only begs the question of why a rising level of healthy young males should be associated with a rising background.

Urbanization has been associated with increasing malignancy mortality,[14,26] but these groups are, at best, mixed in this respect (line 9). In any case, state-by-state comparisons showed little strength or consistency[26] in this

Table 4. U. S. Low and High Background White Populations, 1950-1967

No.	Characteristic	A	B	U. S.	C
1	Natural background, mrem/yr	210	170	130	118
2	White population, thousands	5735	16,897	158,051	59,683
3	r, Mn 140-159	42.9	45.6	52.4	50.3
4	r, Mn 160-164	15.8	16.9	22.3	23.4
5	r, Mn 170-181	36.8	38.2	41.5	40.1
6	r, Mn 190-205	30.8	31.5	33.3	33.0
7	r, All malignancies	126.3	132.2	149.5	146.8
8	Residence altitude, ft	4510	2650	900	730
9	Urbanization, %	63	57	69	74
10	Per capita personal income, $	2021	1922	2215	2255
11	Median family income, $	5000	5400	5660	5650
12	Physicians/1000 population	1.27	1.25	1.49	1.49
13	Hospital beds/1000 population	8.24	8.82	9.49	8.76
14	Median years of school completed	11.8	11.7	10.9	10.8
15	Poor diet households, %	16.5	21.2	19.1	19.1
16	Population on Federal Food Assist, %	2.6	3.2	3.2	2.5
17	Unemployment, %	4.3	3.9	3.9	3.3
18	Accepted, Military Selective Service	65	63	56	53
19	Life expectancy, male	67.7	67.7	67.6	67.5
20	Life expectancy, female	74.5	74.7	74.2	74.3
21	Urban air, particulates, $\mu gm/m^3$	129	119	115	116
22	Urban air, benzene soluble, $\mu gm/m^3$	10.1	9.3	9.5	9.6
23	Urban air, radioactivity, pCi/m^3	8.5	7.7	6.8	6.3
24	Urban air, beta, pCi/m^3	5.5	5.2	4.4	4.2

Table 4. (Contd.)

No.	Characteristic	A	B	U. S.	C
25	r, Mn 140-205, age 0-9	8.11	8.31	8.54	8.31
26	r, Mn 140-205, age 10-19	6.80	6.61	6.82	6.72
27	r, Mn 140-205, age 20-29	10.46	10.73	11.09	11.19
28	r, Mn 140-205, age 30-39	27.61	28.39	31.45	32.27
29	Mortality rate, all causes	892.0	893.2	928.5	903.9
30	U. S.-group, all causes	36.5	35.2	-	24.6
31	U. S.-group, malignancy	23.2	17.3	-	2.7
32	r, Stomach, 151	11.7	11.6	11.8	11.0
33	r, All G. I., 150-159	40.7	43.0	49.0	46.7
34	r, Lung, 163-164	14.5	15.5	20.4	21.5
35	r, Breast, female, 170	21.5	22.6	25.3	24.4
36	r, Thyroid, 194	0.055	0.054	0.057	0.054
37	r, Bone, 196	0.92	1.03	1.12	1.07
38	r, Leukemia, 204	7.03	7.23	7.13	6.91

area.[24] In addition, comparisons of 16 Standard Metropolitan Statistical areas in groups A, B and C gave the same results as those of the groups *in toto*, i.e., urban areas in A or B showed much lower rates than those in C, even for areas of common socioeconomic and ethnic factors.[24] In short, if any associations existed at all, they were in such a direction as to lead to *increased* rates for A and B. In the face of all this it was difficult see how the multiplicative model could be maintained or, in fact, any model which predicted an increase of malignant mortality with increasing background.

Then, too, the greatly increased sensitivity of the young should lead to a marked increase in malignant mortality.[2,3] Even if the low spontaneous rates of the very young should somehow mask this, combined with the predominantly embryogenic character of childhood malignancy, an increase should certainly be evident by middle age. In fact, the reverse is the case (lines 25-28). The observed rates among the young of groups A and B are decreased relative to those of U. S. and group C, and in much the same ratio as the total malignant mortalities. Even more than the total observations, this left either the additive or the multiplicative model without the attributes of a viable epidemiologic model.[26]

Giving consideration to the fact that radiation cannot be the only carcinogen, that we have far from exhausted the existing low-mortality groups at high backgrounds, even for the U. S., and that we have probably understated the actual exposures of the population by ignoring full medical and dental exposures, weapons, fallout, and regions of high local background, it appears that the actual carcinogenic effects of low-level, low dose-rate radiation are very much less than those predicted from higher level and rate studies,[1-3,13,15,20-22,26,31,34,40] if, in fact, there are any at all. In short, extrapolation from high-rate, small-sample studies predicts a marked increment in malignant mortality rate due to the natural radiation background. Observation of the actual populations at risk shows not only no increment, but an actual decrement.

While identification of the factors at work remains a fascinating exercise for the future, it is not necessary to await such identification to conclude that low-level, low-rate, low LET radiation constitutes a negligible environmental carcinogen. If it is permissible to extrapolate data from Japanese bomb victims, British spondylitics, uranium miners, etc. to the U. S. population at large, then it is certainly far more permissible to extrapolate portions of the U. S. population to itself. This done, the present evidence is quite incompatible with an increase of malignant mortality with increasing background. For a model of background carcinogenesis to remain viable it will be necessary not only to identify reversing factors, but to quantify them with sufficient precision to be certain that they are, indeed, significant factors, and not merely possibilities without quantitative pertinence.

Indeed we claim no novelty for the tests applied above. The existence of geographically contiguous populations of malignant mortalities low enough to place an upper bound on the involvement of uniformly distributed carcinogens has been previously noted,[26,29,41,43] as has the low malignant incidence in at least one high-background city.[30] All that we have done is to quantify these to the point where simple statistical causes can be ruled out.

Certain possibilities, for example, were ruled out by the nature of the observed mortality pattern. Thus, if the decedent populations of groups A or B above were to contain significantly large numbers of immigrants from other parts of the U.S., (i.e., the decedents had not been exposed to the high backgrounds until late in life), one would have expected the rates in groups A and B to be higher than those of the remaining states. This because the Mn rates of the remaining states are much higher than those of A or B, e.g., 150.4 for the U. S.-minus-A, and 151.6 for the U. S.-minus-B. Instead, the reverse was true. Accordingly, if short-term residents are a factor, the true rates for the long-term residents must be even lower than those given in Table 4.

In this context, too, the problem of competing risks arises.[2,46] Thus, are the populations of groups A and B dying of some other cause, so that their members are removed before malignancy can become manifest? Fortunately, the data were at hand to answer this. The age-adjusted mortality rates for all causes could easily be computed, and these are presented in Table 4 (line 29), along with the decrements of each population, relative to the U. S., for all causes (line 30), and for malignancy alone (line 31). All three of these groups are slightly lower than the U. S. in total mortality rate, and by about the same amount. The malignant decrement, however, decreases rapidly with decreasing background. Indeed, in the very highest background group, the malignancy decrement is very nearly equal to the total decrement. This is just the reverse of a case of competing risks, at least in the sense given above.

Again the possibility existed that the residents of group A or B were characterized by much lower radiographic exposures than those of the U. S. This, however, fell of its own weight, since it would have required an excess of over 300 mrem/yr in the rest of the U. S. to account for the difference in group A, and another 80 mrem/yr to account for the expected increase of A due to its high background.[2,3] Such values are hardly credible in view of what is known for U. S. radiographic exposures.[4,12,16,32]

In addition, a multiplicative model predicts a radiation independent rate, r', given by $r' = r/m$. Here r is the observed rate, and $m = (D/DD) + 1$, where D is the total background dose, and DD the doubling dose.[2,3] If this expression truly represented the case one would expect the dispersion of the corrected rates, r', to decrease relative to the dispersion for r.[42] This is the case for such factors as urbanization in total malignant mortality, solar exposure in skin cancer mortality for whites, etc.,[24] so that it should easily be the case for a factor which accounts for 1% to 30% of the total U. S. rate.

Following previous authors[2,3] we took DD = 50 rem for the general population and D as equal to 30 times the total annual background for the State.[6] The 1950-1967 rates, r, were from Burbank[5] as before. For r the coefficients of variability, V, for white males, nonwhite males, white females and nonwhite females were 11.44%, 21.52%, 8.98% and 12.26%,[45] respectively. After correction to r', as above, these values increased to 12.23%, 22.08%, 9.55%, and 12.25%,[45] respectively. Regardless of which of the suggested values[2,3,32] we used for D or DD, V invariably increased, i.e., the results were always the opposite of what would have been expected if the model represented a real factor in U. S. malignant mortality. Furthermore, this increase in V was

found to hold for essentially all U. S. malignancies, even for leukemia, the classic of radiogenic malignancies. Thus, we seemed to be left without statistical support for a multiplicative model, either for all malignancies (pancarcinogenesis), or even for specific ones.

OTHER MODELS

Most of the foregoing would apply with equal force to any model of radiation carcinogenis that predicted increased mortality below the 300 mrem/yr or so that characterizes the highest U. S. populations. However, two such models have been proposed that deserve explicit mention, if only because their tenets are so reasonable.[22,34]

For many forms of cancer the spontaneous incidence rises exponentially with age, i.e., as exp(at), where a is a constant and t is time. It is often postulated that the effect of radiation exposure might be to shift the exponential so that incidence now depends on exp[a(t + b)], where b is a variable depending on radiation dose. This can be understood as either a multiplying effect of radiation or as an aging effect, depending on which axis is shifted to superimpose results from irradiated and unirradiated populations. In either case, though, this predicts a general increase in mortality with increasing background, as well as prominent effects occurring at the earlier ages.[22] Neither of these predictions is compatible with the results presented in the previous section.

A yet more recent model[34] addresses the question of differences in individual sensitivity. Thus, the most sensitive subgroups respond at the lowest doses, so as to cause a steeper-than-linear slope in the low-dose portion of the dose-effect. However, this model predicts even higher mortalities at very low doses than the linear models treated in the previous sections, so that it, too, is incompatible with their results.

Any number of equally reasonable models could certainly be erected but, in the last analysis, reasonableness is not the ultimate criterion of a model. It's not that the models are unreasonable, but that Nature seems to be!

Well, reasonable or not, was she "straight", i.e., linear? We further examined rates for the 7 malignancies that have provided most of the date for present extrapolations.[32] These are arraigned in Table 4, lines 32-38 and, again, the verdict seems to be "not guilty".

For thyroid carcinoma the mortality fraction is so low that incidence is a more useful measure than mortality.[32] The most recent estimates have given 3400-6800 new cases per year, at 170 mrem/yr, over the U. S. population,[32] i.e., a *radiogenic* rate of about 2.6. Against this may be compared Scandinavian[37] and U. S.[8] *total* rates of 0.6-1.3, with several metropolitan populations at 0.0.[8]

Thus, Nature has been anything but "straight" with us, and linearity seems to be, if not deceased, at least moribund.

FUTURE MODELS

Now, statistical analysis can certainly demolish a hypothesis, and this is part of the science of epidemiology. But it can hardly propose one, nor yet can it establish causality.[46] These latter exercises are, really, the "art" of epidemiology and, in the last analysis, its ultimate justification. Hence, we would feel derelict if we did not hazard some opinion as to the direction future models might take.

Examination of generalizations (1.) through (5.) revealed nothing unreasonable and little that was not consonant with what is known. The toxicological dictum that "all men are brothers" has well stood the test of time. And, if radiation at high dose-rates is not pancarcinogenic, it is, at least, polycarcinogenic. The remaining generalizations are primarily numerical, and could be easily modified if need arose.

Perhaps the simplest accommodation would be to abandon the assumption of simple linearity and to substitute some function more dependent on dose rate and dose level, as suggested by parts of the BEIR Report.[32] Certainly the "dose-rate effect" has been known for many years[15] and is, in fact, the basis of the fractionation procedures common in radiotherapy, as well as the low-level, whole-body irradiation methods being attempted for the therapy of some malignancies.

Furthermore, dose rate factors of the order of 5-15 have been reported for large populations of mice irradiated for life at modest rates,[16] and a very strong dose-rate effect has been reported for mutagenesis in mice.[48] A nonlinear model for such diminuition of effect, and even for its reversal,[50] has recently been erected and partially tested.[17] Since linearity has always been an inference rather than an observation,[13,32,53] and since extrapolation over many orders of magnitude[51] has always been a dubious procedure at best,[52] this path of hypothesis rejuvenation appears the most attractive *a priori*. No matter which hypotheses are advanced, however, we hope that the availability of epidemiological techniques and data will come as an "aid and comfort" to the beleaguered radiobiologist in his search for reliable estimates of the effects of radiation on human populations.

CONCLUSIONS

In an attempt to uncover some secondary association between rising background and falling malignancy rate, regressions were run for additional factors beyond those shown in Table 4. These are summarized in Table 5. Again, no meaningful associations were found beyond the obvious ones, i.e., high backgrounds tend to be associated with high altitudes because of the increased cosmic ray component. However, in the U. S., high terrestrial backgrounds accompany high altitudes because of the particular geology of mountainous regions.[6,23,35] Thus, as seen on lines 1 and 2, both components decrease going from group A to group C. Thus, as one would expect, groups A and B tended to be drier, cooler, higher, sunnier, (lines 3-7) than group C, and to require more domestic heating (line 8). (The latter may account for the higher air pollution levels noted in lines 21 and 22 of Table 4.) We know of no observation, or even hypotheses,[7-10,25,26,41,43,44,46] that would causally link these factors with decreased malignancy. Thus, we were forced to conclude that their association with increased backgrounds was an accident of altitude and geology, and not significant in the observation of decreasing malignant mortality rates with increasing background.

A number of other regressions of possible socioeconomic indicators were also run and are presented in Table 5. However, none of these provided any secondary associations that might be expected to lead to decreased malignancy. At best, the groups were mixed, and showed no correlation. At worst, the correlations detected were in a direction that should have led to increased malignancy in groups A and/or B.

Although the dose estimates used[6,35] are the result of careful, independent and prolonged studies, and certainly the best values available, we reexamined them for the possibility that the results which we obtained were an artifact of dose estimates. This did not appear to be the case. Groups A and C, especially, consisted of states where simple altitude and geological considerations, coupled with repeated measurements over the years, have shown these states to be quite disparate in background. The majority of these states, in fact, have been among the classical examples of high and low background states for decades. In addition, study of the sources cited suggested the true backgrounds in groups A and B were very probably higher than those used in that houses were most often made of stone or concrete of high radioactivity, indoor air had often been shown to be very high in radon, and the population spent a larger fraction of time indoors because of the relatively inclement weather compared to the rest of the U. S. or to group C. Thus, if anything, the background of groups A and B had been underestimated relative to the U. S. or to group C.

The current models of radiation carcinogenesis have all derived their data from small, selected, populations, at high dose rates and generally at high dose levels. They have assumed monotonic extrapolation to zero dose, even though this assumption has always been without observational basis.[13,32] They have also assumed such extrapolation can be made without consideration of dose rate, an assumption not only without observational basis, but one contradicted by a large body of radiological and toxicological

Table 5. Characteristics of U. S. White Populations at Low and High Backgrounds

No.	Characteristic	A	B	C
1	Cosmic ray dose, mrem/yr	105	72	42
2	Terrestrial dose, mrem/yr	80	72	51
3	Mean altitude, feet	5400	2900	1300
4	Annual mean temp., °F	50	51	59
5	Annual precip., inches	14	25	36
6	Days/yr with precip.	86	97	99
7	% of possible sunshine	69	65	63
8	Annual degree-days (65°F)	6300	6100	3550
9	Murder, per 10^5 pop.*	3.9	4.0	4.9
10	Rape, per 10^5 pop.	9.9	7.6	9.7
11	Robbery, per 10^5 pop.	39.8	30.2	49.2
12	Agg. assault, per 10^5 pop.	37.2	64.9	82.0
13	Burglary, per 10^5 pop.	476	399	574
14	Larceny, per 10^5 pop.	331	260	297
15	Auto theft, per 10^5 pop.	218	167	209
16	Lawyers, per 10^5 pop.	146	125	133
17	Marriages, per 10^5 pop.	993	1114	783
18	Divorces, per 10^5 pop.	276	242	237
19	Urban births, per 10^5 pop.	1563	1381	1423
20	Rural births, per 10^5 pop.	1025	1082	818

*Lines 9-18 are for total populations.

data.[16-17,24] On these bases predictions have been made of significant increments in malignant mortality rates due to the radiation background. Observation of actual populations at risk shows not only no increment but an actual decrement, and these predictions are left quite without observational support. Thus, it appears that one or both of the above assumptions is invalid and that background radiation does not constitute an environmental carcinogen of significance. By the same token, the radiation added by nuclear power plants cannot be a carcinogenic hazard either, since it has the same radiobiological character as the current background, but is much lower in both dose level and rate.

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Health Survey in High Background Radiation Areas in China

High Background Radiation Research Group, China

Since 1972 we have studied the health status of a large number of people living in some parts of Yangjiang County, Guangdong Province, China, where the radiation level is about three times that in the neighboring areas. We hope that this investigation of a large population whose families have been continuously exposed to a low dose rate of ionizing radiation for many generations may provide some information for evaluating whether any detrimental effect of such exposure exists.

High-Background and Control Areas

The high-background areas in Yangjiang County are two regions separated by a short distance, which cover a total area of about 540 square kilometers. The source of background radiation in the first region (Dong-anling) is a hill whose surface rocks are granites, from which fine particles of monazite are washed down continually by rain and deposited in the surrounding basin region. A similar mechanism operates in the second region (Tongyou): monazites washed down from a mountain near Tongyou are deposited in another basin region and elevate its background radiation level.

More than 80,000 people live in these two regions. For the purpose of this investigation, those who live at the borders of the high-background areas were not examined; therefore, only about 73,000 people were investigated.

Two control regions were selected not far from the high-background areas (the closest points are about 10 km apart). Both types of regions are at comparable altitudes, less than 50 meters, and there are 77,000 inhabitants in the control regions (Fig. 1).

Nationality, occupation and history of inhabitation. From the general records of these counties, the family trees of the inhabitants, and interviews with aged people, we learned that the overwhelming majority of the inhabitants of the regions studied are of the Han nationality and most of them are peasants and their families. They have lived there for many generations. The inhabitants whose families have lived in the high-background areas for six or more generations comprise 90.6 percent of the total (see Table 1).

Medical exposure. During the investigation, inhabitants who had been occupationally exposed to ionizing radiation were not examined. A survey of medical exposure was carried out (Table 2).

Results of the investigation carried out from 1972 to 1975. In the period 1972 to 1975 we investigated only some of the inhabitants and their environments. In the Dong-anling (high-background) region, 20,154 people were studied; in the Sanhe (control) region, 21,235 people. The investigation included radiation level and dosimetry; chromosomal aberrations of peripheral lymphocytes; frequencies of hereditary diseases and deformities; frequencies of malignancies; growth and development of children; status of spontaneous abortions; and some nonstochastic effects.

Table 1. Distribution of inhabitants whose families lived in investigated areas for various numbers of generations.

Generations	High-background areas		Control areas	
	Persons	Percent	Persons	Percent
2 to 5	6,912	9.4	25,126	32.6
6 to 10	25,737	35.0	39,220	50.9
11 to 15	17,501	23.8	9,172	11.9
16 or more	23,384	31.8	3,545	4.6
Total	73,534	100.0	77,073	100.0

Table 2. Status of medical exposure (person-times per 10^3 persons per year).

Areas	Fluoroscopic examination		
	Chest	Gastrointestinal	Pelvic
High-background	36.2	1.0	27.7
Control	28.7	2.5	19.7

Table 3. Contents of natural radionuclides in surface soil.

Areas	Uranium (10^{-3} g/kg)	Thorium (10^{-3} g/kg)	^{226}Ra (10^{-9} Ci/kg)	^{40}K (mg/kg)
High-background	5.8 to 9.2	35.0 to 49.7	1.7 to 2.0	1.3 to 2.5
Control	1.5 to 3.3	4.3 to 10.7	0.3 to 0.9	0.2 to 0.5

Table 4. Cumulative exposures measured by $CaSO_4$ and fluoroglass dosimeters. Values are means ± standard deviation (S.D.).

Areas	Exposure (mR/month)		
	Field	Indoor	Individual
	$CaSO_4$ dosimeters		
High-background	14.5 ± 2.5	28.7 ± 3.6	25.0 ± 4.0
Control	5.9 ± 1.2	9.4 ± 2.2	9.3 ± 2.3
	Fluoroglass dosimeters		
High-background	19.5 ± 2.4	28.5 ± 3.9	25.0 ± 2.7
Control	7.5 ± 1.8	10.5 ± 2.2	9.2 ± 1.9

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Radiation Level and Dosimetry

Contents of natural radionuclides in the surface soil were examined by radiochemical analyses. The results are shown in Table 3.

Cumulative exposure to radiation. Both calcium sulfate and fluoroglass dosimeters were used to determine the cumulative exposure of the inhabitants to their environments. According to the exposure distribution in the investigated regions, which was measured with radiation detectors, 14 villages in the high-background area and 16 villages in the control area were selected; their inhabitants comprised approximately one-fourth of the total number investigated. By a random process, 408 people in the high-background area and 417 in the control area were selected to wear the dosimeters: 56 in the high-background area and 64 in the control area wore both types of dosimeters, the others wore calcium sulfate dosimeters only. They wore the dosimeters around their waists in the daytime and put them on their beds at night for a period of 2 months. Meanwhile, dosimeters were also placed on stakes at approximately 1 m above ground level in the rooms and fields of these villages: 1019 calcium sulfate dosimeters and 314 fluoroglass dosimeters were issued for this purpose, and 940 and 312 dosimeters, respectively, were returned to us in readable condition. The results are shown in Table 4.

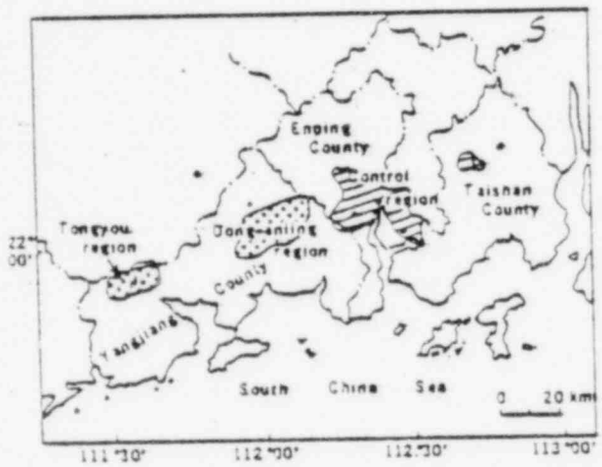

Fig. 1. Locations of the high-background and control regions.

The results obtained with the two kinds of dosimeters are almost identical. Doses absorbed by the gonads, whole body, red bone marrow, and endosteal cells were calculated. The average dose absorbed per year from external radiation was 196 millirads for those in the high-background area and 72 millirads for those in the control area.

Internal radiation. To ascertain the daily intake of natural radionuclides, a dietary investigation was carried out. Samples of local foodstuffs, vegetables, and drinking water were collected and analyzed by radiochemical procedures. Body burdens of natural uranium, natural thorium, ^{226}Ra, ^{228}Ra, and ^{40}K were calculated and the doses they contribute annually to the endosteum, bone marrow, and gonads were estimated. In addition, the concentrations of ^{220}Rn, ^{222}Rn, and their daughter products in the air indoors and outdoors at various times of day were measured and the annual doses to the lungs were estimated; these are shown in Table 5.

Table 5. Annual dose equivalents (millirems per year).

Exposure	High-background areas				Control areas			
	Whole body	Gonads	Endosteum	Bone marrow	Whole Body	Gonads	Endosteum	Bone marrow
External radiation	196	196	196	196	72	72	72	72
Internal radiation								
^{226}Ra	4.0	4.0	58.8	10.7	1.3	1.3	19.1	3.5
^{228}Ra	10.5	10.5	156.5	28.5	2.7	2.7	40.9	7.4
^{40}K	19	19	15	15	19	19	15	15
Daughter products of ^{222}Rn	1.3	1.3	1.5	1.5	0.7	0.7	0.9	0.9
Daughter products of ^{220}Rn	0.5	0.5	8.9	8.9	0.3	0.3	4.7	4.7
Total	231	231	437	261	96	96	153	104

Table 6. Incidence of rings and dicentrics.

Areas	Cases	Cells scored	Rings + dicentrics per 100 cells	P
		1973		
High-background	95	9,642	0.031	> .20
Control	95	12,757	0.024	
		1975		
High-background	106	21,144	0.019	> .10
Control	104	20,778	0.005	

Table 7. Incidence of chromatid and chromosomal aberrations of peripheral lymphocytes (1975).

Areas	Cases	Cells scored	Number of		
			Cells with chromatid or chromosomal aberrations	Chromatid aberrations	Chromosomal aberrations
High-background	106	21,144	232 (1.097)*	190 (0.899)	57 (0.269)
Control	104	20,778	205 (0.987)	154 (0.741)	57 (0.274)

*Figures in parentheses are changes per 100 cells.

Results of Health Survey

Cytogenetic study. Before blood sampling, clinical examinations of a large number of inhabitants of both areas were performed to rule out those who suffered from diseases associated with chromosomal aberrations and those who had been exposed to medical x-rays and agricultural insecticides not long before the examination. Thus, in the first period (1973 and 1974) 95 inhabitants each from the high-background and control areas, and in the second period (1975) 106 inhabitants from the high-background area

and 104 from the control area were selected for analysis of lymphocyte chromosomal aberrations. Since the durations of blood culture in the two periods were different (72 hours in 1973–1974 and 54 to 56 hours in 1975), the results are listed separately (Tables 6 and 7).

The results showed that the frequencies of chromatid and chromosomal aberrations among inhabitants of the high-background and control areas are almost identical, within the normal ranges. The incidence of chromatid and chromosomal aberrations in 1975 is shown in Table 7.

Hereditary diseases and congenital deformities. Infants and children below 12 years of age in both types of areas were examined clinically by experienced physicians, pediatricians, dermatologists, and gynecologists for 31 kinds of diseases and defects. Diagnoses were checked once or twice whenever necessary. The chromosome number of some patients who suffered from Down's syndrome was determined to confirm the diagnoses.

The results of these examinations are shown in Table 8. The frequency of hereditary diseases and congenital deformities in the high-background area is somewhat lower than that in the control area, but the difference is not statistically significant. The incidence of Down's syndrome in the high-background area (1.71 per thousand) is significantly higher than that in the control area (zero). However, the size of population investigated is quite small to show this difference. It will be necessary to examine more children in further investigations.

Spontaneous abortion rate. A retrospective survey of the incidence of spontaneous abortions was carried out in both areas. In the high-background and control areas, respectively, 1551 and 1716 married women were interviewed about the courses of their pregnancies from 1963 to 1975. Spontaneous abortion was defined as termination of any pregnancy within 28 weeks after conception, excluding artificial abortion. Results obtained from both areas were almost identical (Table 9).

Frequency of malignancies. Both general clinical examinations and 5-year (1970 through 1974) retrospective surveys were carried out to determine the frequency of malignancies in the high-background and control areas. Materials and information were collected from demographic survey and hospital records. Death rates were standardized according to the distributions of age and sex of the inhabitants in both areas. Results are shown in Tables 10 to 12.

Neither the morbidity rates from general clinical examinations nor the mortality rates from retrospective surveys showed significant differences between inhabitants living in the high-background and control areas. It was reported that the average annual mortality rate from malignancies between 1970 and 1972 in the whole Guangdong Province was 50.90 per 10^5 persons; in the Hupei Province between 1971 and 1973 it was 68.34 per 10^5 persons. The mortality rates in both investigated areas were comparable with that in Guangdong Province, but lower than that in Hupei Province.

Table 8. Frequencies of 31 kinds of hereditary diseases and congenital deformities of children in high-background and control areas.

Areas	Persons examined	Persons diseased	Frequency (per 1000)	P
High-background	3504	48	13.70	> .05
Control	3170	46	14.51	

Table 9. Spontaneous abortion rate from 1963 to 1975.

Areas	Pregnancies	Abortions	Abortion rate (per 1000)	P
High-background	3896	288	73.9	> .05
Control	3062	222	72.5	

Table 10. Frequencies of malignancies observed in 1975.

Type of malignancy	High-background area (N = 20,154)		Control area (N = 21,235)	
	Cases	Frequency (10^{-5})	Cases	Frequency (10^{-5})
Cancer of nasopharynx	0	0	3	14.13
Cancer of esophagus	0	0	1	4.71
Cancer of liver	3	14.85	2	9.42
Cancer of intestine	2	9.92	1	4.71
Cancer of breast	0	0	1	4.71
Cancer of cervix	0	0	3	14.13
Other cancers	2	9.92	3	14.13
Total	7	34.69	14	65.94

Table 11. Mortality rates from malignancies in high-background and control areas (1970 through 1974, retrospective survey).

Areas	Person-years	Deaths (No.)	Mortalities averaged (10^{-5})	Mortalities standardized (10^{-5})
High-background	96,533	45	46.61	45.69
Control	122,554	55	44.87	44.83

Table 12. Mortality rates from various malignancies in high-background and control areas (1970 through 1974, retrospective survey). Data were standardized according to age and sex.

Type of malignancy	High-background areas		Control areas	
	Deaths (No.)	Mortality (10^{-5})	Deaths (No.)	Mortality (10^{-5})
Cancer of liver	12	12.10	21	16.83
Cancer of nasopharynx	11	11.37	12	9.34
Cancer of stomach	6	6.39	1	0.77
Cancer of cervix	3	3.19	1	0.69
Cancer of esophagus	2	1.60	3	2.07
Cancer of lung	1	0.23	4	2.94
Cancer of intestine	0	0	3	4.20
Cancer of breast	0	0	1	2.57
Leukemia	5	5.31	5	4.14
Other cancer	5	4.71	4	3.09

The mortality rates from leukemia, about which we were more concerned, were not significantly different between both areas. Nevertheless, the size of the population investigated in this period was relatively small, and further study is necessary.

Growth and development of children. Measurements of head circumference, body weight, and height of the children below 12 years of age in the high-background area (3239 persons) and the control area (2991 persons) showed that differences in growth and development of children between these two areas were not statistically significant.

Conclusions

The radiation level in some regions of Yangjiang County, Guangdong Province, is about three times that in the neighboring control areas, but lower than that in some parts of high background radiation areas in India and Brazil. However, the distribution of exposure rates in the investigated regions is relatively even, and there is a high density of people whose families have lived there for many generations. Results of the health survey carried out between 1972 and 1975, which did not demonstrate any significant difference between inhabitants living in the high-background and control areas, suggest that the size of the population investigated may be not large enough to reveal minor increments of detrimental effects at such a low dose range of ionizing radiation. Or there might be a practical threshold dose; that is, the possibility that the dose-effect curve had a zero slope at these doses cannot be ruled out. For the reasons given above, further investigation of a larger population is necessary.

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Innovation and Scientific Funding

Richard A. Muller

It is difficult to judge the performance of scientific funding agencies, for, like physicians, they often bury their mistakes. Rejected proposals usually mean doomed projects. If the projects survive rejection and succeed, it is rare that they achieve recognition soon enough to alert the funding agencies that mistakes are being made. In 1978 I was given the Alan T. Waterman Award of the National Science Foundation and the Texas Instruments Foundation Founders' Prize for research that initially had been rejected for funding by the National Science Foundation (NSF), the Department of Energy (DOE), the National Aeronautics and Space Administration (NASA), and the Department of Defense. I felt an obligation to make my experience known, not because I thought it unique, but because of my unique position as the recipient of the awards. A discussion with Dr. Frank Press of the White House Office of Science and Technology Policy led to meetings with agency heads and testimony before the Committee on Science and Technology of the U.S. House of Representatives. This article is an adaptation of that testimony.

I was able to proceed with the rejected projects by "circumventing the system." I had been advised by my mentor, Luis Alvarez, to spend money designated for other projects on the unfunded work. He said that if the projects were successful, nobody would question the propriety of having done this. I was helped by our NASA funding monitor, who allowed us to designate a fraction of one of our grants as "seed money" for new projects, as long as the amount was small and remained "low profile." In addition, I was able to obtain some seed money from the Lawrence Berkeley Laboratory, although those involved felt that they were taking a risk, since the projects were not immediately relevant to the DOE's mission.

It is well known in the research community that one cannot expect a proposal to be funded until a considerable amount of work has been done on the project. When I began research in 1965, our research group often received more than the minimum support necessary for our projects, and the excess money was used to seed new ideas. Only a small fraction of these ideas led to a formal proposal. If the proposal was funded, it could provide seed money for the next idea.

This situation gradually changed. By 1972 our proposals were scrutinized to ensure that we received no more than the necessary minimum. Rarely did we receive the total requested. By 1976 few of our proposals received enough money even to sustain a project, and we had to obtain support from more than one agency. Much of the time we had devoted to thinking about new projects was now spent writing and polishing proposals. Tight funding, increasing overhead, and additional constraints on spending have made it more and more difficult to begin new projects. Fortunately, the Lawrence Berkeley Laboratory has continued to provide seed money, making it possible for our research program to continue to evolve.

Innovation

I have originated several project termed innovative by the award committees and others. The periods when

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EVALUATION OF THE LONG-TERM EFFECTS OF HIGH BACKGROUND RADIATION ON SELECTED POPULATION GROUPS ON THE KERALA COAST*

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Abstract–Résumé–Аннотация–Resumen

EVALUATION OF THE LONG-TERM EFFECTS OF HIGH BACKGROUND RADIATION ON SELECTED POPULATION GROUPS ON THE KERALA COAST.

Investigations on human populations which are subjected to high levels of natural background radiation offer possibilities for evaluating the long-term effects of chronic radiation exposure in man. Monazite-bearing high-radiation areas exist along the south-west coast of India. Here a strip of about 55 km has definable geographical landmarks, includes the most concentrated distribution of monazite and supports a high density of population. Demographic data, including detailed fertility history for 13 355 households (about 70 000 individuals) of this strip were collected. Dosimetric survey of 20% of this population using calcium fluoride thermoluminescent dosimeters was completed. Processing of a representative sample of the dosimetric data revealed that about 20 600 individuals in this strip are likely to be receiving radiation dose between 5 to 10 times normal background. These data are being analysed to yield information on: (i) classification of population into sub-samples according to radiation exposure; (ii) sex-ratio among offsprings in each sub-sample; (iii) ratio of abortions and still-births to total pregnancies; (iv) ratio of congenital abnormalities to total live-births; and (v) infant mortality rates in each sub-sample. Studies of chromosomal patterns in new borns in this population and interlinking with radiation exposure history of parents have been initiated.

EVALUATION DES EFFETS A LONG TERME D'UNE HAUTE RADIOACTIVITE NATURELLE SUR DES GROUPES SELECTIONNES DE LA POPULATION DES COTE DE KERALA.

Une enquête sur les populations qui sont exposées à une haute radioactivité naturelle permet d'évaluer les effets à long terme de l'exposition chronique aux rayonnements chez l'homme. On trouve des régions à haute radioactivité naturelle, due à la présence de monazite, sur la côte sud-ouest de l'Inde. Une bande d'environ 55 kilomètres, géographiquement bien délimitée et à forte densité de population, a les concentrations les plus élevées en monazite. Les données démographiques, comprenant des détails sur la fécondité de 13 355 familles (environ 70 000 personnes) de cette région, ont été recueillies. L'étude dosimétrique de 20% de cette population a été effectuée à l'aide de dosimètres thermoluminescents au fluorure de calcium. Le traitement d'un échantillon représentatif des résultats dosimétriques a montré qu'environ 20 600 individus de cette région sont exposés à des doses de 5 à 10 fois supérieures au rayonnement naturel normal. On analyse ces renseignements en vue d'établir: i) la classification de la population en sous-échantillons selon la radioexposition; ii) la proportion des sexes dans la progéniture pour chaque sous-échantillon; iii) la proportion des avortements et des mort-nés en fin de grossesse; iv) la proportion des difformités congénitales pour la totalité des enfants nés vivants; v) les taux de mortalité infantile dans chaque sous-échantillon. On a commencé des études sur les types-chromosomiques parmi les nouveau-nés de cette population et sur l'influence des antécédents de radioexposition des parents.

* This work has been supported in part by WHO under an Agreement dated 23 Dec. 1965.

ОЦЕНКА ДЛИТЕЛЬНОГО ВОЗДЕЙСТВИЯ ВЫСОКОГО ФОНОВОГО ИЗЛУЧЕНИЯ НА ОТДЕЛЬНЫЕ ГРУППЫ НАСЕЛЕНИЯ ПОБЕРЕЖЬЯ КЕРАЛА.

Исследования популяций человека, подвергшихся воздействию высокого фонового природного излучения, открывают возможности для оценки длительного влияния облучения на человека. Вдоль юго-западного побережья Индии расположены залежи монацита высокой активности. Полоса, длиной около 55 км, имеет наиболее концентрированные месторождения монацита и высокую плотность населения. В этом районе были собраны демографические данные, включая подробные данные о рождаемости, у 13 255 семей (примерно 70 000 индивидуумов). Было проведено дозиметрическое обследование 20% населения с помощью термолюминисцентных фтористо-кальциевых дозиметров. Обработка большого количества дозиметрических данных показала, что 20 000 индивидуумов в данном районе, по всей вероятности, получили дозу облучения, в 5-10 раз превышающую обычный фон. Эти данные анализируются, чтобы получить информацию по следующим пунктам: i) классификация населения на подгруппы по дозе облучения; ii) соотношение полов среди потомков каждой подгруппы; iii) отношение выкидышей к мертворожденным детей к общему количеству беременностей; iv) отношение врожденных аномалий к общему числу нормально рожденных детей; v) процент смертности детей в каждой подгруппе. Были начаты исследования хромосомных моделей новорожденных этой популяции и их взаимосвязи с историей облучения родителей.

EVALUACION DE LOS EFECTOS A LARGO PLAZO DE FONDOS RADIACTIVOS CONSIDERABLES EN LA COSTA DE KERALA SOBRE GRUPOS SELECCIONADOS DE POBLACION.

Las investigaciones sobre las poblaciones humanas que están sometidas a fondos radiactivos naturales elevados ofrecen la posibilidad de evaluar los efectos a largo plazo de la exposición crónica a la radiación en el hombre. A lo largo de la costa sudoccidental de la India existen zonas de radiación elevada, por su contenido en monacitas. En este lugar, una franja de unos 55 km con límites geográficos definidos, presenta la mayor concentración de monacitas y tiene una gran densidad de población. Se han recogido datos demográficos, un historial detallado de la fertilidad en más de 13 255 círculos familiares (unos 70 000 individuos) de esta franja. Se ha finalizado un estudio dosimétrico del 20% de esta población utilizando dosímetros termoluminiscentes del fluoruro cálcico. La elaboración de los datos dosimétricos de una muestra representativa reveló que unos 20 000 individuos de esta franja está recibiendo muy probablemente dosis de radiación comprendidas entre 5 y 10 veces el fondo normal. Estos datos se están analizando para obtener información sobre: 1) la clasificación de la población en muestras estratificadas de acuerdo con la exposición a la radiación; 2) la relación de sexo en la descendencia de cada estrato muestral; 3) la relación de abortos y nacidos muertos a embarazos totales; 4) la relación de anormalidades congénitas al total de nacidos vivos, y 5) las tasas de mortalidad infantil en cada estrato muestra. Se han iniciado estudios de los modelos cromosómicos en los recién nacidos de esta población y su conexión con el historial de exposición a la irradiación de los padres.

1. INTRODUCTION

Ever since the demonstration that ionizing radiations are mutagenic in lower forms of life, there has been an increased tendency on the one hand to regard all unwanted exposures to these radiations as harmful and on the other to identify areas where human populations are subjected to high levels of natural background radintions. It has been the fervent hope that investigations on such populations may perhaps lend themselves to critical analysis and thus provide more meaningful answers to yet unresolved questions: Do chronic exposures of human populations indeed result in genetic harm? And are estimates of risk based on animal experiments indeed valid for the human situation? The task we have set ourselves is complex and direct answers to these questions are unlikely to emerge in view of the many imponderable variables in the nature and styles of life of the populations studied.

One such geographical area, where large human populations are involved, lies along the west coastal region of Kerala and Tamil Nadu (Fig. 1).

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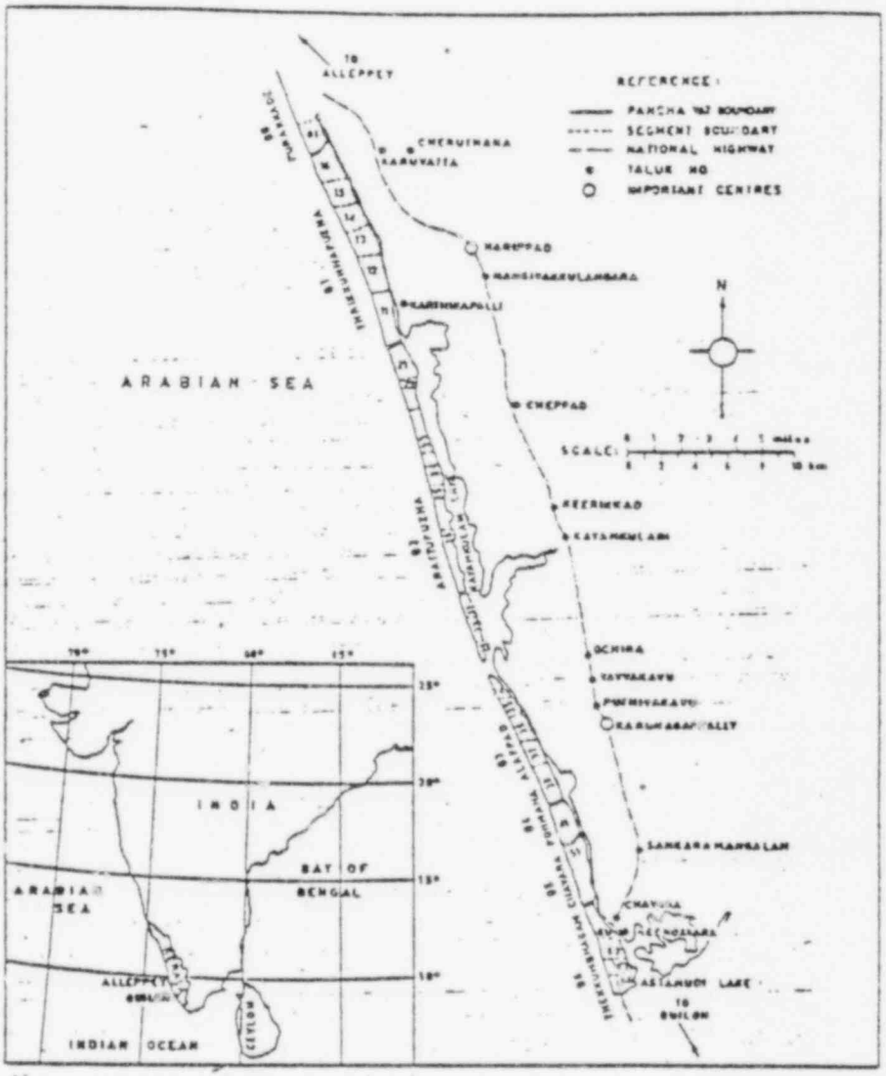

FIG.1. Monazite bearing area surveyed in Kerala.

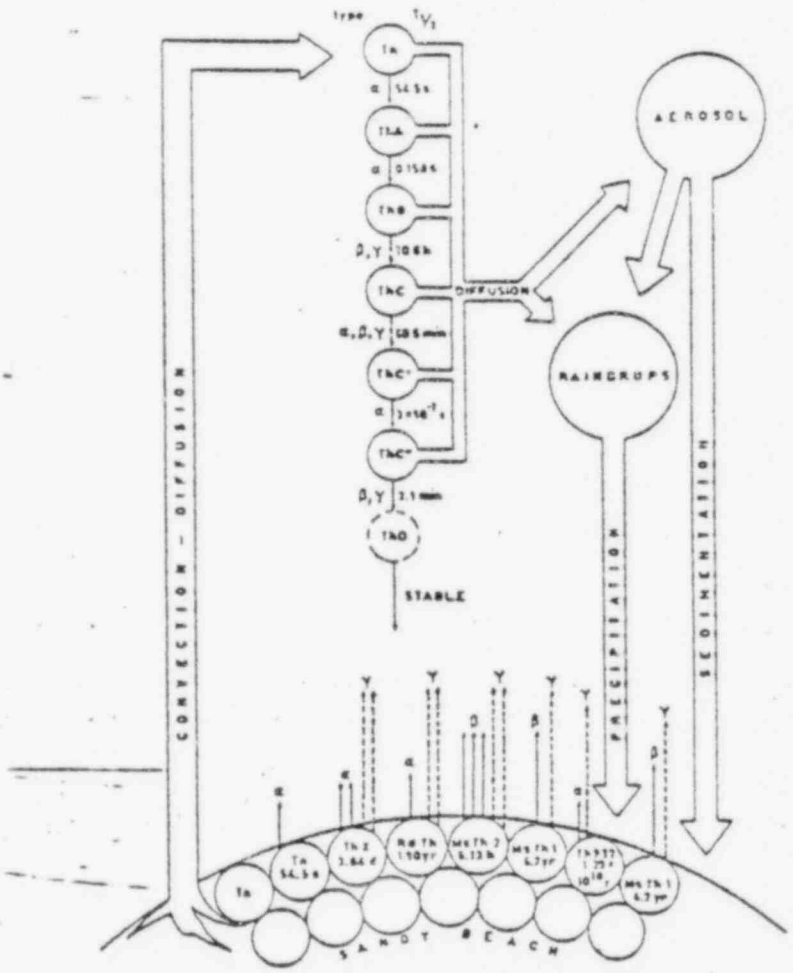

FIG. 2. Schematic representation of the decay chain of thorium and its daughter products.

Here over a stretch of about 60 kilometres the coastline is characterized by patches of radioactive sand. For the purposes of our investigations we have selected a coastal strip of about 55 km extending from Thekkumbhagum in Quilon District in the south to Purakkadu in Alleppy District in the north. This area has certain features: (i) the area includes the most concentrated distribution of monazite; (ii) it has definable geographical land marks with the backwaters separating this strip from the mainland; (iii) it carries a high density of population.

The monazite deposits admixed with ilmenite, rutile, zircon and other rare earths are widely distributed in the beach sands and in the adjoining areas along this coastal strip. Thorium and its radioactive decay products contribute to the high background radiations. Figure 2 presents a schematic representation of the decay chain of thorium and its daughter products. It is evident that during the decay of thorium, alpha, beta and gamma radiations are released – as is also radioactive thoron, which diffuses out of the sands and contributes to the contamination of the air. Therefore, the human populations living in these areas are subjected to radiation exposures from: (i) external radiations caused by beta and gamma radiation from natural uranium and thorium contained in monazite; (ii) beta and gamma radiations from radon, thoron (gaseous products) and their decay products in the air; and (iii) internal exposures from deposition of these radioactive materials in the body through ingestion and inhalation.

This paper reports on the present status of our studies in the region. During the current phase of the project, two aspects have been taken up:

(a) An ad hoc demographic survey;

(b) Radiation dosimetric measurements of households and personnel.

A preliminary progress report on these investigations was published during 1970 [1]. The data reported therein which pertain to a small sub-sample of the households and population in the monazite belt, are incorporated in the present paper.

2. MATERIALS AND METHODS

2.1. Demographic survey

The demographic survey was completed in January 1970 with the assistance of the Bureau of Economics and Statistics, Kerala State. The region under study is divided into 7 areas and each area is subdivided into segments, and each house in a segment is given an individual house number. In all 13 355 households are included in the region and nearly 70 000 persons representing all age groups and different religions are covered. A batch of investigators visited each household and the demographic data were collected in computer-compatible formats [2]. The population has been divided into three occupational groups representing: (I) individuals employed outside the area; (II) individuals totally employed within the area; and (III) fishermen. The occupational Group I includes children going to school and other individuals who spend a good part of the day outside the area, but does not include fishermen. Occupational Group II includes male members who normally work in the area, housewives, and children who do not go to school but spend the day in and around the house. Occupational Group III consists of all persons who go fishing and spend the rest of their time in and around the area. This classification is important since human beings are mobile and the fact that they are resident in a high background area may not necessarily imply that they always receive high exposures. For each married couple in a household, a detailed fertility history has also been obtained.

A preliminary inquiry in the region revealed that there are various religious groups clustered discretely along the coast and it was difficult to get a suitable control population which would meet all requirements outside the region. Further, information obtained from our earlier radiometric surveys [3-6] indicated that along the coast the high radiation belt is frequently interrupted by stretches with normal background radiation levels. In view of this unique situation, it was felt that a sizeable fraction of the population would be receiving low or near normal background levels of radiation exposure which could justify this fraction being considered as a suitable control group.

2.2. Dosimetric survey

Our earlier radiometric surveys have demonstrated the patchy distribution of monazite sand in the Thekkumbhagam to Purakkadu region. In addition, the radioactivity in a given zone is relatively high near the shoreline and generally falls off as one moves inland by about 400 m [5]. These measurements were made by the conventional G-M survey meters. In the absence of reliable personnel integrating dosimeters, no measurements on individuals residing in the area could be carried out earlier. These studies however highlighted the complexity of the situation and also indicated that the computation of per capita annual dose on the basis of a single G-M measurement was likely to overestimate the actual doses received by the individuals in the population.

2.2.1. Dosimetric system

The development of thermoluminescent dosimeters, and especially calcium fluoride dosimeters at the Bhabha Atomic Research Centre, has now made it possible to measure the radiation dose received by individual members of the population with a reasonable measure of accuracy, reliability and reproducibility.

Dosimeter design and reader system have been reported earlier [7-9]. The dosimeter consists of a 40 mm × 12.5 mm × 0.25 mm Al variety kanthal strip at the centre of which fluorite TLD powder (50 to 100 mg, mesh 120) is deposited and secured by a silicone resin. For reading the light output from an exposed dosimeter, an EMI type 9514S photomultiplier tube is used, whose output is recorded when the dosimeter is heated under its photocathode. Minor modifications were made on this dosimeter for its use in the present work: A 15 mm × 10 mm × 0.5 mm depression was embossed at the centre of the kanthal strip, ensuring firm deposition of the fluorite. The kanthal strip dosimeter was enclosed in a light-tight black PVC bag which was in turn finally sealed inside another PVC packet.

Figure 3 shows the dosimeter which was supplied to the individuals. Female members of the population wore these dosimeters around the neck, children and male members wore them around their waist. The effect of variations in the geometry of these dosimeters on the exposure recorded was not significant.

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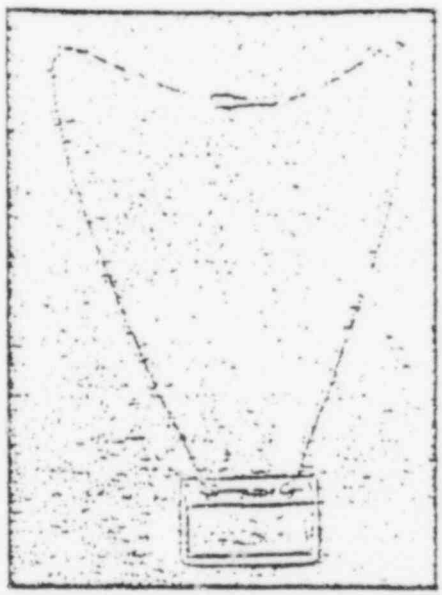

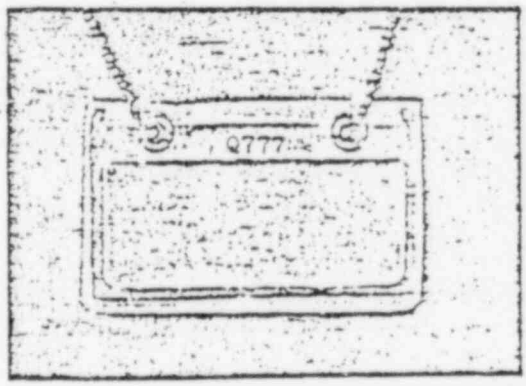

FIG.3. A typical CaF_2-TLD with chain.

2.2.2. Calibration procedure

Calibration of the dosimeters was carried out using a 9.14 mCi standard radium source enclosed in a 0.0625 in (1.588 mm) thick Perspex capsule. Dosimeters were arranged along the circumference of a 10 cm radius ring with the source at the centre. Twenty-eight dosimeters were placed in the calibrating ring at a time. Initially full range calibration was done for a few dosimeters and the range of linearity

was established. Linearity extends from 10 mR to 10^4 R [7]. There was no change in the calibration of the dosimeters, even after repeated use. In view of the large number of dosimeters used in this study, each dosimeter was calibrated at two exposure values, 20 mR and 100 mR, though even a single point calibration would have been adequate. A calibration line was drawn on a log-log graph with a slope of unity. If both the calibration points did not fall on the unity slope line, the calibration was repeated or the dosimeter rejected. In addition, 3% of dosimeters returned from the field were recalibrated and no change was detected in most batches.

An important factor affecting the calibration is the energy dependence of dosimeters. Calcium fluoride being of comparatively high effective atomic number has an energy dependent response [7, 8]. This problem has been overcome in the present work by using radium gammas for calibration. The comparative response values of CaF_2 TLD per roentgen of exposure from the radium source was found to be very close to that from the gamma rays of the thorium series which predominate in the monazite belt. The weighted average of TLD response per roentgen was 1.42 for the radium source and 1.40 for the thorium series gamma rays.

2.2.3. Mode of dosimetric survey

About 20% of the households in each of the areas in the region were covered by the dosimetric survey. The selection of the households was made from the table of random numbers. In all 12 716 dosimeters were issued. Of these, 10 887 dosimeters were received back in good condition for reading. The remaining were either damaged or lost. The dosimeters were worn by the individuals throughout the day and night for a period of two months. One dosimeter was also placed at approximately 3 ft above ground level in each of the households surveyed. All dosimeters were run through the reading cycle just prior to despatch to the field in order to erase previously accumulated background exposure. A set of dosimeters from each batch was returned to the main laboratory at Bombay immediately on receipt in Kerala to correct for the exposure during transport by air. Another set of control dosimeters was maintained at the field headquarters of the project located at Trivandrum outside the monazite belt. At the end of two months, all dosimeters including the controls were collected and read. The household and personnel radiation exposures reported here were corrected for the exposure recorded by the control dosimeters during their transit from Bombay to Trivandrum and back.

2.3. Analysis of data

The dosimetric and demographic data have been analysed to yield the following information:

(a) Classification of the households and population into sub-samples according to the radiation environment and personal dose received;
(b) Fertility index;
(c) Sex ratio among the offspring in each of the sub-samples;
(d) Ratio of abortions and still-births to total pregnancy;
(e) Ratio of congenital abnormalities to total live births;
(f) Infant mortality rates in each of the sub-samples.

MONAZITE SURVEY PROJECT - BARC

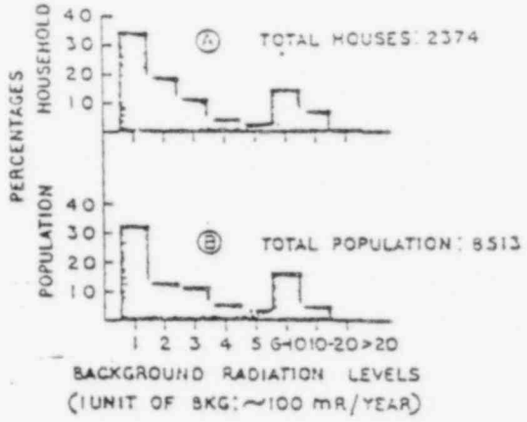

FIG.4. A. Household exposure distribution in all areas under survey;
B. Personal exposure distribution in all areas under survey.

3. RESULTS AND DISCUSSION

Data on the distribution of the 2374 households covered by the survey according to radiation exposure levels are presented in Fig.4A. For the purpose of the present study, one unit of background radiation exposure level has been taken to be equal to 100mR/yr. This value is based on the exposures recorded in Bombay and Trivandrum which have normal natural background radioactivity.

It is evident from Fig.4A that 24.6% of the households recorded levels greater than five times the normal natural background radiation level. 8.8% of the households received more than 10 times the normal radiation level, and 1.1% recorded more than 20 times the normal levels.

Details of the household exposure levels in each of the seven areas within the region under investigation are shown in Fig.5. Data indicate that of the total households in each area those recording greater than 5 times normal natural background level constitute 49.9% and 30.3% in Thekkumbhagum and Chavara in the south, 16.8%, 20.5% and 1.2% in Ponmana, Allappadu and Arratupuzha in the middle of the coastal strip and none in Thirukkunnāpuzha and Purakkadu in the north. Thus, as one proceeds from Purakkadu in the north to Thekkumbhagum in the south the fraction of households in each area receiving greater than 5 times normal background radiation exposure levels gradually increases with the maximum peak value in the Chavara area. Further, the percentages of households receiving greater than 10 and 20 times the normal levels are also observed to be highest in the Chavara and Thekkumbhagum areas.

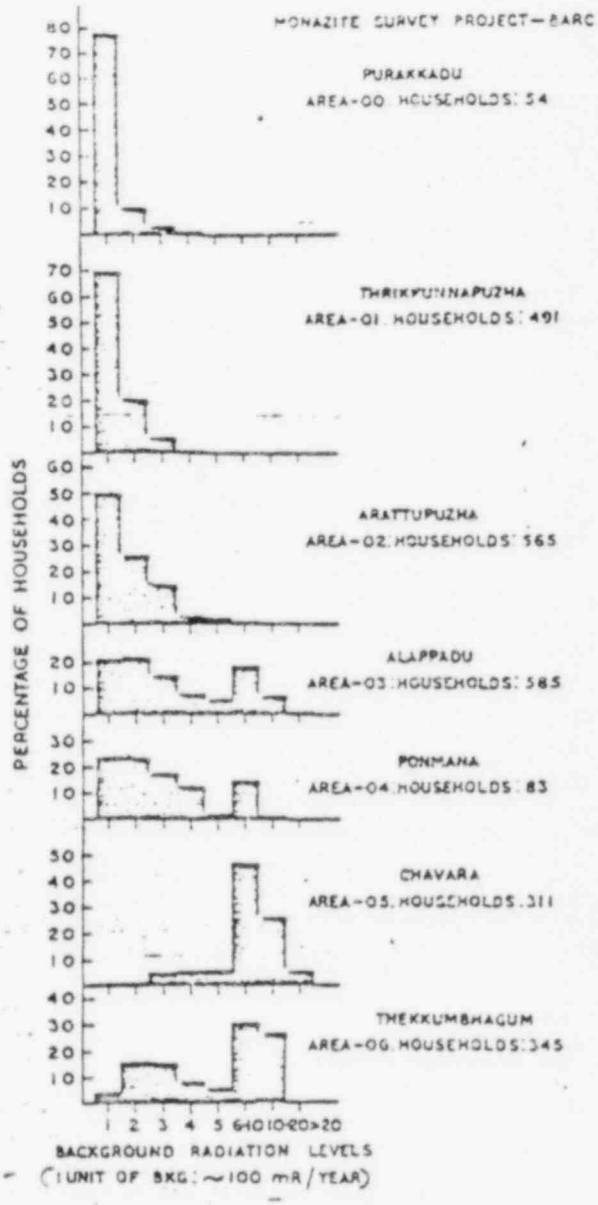

FIG. 3. Exposure distribution patterns in 7 different areas.

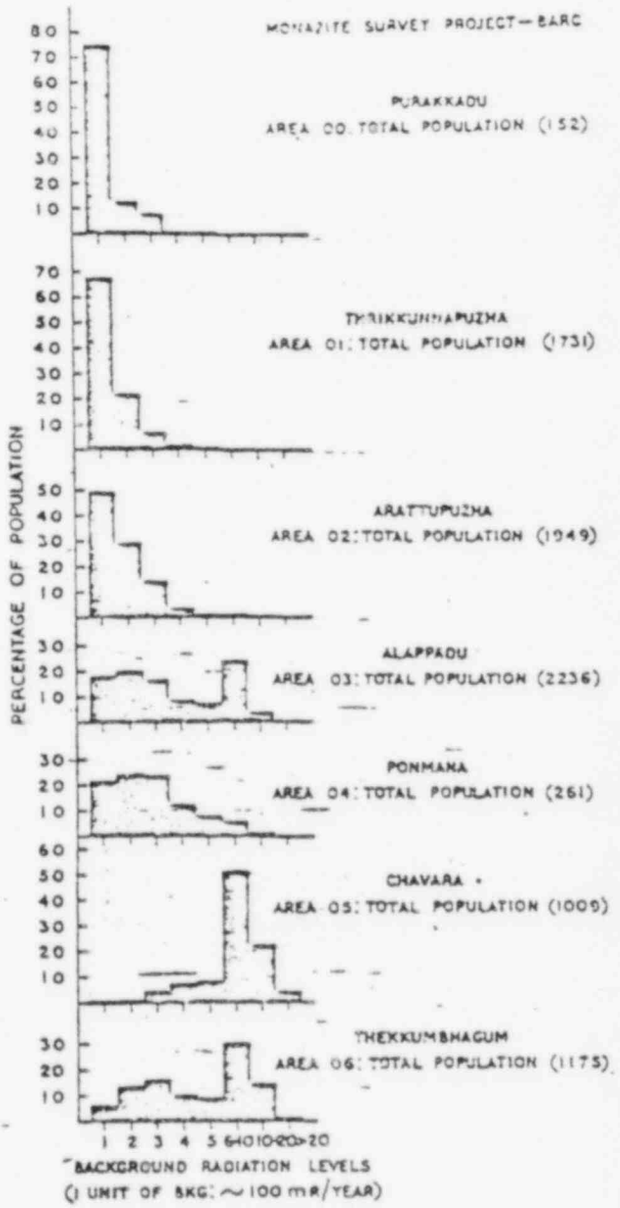

FIG. 8. Personal exposure data of all occupational groups included in the survey.

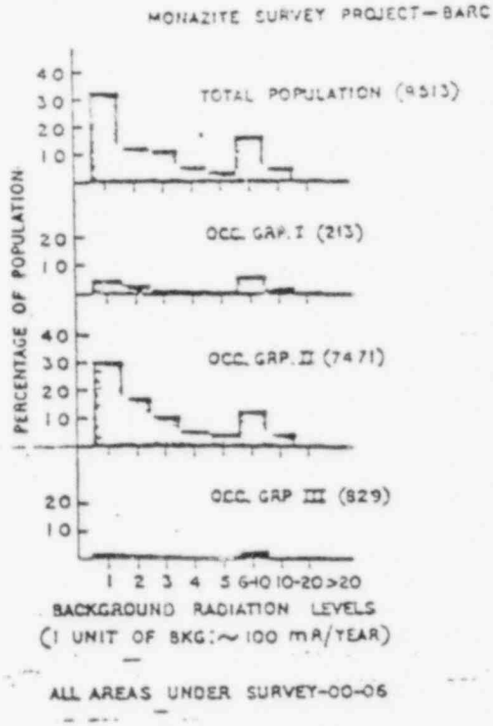

FIG. 7. Personal exposure patterns according to occupation.

We have also examined the individual household measurements in each of the seven areas; the exposure data reveal significant variations between adjacent households in a given area. This indicates that even in a specific area the distribution of monazite deposits is non-homogeneous.

Personal radiation exposure patterns of the total population of 8513 individuals included in the present study as well as the exposure pattern in each of the seven areas are shown in Figures 4B and 6, respectively. The exposure distribution profiles according to different occupational groups within the total population are presented in Fig. 7. Further, the areawise radiation exposure profiles for occupational Group II, which constitutes about 90% of the total population surveyed, have also been computed separately and are given in Fig. 8.

The main feature of personnel dosimetric results is that the radiation exposure profiles for the population taken as a whole (Fig. 4B) as well as in each of the seven areas (Fig. 6) reveal a close similarity to the household exposure profiles (Figs 4A and 5). Between different occupational groups, the exposure profile for Occupational Group II shows the closest fit with the total population profile (Fig. 7). Further, the areawise exposure distribution for individuals in Occupational Group II (Fig. 8), who are

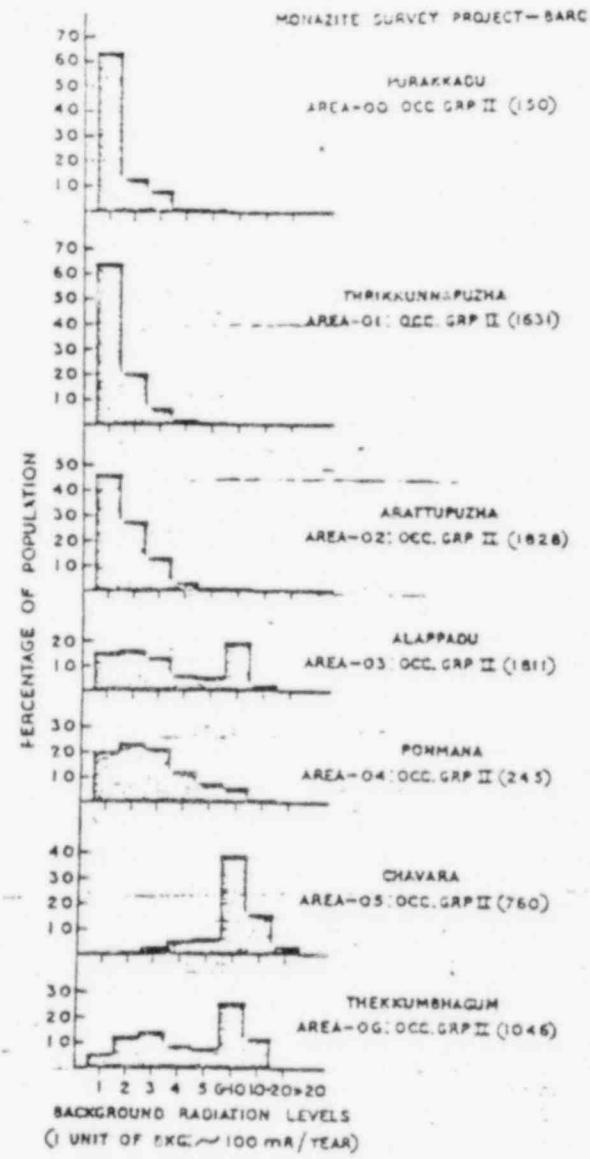

FIG. 8. Personal exposure data of Occupational Group II included in the survey according to the different areas of residence.

TABLE I. SUMMARY DATA ON FERTILITY AND PREGNANCIES OF MARRIED COUPLES RESIDENT IN MONAZITE BELT INCLUDED IN THE DOSIMETRIC SURVEY

BKG Levels	No. of couples	No. of living children			Children born alive, since dead			Children born alive	No. of still-births				Abortions and miscarriages	Total pregnancies	Twins	Gross abnormalities
		M	F	T	M	F	T		M	F	U	T				
1 to 5	1687	2933	2721	5686	1188	1098	2286	8933	318	123	28	384	502	10273	66	33
6 to 10	682	896	795	1691	202	196	398	2088	36	36	8	76	102	3244	31	12
11 to 20	384	518	549	487	81	47	88	563	1	7	2	10	30	889	6	3
> 20	22	46	38	83	9	12	22	104	3	3	1	7	4	334	1	-
Total	9439	5114	4811	9925	1439	1354	2794	12709	339	167	47	475	672	13730	84	34

1 unit of BKG: ~ 150 mR/year.
M = males; F = females; U = sex unknown; T = total.

totally employed in the region, can be seen to mirror the areawise household radiation exposure profiles (Fig.5). The latter observation lends a degree of confidence to the dosimetric data and the classification of the population according to occupation adopted in the demographic survey.

On the basis of the dosimetric data reported in the present paper, 2020 out of the 8513 individuals surveyed received exposures greater than 500 mR/yr. Of these, 551 individuals received exposures greater than 1R/yr while 57 individuals received a dose greater than 2R/yr. If these data are representative of the total population in the region, one could estimate that, as a first approximation, of the 70 000 individuals living in the region covered by the demographic survey about 16 600 are likely to be receiving a dose exceeding 500 mR/yr. This estimate would be the likely maximum value since, as discussed earlier, the individual household and hence the personnel exposure values between adjacent households in the same area show large variations chiefly due to the non-homogeneous distribution of the monazite deposits in the coastal belt. The per capita dose for the entire population resident in the region has been computed on the basis of the sample surveyed and it works out to 397 mR/yr.

3.1. Statistics of the population covered by the survey

Data on the pregnancies and their termination in the population covered by dosimetric measurements are presented in Table I. In addition, the raw data on the subsamples in each of the areas are also presented, in Table II. 2420 married couples with 13 720 pregnancies have been analysed. The couples are grouped according to the personal exposure received by the female member, since in general the maternal dose recorded was higher than the paternal dose.

Fertility index. The number of pregnancies per couple (Fertility index) varied from 5.76 to 6.07. The differences between groups receiving different levels of background radiations are not statistically significant. The fertility indices of the subsamples are presented in Table III. It is observed that there is a tendency to lower values as one proceeds from Purakkadu in the north to Thekkumbhagum in the south. In addition, it is noticed that in two of the areas for groups where maternal radiation exposure levels are in excess of 20 background (BKG) units the fertility index of 4.99 is recorded, which is the lowest for the population surveyed. The number of couples falling in this group is only 22 and the data are not adequate for drawing definite conclusions.

Sex ratio among the off-spring. The sex-ratios of the offspring have been computed for three groups, living, still-born and infant mortality. The data are presented in Table IV. The values obtained for the various groups show wide variations and no clear cut patterns have emerged. On the basis of genetic considerations, maternal exposure would result in fewer males among the living offspring and a greater loss of males through still-births and early deaths during the first year after birth. Though the number of living male children in the group receiving 11 to 20 BKG units are significantly lower than in those groups receiving lower background levels of radiation, it is observed that the group receiving over 20 BKG units does not show this trend. In fact, there appears to be a small excess

TABLE II. PRIMARY DATA ON FERTILITY AND PREGNANCIES OF MARRIED COUPLES RESIDENT IN MONAZITE BELT INCLUDED IN THE DOSIMETRIC SURVEY

BRC levels	Area code	No. of couples	No. of living children			Children born alive, since dead			Children born alive	No. of still-births				Abortions and miscarriages	Total pregnancies	Twins	Gross abnormalities
			M	F	T	M	F	T		M	F	U	T				
	80	75	172	164	348	69	51	120	468	5	3		8	30	545	1	
	81	833	1081	1038	2119	253	227	520	2639	58	23	7	88	162	3018	18	15
	82	541	1104	970	2074	249	218	469	2724	67	44	16	127	134	3278	22	9
1 to 5	83	448	921	943	1864	230	174	404	2418	54	28	11	103	122	2103	13	7
	84	77	151	128	279	33	28	60	339	16	11	3	33	18	352	4	1
	85	65	132	106	231	28	32	51	282	1	3		4	11	297	4	1
	88	183	362	368	731	74	67	141	872	16	8	3	32	38	939	4	
	82																
	81																
	62	8	18	15	33	6	3	9	42		3		3	3	48		
6 to 10	82	143	334	277	611	78	75	150	761	21	11	3	34	30	797	8	3
	81	8	18	16	34	3	8	11	45	3			3	3	49		1
	83	133	293	249	541	63	65	128	669	15	13	4	31	33	726	6	4
	86	217	343	338	681	84	47	131	812	9	6	3	17	35	864	7	

Age in	Sex	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
	04	6	16	15	26	0	0	11	45	1			1	3	43		
	15	133	270	849	341	62	63	176	649	15	19	4	21	28	128	8	4
	06	115	548	538	486	64	67	101	881	0	0	0	11	35	836	7	
11 to 19	00																
	01																
	02																
	03	31	45	47	95	7	0	13	110					4	114		1
	04	1	4	1	0	1		1	8						6		
	05	49	89	114	203	31	53	64	561	1	3		4	8	563	0	2
	06	33	71	87	164	13	18	38	103		4	5	6	8	206		
> 20	00																
	01																
	02																
	03	3	6	5	11		3	3	13	1			1	1	11		
	04																
	05	11	33	18	63	4	3	7	63	1	3	1	3	1	34	1	
	06	9	17	18	30	0	0	13	43	1			1	2	43		
Total		1430	1115	4813	2726	1430	1354	2184	10719	829	167	49	475	629	13158	34	34

1 unit of BCG : = 100 mR/year

00 - Panikkade; 01 - Thikkunnapuzha; 02 - Arattupuzha; 03 - Aryapadu; 04 - Panmana; 05 - Chavara; 06 - Thirumbhagom

M = males; F = females; U = sex unknown; T = total

TABLE III. FERTILITY INDEX OF MARRIED COUPLES IN THE SAMPLED POPULATION

Area	Radiation exposure levels (BKG Units)[a]			
	1 - 5	6 - 10	11 - 20	> 20
0	6.7	-	-	-
1	5.74	-	-	-
2	5.49	5.75	-	-
3	5.85	5.57	5.43	7.5
4	5.54	8.1	6.00	-
5	5.43	5.45	5.40	4.99
6	5.63	5.35	6.24	5.0
Total ± S.E.	5.76 ± 0.16	6.04 ± 0.67	5.77 ± 0.15	5.83 ± 0.8

[a] BKG Unit: = 100 mR/year

TABLE IV. MALE: FEMALE SEX-RATIO AMONG THE OFFSPRING IN THE SAMPLED POPULATION

	Radiation exposure levels (BKG Units)[a]			
	1 - 5	6 - 10	11 - 20	> 20
Living	1.06	1.12	0.87	1.2
Stillborn	1.7	1.2	-	1
Infant mortality[b]	1.06	1.03	1.08	0.69

[a] BKG Unit: = 100 mR/year
[b] National average for rural India: 1.09 [10]

of males in this group as compared to others. Again the sex-ratio among the still-born shows a large excess of males among those couples receiving lower radiation exposures. The pattern of infant mortality also does not indicate any definitive trends and the figures for all groups except those falling above 20 BKG units are comparable to 1.09, a value computed from the data published by Registrar General, India [10] for infant mortality rates for rural India. The lower value observed in those couples receiving radiation exposures in excess of 20 BKG units may perhaps be explained as a compensatory phenomenon to off-set the reduced fertility index in this group, since surviving male children are regarded as potential wage earners to the family. The estimates of sex-ratio among the offspring have not provided any clue from which conclusions on the possibility of genetic damage to the population resident in the monazite bearing areas could be drawn.

TABLE V. INFANT MORTALITY RATE PER 1000 LIVE BIRTHS IN POPULATION

Area	Population exposure levels (BKG Units)[a]			
	1 - 5	6 - 10	11 - 20	> 20
0	225	-	-	-
1	253	-	-	-
2	241	166	-	-
3	216	199	136	153
4	171	244	168	-
5	180	191	210	143
6	162	174	146	309
Total ± S.E.[b]	208 ± 12.3	198 ± 9.7	164 ± 14.9	201 ± 52.4

[a] BKG Unit: ~ 100 mR/year

[b] National average for rural India: 145.9 (10)

Infant mortality rate. The data on the infant mortality rate are presented in Table V. It is observed that the values obtained are slightly in excess of 145.9, a figure estimated by the Indian National Survey for rural areas of India in 1963 [10]. A more recent estimate based on data of continuous enumeration on annual samples in 1968 gives an infant mortality rate of only 111 for the whole country. It is well known that infant mortality is one of the sensitive indicators of the living standards of a community and is particularly sensitive and responsive to improvements and deterioration in the environment in which the infant lives. The infant mortality rates computed from the data collected in our study gives a mean figure of 184. Such high values are recorded in other parts of India, notably in the state of Uttar Pradesh in northern India. Considering the primitive medical facilities available in the monazite bearing areas, inadequate sanitation and exposure to vagaries of nature, the high infant mortality rates seen in this population are not surprising. Again the differences in the infant mortality rates between the high and low background exposure levels are not statistically significant. However, it may be mentioned in passing that the highest value of 309 is recorded in the group of 9 couples living in Thekkumbhagum receiving an exposure greater than 20 BKG units, in whom 13 of the 42 children born died in the first year of their life.

Pregnancy terminations, abnormalities and multiple births. The data are presented in Table VI. It is observed that on an average 4.5% of pregnancies ended in abortions. The frequency of abortions in the four radiation exposure categories are not different. The frequency of still births in the group receiving a radiation exposure in excess of 20 BKG units is significantly higher than in other groups. As stated earlier, the infant mortality rate in this group was also the highest. It thus appears that the total loss of offspring in this group is significantly higher than in those

TABLE VI. PREGNANCY TERMINATIONS, ABNORMALITIES AND MULTIPLE BIRTHS IN THE SAMPLED POPULATION[a]

Parameter	Radiation exposure levels (BKG Units)[b]			
	1 - 5	6 - 10	11 - 20	> 20
Abortions (%)	4.68	4.54	3.40	3.50
Stillbirths (%)	2.60	3.30	1.70	6.14
Gross abnormalities (%)	0.36	0.53	0.50	-
Twins (%)	0.61	0.93	1.01	0.87

[a] As percent of total pregnancies
[b] BKG Unit: ~ 100 mR/year

receiving lower radiation exposures. It is proposed to study a larger group falling in this category in the near future. The frequency of gross abnormalities do not show any significant differences between the various groups.

4. SUMMARY AND CONCLUSIONS

In an attempt to evaluate possible genetic effects of chronic irradiation in man, demographic and radiation dosimetric surveys of the population resident in the monazite bearing high radiation areas of the Kerala coast in south-west India were undertaken. Demographic data including detailed fertility history of married couples of nearly 70 000 individuals resident in 13 355 households in a 55 kilometre strip along the monazite belt were recorded. Radiation exposure levels received by a randomly selected sample population of 8513 individuals resident in 2374 households in the coastal strip were also recorded, using calcium fluoride thermoluminescent dosimeters.

Dosimetric data revealed that about 25% of the households covered by the survey recorded greater than 5 times the normal natural background radiation level. The fraction of households receiving greater than 5 times the normal background exposure gradually increased from north to south along the strip. The percentage of households recording greater than 10 and 20 times the normal levels was also highest in the southern part of the strip.

The radiation exposure profiles for the population revealed a close similarity to the household exposure profiles. The profiles for individuals in Occupational Group II, who are totally employed in the region and which constitutes about 90% of the total population surveyed, was seen to mirror the household radiation exposure profiles. In all, 2020 out of the 8513 individuals surveyed received exposures greater than 5 times the normal levels (500 mR/yr). Of these, 551 individuals received exposures greater than 1 R/yr while 57 individuals received a dose greater than 2R/yr. On the basis of the data reported here, it can be estimated that of the

70 000 individuals living in the strip above 10 000 are likely to be receiving a dose exceeding 500 mR/yr. The per capita dose received by this population is estimated to be 307 mR/yr or nearly four times the normal background radiation level. Analysis of the demographic data indicated no statistically significant differences in fertility index, sex-ratio among offspring, infant mortality rate, pregnancy terminations, multiple births and gross abnormalities between population groups receiving different levels of radiation exposure. However, the lowest value of fertility index and the highest value of infant mortality rate were recorded for a group of married couples who received radiation exposures greater than 20 times normal background levels; the total loss of offspring in this group was significantly higher than in those receiving lower radiation exposures.

It is very likely that the various parameters which have been analysed in Phase I of this study are not sensitive enough to reveal any differences which are statistically significant. In view of the recent observations of radiation damage to chromosomes of human cells irradiated in vitro, it is hoped that human chromosomal studies of the population resident in the area would throw more light on the effects of chronic high background radiation on man. Phase II of the investigations would include, among others, detailed studies on blood lymphocytes from the new born, children of various age groups and adults on samples drawn from the various radiation exposure groups.

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Oral presentation. The influence of bone remodelling on the effectiveness of DTPA therapy for [illegible] Pu. A. C. JAMES and N. F. KENNEY, Department of Medical Physics, Royal Free Hospital School of Medicine, London (UK).

[illegible] presented illustrating the role of local bone [illegible] in the removal of ^{239}Pu deposited in the [illegible] of immature rats following intravenous ex[illegible]. The effects of various treatments with [illegible] on the activity of plutonium deposits on [illegible] surfaces undergoing various types of remodelling [illegible] were studied. Chelation of plutonium bound in [illegible] matrix can occur effectively only if the [illegible] is first liberated into soft tissue by osteo[illegible] action in the process of bone resorption. The [illegible] of the total bone surface area undergoing [illegible] during DTPA therapy thus dictates the [illegible] of delayed therapy (commenced after the [illegible] of skeletal plutonium uptake) in reducing [illegible] burden. The effectiveness of promptly [illegible] DTPA is largely due to the chelation [illegible] in the blood, thus preventing skeletal [illegible]. The results of measurements of the [illegible] particle dose to the osteogenic cells under the [illegible] conditions of remodelling activity and DTPA [illegible] are also presented. The relevance of this [illegible] to the problem of human therapy is discussed.

Oral presentation. Rationale for the use of DTPA in the treatment of plutonium contaminations [illegible]. A. M. FREEZ and G. W. DOLPHIN, UKAEA Health and Safety Branch, Harwell (UK).

[illegible] complexed with proteins and smaller [illegible] in the blood mainly becomes deposited [illegible] bone and liver. However, a small fraction [illegible] and 10% of the total amount in the blood [illegible] daily in the urine. As plutonium deposited [illegible] these organs is retained for long periods, [illegible] treatment of contaminated humans is [illegible] increasing the urinary excretion and [illegible] diminishing the amount deposited in [illegible] and liver. To this end DTPA (Diethylene[illegible]triaacetic acid) has been used in a number [illegible] where internal contamination of workers has [illegible].

[illegible] the therapeutic application of DTPA may [illegible] more widespread the authors have attempted [illegible] a rationale for its use. The mode of entry [illegible] into the body is by wound or inhalation [illegible] In [illegible] cases there is considerable variability [illegible] amount and rate of transfer of plutonium to the [illegible]. Although external counting over the lung or [illegible] may indicate the total amount at the site of [illegible] the amount and rate of transfer to the blood [illegible] depend upon the physico-chemical state of the plutonium. The DTPA regime depends more on the rate of transfer to the blood rather than the amount present at the site of entry.

As DTPA injections are known to clear the blood of complexed plutonium, it is possible to investigate the rate of transfer of plutonium from the site of entry to the blood by measurement of the urinary excretion following a planned series of DTPA injections. Several initial DTPA injection regimes have been tested in a simple model with the object of establishing an optimum regime for evaluating the rate of transfer of plutonium to the blood and its variation with time. The indications for continuous DTPA treatment are discussed.

287 *Oral presentation.* A review of the use of DTPA in the treatment of internally deposited plutonium. G. B. SCHOFIELD, UKAEA Windscale Works, Sellafield (UK).

Attempts have been made over the years to immobilize internally deposited or circulating plutonium by the use of chelating agents. These have included EDTA, zirconium malcate, sodium citrate, desferrioxamine, penicillamine and DTPA. This latter has proved to be the most effective agent and has for many years been the only therapeutic compound used at the Windscale Works of the Atomic Energy Authority in the treatment of cases of internally deposited plutonium.

This paper reviews the efficiency of treatment by this compound in 17 people who have been internally contaminated during the period 1963–1969. These patients include six with plutonium contaminated wounds, ten inhalation cases and one whose internal contamination was of unknown origin. It is further suggested that DTPA may be used as a diagnostic as well as a therapeutic agent, since any resulting enhancement of plutonium urinary excretion will indicate the presence of circulating and available plutonium.

RADIATION EFFECTS IN MAN II

288 *Oral presentation.* Dosimetric and cytogenetic studies in Brazilian areas of high natural activity. T. L. CUTLEN, Instituto de Fisica, Pontificia Universidade Católica, Rio de Janeiro and J. C. CABRAL DE ALMEIDA, Instituto de Biofisica, Universidade Federal, Rio de Janeiro (Brazil).

In Guarapari the population of 6000 lives in a radioactive environment approximately 10 times normal levels. After radiation levels were mapped, lithium fluoride dosimeters were randomly distributed and 340 were recovered. The air was mensured for radon, thoron and long lived content, and food

and water were radiochemically analysed. A random sample of 107 people living in the area for at least 10 yr were chosen for a cytogenetic study and compared with a control group of 82. From the exposed population 7711 metaphases were scored for chromosomal aberrations and compared with 6091 metaphases from the control group. The results showed no statistically significant difference.

289 *Oral presentation.* **Incidence des leucemies tardives survenant chez les cancéreux irradiés.** S. SIMON, Centre Anticancéreux de l'Université de Bruxelles (Belgium).

Etude portant sur des malades ayant reçu une irradiation totale au cours d'un traitement par rayons X ou par radium et ayant eu une survie supérieure de 5 a. Les traitements ont été effectués entre 1925 et 1962.

L'enquête porte sur 1438 cas de cancers utérins traités par radium, 2042 cas de cancers du sein traités par rayons X, 1356 cancers de la tête traités par appareils moulés radifères et 1064 cancers de la tête traités par curiepuncture. Comme élément de comparaison, nous disposons de 1464 épithéliomas superficiels de la face traités par radiothérapie à bas voltage n'entrainant aucune irradiation générale.

L'ordre de grandeur des doses reçues en irradiation totale est calculé pour les quatre catégories.

Le relevé des "seconds cancers" observés dans les différents groupes, après un délai minimum de 5 a, montre que les leucémies apparaissent en nombre significativement supérieur à la normale dans les trois premiers groupes: le dernier groupe et les témoins n'ont pas présenté de leucémie. Il semble donc qu'il y a un seuil d'action qui n'est pas atteint lorsque l'irradiation totale résulte d'une curiepuncture au niveau de la tête; dans ce cas, les doses sont comprises entre 1 rem et 6 rem au niveau du pubis et 5 rem et 30 rem au bord supérieur du sternum. Les autres modes d'irradiation donnent des valeurs d'irradiation totale plus élevées.

L'étude du groupe fort important des "seconds cancers" localisés dans le tube digestif ne montre aucune différence entre les cinq groupes envisagés, et donne des valeurs équivalentes à celles fournies par les statistiques belges de mortalité, pour les tranches d'âge correspondantes.

Incidence of delayed leukaemia in irradiated cancer patients.

Study of patients who have received total irradiation in the course of X-ray or radium treatment and having survived for more than 5 yr. Treatment was carried out between 1925 and 1961.

The investigation deals with 1438 cases of uterine cancers treated with radium, 2042 cases of breast cancers treated with X-rays, 1356 cancers of the head treated with moulded apparatus containing radium and 1064 head cancers treated by curiepuncture. As a comparison factor, we have available 1464 surface epithelial cancers of the face treated with low voltage radiotherapy involving no general irradiation.

The order of magnitude of the doses received in total irradiation is calculated for the four categories.

The records of "second cancers" observed in the different groups, after a minimum period of 5 yr, show that leukemias appear in numbers that are significantly higher than the normal in the first three groups: the last group and the controls have not exhibited leukemia. It would appear that there is a level of activity which is not reached when total irradiation results from a curiepuncture at the head level; in this case, the doses are between 1 rem and 6 rem at the level of the pubis and between 5 rem and 30 rem at the top edge of the sternum. The other methods of irradiation give higher total irradiation levels.

The study of the very important group of "second cancers" located in the digestive tube shows no difference between the five groups considered and gives values equivalent to those furnished by Belgian mortality statistics, for the corresponding age groups.

290 *Oral presentation.* **Observation of radiation induced cancers in an exposed environmental population.** G. W. DOLPHIN, UKAEA Health and Safety Branch, Harwell (UK).

Risks of radiation induced cancer have previously been evaluated by the author in studies of data from exposed groups such as radiotherapy patients and atomic bomb survivors. On the assumption of a linear dose effect relationship the risk of cancer induction was found to be $100/10^6$ man-rads. The validity is discussed of using this risk value at doses lower than 10 rads in estimating the number of induced cancers in an environmental population following an accidental release of radioactivity from a nuclear installation. Calculations have been made at several dose levels to show the size of an environmental population in which the radiation cancers may be observed as a statistically significant increase over the natural incidence. The effect of other factors on the detection of radiation induced cancers in a population are also discussed.

A-79-25
IV-D-90

Department of Energy
Washington, D.C. 20585

July 15, 1981

Ms. Kelsey Selander
U.S. Environmental Protection Agency
401 M Street, S.W.
Washington, D.C. 20460

Dear Ms. Selander:

This is the Department of Energy's response to the Agency's request for formal comments on the Draft Environmental Impact Statement (DEIS) for Remedial Action Standards for Inactive Uranium Processing Sites (EPA 520/4-80-011, December 1980), and the proposed standards that were published in the Federal Register on April 22, 1980 and on January 9, 1981. For the past year and a half we have expressed our concerns to the Agency in letters and through meetings with its staff.

Of particular concern is the interim cleanup standards that the Agency has published. Implementation of these standards (in other cleanup programs) has been shown to be costly and technically difficult. The major reason for these difficulties is that the proposed extremely low single-valued standards must be applied to open lands and structures where there are variations in the natural occurrence of radioactivity, and where the proposed standards are near or below background in many cases. The concept of "reasonable assurance" contained in the preamble to the standards is too vague to be effective for assuring compliance, and we want to avoid any use of the criteria for exceptions wherever possible. If the remedial action program is to be conducted at the lowest possible cost to the taxpayer without jeopardizing public health, there must be a reasonable range of numerical values in the standards.

Flexibility is needed in the final standards not only to facilitate the cleanup at the lowest possible cost of the inactive uranium processing sites and vicinity properties that are covered under Pub. L. 95-604, but also to facilitate cleanup of radioactivity contaminated sites under two other cleanup programs the Department conducts. The mill tailings standards will set a precedent, for the cleanup of residual radioactive material at sites where nuclear operations were formerly conducted for the Manhattan Engineer District and the Atomic Energy Commission, at surplus radioactively contaminated DOE-owned facilities, and at private properties in their vicinity. If the proposed standards become applicable, the necessity for compliance will increase the cost and time required for these cleanup programs.

We are, of course, as concerned as you are about the protection of the public from exposure to levels of radon and its decay products that may pose potential and significant health hazards. The Department will make every effort to eliminate any potential hazards and to concomitantly keep the costs of conducting radiological surveys and remedial action operations as low as possible. However, we feel it will become difficult to justify requests for Congressional appropriations for a program to meet standards that according to the Agency's estimates will only prevent about two deaths per year as a result of radon emanation from the 25 inactive uranium processing sites over the present rate of about 92,000 deaths per year from lung cancer.

The following is a discussion of our specific concerns regarding the proposed standards and the accompanying Draft Environmental Impact Statement. Suggested alternatives to the proposed standards which, if adopted, would allieviate our concerns are included. Our comments here parallel and supplement those we have made to the Agency since January 1980 and are also provided in a capsulized form in Enclosure 1.

Radon Flux and Dispersion

The mathematical models the Agency used to estimate radon dispersion from unstabilized piles are in disagreement with field measurements of radon concentrations versus distance from the piles. All the data we have seen indicate that radon concentrations are essentially the same as natural background levels at about 1/4 to 1/2 mile from the boundary of the site. As an example, Enclosure 2 indicates average contours of constant outdoor radon concentrations (pCi/l) in the vicinity of the inactive processing site in Canonsburg, Pennsylvania, where the Department is maintaining a radon monitoring network. The highest radon concentration is about 0.7 pCi/l near the boundaries of the site and decreases to average background for the vicinity (0.3 pCi/l) in about a half mile. We see no evidence of any potential health impact to the public because the highest measured concentration is only 25 percent of the Nuclear Regulatory Commission's allowable value of 3 pCi/l for nonoccupational exposure. Additionally, data collected in aerial radiological surveys at uranium processing sites in the West indicate that during the flights radon concentrations in the immediate vicinity of the sites were essentially at background levels. Further evidence that radon concentrations in the vicinity of unstabilized sites are much lower than the Agency's estimates was provided in Dr. Robley D. Evans' letter of May 27, 1981, to Dr. William A. Mills of your staff.

Because radon dispersion from unstabilized piles does not produce measurable effects even at short distances from their boundaries, we cannot support the proposed 2 pCi/m^2-sec radon flux standard, which is very close to or lower than background in many parts of the nation. As an alternative, we suggest a maximum average annual concentration of 30 pCi/l of radon at 3 feet above the surface of a stabilized pile or disposal site and 3 pCi/l at the fenced boundary of the property, and that the standard itself specify that the average is to be determined by reasonable statistical measurement protocols. Our suggested alternative is consistent with the Commission's radiation protection standards of 30 pCi/l for occupational exposure, which, for example, will protect workers, repairing a stabilized site, and 3 pCi/l for nonoccupational exposure, which will protect the public if they are exposed at the fenced boundary.

The stabilized disposal locations for tailings are to be government-owned, fenced, and licensed by NRC. It is the intent of the Act that the Government both monitor and regulate the use of the property and its perimeter. The appropriate established limits for occupational exposure to radon at a DOE operating facility is 30 pCi/l of air (which could apply on the stabilized tailings and be monitored at an elevation three feet directly above the surface) and the established fence line limit generally recognized as acceptable exposure to an exposed individual at the point of control is 3 pCi/l.

The measurement of radon flux is extremely difficult due to its variability over a large area such as a tailings pile. We therefore suggest that the standard for radon control of stabilized tailings be based upon measured radon concentration in air.

We believe that EPA may have overestimated the potential health effects from radon by a factor of 10. Even assuming that EPA's analysis of the potential health effects is correct, the Draft Environmental Impact Statement estimates that 78 percent of the total number of health effects can be avoided by stabilizing piles at the 100 pCi/m^2-sec flux value, which roughly corresponds to 30 pCi/l of radon three feet above the surface. We estimate that a potential savings in remedial action costs of $80,000 to $120,000 per acre, or a total savings of $80 to $120 million dollars could be realized if piles and disposal sites are stabilized at our suggested radon concentration values rather than EPA's proposed 2 pCi/m^2-sec radon flux standard.

Radon Decay Product Concentration Standard

The proposed standard for radon decay product concentration in structures is exceeded by, or is close to background concentrations in a significant fraction of homes that are not associated with mill tailings or other residual radioactive material. The results of some American sampling programs shown in Enclosure 3 indicate the variations that can be expected. The results of extensive Canadian studies of radon and progeny concentrations in residences summarized in Enclosure 4 parallel the naturally occurring variations found in American structures. These data lead us to conclude that an inflexible 0.015, including background, working level (WL) standard will in many cases require unnecessary and expensive removal of material to background levels or even below background.

The preamble to the standards indicates that if the allowable working level is still exceeded after all apparent tailings have been removed or otherwise prevented from affecting the interior of the structure, then the proposed standard does not require further remedial measures. However, we will have to certify during the conduct of remedial action that all apparent tailings materials have been removed. Substantiation of removal will have to ultimately provided by indoor radon decay product concentration measurements that can be influenced by sources other than tailings. Potential difficulties are indicated (a) by a recent review of post-remedial action surveys in Grand Junction indicating that 32 percent of decontaminated structures now have working levels greater than the proposed standard, and (b) by preliminary results of an analysis of radon daughter concentrations in residences that is tending to suggest that 55 percent of basements and 30 percent of first levels in U.S. homes exceed 0.015 WL.

We face the reasonable possibility of post-remedial action radiological surveys in structures indicating that certification conditions have not been met. A dilemma will exist because we will not be able to determine if the allowable radon level is exceeded because contaminated material derived from a processing site has not been removed, or because of the presence of natural radioactivity in construction materials and natural background radiation. The only alternatives will be either to remove additional materials including natural radioactivity from other parts of the structure such as from under floors and footings, or to request an exception from the Agency.

The number of significant figures in the 0.015 WL standard is questionable. Some, but not all, available instrumentation can probably determine working levels to three significant figures under controlled conditions at a given time and place. However, working level in a structure varies significantly with the time of day, the season of the year, ventilation conditions, and other factors. The collection and analysis of samples under such variable conditions over a period of a year to calculate an average annual working level is a statistical procedure. We have no evidence to convince us that 0.015 WL can be distinguished from 0.02 WL and we seriously question whether an average of 0.02 WL can be distinguished from 0.04 WL. Recognition of these real-world limitations may have influenced the formulation of the Surgeon General's flexible guidelines for remedial actions in Grand Junction, Colorado. The statistical nature of determining an annual average working level value has not changed since those guidelines were written, and we must have the flexibility to avoid the need for unnecessary and costly removal of material.

As an alternative we suggest that the standard require remedial action if the working level is greater than 0.05 WL above background and is caused by the presence of mill tailings. If the working level is between 0.01 WL and 0.05 WL above background and is caused by the presence of mill tailings, the Department would decide if remedial action is required, utilizing the so-called ALARA approach that reduces radon concentrations to a level as low as reasonably achievable, taking into account social, economic and technical considerations. If the working level is less than 0.01 WL above background, remedial action is not required. We feel that this standard (a) will impose no measurable difference in health effects relative to the Agency's proposed standard, (b) will significantly reduce implementation and certification costs, and (c) will allow us to avoid the exceptions procedure.

Radium in Soil

The proposed 5 pCi/g standard for radium-226 in soil is a level that can probably be measured with available field laboratory equipment, but at a cost. The proposed standard is not directly related to potential health effects, and we see no loss of health benefits if 10 or perhaps even 20 pCi/g were specified. Higher values would save time and cost required for coring and analyzing samples taken from large areas, and would significantly reduce the volume of contaminated natural soil that must be collected, moved and disposed of with any remedial action. We estimate for example that 5 pCi/g material could exist in soil as deep as 25 feet beneath the ground level of the Salt Lake City tailings pile. The removal of each foot of soil in decontaminating that site requires the removal, transport, disposal and backfilling of 177,000 cubic yards of soil at an estimated incremental cost of $4,600,000.

The word "any" in the specification of the occurrence of radium-226 in soil is also a major source of additional time and costs for radiological measurements and cleanup. Enclosure 5 illustrates the substantial impact of the word "any" on the time and cost to certify compliance with radium-in-soil standards. The chart is based on the most recent experiences of one of our radiological survey groups in a DOE National Laboratory. The manpower and cost estimates in the table relate to two protocols for certifying that after cleanup of one acre of ground the amount of radium-226 in the soil does not exceed 5 pCi/g. The first protocol is necessary to assure that the "any" sample specification required by the standard is satisfied, and the other protocol is a realistic statistical averaging of samples. The percentage difference in cost between the "any" and averaging protocols is significant; the "any" specification requires 68 percent more cost at 5 pCi/g than the statistical averaging protocol. There is a savings of $3,900 per acre if reasonable averaging rather than the proposed "any" sample were specified. A reasonable specification of averaging as opposed to "any" in the standards could save about $3,900,000 for certification of the cleanup of the tailings piles. This estimate of potential savings does not include the areas of wind-blown and other dispersion tailings, and vicinity properties, which might altogether triple the savings to perhaps $12,000,000 in certification costs. The savings will probably increase substantially as experience with quality assurance operations is gained during the conduct of remedial action operations.

These additional costs could be avoided if the word "any" is deleted from the standard and the standard itself is replaced by an average value of 15 pCi/g for radium in soil utilizing the ALARA approach, and the statement that "the necessary measurements are to be performed within the accuracy of available field and laboratory instruments used in conjunction with reasonable survey and sampling procedures." We believe that no measurable increase in health effects will result from adoption of the above procedure.

1,000 Year Disposal Site Compliance

The concept of "reasonable assurance" included in the proposed standard is not clearly defined. Computer codes that have been confirmed by field measurements made over time, and expert opinions are inadequate substitutes for experience. Disposal site technologies are being developed and evaluated and cost effective systems may not be available for some years with a confidence level of centuries. The alternative we suggest takes advantage of the fact that the sites are to be Government-owned and fenced, and licensed by the Commission for the indefinite future. This will allow sites to be

carefully monitored and evaluated annually for 10 or 20 years to confirm the efficacy of the stabilization, and to make any necessary repairs or additions to the stabilization.

Exceptions Procedure

Flexibility should be provided in the standards themselves rather than in the preamble to the standards and in the exceptions procedure. An exceptions procedure will be costly, time consuming, and difficult to implement. The procedure could undermine public confidence in the remedial action program because significant number of exceptions is anticipated for the present form of the standards, and the public would perceive that residual health hazards exist if the standards were exceeded. Flexible standards will enable the Department to pursue the ALARA objective, minimize costs, and adjust the remedial action without measurable loss of health benefits, because implementation decisions can be based on an adjustment of design parameters rather than a requirement for meeting a single valued standard.

Water Standards

An extensive sampling program could be required to establish the concentration of substances that are now present in water in the vicinity of potential disposal sites and what concentrations are released after the disposal is completed. An even greater concern to us is the concentration of substances in ground water at sites were tailings piles will be removed. We do not have specific alternatives at this time, but we want to discuss with the Agency the basis for and potential impact of the water standards.

Cost-Benefit Analysis

A review of the cost figures presented in the DEIS by Sandia National Laboratories indicates that the EPA estimate is low by at least a factor or two, and, more likely, by a much larger factor. The Sandia analysis also illustrates the extremely high remedial action cost per health effect eliminated that can result from the conduct of extensive remedial action at remote sites. It should be noted, however, that the cost-benefit ratio is still very high if the calculation is performed just for sites in the more populous areas.

Additionally, the DEIS presents very limited and, in our view, inadequate consideration of alternatives to the standards proposed. Health effects are not estimated for any of the standards except radon flux. We feel that the final DEIS should compare the cost effectiveness sensitivity of each of the standards that are promulgated, and should include

assessment of the cost effectiveness of both more or less restrictive standards as part of its justification for the selection of the standards being proposed.

We will be pleased to discuss our comments in greater detail with the Agency.

Sincerely,

Stephen H. Greenleigh

Stephen H. Greenleigh
Acting Deputy Assistant Secretary
for Environment, Health and Safety

5 Enclosures

cc: Sheldon Meyers

Enclosure 1

EPA Proposal	Impact on DOE Remedial Action Programs/Health	Suggested Alternative	Effect of Change
1. Radon Dispersion and Radon Flux Standard - 2 pCi/m^2 above background.	Program Stabilization costs will be significant to meet 2 pCi/m^2 - sec. standard on the tailings piles. Health The resulting additional benefit to public health over that resulting from a less stringent standard is questionable since: a. The value of 2 pCi/m^2 --sec is near or below background in some areas unaffected by tailings; b. Radon dispersion from unstabilized piles is overestimated by EPA computer codes; and, c. Aerial radiological surveys and DOE ground monitoring data indicate that radon concentrations in the immediate vicinity of inactive uranium processing sites (about 1/2 mile away) are essentially at background levels.	If stabilization is necessary, use 100 pCi/m^2 --sec or NRC's 3 pCi/l near the boundary line of the site.	Program Reduced costs and remedial action time. Health About 78% of EPA estimated radon health effects are avoided at the high priority sites (DEIS, page 6-7).
2. Radon 2. Radon Decay Product Concentration Standard Avg Annual Value of 0.015 WL including background.	Program Annual average working level is a statistical quantity which cannot be determined to 3 significant figures nor can average annual values be distinguished between 0.02 and 0.04 WL. Remedial action costs may increase as DOE may have to dig under footings, etc., because WL standard is exceeded and "apparent" tailings cannot be found.	There should be a range of working levels of: . WL $<$ 0.01 above background remedial action is not required. . 0.010 $<$ WL $<$ 0.05 above background. DOE will decide if remedial action is required (if working levels are in this range), utilizing an ALARA approach. . WL $<$ 0.05 above background, remedial action is required.	Program Implementation and certification costs are reduced. The exceptions procedures will be avoided. Health No measurable increase in health effects.

EPA Proposal	Impact on DOE Remedial Action Programs/Health	Suggested Alternative	Effect of Change
2. (cont.)	(cont.) Compliance with standards may not be possible in all cases e.g., 32% of structures in Grand Junction that have had at least one remedial action still exceed 0.015 WL. Health The standard is near or below background levels in many structures not affected by tailings, and the resulting added benefit to public health over that resulting from a less stringent standard is questionable.		
3. Radium in Soil standard - 5 pCi/gm in "any" layer.	Program Extensive sampling at increased cost and time will be required since "any" is equivalent to "all" layers. Health The standard is not related to potential health effects	An average of 15 pCi/g and the standard itself (not the preamble) states that the necessary measurements are to be performed within the accuracy of available field and laboratory instruments used in conjunction with reasonable survey and sampling procedures.	Program Implementation and certification costs will be markedly reduced - e.g., 95% confidence certification that "any" soil sample does not exceed 5 pCi/g costs 41% more per acre to certify than a 90% confidence certification based on a reasonable statistical averaging procedure. Health No measurable increase in health effects.
4. Gamma Radiation Standard - 0.02mr/hr above background.	_____	DOE has no objection	_____
5. Exceptions procedure.	Program Costly, time consuming and difficult to implement.	Flexibility should be provided in the standards rather than in the exceptions procedure or the preamble to the standards.	Program DOE will save time and cost because remedial action implementation decisions can be made in the field rather than in Washington.

EPA PROPOSAL	IMPACT ON DOE REMEDIAL ACTION PROGRAMS/HEALTH	SUGGESTED ALTERNATIVE	EFFECT OF CHANGE
5.(cont).	(cont.) The procedure could undermine public confidence in the remedial action program because a significant number of exceptions are anticipated and the public may perceive a residual health hazard to exist if the standard is exceeded.		
	Program		Program
6.Disposal Sites-Standards will be met for 1,000 yrs.	The standard can't be implemented because the concept of "reasonable expectations" included in the proposed standards is not clearly defined. Institutional control of disposal sites will be required for an indefinite period of time. Disposal technology will have to be developed.	Monitoring of the sites annually for ten or 20 years to establish a reasonable guide for expected longevity. The sites will be Federal property and will have to be licensed and fenced.	An assessment of the effectiveness of disposal technologies over time can be accomplished. Health Public health will not be adversely affected because of Federal ownership and fencing of the sites.
	Program		
7.Water Standards	An extensive sampling program could be required to establish the "present conditions" that are not to be exceeded. This program will be costly	DOE has no specific alternative at this time but would like to discuss the water standards with EPA.	

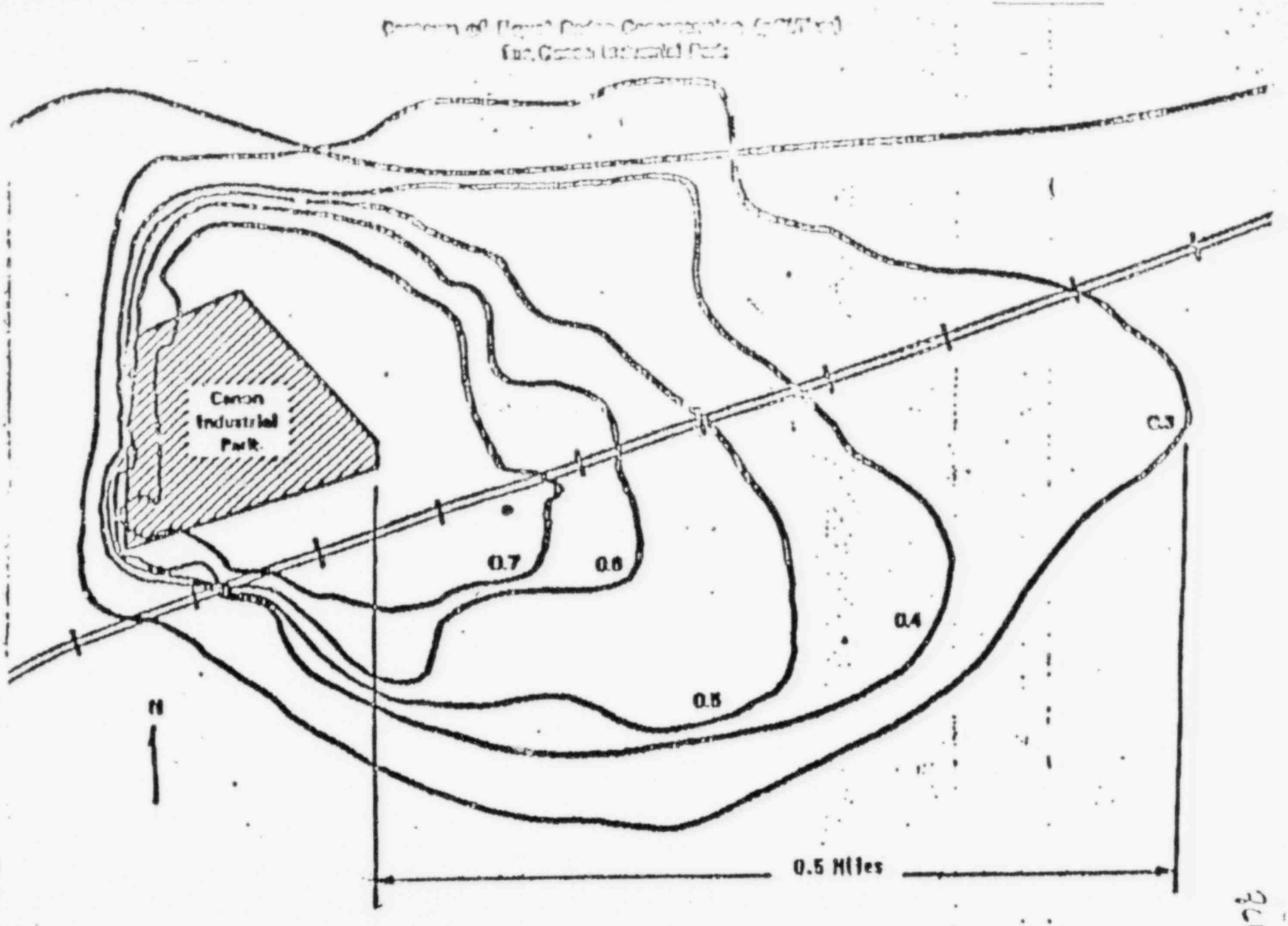
EPA PROPOSAL	IMPACT ON DOE REMEDIAL ACTION PROGRAMS/HEALTH	SUGGESTED ALTERNATIVE	EFFECT OF CHANGE
3.Stabilization or removal of a disposal site.	Program Will involve large sums of money and very high costs.	DOE is considering the potential implications of stabilizing only the high priority processing sites, and fencing and control access to the medium and low priority sites.	Program Significantly reduced implementation costs.
	Health Resulting benefit to public health is not justified by the costs of meeting the standard.		Health At least 58-71% of the EPA estimated health effects per century (excluding vicinity properties) will be eliminated. (See the table below.)

Fatal Cancers Per Century - Local Populations Only (1)

Certain High Priority Sites(2)	Relative Risk	Absolute Risk
Grand Junction, Colorado	29	18
Gunnison, Colorado	3	2
Rifle, Colorado (1 site)	1	1
Shiprock, New Mexico	4	3
Canonsburg, Pennsylvania	29	17
Salt Lake City, Utah	72	79
TOTALS	138 (58% of Deaths per century)	120 (71% of estimated 170 deaths per century)

(1) Data taken from EPA's DEIS

(2) Local population effects were not presented for the high priority sites at Durango, Colorado, and Riverton, Wyoming in the DEIS.


Canon
Industrial
Park
0.7
0.6
0.5
0.4
0.3
N
0.5 Miles

Enclosure 3

INDOOR RADON AND RADON DECAY PRODUCT LEVELS FROM VARIOUS REGIONS IN THE UNITED STATES[1]

HOUSE LOCATION	APPROPRIATE NUMBER OF HOMES	AVERAGE RADON CONC (pCi/L)	AVERAGE WL	MEASUREMENT CONDITIONS	% ABOVE .01 WL	% ABOVE .02 WL
NY/NJ - EML						
(basements)	18	2.0	0.01	YR*	39%	17%
(first floors)	18	1.0	0.007		17%	0%
Grand Junction, CO - CDH						
(background)	29	(1.1)**	0.007*	YR	25%	0%
Florida						
(phosphate) - EPA & CDHS	100	(2.5)	0.015	YR	40%	25%
(background) - EPA	29	(0.5)	0.0033	YR	3%	0%
(background) - CDHS	28	(0.65)	0.004	YR	4%	0%
(background) - UF	13	0.8+	0.004+	YR	14%	14%
Butte, MT - EPA & MBMG	56	(3.3)	0.02	YR	75%	30%
Anaconda, MT - EPA & MBMG	16	(2.6)	0.013	YR	56%	25%
Alabama and Neighbor States - TVA						
(phosphate slag, first floors)	9	(2.8)	0.017	Jan-May	40%	40%
(phosphate slag, basements)	17	(3.6)	0.018	Jan-May	76%	35%
(control, basements)	5	(2.8)	0.014	Jan-May	60%	40%
San Francisco Region - EML	25	0.3	(0.002)	Grab D&W Closed++	0%	0%
Energy Efficient Homes (various locations) - EML	17	4.0	(0.027)	Grab D&W Closed	76%	30%
Soda Springs, ID - IDHW (phosphate slag, basements)	100	1.6+	0.006+	Grab D&W Closed	25%	2%
Illinois - ANL (with unpaved crawl space)	22	6 houses > 10.0 pCi/l of Rn 9 houses > 5.0 pCi/l of Rn		Grab Sample D&W Closed		(41% > 0.03 WL)

[1] Data obtained from Radiation Policy Council Task Force, position paper on Radon in Structures, August 15, 1990.

* YR means year round average under occupied conditions (air pump integrated measurements).

\+ Geometric mean.

** Values in parenthesis are not direct measurements, but are calculated using a characteristic radon decay products/radon equilibrium ratio of 0.5 for basements and 0.61 elsewhere.

++ Doors and windows of structure were closed for a time before taking a grab sample or continuous measurement.

CANADIAN RADON & RADON DECAY PRODUCT LEVEL WITHIN STRUCTURES

Enclosure 4

26-1

CITY	AVERAGE CONCENTRATION: RADON	AVERAGE CONCENTRATION: RADON DAUGHTERS	NUMBER (%) OF STRUCTURES ABOVE 0.014 WL	NUMBER (%) OF STRUCTURES ABOVE 0.020 WL	NUMBER OF STRUCTURES SURVEYED
Calgary, Alta.*	0.31pCi/l	0.0019 WL	23 (3%)	2 (0%)	900
Charlottetown, P.E.I.*	0.41pCi/l	0.0018 WL	25 (3%)	2 (1%)	814
Fredericton, N.B.*	0.66pCi/l	0.0032 WL	51 (11%)	15 (3%)	464
Halifax, N.S.*	-	0.0021 WL	147 (17%)	46 (5%)	881
Montreal, P.Q.*	0.25pCi/l	0.0014 WL	37 (5%)	5 (1%)	800
Quebec, P.Q.*	0.28pCi/l	0.0013 WL	30 (5%)	12 (2%)	584
St. John, N.B.*	0.27pCi/l	0.0018 WL	64 (7%)	24 (3%)	867
Charlottetown, P.E.I.*	0.15pCi/l	0.0022 WL	104 (11%)	57 (6%)	903
St. John's, Nfld.*	0.20pCi/l	0.0015 WL	29 (5%)	4 (1%)	583
St. Lawrence, Nfld.*	0.25pCi/l	0.0017 WL	45 (10%)	18 (4%)	435
Sudbury, Ont.*	0.17pCi/l	0.0036 WL	139 (18%)	53 (7%)	772
Thunder Bay, Ont.*	0.14pCi/l	0.0025 WL	39 (6%)	14 (2%)	627
Toronto, Ont.*	0.31pCi/l	0.0015 WL	39 (4%)	7 (1%)	781
Vancouver, B.C.*	0.14pCi/l	0.009 WL	1 (10%)	0 (0%)	823
Marsh Township, Ont.**	0.22pCi/l	0.014 WL	85 (25%)	46 (13%)	343
Long Harbour, Nfld.**	-	0.001 WL	2 (2%)	0 (0%)	179
Castlegar-Trail, BC**	-	0.011 WL	79 (32%)	43 (17%)	251
Port Hope, Ont.***	0.08pCi/l	0.003 WL	-	• 89 (3%)	2961
Deloro Ltd.**	-	0.013 WL	-	• 86 (16%)	532
Elliot Lake**	-	0.009 WL	-	•307 (16%)	1921
Uranium City**	-	0.004 WL	-	• 9 (13%)	66
Bancroft Area**	-	0.007 WL	-	•197 (17%)	1162
Glace Bay**	0.60pCi/l	0.0015 WL	-	8 (3%)	97

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*** Culgan, E.E. and C.E. Rizzardo (Atomic Energy Control Board), "Modification of the Natural Radionuclide Distribution by Some Nuclear Activities in Canada," a paper presented at the Third International Symposium on the Natural Radiation Environment, Houston, TX, April 23-28, 1978.

NOTES:

1 Homes built on uranium ore bodies

2 Uncrushed fluoride precipitate waste slag used in building materials

3 Castlegar-Trail chosen due to high radon in drinking water; (Radon daughter average for the 247 homes under 150 WL)

4 Concentration from mill tailings

5 A cobalt smelter is the area handled arsenical ore and Port Hope residue

6 Control area used in Canadian Study to represent typical background values

Table 5

ESTIMATES OF TIME AND COST PER ACRE (ABOUT 4,000m^2) TO CERTIFY COMPLIANCE WITH 5pCi/g RADIUM-IN-SOIL STANDARDS USING AVAILABLE MOBILE FIELD LABORATORY INSTRUMENTATION (1)

	5pCi/g
o 95% Confidence that "Any" sample does not exceed pCi/g of Radium-226 in soil	- 1 sample taken from each m^2 (4,000 total) - Blend and analyze composite of 25 samples from each 25 m^2 (160 total) - 1 of every 4 composite samples confirmed at permanent lab - TIME: 28 person-days - COST: $9,600
o 90% Confidence that a statistical average of samples does not exceed 5 pCi/g of Radium-226 in soil	- An average of 4 samples taken at random from each 100m^2 (2) (160 total) - Analyze each sample - 1 of every 10 samples confirmed at permanent lab - TIME: 16 person-days - COST: $5,700 (3)

(1) Mobile laboratory systems included a gamma ray spectrometer

(2) Typical ground area for a house

(3) The savings provided by statistical averaging will increase as operational quality assurance experience is gained.

Health Physics Pergamon Press 1969. Vol. 17, pp. 77–88. Printed in Northern Ireland

EVALUATION OF ATMOSPHERIC RADON IN THE VICINITY OF URANIUM MILL TAILINGS

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Abstract—A field study is currently being concluded at Grand Junction and Durango, Colorado, and at Monticello and Salt Lake City, Utah, to evaluate the public health aspects of atmospheric ^{222}Rn in the vicinity of uranium tailings piles. Air is collected continuously for 48 hr periods from each station in order to average out diurnal fluctuations. The integrated sample is then analyzed for ^{222}Rn. A total of 57 stations is in operation. Each station is re-sampled every three weeks for one calendar year to include seasonal fluctuations and to permit a more realistic estimate of the yearly dose to people in the immediate vicinity.

This paper presents data and conclusions from the 1 yr study. Of the 13 stations directly over tailings, average radon concentrations ranged from 3.5 pCi/l. to 16 pCi/l. Of the 44 stations not over tailings, only two averaged greater than 1 pCi/l. The results indicate negligible radiation exposure of the surrounding population from this source.

INTRODUCTION

The impact of uranium milling operations on the air and water environment has been studied by a number of Federal and State agencies as well as private industry since the middle 1950's. There are numerous references in technical literature to these studies. Most of the investigations to date have been concerned with potential water pollution from the milling operations. Increased attention has been focused during the past several years, however, on the atmospheric effects of such operations, particularly the influence of unstabilized mill tailings piles on the atmospheric radiation levels in surrounding communities.

Limited atmospheric grab sampling in the vicinity of several uranium mills in 1966, indicated that the ^{222}Rn emanation from the tailings might be increasing environmental levels significantly. However, the sampling program was minimal and the results were limited in their interpretation. Because of increasing public interest, a more elaborate joint study described herein was initiated.

OBJECTIVES

In 1966 a joint Federal position regarding control of uranium mill tailings was agreed to by the U.S. Department of Interior (USDI), U.S. Department of Health, Education, and Welfare (USDHEW), and U.S. Atomic Energy Commission (USAEC). On March 3, 1967, it was agreed that the U.S. Public Health Service (USPHS) and USAEC should act jointly to provide technical advice and assistance to states and industry in evaluating the public health aspects of atmospheric concentrations of radon due to the piles and in evaluating the effectiveness of the stabilization and containment of uranium mill tailings piles in controlling radon emanation. This goal is divided into several specific objectives:

1. To develop techniques for taking integrated air samples for radon near uranium tailings piles

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2. To evaluate atmospheric concentrations of radon in areas near piles as an index to radiation exposure of population

3. To determine the effect of stabilization and covering on emanation of radon gas from piles

4. To develop joint recommendations (or guidelines) if necessary to control exposures based on radon concentrations found

5. To make the above information available to states through technical assistance and published reports.

Radon-222 criteria have been developed by the National Committee on Radiation Protection (NCRP) and the International Commission on Radiological Protection (ICRP) for individuals occupationally exposed. The maximum permissible concentration (MPC_a) of 30 pCi/l. for a 40 hr week and 10 pCi/l. for a 168 hr week, respectively, have been set for occupational exposure to ^{222}Rn plus its daughter products in equilibrium. It is permissible to adjust these concentration limits if it is found that ^{222}Rn is not in equilibrium with its daughter products.

The NCRP (NBS Handbook 69, p. 6) states that the maximum permissible average body burden of radionuclides in persons outside of the controlled area and attributable to the operations within the controlled area shall not exceed one-tenth of that for radiation workers (i.e., based on continuous occupational exposure for a 168 hr week), and that this recommendation is primarily for the purpose of keeping the average dose to the whole population as low as reasonably possible, and not because of the likelihood of specific injury to the individual.

Recommendations of the ICRP state that the annual radiation dose limits for members of the public shall be one-tenth of the corresponding annual occupational values for continuous exposure. Application of these recommendations to the 168 hr value for occupational exposure leads to a value of 1 pCi/l. for ^{222}Rn, with daughter products present at equilibrium concentrations, for continuous exposure to individuals in the general population. This concentration is in addition to natural background radon levels. Thus, for the purpose of this study, 1 pCi/l. of ^{222}Rn in air was used as the screening value below which no further attention would be given to the concentrations involved. Should data obtained from the survey indicate average annual exposures to concentrations of ^{222}Rn in air in excess of 1 pCi/l. above background, it would be necessary to consider the extent of radioactive disequilibrium between radon and its daughter products.

While Federal regulations generally[1] permit average concentrations of ^{222}Rn up to 3 pCi/l. in air leaving the boundary of a restricted area of a licensed facility, quantities permitted to be released from the restricted area are subject to reduction if it appears that they might result in the exposure of a suitable sample of an exposed population group to average concentrations of ^{222}Rn in excess of 1 pCi/l.

DESCRIPTION OF STUDY

Station locations

In order that the study areas reflect conditions at both operating and inactive uranium mills, locations were chosen initially at Grand Junction, Colorado (operating mill, uncovered tailings), Durango, Colorado (inactive mill, uncovered tailings), and Monticello, Utah (inactive mill, covered tailings). Salt Lake City, Utah (operating mill with only vanadium recovery presently being carried out, uncovered tailings), was added to the study in October 1967. At each of these study areas, a number of sampling sites were selected which were representative of on-pile, near-pile and general-community conditions. Locations were chosen taking into consideration the prevailing wind patterns, population densities, geographical factors, etc. At Grand Junction, 25 sampling sites were chosen with 5 being located directly on the tailings pile; at Durango, 8 sites were chosen, 2 of which were on the tailings pile; at Salt Lake City, 12 sites were chosen with 1 station being on the tailings pile and another immediately adjacent; and at Monticello, 12 stations were established, 4 of which were on the tailings area. Therefore, at the 4 study areas a total of 57 sampling sites were selected, 13 of which were directly over tailings material.

Sampling techniques and frequency

The equipment used to collect the samples has been described by Still.[2] A small aquarium aerator pump forces filtered air through a precision needle va
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precision needle valve into a 40 l. laminated Mylar bag at 10 ml/min to give a volume of 30 l. in each 48 hr sampling period. At each sampling location, the pump and bag were placed in a protective weather enclosure. The pump inlet was at a height of 3 ft above the ground or tailings. Sampling was carried out over a 48 hr period in order that 2 diurnal cycles might be covered. The sampling schedule was designed such that each location would be sampled once every 3 weeks over a 12 month period. By sampling in this manner any bias due to daily or seasonal meteorological variations would be minimized.

Analytical procedures

After an extensive intercomparison period, half of the samples collected were analyzed at each of the two laboratories by accepted analytical procedures. The analytical technique consisted of passing a known volume of sample through a gas separation apparatus and absorbing the radon on activated charcoal at low temperature. The charcoal was then heated and the radon transferred to an alpha-sensitive scintillation cell for subsequent counting.

In order to determine the consistency and agreement between the two laboratories' results, all samples collected during the first six weeks of the study were analyzed in duplicate at both laboratories. The data obtained demonstrated that both laboratories' results were the same within the uncertainty of the counting statistics involved. Following this initial extensive intercomparison, about 5% of the remaining samples were analyzed in both laboratories as a continuing cross-check on quality control. All counting data were corrected for radioactive decay to the midpoint of the respective sampling periods. In addition, the concentrations as determined in the laboratory were calculated to the mean annual pressure and temperature at each of the study locations. These are as follows: Grand Junction, 638 mm, 11°C; Durango, 599 mm, 8°C; Monticello, 587 mm, 15°C; and Salt Lake City, 654 mm, 11°C, respectively.

RESULTS

During the period of sampling for this program, a total of 892 samples was collected, 209 of which were on-pile samples and 683 were off-pile samples. The numbers of on-pile and off-pile samples by study area are respectively, 85 and 320 in Grand Junction, 30 and 82 in Durango, 31 and 150 in Salt Lake City, and 63 and 131 in Monticello. The concentration of ^{222}Rn found at each station in the four study sites is given in Table 1. The data shown in Table 1 covers the following dates at each of the four study areas:

Grand Junction	June 6, 1967–August 20, 1968
Salt Lake City	October 27, 1967–October 8, 1968
Monticello	August 8, 1967–July 29, 1968
Durango	August 3, 1967–July 30, 1968

As stated earlier, each of the 57 sampling stations was re-sampled at three-week intervals throughout the study.

Table 2 presents an overall summary of the

Table 1. Average concentrations of ^{222}Rn found at the four study areas

Station	Number of samples	Concentration of ^{222}Rn, pCi/l. Average	Standard deviation	Range
GJ 11*	17	11	6.7	3.3–28
12*	17	4.7	1.7	2.2–7.1
13*	17	9.0	3.6	4.6–21
14*	17	5.7	2.4	1.1–9.7
15*	17	8.7	3.9	3.5–16
16	17	1.1	0.33	0.50–2.6
21	16	0.77	0.35	0.43–1.8
22	17	1.3	0.67	0.53–2.3
23	17	3.4	3.0	0.60–13
24	17	0.78	0.45	0.31–2.2

Table 1 cont.

Station	Number of samples	Concentration of ^{222}Rn, pCi/L Average	Standard deviation	Range
25	17	0.84	0.45	0.28–1.9
26	17	0.82	0.50	0.30–2.2
31	16	1.1	0.88	0.42–3.3
32	16	0.75	0.41	0.43–1.8
33	17	1.8	1.1	0.50–4.5
34	15	0.84	0.39	0.46–2.2
35	17	0.88	0.51	0.32–2.1
36	17	0.88	0.41	0.33–1.9
41	16	0.69	0.30	0.13–1.2
42	16	0.70	0.27	0.27–1.1
43	15	0.78	0.48	0.17–1.9
44	16	1.1	0.93	0.48–4.4
45	16	0.82	0.33	0.40–1.3
46	15	0.74	0.43	0.34–2.1
47	10	0.79	0.48	0.29–1.9
SLC 81*	16	10	6.4	1.6–22
82†	15	4.2	1.4	2.3–6.6
83	16	0.43	0.31	0.09–1.3
84	15	0.39	0.24	0.21–0.85
85	13	0.68	0.30	0.23–1.4
86	13	0.28	0.14	0.11–0.54
91	15	0.44	0.30	0.15–0.99
92	14	0.42	0.24	0.17–0.85
93	14	0.24	0.12	0.06–0.55
94	17	0.44	0.26	0.07–0.94
95	17	0.29	0.16	0.13–0.60
96	16	0.22	0.12	0.06–0.45
M 61*	17	4.1	2.0	1.1–8.1
62*	15	2.4	1.2	0.55–4.4
63*	15	4.5	3.1	1.1–12
64*	16	3.1	1.8	0.89–6.1
65	17	0.52	0.29	0.18–1.3
66	16	0.41	0.17	0.12–0.64
71	15	0.34	0.18	0.10–0.72
72	17	0.24	0.13	0.03–0.53
73	16	0.29	0.17	0.06–0.59
74	17	0.24	0.13	0.06–0.59
75	16	0.31	0.18	0.14–0.87
76	17	0.40	0.20	0.11–94
D 51*	16	19	7.2	3.8–34
52*	14	12	9.1	7.5–32
53	15	0.49	0.17	0.25–0.75
54	10	1.4	0.67	0.44–2.3
55	17	0.52	0.27	0.11–1.3
56	14	0.47	0.17	0.22–0.78
57	13	0.59	0.35	0.17–1.2
58	13	0.47	0.34	0.09–1.3

GJ = Grand Junction; SLC = Salt Lake City; M = Monticello; D = Durango.
* On-pile stations.
† This station is not actually on-pile, but adjacent to pile at sewage plant.

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Table 2. Summary of on-pile and off-pile stations at the four study areas

Area	Number of stations	Number of samples	Concentration of ^{222}Rn, pCi/l. Average	Range
Grand Junction				
On-pile	5	85	7.8	1.1–28.0
Near-pile*	4	68	1.9	0.50–4.5
Other	16	252	0.83	0.13–4.4
Durango				
On-pile	2	30	16.0	3.8–34.0
Station 54	1	10	1.4	0.44–2.3
Other	5	72	0.51	0.09–1.3
Monticello				
On-pile	4	63	3.5	0.89–12
Off-pile	8	131	0.34	0.03–1.3
Salt Lake City				
On-pile	2	31	7.2	1.6–22
Off-pile	10	150	0.38	0.06–1.4

* Near-pile stations: 16, 22, 23, 33.

on-pile and off-pile radon concentrations for the four study sites. Four of the off-pile stations in Grand Junction, and one of the off-pile stations in Durango, have been shown separately from the other off-pile stations. The reasons for this will be explained below. The averages are also shown in Figs. 1–4 with the corresponding sampling location at each of the four study areas. For the sake of clarity, only the principal city and residential areas have been shown. In Fig. 1, the average of the on-pile stations is shown instead of the individual averages for clarity.

DISCUSSION OF RESULTS

The conclusions and trends are presented below for each of the four study areas:

Grand junction

It is apparent when examining these data that there are three distinct groups: (1) on-pile stations; (2) near-pile stations in the prevailing wind patterns; and, (3) all other stations.

The wind patterns shown as dotted lines on Fig. 1 represent conditions which occur about 80% of the time. The early daytime winds are from the southeast while the afternoon and evening winds are from the northwest. The shift is about equally divided in the two directions, being about 53% of the time from the southeast and 47% of the time from the northwest.

It is interesting to note that station 11, which has the highest average ^{222}Rn concentration, is positioned at the northwest corner of the main tailings pile at the foot of a 25 ft bluff in the prevailing wind envelope for the nocturnal inversion conditions. Thus, this station can be expected to intercept the drainage of radon from the pile down the slope during periods of highest concentration. In contrast, the lowest value of the on-pile stations is obtained at station 12 which is almost in the geometric center of the pile. The pile is slightly conical with the station near the apex. Apparently, except under conditions of strong vertical mixing, the radon drains down the slope and away from the sampler, minimizing the concentration available at a height of 3 ft where the inlet is located.

As stated above, it was considered valid to consider stations 16, 22, 23 and 33, as a separate group from the other remaining off-pile stations. There are several reasons for this: (1) they are all about a half mile from the pile; (2) they are in the predominant wind envelopes; (3) the area is characteristically industrial; and, (4) they are all on the mill side of the river. In

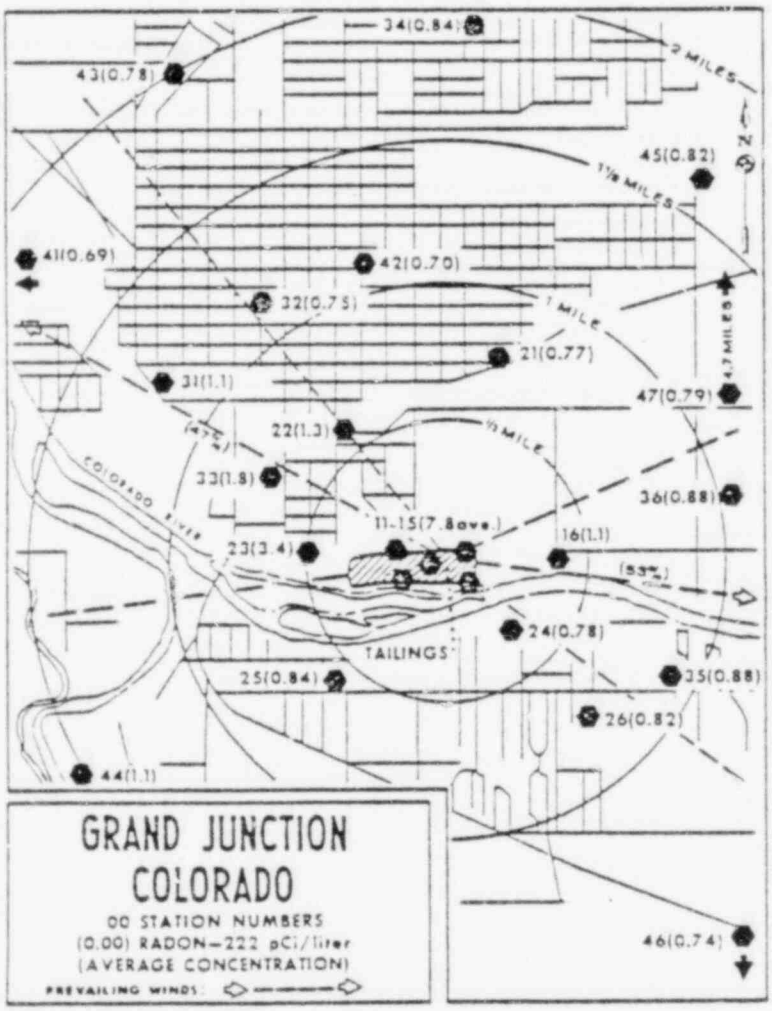

FIG. 1

addition, standard *t*-test and analysis of variance comparisons of station averages clearly showed that stations 23 and 33 were significantly different from the other off-pile station averages. Stations 16 and 22, while exhibiting higher averages than off-pile stations, were not determined to be significantly higher, but were considered, because of the reasons stated above, to fall into the same "population group" as stations 23 and 33.

Station 24 was not included in this group because, while it is less than one half mile from the pile and is in the predominant wind envelope, it is situated across the river close to the edge of a mesa about 80 ft higher than stations 16, 22, 23 and 33. Station 23, as can be seen on Fig. 1, is the closest off-pile station and has the highest average.

The remaining sixteen off-pile stations were considered as a third group of stations. For these stations, the individual station averages were compared utilizing standard *t*-test and analysis of variance comparisons of the averages. The results of these statistical techniques showed

FIG. 2

that none of the individual station averages could be considered significantly different (at the 95% confidence level) from each other and, therefore, would represent data from the same "population group."

The above conclusion is quite significant when comparing station 47 with the others. Previous to 1966 a quantity of tailings material was distributed widely throughout the Grand Junction community. These tailings were used as fill material, sub-grade material under buildings, and other uses. It was felt, therefore, that the tailings pile at the mill might not represent a single point source of radon release to the general environment, and that the distributed tailings might influence the radon concentrations at outlying stations. To eliminate this possible variable, it was decided to set up a station at a considerable distance from the main tailings pile in an area where it was known for certain that no tailings had been distributed. Therefore, station 47 was located at a distance approximately five miles north of the main tailings pile. This station was placed in operation in early February 1968. As can be seen from Table 1, the average radon concentration

FIG. 3

for the study period was 0.79 pCi/l. This average is not significantly different from the overall average of the off-pile stations of 0.83 pCi/l. It is thus concluded that the radon concentrations at station 47 are indicative of natural background levels in the Grand Junction area. In comparing this station's average with the other off-pile stations (exclusive of stations 16, 22, 23, and 33), it appears reasonable to conclude that the distributed tailings, as well as the main tailings pile, are not increasing appreciably the general atmospheric radon concentrations in Grand Junction.

Because of lack of correlation of ^{222}Rn concentration with distance from the pile, and because a known background station indicates a value of at least 0.79 pCi/l., it appears reasonable to consider that the natural background concentration of atmospheric ^{222}Rn for Grand Junction as a whole is 0.8 pCi/l. If this figure is subtracted from the other values, no off-pile stations are above the value of 3 pCi/l. permitted by Federal regul areas. Stations 23 above, the screenin mended for this stud are well within eith

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Durango

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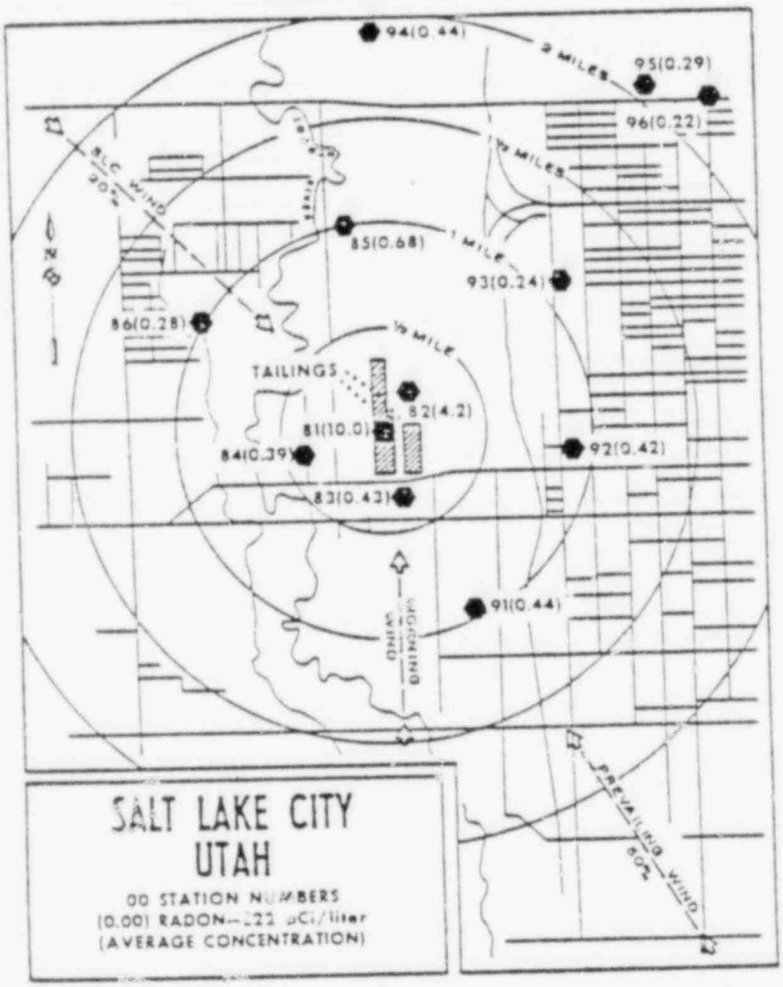

FIG. 4

by Federal regulations[4] for uncontrolled areas. Stations 23 and 33 are at, or slightly above, the screening level of 1.0 pCi/l. recommended for this study. All other station averages are well within either of these two figures.

No definitive quantitative correlation of radon concentrations with distance from the tailings pile could be obtained.

Durango

The on-pile station averages at Durango are considerably higher than the other study locations. The most likely reason for this is the physical location of the sampling sites. Due to the very steep side slopes of the tailings pile as well as its location against the side of a mountain, it was not convenient to locate the sites directly on top of the pile. Instead, the samplers were located on tailings material at the base of the tailings dike. It is likely that the radon diffusing out of the entire side slope of the pile will drain directly down to the sampler at the base of the pile under almost all meteorological conditions, ensuring a high concentration of radon at all times.

The wind patterns at Durango are shown

as dotted envelopes in Fig. 2. The daytime winds are from the southeast and split toward the northeast and west at about the mill area. The nighttime winds are just the reverse.

Standard *t*-test and analysis of variance comparisons of the averages of stations 53 through 58 showed that station 54 was significantly higher than the others and that all other stations were not significantly different from each other. Thus, Table 2 shows station 54 separately from the other off-pile stations. Station 54 is only one-quarter mile southeast of the pile inside the wind envelope for the nocturnal inversion conditions, and is across the river at the sewage treatment plant. It is reasonable to conclude that the average of all off-pile stations with the exception of station 54 represents the natural background levels for this area. This average, as shown in Table 2, is 0.51 pCi/l. If this figure is subtracted from the average at station 54, it is determined that the resulting net average is 0.89 pCi/l. or less than the screening level of 1 pCi/l. As in Grand Junction, no correlation of radon concentration with distance could be made with the Durango data.

Monticello

During its operational period, the Monticello mill was owned by the USAEC. Following termination of operation, the tailing areas were leveled and covered with earth and a grass cover planted. This stabilization was completed in 1962. As stated earlier, one objective of this study was to determine the effect of stabilization and covering on emanation of radon gas from piles. For this reason, Monticello was selected as one of the study locations. As seen in Fig. 3, there are four distinct tailings areas at Monticello. It was deemed desirable to locate a sampling station on each of these areas. Other sampling locations were located taking into consideration prevailing wind patterns.

Standard *t*-test and analysis of variance comparisons were made on all the off-pile stations and the results showed that none of the off-pile station averages was significantly different from the others. This indicates that the tailings are not having any significant effect on the environmental radon levels off the tailings area. The average of all off-pile stations is 0.34 pCi/l. and this figure can be considered to represent background for the Monticello area. If this value is subtracted from the other results, even the on-pile net average concentration of 3.2 pCi/l. is only slightly above the guideline permitted by regulation[1] for continuous exposure in uncontrolled areas.

Salt Lake City

For purposes of averaging, stations 81 and 82 are considered as on-pile stations although station 82 is actually closely adjacent to the tailings at the sewage treatment plant which is occupied only 40 hr a week. The off-pile station averages were compared using a standard *t*-test and an analysis of variance, and it was found that there was no significant difference among stations.

As in Monticello, the off-pile station average of 0.38 pCi/l. was considered to be the most reasonable estimate of the natural background concentration in Salt Lake City. Subtracting the background gives values for the on-pile station and the sewage treatment plant of about 3 and 1¼ times the regulatory guideline, respectively.

Inter-city comparison

Because of the significant differences in the distances and orientation of samplers with respect to the piles, differences in meteorological variables, and the shape, size and condition of the piles themselves, it is extremely difficult to make comparisons among cities from the present data. There is certainly no evidence of significant contribution of radon to any of the cities beyond 0.5 mile from the piles. Even comparison of the piles themselves is imprudent. Estimates of the ^{226}Ra content of the tailings material shows the concentrations to be almost identical for all four cities at about 900 pCi/g. Consequently, concentrations of ^{222}Rn above these tailings might also be expected to be quite similar. The data of Table 2 show this to be true for the piles at Grand Junction and Salt Lake City. It is tempting to postulate that the concentration at Durango would also have been similar if it had been possible to locate the sampling station on top of the pile and if the pile was essentially flat as with the other two.

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placed on tailings material will result in some decrease in the radon emanating from the tailings if for no other reason than the additional radiological decay resulting while the radon is diffusing through the covering. Based on the similarity of the tailings at the four study locations, and the actual on-pile radon concentrations obtained thus far, it is tempting to conclude that the type of stabilization carried out at Monticello has been effective in reducing the radon concentration over the tailings by approximately 50%. However, this is a tenuous conclusion at best. The Monticello piles contain about half as much tailings spread over a much larger area than the other three piles and the shapes are significantly different. Variations in vertical mixing due to meteorological conditions are undoubtedly more important in determining air concentration above the piles than a small variation in specific activity of the tailings or type of surface.

Upon comparing the off-pile data among the four study areas, it is reasonable to conclude that the radon concentrations in Durango, Monticello, and Salt Lake City, are quite similar and are about one-half that in Grand Junction. Thus, it is apparent that the general Grand Junction area has a naturally higher ^{222}Rn background than the other locations studied.

Thermoluminescent dosimeter results

During the latter part of this study it was considered desirable to obtain information on external radiation exposure at a number of the radon sampling stations. Accordingly, thermoluminescent dosimeters (TLD's) were placed at each of the four study sites.

The TLD's used were of the CaF_2:Mn type. They were shipped from the Las Vegas laboratory and placed at a number of stations. At each station three TLD's were placed at the same height above the ground as the air intakes of the radon samplers. They were left exposed for thirty days and returned to Las Vegas for readout. The dates of exposure were June 17–July 17, 1968.

The net average exposure results obtained are given in Table 3.

Those stations situated directly over tailings (with the exception of Monticello) range from 0.2 to 1.1 mR/hr. This range agrees well with values found during previous environmental surveys at the Tuba City, Arizona, and Mexican Hat, Utah tailings areas. The Monticello site (covered pile) shows exposures of 0.03 and 0.06 mR/hr on the pile. These values agree with those presented by Paas.[3] All other stations show normal background levels of exposure. The difference between Grand Junction off-pile stations (0.02 mR/hr) and the other city off-pile stations (0.01 mR/hr) is insignificant.

Table 3. Thermoluminescent dosimeter results

Location	Station	Net average exposure (mR/hr)
Grand Junction, Colorado	11*	0.2
	12*	0.4
	16	0.02
	33	0.02
	42	0.02
	47	0.02
Durango, Colorado	51*	0.4
	55	0.02
	58	0.01
Monticello, Utah	63*	0.03
	64*	0.06
Monticello, Utah	72	0.01
	74	0.01
Salt Lake City, Utah	81*	1.1
	85	0.01
	86	0.01
	91	0.01
	96	0.01

* On-pile stations.

SUMMARY AND CONCLUSIONS

This paper presents the results of a joint PHS-AEC year long study to evaluate the atmospheric ^{222}Rn concentrations in the vicinity of uranium mill tailings piles. Samples were collected from 13 on-pile and 44 off-pile stations in the 4 study cities of Grand Junction and Durango, Colorado, and Monticello and Salt Lake City, Utah. The tailings pile at Monticello has been covered and stabilized and the other three are uncovered and unstabilized. The side slopes of the pile at Grand Junction are partially stabilized with a grass cover.

On the basis of the data which have been

gathered at the four study sites, it is possible to reach the following conclusions:

1. The natural background ^{222}Rn concentration at Grand Junction averages 0.8 pCi/l. and is approximately double the background concentrations at the other three study sites.
2. The tailings which have been widely distributed throughout the community of Grand Junction do not raise significantly the general environmental levels of radon.
3. The tailings at Grand Junction are not affecting the atmospheric radon concentrations beyond a distance of 0.5 mile in the prevailing wind directions. At the other three study locations the effect of tailings is not observed at distances greater than one-quarter to one-half mile.
4. Of the 44 off-pile stations, only 2 exhibited an average radon concentration (exclusive of background) equal to or higher than 1 pCi/l. screening value adopted for this study.
5. Due to diffusion of radon through the covering, the type of stabilization carried out at Monticello has probably reduced the on-pile radon concentrations by about half, although any real definitive percentage reduction would have to be based on before and after stabilization sampling.

In conclusion, some attempt should be made to interpret these data in terms of the radiological dose actually received. The MPC for ^{222}Rn assumes complete equilibrium with its short-lived daughters which actually contribute about 95% of the total dose. These conditions would be obtained only directly over tailings under completely stagnant weather conditions or at considerable distances from the pile. With a 10 mph wind, the composition of air at the 0.5 mile are would correspond to the so-called "3 min air", i.e., air aged sufficiently to build up 50% of the equilibrium activity of 3.05 min ^{218}Po and little else. Consequently, the actual dose received will generally be far less than that inferred from the concentration of ^{222}Rn alone. At distances more remote than 0.5 mile, the daughters might more nearly approach equilibrium conditions but the total concentrations will have ben so diluted as to be virtually negligible.

Acknowledgment—Grateful appreciation is expressed by the authors to many individuals of the USPHS, the USAEC, Lucius Pitkin, Inc., Climax Uranium Co., and the Health Departments of the states of Utah and Colorado, for their considerable contributions and assistance to this study. Without their help, the study would not have been possible.

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BEFORE THE

UNITED STATES DEPARTMENT OF LABOR

ASSISTANT SECRETARY OF LABOR FOR

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION

WASHINGTON, D.C.

In Re: PROPOSED REGULATIONS FOR IDENTIFICATION, CLASSIFICATION AND REGULATION OF TOXIC SUBSTANCES POSING A POTENTIAL OCCUPATIONAL CARCINOGENIC RISK	OSHA DOCKET NO. H-090

DIRECT TESTIMONY OF
RICHARD WILSON

TABLE OF CONTENTS

The Concept of Risk

1. Introduction

The principal theme in this testimony is a better definition of the phrase "lowest feasible level" with respect to occupational exposure. Until it is defined, the aim of OSHA of simplifying the hearing process will be nullified. If "lowest feasible level" is to be defined as essentially zero or whatever level is technically possible, then the whole proposal of OSHA is unworkable and it will be attacked in all possible ways. Scientifically, one can reduce almost any exposure without limit—at increasing expense. But we can't afford to spend the whole Gross National Product on one chemical alone.

If, as I propose, the phrase is defined in a common sense manner—and more formally in terms of a risk analysis—then there is a clear working procedure for all parties to follow. I summarize my proposed procedure for risk calculation:

1) Human data should be used whenever possible, but animal data, in at least two different mammalian species, may be used as a surrogate for human data. If animal data in only one mammalian species or mutagenesis data exist and show a very high carcinogenic potency, this can be used to signal an immediate need for more data and the limited data can be used in the interim for limited purposes.

This listing demands slightly more proof of carcinogenicity than that of OSHA's group I. I believe this is appropriate and seems to agree better with recommendations of government committees.

2) We need to know the risk at low exposure levels, and it is hard to obtain statistically significant data at low exposure levels.

therefore, data at high exposure levels must be used and an interpolation made between these data and the point with zero effects at zero exposure. The preferred technique for simplicity and for a prudent (conservative) public policy is a linear interpolation with no threshold.

3) Data on exposure of humans or animals over a lifetime should be used when possible. When data is only available which covers part of a lifetime, then the risk can be estimated for a full lifetime using a reasonable theory.

4) The cancer risks we ask workers to accept should be comparable to other risks we ask workers to accept and hopefully progressively lower as civilization proceeds. All risks must be reduced, and it is appropriate to reduce the largest risks first, and those risks which are the least costly to reduce. But it is unrealistic to demand that risks due to carcinogens be reduced much more than risks due to other causes.

5) Associated with a linear no threshold theory is usually the statement that at low doses we should measure a long term average exposure to calculate the carcinogenic risk. Fluctuations about this average, while they might affect the actual risk if a threshold is assumed, will not affect the risk if the linear theory is used. This theory remains a conservative upper bound to the risk even in the presence of exposure fluctuations. A more detailed summary of my views on these matters is in reference 1.

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[1] "The Risks of Low Levels of Pollution," Richard Wilson, Yale Journal of Biology and Medicine, Jan/Feb, 1978.

2. The risk/benefit concept

Once it is decided that a chemical is a carcinogen and poses a risk, it remains to decide what to do about it.

It would be nice if it were inexpensive and easy to reduce the exposure to the chemical to such a level that the risk were zero. But life isn't that simple—or at least we cannot prove that it is that simple. As noted later, although there are distinguished scientists who believe that there is a dose below which there is no carcinogenic risk in a human lifetime, there are other scientists who do not; there is no way experimental evidence can directly distinguish the two cases and the argument remains theoretical. However, as noted later, there are few who believe that the cancer incidence is worse than linear with dose, so that a bounding, reasonably conservative, estimate of risk can be made.

If it were possible to reduce all exposure to zero we could reduce all risks to zero. But there are many risks in life—most of them fortunately small—and we cannot reduce all of them to zero simultaneously. We must therefore compare the risks of different actions to cause the same benefits, of different actions to reduce overall risk and then compare the risk and benefit of each action. This is not stated in the OSEA proposal, but it is stated by many advisory boards, including most of those quoted by OSEA in support of the OSEA classification proposal. For example, the National Cancer Advisory Board Subcommittee on Environmental Cancer[2] says:

[2] "General Criteria for Assessing the Evidence for Carcinogenicity of Chemical Substances: Report of the Subcommittee on Environmental Carcinogenesis," National Cancer Advisory Board, Journal of the National Cancer Inst. 58, 461 (1977).

"In those cases where a compound has been proved to be carcinogenic, there remains a decision to what extent the possible risks to man are counterbalanced by the possible social, economic, or medical benefits of that substance. Scientists must play a major role in these decisions by providing the available data. The final decision, however, must be made by society at large through informed government regulatory and legislative groups."

In a statement on May 22, 1976, Russell Train,[1] Administrator of EPA said:

"I believe that it is important to emphasize the two-step nature of the decision-making process with regard to the regulation of a potential carcinogen. Although different EPA statutory authorities have different requirements, in general two decisions must be made with regard to each potential carcinogen. The first decision is whether a particular substance constitutes a cancer risk. The second decision is what regulatory action, if any, should be taken to reduce that risk."

"In other regulatory areas, for example those under the Clean Air Act, the Federal Water Pollution Control Act, or the Safe Drinking Water Act, where a large number of suspect carcinogens may exist in the atmosphere or public water supplies, the detailed risk benefit assessment will, because of limited Agency resources, necessarily have to be carried out on a priority basis in terms of which agent appears to be the most important."

"Once the detailed risk and benefit analyses are available, I must consider the extent of the risk, the benefits conferred by the substance, the availability of substitutes and the costs of control of the substance. On the basis of careful review, I *may* determine *that the risks are so small or the benefits so great that no action or only limited action is warranted.* Conversely, I may decide that the risks of some or all uses exceed the benefits and that stronger action is essential." (my italics)

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[1] Federal Register, 41102, Tuesday, May 25, 1976.

The first application of the risk/benefit approach was probably in the burgeoning radiation industry in the 1920s. The recommendations of advisory committees in this field are therefore the most sophisticated. A National Academy Committee says:[4]

> "Logically the guidance or standards should be related to risk, whether we regard a risk as acceptable or not depends on how avoidable it is, and to the extent not avoidable, how it compares with the risks of alternative options and those normally accepted by society."
>
> "a) no exposure to ionizing radiation should be permitted without the expectation of a commensurate benefit.
>
> b) the public must be protected from radiation but not to the extent that the degree of protection provided results in a worse hazard for the radiation avoided. Additionally there should not be attempted the reduction of small risks even further at the cost of large sums of money that spent otherwise would clearly produce greater benefit."

The committee goes on to say:

> "When the risk from radiation exposure from a given technological development has been estimated, it is then logical for the decision-making process that comparisons be made and considerations given to (a) benefits to be attained, (b) costs of reducing the risks, or (c) risks of the alternative options including abandonment of the development."[5]

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[4] The effects on populations of exposure to low levels of ionizing radiation. Pages 2-3. Report of the Advisory Committee on Biological Effects of Ionizing Radiation (BEIR) National Academy of Sciences November 1972.

[5] Ibid. Ch. II p. 7. (Needs of the Times)

Dr. Philip Handler, President, National Academy of Sciences introduced a National Academy Symposium on the subject. Among other statements, he makes the following remarks:[6]

"The burden on the scientific community is to provide adequate basis for such decisions in the future."

"The second difficulty lies in the calculus.... in many cases the dimensions on the two sides of the equation are non-equivalent... with dollars on the one side and on the other human lives or less quantifiable social amenities.... But until we settle that question we will be unable to engage in logical decision making in many instances."

In a recent report[7] a World Health Organization group concludes:

"In those situations where carcinogens are unavoidable, or where the banning of a substance would impose a hardship or an unrealistic economic burden, the toxicologist must assess the risks associated with different levels of exposure."

The fact that carcinogens can vary in their potency by a factor of 10 million—from aflatoxin to saccharin—suggests a graded response to risks. Thus Schneiderman[8] says (and I agree) "materials should be assessed in terms of human risk rather than as safe or unsafe."

Others use the phrase an "acceptable level or risk." Thus Dr. Kraybill[9] of the National Cancer Institute says:

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[6]"How Safe is Safe?—the design of policy on drugs and food additives. National Academy of Sciences (1974) pp. 3, 4.

[7]"Assessment of the Carcinogenicity and Mutagenicity of Chemicals," WHO Technical Report Series 546 (1974).

[8]"Establishing Cancer Risks to a Population," M.A. Schneiderman and C.C. Brown, Environmental Health Perspectives (1977).

[9]"Pesticide Toxicity and Potential for Cancer: A Proper Perspective," H.F. Kraybill, Pest Control, page 9, Dec. 1975.

"One should not exhaust one's energy and resourcefulness in the unexorable task of looking for a 'zero' exposure, the 'no effect level' but should view the problem in the context of an 'acceptable level of risk.' This concept should hold for a carcinogenic or noncarcinogenic event."

Sir Edward Pochin, M.D. of the Medical Research Council, devotes a whole paper[10] to discussing risks which society has chosen to accept. Pochin's paper emphasizes that the question of what risks are acceptable is a question for society as a whole: for common man to decide with the facts placed before them in a commonplace way. The scientist can present the facts, his interpretation, and his recommendation based upon what society has decided, implicitly or explicitly in previous situations.

That it is a political decision is emphasized by John Higginson, M.D., Director of the International Agency for Research on Cancer in Lyon, France[11] who warns also that it can be an elitist one:

"However undesirable, 'political oncology' exists and must be accepted by oncologists and public health officials as a fact of life. Nonetheless perfect environmental control at the expense of the material environment is essentially a concept of the wealthy society. It ill behooves those who have benefited from the industrial society to deny less privileged communities the same material benefits unless the reasons are clear cut and impelling."

In discussing some of the political effects, Higginson goes on to say:

"the concept of acceptable risk is widely accepted in some form or another."

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[10] E.E. Pochin, Brit. Med. Bull., 31, 184 (1975)

[11] "A Hazardous Society? Individual versus community responsibility cancer protection," Third Annual B. Rosenhaus Lecture, J. Higginso Am. Journal Public Health, 64, pp. 361, 363 (1976).

> "In accepting [it] we should be guided by common sense and honesty. We should not subject others knowingly to risks that we would not accept for ourselves or for our families. The decisions on socially acceptable risks which imply the calculation of costs/benefits should not necessarily be confined to an elite group but rather be established through a consensus of society as a whole and/or its respresentatives *assisted by experts.*" (my italics)

The important role of balance in the political decision is stressed by those actually responsible for environmental protection. Thus K. Mellanby, Director of the Monks Wood Experimental Station of the English Nature Conservancy and Editor of the Journal of Environmental Pollution, writes:[12]

> "Some ecologists harm their cause by overstating their case and by condemning any industrial development even if they do not hesitate to make use of the products of that industry! We need to recognize 'real' risks, and to concentrate on eliminating them while at the same time using our technology properly for the benefit of mankind."

One of the advantages of a logical procedure of risk analysis is that it can reduce polarization. John Dunster, Deputy Director General of the (UK government) Health and Safety Executive, says:[13]

> "Some risks are clearly so unacceptable that they must be eliminated. Others, less severe or less likely should be reduced to the point where the benefits of the risky activity balance the costs of the ill effects. Striking the balance invariably involves compromise."

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[12] K. Mellanby, "Unwise Use of Chemicals," Keynote paper in 1st International Conference on the Environmental Future, Finland 1971, p.143, Barnes & Noble, Inc., Ed. N. Polunin

[13] John Dunster, "The Risk Equations, Virtue in Compromise" The New Scientist, 10 May 1977.

We can base an acceptable risk on what is already present. A group of World Health Organization advisors quoted by Truhaut,[14] states:

> "As or where sensitive and reproducible quantitative measures become available, it will be possible to define levels of carcinogens naturally and undeniably present in our environment. From such knowledge it may then be possible to establish 'socially acceptable levels of risk' for carcinogens in the work place and in the general environment."

This suggests an attempt to allow carcinogens if they only add a little to what is already present. For radiation, the internationally regulated tolerance dose is close to the natural background level. But it is possible to do better—to evaluate a risk and to compare the risks. This is the procedure I propose for OSHA.

The Federation of American Scientists, a public interest lobbying group of some distinction, in a report in May 1976, say:

> "There is needed some simple measure of cost and benefit that would make widely different risk situations comparable so as to maintain, in different areas, roughly similar standards for spending government and industrial funds to save lives. Without such a standard, as economists will sense immediately, cancer-avoiding expenditures cannot be spent efficiently. And, in addition the public will have the greatest difficulty distinguishing minimal risks from large ones."

Scientists active in public causes also discuss risk analysis. Barry Commoner[15] points out that it is a societal decision

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[14] "Can Permissible Levels of Carcinogenic Compounds in the Environment Be Envisaged?" Ecotoxicology and Environmental Safety 1, 11 (1977)

[15] "Saccharin and Cancer," Washington Post, Sunday, March 27, 1977.

> "Balancing the benefits against the risks belongs not in the domain of science but to society. The judgment is a value judgment--a social rather than a scientific decision."

Later he says:[16]

> "Based upon widespread concern about health and environment problems the public appears to be ready... to determine what balance between the hazards and benefits is acceptable."

As I look through the scientific literature I find no author who states that a risk comparison is not the way to proceed, although some believe that the "public" is not ready to accept such analyses. Part of this testimony is to show, by comparative analyses, how the facts for decision can be put in a form which is easy to understand so that acceptance is easier.

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[16] Barry Commoner, N.Y. Times Magazine, September 25, 1977, p.73.

3. Risk/benefit analysis

There are several stages to a risk/benefit analysis:

1) The risk must be evaluated. This risk contains two factors: the exposure to the carcinogen, and the carcinogenic effect at this exposure This will be a simple product if it is assumed that the dose-response relationship is linear. The slope of this dose-response relationship is the potency and we obtain a simple relationship at low doses.

Fractional tumor incidence = potency x exposure and at high doses, since the tumor incidence cannot exceed 100% fractional tumor incidence = 1 -exp [-potency x exposure]. (See Figure 1b later.) The carcinogenic risk is the fractional tumor incidence in a large body of people.

Exposure is typically measured in milligrams of pollutant ingested daily or in milligrams per kilogram of body weight when we wish to compare carcinogenic potency between species.

Since the risk, or the fractional tumor incidence, is a number without dimensions, the dimensions of potency are the reciprocal of the dimensions of exposure, or typically 1/(mg ingested daily) or 1/(mg ingested per kilogram body weight). As I will show, this is a conservative estimate (overestimate) of the risk and as such is suitable for a prudent public policy.

2) The benefit of the product must be evaluated.

a. The benefit to society as a whole must exceed the risk. However, the analysis must not stop here. In the 1920's physicians using x-rays for diagnosis correctly argued that the great benefit

outweighs the risk. Cautious scientists pointed out that the same benefit can be achieved at much less risk by inexpensive and simple measures (dose reduction, shielding, film sensitizers, etc.) which are now adopted. This makes clear that even if the benefit to society exceeds risk we must still proceed to ask—

b. Can we obtain the same benefit with less risk by using another chemical or other substitutions. This question is not now within the province of OSHA. Moreover, only if the other chemical is an easy one to use, or a cheap one, is the question simple. Then the question become a subsidiary of questions.

d. Can we reduce the risk at reasonable (to be defined later) expense?

d. Finally, we must ask are the benefits properly disaggregated?—meaning do enough benefits accrue to those directly undertaking the risk? This can, in an extreme case be by compensation or hazard pay.

I will assume that the items 2(a) and 2(b) have already been decided—probably by the marketplace. I will focus on items (c) and (d) in my testimony. A listing of benefits and risks to be considered are in an NAS report[17] from which I take the following table.

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[17] "Principles of Evaluating Chemicals in the Environment," Report of the Committee for the Working Conference on Principles of Protocols for Evaluating Chemicals in the Environment. National Academy of Sciences, 1975, Chapter III, Benefits.

TABLE I

BENEFITS	RISKS
1 Value to the consumer a) practical utility b) aesthetic value	1 Adverse effect on health a) well being and general health b) death
2 Conservation of natural resources and energy	2 Environmental damage a) air, water, and land pollution b) wildlife c) vegetation d) aesthetic e) property damage
3 Economic a) Employment b) Regional Development c) Balance of Trade	3 Misuse of natural resource and energy sources

Finally, I refer to some summaries of the risk/benefit analysis and its public perception [18, 19, 20, 21, 22] which discuss risk/benefit analysis in other situations.

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[18] W.W. Lawrence, "On Acceptable Risk" Kaufman (1976).

[19] E.M. Clark & A.J. Van Horn, "Risk Benefit Analysis and Public Policy", a Bibliography. Informal report by Energy & Environment. Policy Center, Harvard University for Brookhaven National Labora BNL 22285, (1976) (Dec.)

[20] "Perspectives on Benefit-Risk Decision Making," National Academy Engineering (1972)

[21] A.J. Van Horn and Richard Wilson, "The Status of Risk Benefit Analysis," Informal report by the Energy and Environmental Policy Center, Harvard University for Brookhaven National Laboratory. BNL 22252, Dec. 1976. Printed in Science Policy Implications of DNA Recombinant Molecule Research, Hearings before the House Subcommittee on Science and Technology, U.S. House of Representati No. 24, page 751, 1977.

[22] A.J. Van Horn and Richard Wilson, "Factors Influence the Public Perception of Risks to Health and Safety--A Brief Summary Report," Energy and Environmental Policy Center for Brookhaven National Laboratory (1977).

4. Evaluation of the risk--animal and human data

OSHA in the preamble to its October 4, 1977 regulation, spend many pages, citing many authorities, to show that it is necessary to allow animal data alone as a proof of carcinogenesis. This is because it is obviously unacceptable to irradiate humans directly and we want to find out what to do somehow. In many cases where both human data and animal data on carcinogenesis are available (vinyl chloride, radiation) there is moderate agreement--although this is not true of teratogenesis.

I therefore accept the OSHA recommendations that one can accept animal data when human data is unavailable or too inaccurate. Mutagenesis data can also be a useful supplement as a screening test, as I will show later.

I also believe that it is vital to realize that carcinogens vary in their potency and that a mere statement of it is/(is not) a carcinogen is not only unhelpful, but is likely to change as detection sensitivities improve. At the moment this is not recognized in the OSHA proposal.

5. Evaluation of risk--dose-response relationship

The important feature to recognize about a dose-response relationship is that there is one. For example, society used to allow men to work where there were high levels of vinyl chloride in the air--greater than 1 part in 1000 and even up to 10%. Sixty-six cases of liver angiosarcoma have occurred worldwide over 30 years as a result. Now occupational exposures have been reduced a factor of 1000. Will the number of cancers go down by this same factor of 1000 to a level of one cancer in 300 years or more than one in 300 years or less than one in 300 years?

It is only in rare cases that we have data on carcinogenesis in humans suitable for developing a dose-response relationship. But we do know, for example from the work of Doll and Hill,[23] that smoking 40 cigarettes a day gives 10 times the incidence of lung cancer as smoking 4 a day and not smoking at all gives a much lower incidence. This is in agreement with a linear, non-threshold, dose response relationship. For radiation carcinogenesis we also have a linear relationship, with a possible reduction at low dose rates. This reduction is, for example, taken into account by the Nuclear Regulatory Commission in their reactor safety report, where further details can be found.[24] There are also indications from a large scale animal test carried out at the National Center for Toxicolog-

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[23] R. Doll and A.B. Hill, Brit. Med. J., 1, 1399 (1964). See also A. Whittemore and B. Altshuler, "Lung Cancer Incidence in Cigarette Smokers. Further analysis of Doll and Hill's data for British Physicians." NYU Medical Center report.

[24] Nuclear Regulatory Commission, Reactor Safety Survey--WASH 1400/ NUREG 75-014, Appendix VI commonly called the "Rasmussen Report."

Research that the dose-response curve for liver cancers is non-linear at low doses.[25] But in most cases, even if data exist to prove human carcinogenesis enough does not exist to establish a clear dose response relation for humans. We must then rely on animal data and analogy.

In the vinyl chloride case, the animal data suggest that the reduction of a factor of 1000 in occupational exposure reduces the death rate by at least a factor of 1000 to a level one cancer in 300 years or less.

The rule proposed by OSHA on October 4, 1977 implies that exposure to any quantity of a carcinogen involves some risk just as a purely linear dose-response relationship would suggest. It is experimentally impossible to disprove such a concept and we are left with only theoretical concepts to guide us. We might envisage three possible relationships between health effects and dose. Fig. 1(a). Proponents of curve A argue in one of two ways. Some argue that the latent period before a tumor occurs increases as the dose decreases, and that when the latent period equals a human lifetime, there is an effective threshold.[26] Others[27] relate the cancer

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[25] Dr. M.F. Cranmer, Presented at the Nov. 28-29, 1977 Science Advisory Committee Meeting.

[26] R. Doll, "Age Distribution of Cancer: Implications for Models of Carcinogenesis," Journ. of Royal Stat. Soc., 134A, 133 (1971).

[27] R.E. Albert and B. Altshuler, "Considerations Relating to the Formulation of Limits for Unavoidable Population Exposures to Environmental Carcinogens," Proceedings of the 12th Annual Hanford Biology Symposium, pp. 231-253 (1972).
"An Assessment of Environmental Carcinogen Risks in Terms of Life Shortening," Environmental Health Perspectives, 11, 91 (1976).
E.B. Jones and A. Grendon, "Analysis of Mathematical Models Used in Data Extrapolation," Clinical Toxicology, 9,791 (1976).
E.O. Hartley and R.L. Sielken, "Estimation of Safe Doses in Carcinogenic Experiments" Biometrics, 11, 1 (1977).

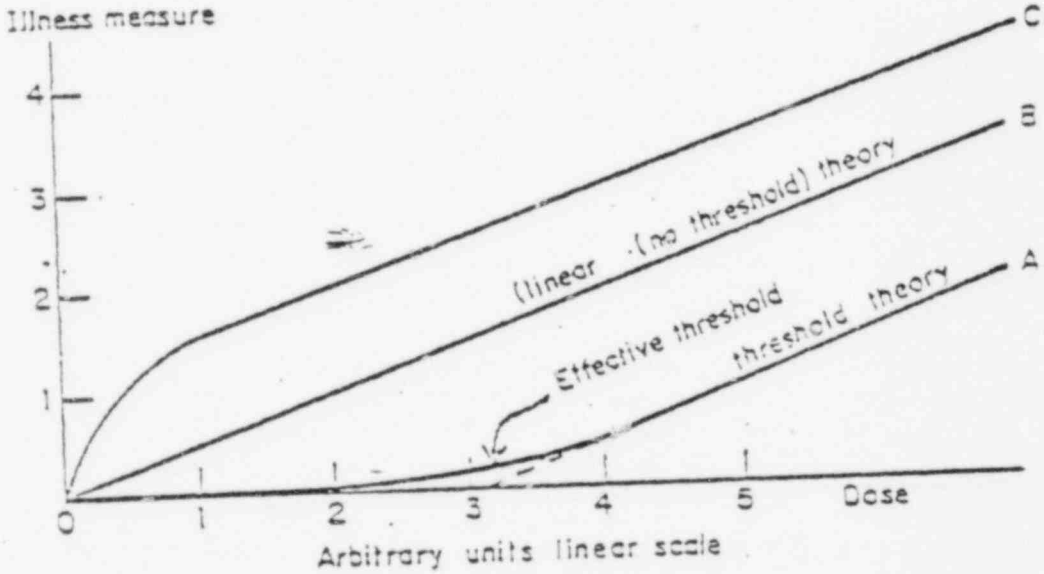
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Fig. 1 (a)

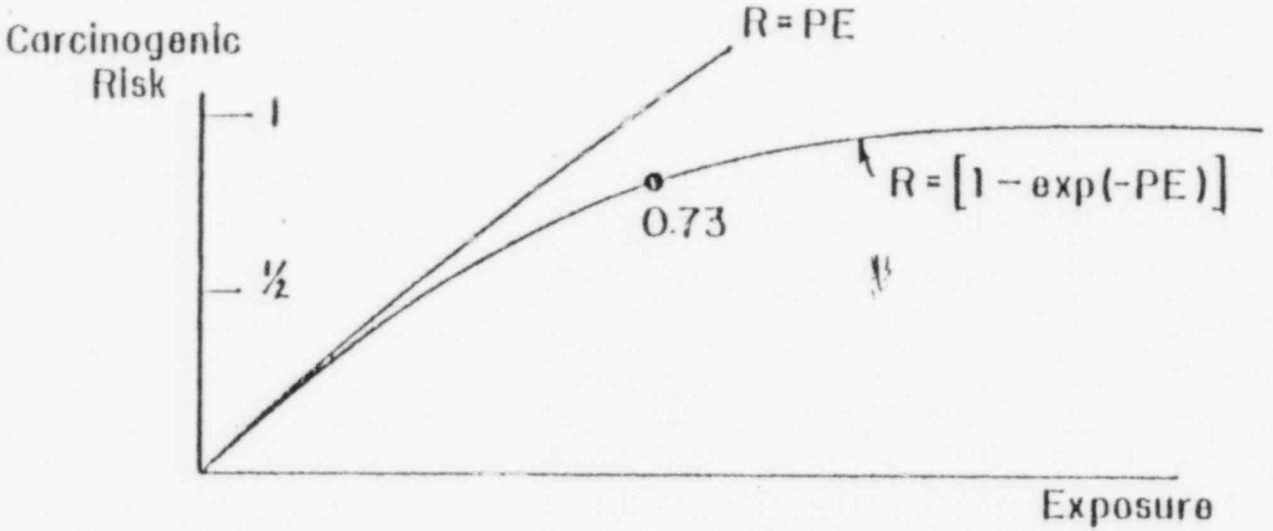

Fig. 1b

induction to some mechanism which implies a threshold. (Curve B comes from the "one hit" theory that the probability of a cancer is random and proportional to the total chemical insult.[28])

The important point, however, is that there is no widespread view that curve C is probable and in this sense the linear, no threshold curve B represents a conservative (pessimistic) hypothetical calculation. It is recommended by many government advisory committees[29] and academic scientists.

I believe curve B should be used for a conservative risk estimation. There should be no need for other safety factors in a risk estimation. It has a further major advantage (as noted earlier) the ease of simple calculation and simple comparison with other risks.

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[28] P.J. Gehring and G.E. Blau, "Mechanisms of Carcinogenesis--Dose Response," Journ. for Env. Path. and Toxicology, 1, 163 (1977). R. Olson, Hearings before the U.S. Department of Labor, OSHA, In Re Proposed Standard for Occupational Exposure to Benzene, OSHA Docket No. H-59 (1977) (Direct Testimony).

[29] International Commission on Radiological Protection, (ICRP), publications 1-10, Pergamon Press, London, Oxford and New York. "The Effects on Population of Exposure to Low Levels of Ionizing Radiation," Report of the Advisory Committee on the Biological Effects of Ionizing Radiation, National Academy of Sciences, November 1972.
"Pest Control, An Assessment of Present and Alternative Technologies," Report of the Consultive Panel on Health Hazards of Chemical Pesticides, Environmental Studies Board, National Academy of Sciences, 1975.
"Estimation of Risks of Irreversibly Delayed Toxicity," D.G. Hoel, D.W. Gaylor, R.L. Kirschstein, U. Saffiotte, M.A. Schneiderman, Journ. Toxicity and Env. Health, 1, 133 (1975).

There may be one or two cases already where the mechanism of carcinogenesis is such that use of a non-linear relationship can be clearly justified; more may appear as more research is conducted. Presumably these could be specifically argued.

It is perhaps interesting to note that this linear hypothesis seems to fit air pollution data[30] and also it is noted by the WHO[31] that incidence of cirrhosis of the liver is directly proportional to the alcohol intake in the country (with a correlation coefficient of 0.93 based on 14 countries with average annual intake varying from 4 to 25 liters per capita).

At low exposure levels the probability of any one person getting cancer in a lifetime is small. Associated with the concept of a linear, non-threshold (or proportional) theory, is the idea that at low doses the important parameter is the dose averaged over a long period of time. Calculation of occupational risks must take this into account.

I note that there is another popular interpolation procedure due to Mantel and Bryan.[32] This relies on a "log normal" distribution and falls between my curves A and B. This fits data as well or better than the linear-non threshold theory. In most cases of interest it leads to a less conservative prediction at low doses. i.e. it suggests a lower cancer risk than the linear theory. For conservative policy the linear theory is to be preferred.

[30] Several curves from Norwegian, Japanese and U.S. data are presented in W.J. Jones and Richard Wilson, Energy, Ecology and the Environment, Academic Press, New York, 1974, Chapter VIII.

[31] WHO Chronicle, 1975.

[32] N. Mantel and W.R. Bryan, "Safety Testing of Carcinogenic Agents," J. Nat. Cancer Inst., 27 455 (1961).

6. The risk assessment—specific successions

On the rare occasions where good data exists on cancer in humans, such as for cancer caused by cigarette smoking, this should be used for a risk analysis. The dose-response curve of health effect versus long-term average dose should be plotted and a straight line taken from the lowest statistically significant point* to the origin. This can be used as the risk. If a lifetime exposure dose is not known, the data may be corrected to a lifetime cancer incidence using cancer statistics, or if these are not available, using the Weibull formula $dN/dt = \lambda t^k$ which was shown by Armitage and Doll[33] to be a good fit to the age distribution of cancer, with $1 < k < 8$ depending upon the site of the cancer, and λ varying with the geographical location or the environment. For the purposes here, I suggest this formula be used as a useful summary of world cancer data.

Thus, if data exist for people exposed for only the first half of their lives a lifetime incidence for continuous exposure comes by increasing the measured incidence by 2^{k+1}.

If the human data are not statistically significant, reasonable upper limits can still be usefully obtained from the data by drawing a straight line from the top of the statistical error bars to the origin.

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[33] P. Armitage and R. Doll, "The Age Distribution of Cancer and a Multistage Theory of Carcinogenesis," Brit. Journ. of Cancer, 8 1 (1954).
R. Doll, "Age Distribution of Cancer, Implications for Models of Carcinogenesis," Journ. Royal Stat. Soc., 134A, 133 (1971).

*I suggest here that statistically significant be taken as when the random statistical error be less than 1/4 of the value of the point.

If (mammalian) animal data are available, the dose response curve should be plotted to get either a value or an upper limit, as suggested for human data. This then should be related to humans by relating an animal's life (2 years for a rat) to the full human life of 75 years and for equal ratios of the daily food intake divided by the body weight. In some early reports, if 10% of all rats developed cancer in their lifetimes when fed 1 part in 1000 of a carcinogen in their diet, it was assumed that 10% of humans would develop cancer when fed 1 part in 1000 of a carcinogen in their diet. This probably overstates the risk to humans as animals eat a far larger fraction of their weight in food than humans do.

If data on mutagenesis (Ames' test) are available, it should be compared to the animal and human data on carcinogenesis in a plot such as that of Meselson[34] (Figure 2). If mutagenesis data suggest a greater potency (and thereby suggests a larger risk) on this plot it should be used as a signal that the animal data may not be adequate. But I would not recommend that mutagenesis data replace animal data, since there exists carcinogens that are not mutagenic in tests[35] presumably because of inadequate metabolite production, and some mutagens may not be carcinogens.

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[34] M. Meselson and R. Russell, Proc. of the Cold Spring Harbor Conference on Origins of Human Cancer, New York (1977).

[35] "Short-term Screening Tests for Carcinogens," Bryan N. Bridges, Nature, 261, 195 (1976).

mg/kg/day giving 50% tumor induction in 24 Months
10^2
10
1
10^{-1}
10^{-2}
10^{-3}
moca
mms
2-naphthylamine
benzidine
4-aminobiphenyl
dibenz (a,h) anthracene
propane sultone
benzo (a) pyrene
sterigmatocystin
aflatoxin B1
10^{-3}
10^{-2}
10^{-1}
1
10
μg per 100 SALMONELLA REVERTANTS

Fig. 2

Finally, these data can be combined with exposure data (averaged over a long time) to get a conservatively estimated risk.

An excellent discussion of the procedure arises in the discussion of the carcinogenicity of saccharin.[36] I agree with this procedure almost in its entirety.

It is also important to realize that, associated with low levels of pollutant and a linear dose-response relationship, we are interested only in levels of pollutant averaged over a long time—and appreciable fraction of a lifetime. Although there are probably good toxicological reasons for preventing short exposures to very high concentrations of carcinogens, these reasons are not taken account of in this present calculational procedure and must be, and are, dealt with separately, by the ordinary rules and regulations for toxic chemicals.

In calculation of a cancer risk it is important to bear in mind that cancer can appear in sites other than the primary one. This is true of cigarette smoking, where only half the cancers caused are cancers of the lung, and for vinyl chloride where human data suggest that, and animal data show that, only half the cancers are liver angiosarcomas. We know also that, for cigarette smoking, heart disease is also prominent, so for prudent public policy the risk of cancer incidence in the primary site should be multiplied by 4 to get a total risk until other data on risks in the other

36. "Saccharin and Its Salts: Proposed Rule and Hearing," 42 Fed. Reg. 19996 (1977).

sites and risk of heart disease are available. Also, as for cigarette smoke, birth defects can be expected.

In order to make a conservative analysis, therefore, I believe it appropriate to multiply the risk, calculated for a cancer at one site only, by 4.

7. Upper limits to a risk

Occasionally the statistical significance of data is not enough to tell us whether the chemical is carcinogenic or not. This may not mean that we know nothing; we can still establish an upper limit to the risk. I illustrate this by the following (hypothetical) example.

Lifetime exposure experiments are performed for a mammalian species at several dose levels; exposure zero (control series); exposure 100 units; 200 unit; 300 units. The data might then look as follows:

Exposure	No. of animals	No. of animals with tumors at end of life
0	500	20
100	500	14
200	500	25
300	500	19

The number of animals (N) with tumors fluctuates by an amount approximately equal to $\sqrt{N}$ which is the fluctuation due to the number of animals used.

I suggest that if the upper limit to the risk comes out less than 1 in 100,000 per year of exposure (10^{-5}/yr) the question can then be ignored by OSHA. If the upper limit comes out to be greater than 10^{-5}, the question of carcinogenicity must be examined further.

This hypothetical set of numbers shows no obvious trend with increasing dose. We can however rule out the postulate that at an expos-

level 300 the number of animals with tumor is 35 because this would be about 3 standard deviations from the mean.

I illustrate this by plotting the data graphically. We see that the horizontal line (risk = 0) fits the data quite well, although the fit leaves two out of four points with an error just greater than one standard deviation. The same data can be fitted with a risk line Risk = 10^{-4}/exposure with a stretch of the error on the control and on the highest exposure point. This gives a probability of less than 1/20, and I take this here as close to an upper limit.

In practice, then, even statistically insignificant data can give an upper limit to carcinogenic potency, which can in turn be put into a risk calculation.

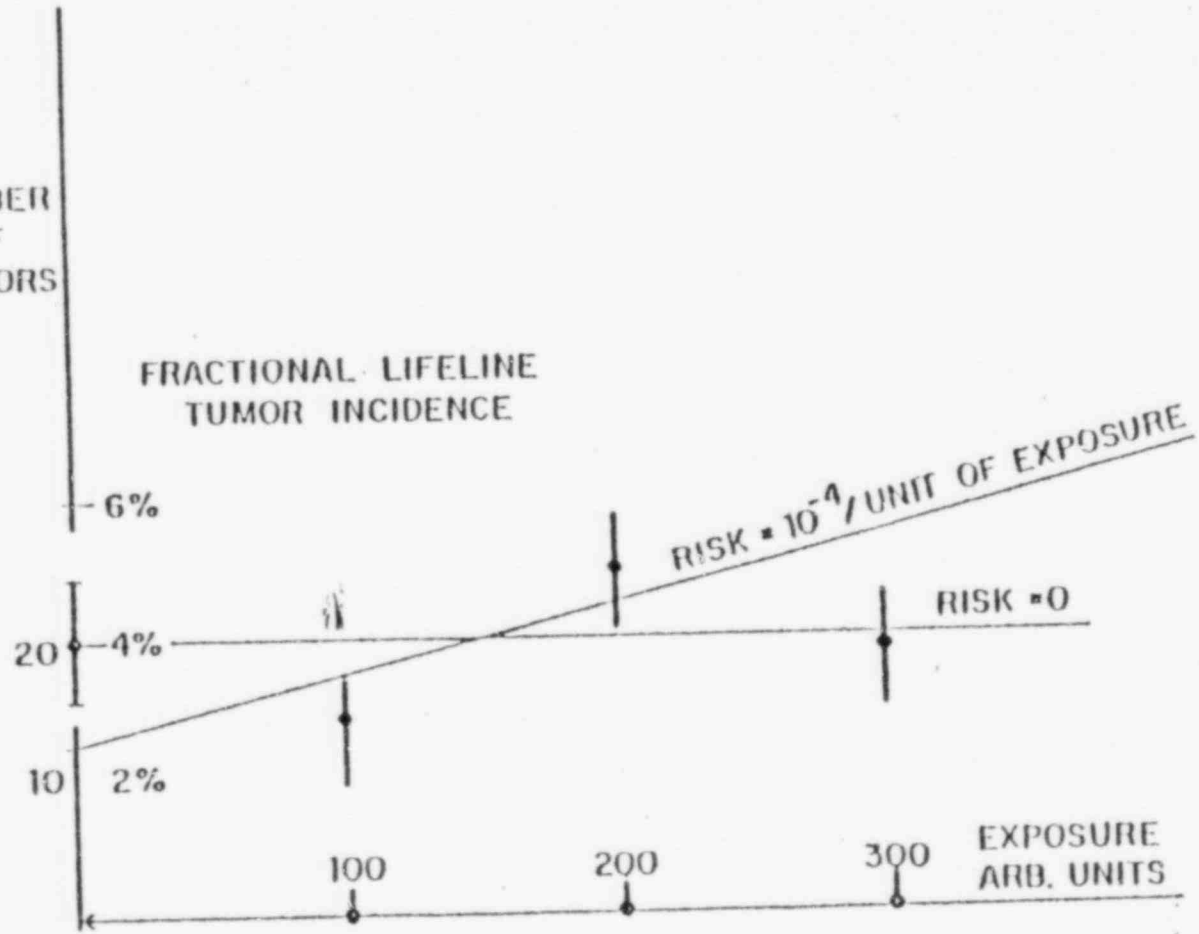
NUMBER
OF
TUMORS
FRACTIONAL LIFELINE
TUMOR INCIDENCE
6%
4%
2%
20
10
RISK = 10^{-4}/ UNIT OF EXPOSURE
RISK =0
100
200
300
EXPOSURE
ARB. UNITS

Figure 3

8. Simplicity . . cost of regulation

It is important that a regulation be simple and easy to administer. There are two reasons. Firstly, it will be enforced, and secondly it will not cost too much to enforce.

The linear dose-response relation with no threshold enables risks to be easily calculated and compared. It probably overstates the risk since it assumes there is no threshold, but—unless we insist on the fruitless search for zero risk—this is likely to be less expensive than regulatory hassles over what the threshold, no effect level, or TLV is for the particular chemical.

It is hard to see how to enforce a criterion on the "lowest feasible level" unless a risk assessment of some sort is made. If it is made implicitly, it becomes subjective, rather than objective. One man can argue it is "feasible" to close down one or two industrial plants; another might not. Interminable arguments would ensue on a chemical by chemical basis.

g. the lowest feasible level

I think the course of action suggested by OSHA is inappropriate because it fails to compare risk and benefit.

OSHA, in the proposed rule of October 1977, suggest that the occupational exposure of all chemicals in group I be reduced to the "lowest feasible level." This phrase is not defined, and to the extent it is undefined, it can allow OSHA flexibility in applying common sense to each situation. However, also to the extent it is undefined, it renders the whole stated purpose of the classification--the saving of time in argument--useless.[37] Furthermore, there is a vague indication that "eventually" the exposure should be reduced to zero and to the extent that zero is unmeasurable, this is meaningless and unworkable.

The International Committee on Radiological Protection (ICRP) also made ill-defined recommendations that "no exposure shall be undertaken without expectation of benefit" and that exposures should be reduced "as low as practicable." This committee was, therefore, an early urger of cost benefit calculations. When nuclear power became important, the Atomic Energy Commission (superseded by the Nuclear Regulatory Commission) found it useful to define the phrase

[37] Debbie Galant writing "Taking Cancer Out of the Workplace," in Environmental Action, a journal of a major lobbying organization, seems to agree. She states: "The problem of determining feasibility may be the clinker in OSHA's cancer policy. A term open to speculation and debate, its vagueness could frustrate the agency's plans."

further and held public hearings over three years.[38, 39]

Likewise, the Environmental Protection Agency is finding problems with enforcement of the ill-defined injunction to use the "best available control technology" and the FDA is struggling with what it means, in these days of sensitive detection methods, to reduce food additives to "undetectable" levels.

I therefore propose that OSHA should define the action to be taken more clearly in terms of risk benefit analysis and for this purpose I suggest a redefinition of category I and a separation of category I into 3 subcategories, (a), (b), and (c), according to the way in which the chemical is used.

Firstly, for all chemicals that are "proven" carcinogens, a risk calculation as outlined above should be mandatory. For new chemicals, or new uses of old chemicals, this should be before the proposed use; for old chemicals, within a stated period --say 5 years--after adoption of the rule.

Secondly, for all chemicals in category I, I would make labeling of risky materials, posting of risky areas and education of workers mandatory. This already happens for one class of worker exposed to carcinogens--radiation workers. It would be an advantage if the procedures were similar between radiation workers and

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[38] The "As low as practicable" hearings, AEC RM-50-1.

[39] The ICRP itself found it useful to change the words. The current form is "all exposures shall be kept as low as reasonably achievable, economic and social factors being taken into account."

chemical workers, because this simplicity would be an aid to understanding. Safety depends on active worker understanding and participation at all levels. An industrial worker can often reduce his risk by his own actions much more easily than his supervisor can. Education and posting of warning signs then would be one of the most important of OSHA actions.

10. Separation of Subcategories

This modified category I can now be separated into its subgroups according to the conservatively estimated risk to the workers. I tentatively propose the following separation and actions to be taken in each group.

Category I(a). Estimated risk of mortality, averaged over a lifetime of continuous work exposure greater than one percent per year (R > 1%/year). In this group steps must immediately be taken to reduce the risk by reducing exposure.

Category I(b). Estimated risk of mortality, averaged over a lifetime of continuous work less than one percent per year but greater than one in one hundred thousand per year (10^{-2}/year > R > 10^{-5}/year). For risks in this region exposure would only be allowed if the cost to reduce them were too great. The cost estimates would no doubt be a subject of discussion between OSHA and industry.

Category I(c). Estimated risk of mortality, averaged over a lifetime of continuous work exposure less than one in one hundred thousand per year (R < 0.00001/year or R < 10^{-5}/year). In these cases the continued worker exposure should be allowed without further question by OSHA at least until other risks are reduced, although OSHA should no doubt continuously monitor the risk, and should probably promulgate maximum exposure levels. Of course, industry might well further reduce the risk without the force of OSHA's regulatory action.

I suggest a figure for the appropriate amount industry should pay for reducing a risk $10 per person per year for a risk 10^{-5}

per person per year ($10 for 10^{-5} risk). This corresponds to a cost of $1,000,000 for every calculated hypothetical life saved. This seems a reasonable number. For example in a plant of 1,000 workers, all of whom are exposed to a cancer risk of 10^{-5}/year, one cancer would be induced every 100 years and if the risk is a hypothetical one and an upper limit there would be none at all. Then to reduce this risk industry should pay $1 million every 100 years or $10,000 per year. The Nuclear Regulatory Commission in their discussions of low levels of radiation to the general public (As Low As Practicable Hearing AEC RM-50-1) suggested $1,000 per man rem of exposure, which corresponds, by a linear, average calculation similar to that suggested here, to $10,000,000 per calculated hypothetical life saved. They considered this figure to be a temporary figure (pending a large public hearing on the specific subject) and reached it as being a round number larger than any number proposed to them at the hearing. I prefer the lower figure and will make comparisons later in this testimony.

This separation of risk levels is similar to one recently recommended in England by the Royal Commission on Environmental Pollution of the United Kingdom in the 6th Report, 1976. This is shown in Reference 39A, from which I take the attached figure.

Professor J.C. Wood of England writing in his book on Industrial law states:[39B]

[39A] W.R. Lee, Brit. Journ. Ind. Med., 34, 274 (1977).

[39B] J.C. Wood, Cooper's Outlines of Industrial Law, 6th Ed., p. 181, Butterworth, London.

"The conduct of any industrial undertaking involves some element of risk to the people employed. An element of danger is something to which in a greater or lesser degree, the employees must get accustomed."

From: Ref 39A

Some critical problems of hazardous substances in the working environment 35

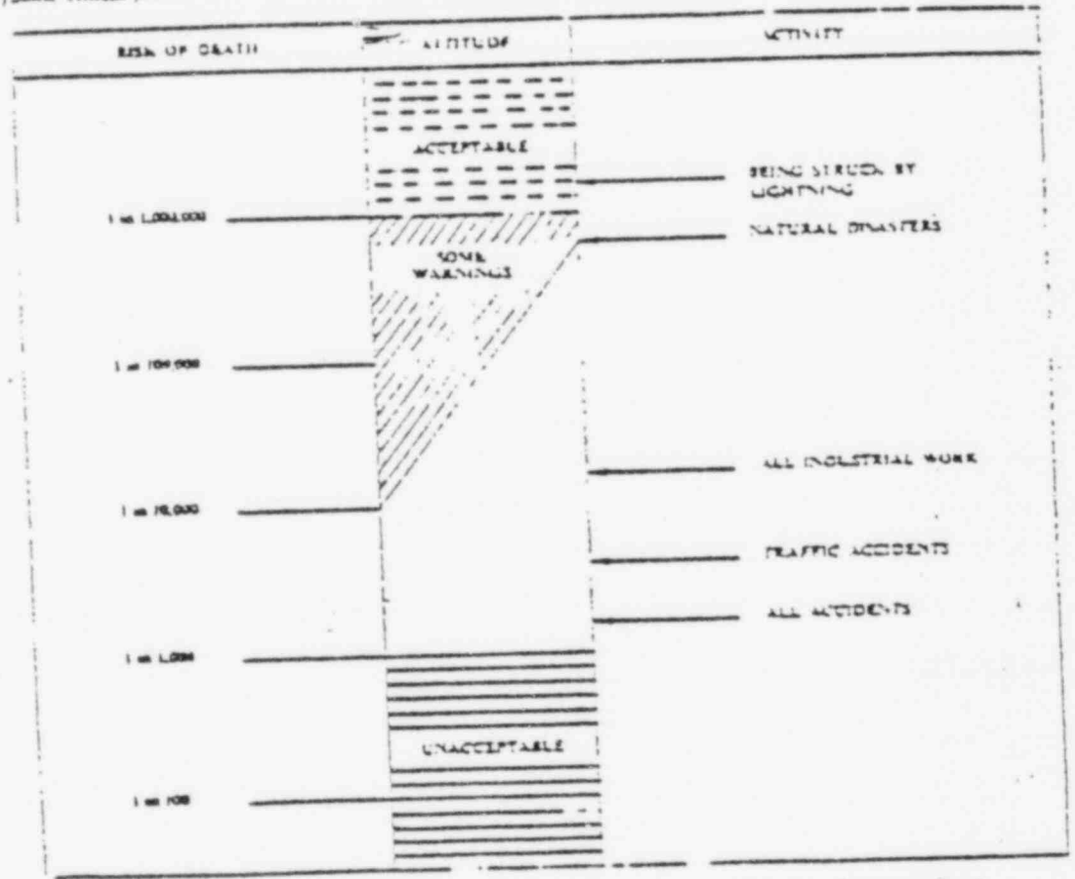

Figure Probability of death for an individual per year of exposure (risks of recreation). (After from Royal Commission on Environmental Pollution, 3rd Report, 1972.)

II. Examples of acceptable risks

As noted above, the acceptability of risks is a political and not a scientific question. I will assume, however, that when properly informed about the risks and their nature the body politic will make sensible decisions. I therefore calculate some risks and show how society in fact accepts many risks. These then are the justification for my suggested limits. I put these in several lists:

1) A list of recreational, voluntary risks (Table II).
2) A set of ordinary involuntary risks (Table III).
3) Since cancer arouses particularly strong emotions, and cancer is the subject of this hearing, I list some cancer risks that are commonplace and presumably accepted (Table IV).
4) A set of occupational risks (Table V).

The recreational risks (Table II) are very hard to quantify because they are so variable. Thus an ardent rock climber will spend more than 40 hours/year in his sport, and be subjected to a higher than average risk. The 100 million bicycles in the U.S. are not ridden with equal enthusiasm. Most people engage in several of these activities—bicycling, sunbathing, fishing, etc. for a recreational risk of 10^{-4}/year; this means that there will be one fatal accident a year for every 10,000 persons engaged in the activity. We watch others, for our enjoyment, who have 20 times larger risks.

Starr[40] pointed out that many of the risks in Table III are voluntary and that people in fact will not accept such large risks if they are involuntary. The table of common non-cancerous risks is mostly involuntary. Purists might insist that driving a car is voluntary (even in the terrible state of U.S. public transportation), but the large risk of being a pedestrian in our car-laden society (4×10^{-5}) is certainly involuntary, and so is urban air pollution.

We come then to the risks of cancer (Table IV). These are separately listed for three reasons. Firstly, an accident leads to an average life shortening of about 30 years[41] whereas black lung disease is an impediment which incapacitates and renders the victim more susceptible to disease but does not kill at once, and cancer also lies latent and kills only late in life. The average life shortening is less--15-20 years. Therefore, the risk might be regarded as less important. The second reason, however, probably outweighs the first. Risks are often perceived by the survivors and not the victims, and the lingering death due to cancer is often more important. Thirdly, many of the cancer risks are uncertain and involve extrapolation to low levels of exposure. These extrapolations are likely to have similar uncertainties (and my cancer risks are probably overestimated as stated in the scientific

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[40] Starr, Chauncey, Science, 165, 1232 (1969).

[41] Baldewicz, et. al., UCLA-ENG-7485, Nov. 1974.

on dose-response relationships) and comparison is therefore easier.

Finally, the set of occupational risks, Table V, is particularly important because this is an OSHA hearing. Workers have traditionally been allowed a higher risk than the general population. For radiation, for example, the maximum level for occupational exposure is 5 R/year (higher for astronauts) whereas for the general public it is 170 mR/year--one thirtieth of the occupational level.

TABLE II

Risks in Sports[42, 43, 44, 45, 46, 47, 48]

		Deaths 1975	Risk/yr.
Football	Averaged over Participants		4×10^{-5}
Automobile racing			1.2×10^{-3}
Horse racing			1.3×10^{-3}
Motorcycle racing			1.8×10^{-3}
Power boating			1.7×10^{-4}
Boxing (amateur)	40 hrs/yr engaged in sports		2×10^{-5}
Skiing			3×10^{-5}
Canoeing			4×10^{-4}
Rock climbing (U.S.)			10^{-3}
Sunbathing, mountain climbing (skin cancer risk/curable)		300,000 cases	5×10^{-3}
Fishing (drowning)	Averaged over fishing licenses	343	1.0×10^{-5}
Drowning (all recreational causes) all over U.S.		4110	1.9×10^{-5}
Bicycling (assuming 1 person per bicycle		1000	10^{-5}

[42] B.G. Ferris, New Eng. J. Med., 268, 430 (1963).

[43] F.D. Sowby, Health Phys., 11, 879 (1965).

[44] C. Starr, Science, 165, 1232 (1969).

[45] K.S. Clarke, J. Am. Med. Assoc., 197, 894 (1966).

[46] Statistical Bulletin, Metropolitan Life Insurance Co., May 1977.

[47] Accident Facts, 1976 edition.

[48] Statistical Abstract of the U.S.

TABLE III

Commonplace and Therefore Accepted
Risks of Death (non-cancerous)

		No. of Deaths in 1974	Risk/Year
Motor Vehicle (in 1975)	Total	46,000	2.3×10^{-4}
	Pedestrian (certainly involuntary)	8,600	4×10^{-5}
Home Accidents (1975)		25,500	1.2×10^{-4}
Alcohol—cirrhosis of the liver (1974)			1.6×10^{-4}
Alcohol—cirrhosis of the liver (moderate drinker)			4×10^{-5}
Air travel: one transcontinental trip/year			3×10^{-6}
jet flying professor			10^{-4}
Accidental poisoning—solids and liquids		1,274	6×10^{-6}
gases and vapors		1,518	7×10^{-6}
Inhalation and ingestion of objects		2,991	1.4×10^{-5}
Electrocution		1,157	5×10^{-6}
Falls		16,339	7.7×10^{-5}
Tornados	Average over several years	160	5×10^{-7}
Hurricanes	Average over several years	118	4×10^{-7}
Lightning	Average over several years	90	4×10^{-7}
Air pollution (total U.S.) estimate (sulphates)		30,000	1.5×10^{-4}
Air pollution (benzo (a) pyrene) urban U.S.—cancer risk			3×10^{-5}
Vaccination for small pox (per occasion)			3×10^{-6}
Living for one year downstream of a dam (calculated)			5×10^{-5}

Sources: Accident Facts, 1976 Edition
Statistical Abstract of the U.S.
Alcohol—detailed discussion in appendix
Air travel—detailed discussion in appendix
Air pollution—detailed discussion in appendix
Dam failure—UCLA report, UCLA-ENG-7421, Payne-Ney, et. al., March 1974

TABLE IV

Commonplace Risks of Daily Life (Cancer Risks)

	Risk/year
Cosmic ray risks	
One transcontinental flight/year	5×10^{-7}
Airline pilot 50 hrs./mo. @ 35,000 feet	5×10^{-6}
Frequent airline passenger	1.5×10^{-5}
Living in Denver compared to N.Y.	10^{-6}
One summer (4 months) camping at 15,000 feet	10^{-6}
Other radiation risks	
Average U.S. diagnostic medical x-rays	10^{-5}
Increase in risk from living in a brick building (with radioactive bricks) compared to wood	5×10^{-6}
Natural background at sea level	1.5×10^{-5}
Eating and drinking	
One diet soda/day (saccharin)	10^{-5}
Average U.S. saccharin consumption	2×10^{-6}
Four tablespoons peanut butter/day (aflatoxin)	4×10^{-5}
One pint milk per day (aflatoxin)	10^{-5}
Miami or New Orleans drinking water	1.2×10^{-4}
1/2 lb. charcoal broiled steak once a week (cancer risk only; heart attack, etc. additional)	4×10^{-7}
Alcohol—averaged over smokers and non-smokers	5×10^{-4}
Alcohol—light drinker (1 beer/day)	2×10^{-4}
Tobacco	
Smoker, cancer only	1.4×10^{-3}
Smoker, all effects (including heart disease)	3×10^{-3}
Person in room with smoker	10^{-5}
Miscellaneous	
Taking contraceptive pills regularly	2×10^{-5}

Sources: See Appendix I

TABLE V

Current Occupational Risks

	Number of Fatalities (in 1975 unless stated)	Risk/yr.
Mining & Quarrying (accident only)	500	6×10^{-4}
Coal mining - accident (average 1970-74)	180	1.3×10^{-3}
- black lung disease (1969)	1135	8×10^{-3}
Agriculture - total	2100	6×10^{-4}
tractor driver (1 driver/tractor)		1.3×10^{-4}
Trade	1200	6×10^{-4}
Manufacturing	1500	8×10^{-5}
Service	1800	9×10^{-5}
Government	1100	1.1×10^{-4}
Transportation & Utilities	1600	3.3×10^{-4}
Airline Pilot		3×10^{-4}
Truck driver (1 driver/truck)	400	10^{-4}
Jet flying consultant & professor		10^{-4}
Steel worker (accident only) (1969-71)	66	2.8×10^{-4}
Railroad worker (1974) (all accidents excluding grade crossing)	688	1.3×10^{-3}
Fire fighters (1971-72 average)		8×10^{-4}

Source: Accident facts, 1976 Edition, p.23,87.
National Safety Council, 444 N. Michigan Ave., Chicago, Ill., 60611
Also, (coal mining black lung, rail worker, steel worker)
W. Baldewicz, et al UCLA-ENG-7485 Nov. 1974)
Airline pilot - see appendix
Statistical Abstract of the U.S., 1976 Ed. Table 1200

12. Justification for levels of risk distinguishing subgroups

Category I(a). There is no risk in my table larger than 10^{-2}. Coal mining risk (black lung plus accident) is close--9.3×10^{-3}. this risk is accepted--but barely so. Society now correctly insists that it be reduced.

It seems reasonable, therefore, to take this figure, and use this as the figure above which society must act. I would also insist on action if the risk for an individual plant or process (as distinct from an industry average) were this high.

Category I(c). As shown in Table V there are many occupations where the risk is one in ten thousand per year (10^{-4}/year) or greater, and neither workers nor society take any particular note of them. There are even some everyday, voluntary, non-occupational risks this large.

I note that according to my suggested distinction between subgroups according to risk, exposure to cigarette smoke in the workplace, whether a factory or an executive office, just comes in category I(b) and demands a study to determine whether the exposure can be reduced at reasonable cost. But a continuous exposure to such cigarette smoke is probably an extreme case--applicable primarily to smoke-filled committee rooms--and for most situations the occupational risk of working with smokers is less than 10^{-5}. If, however, we set the level at which exposure must be reduced (if cost effective) at a risk of 10^{-6}, all smoking in the workplace would have to be banned.

13. Cost for reducing a risk

The important question arises, how much should society pay to reduce a risk and how much is society willing to pay? A lot depends upon how the risk is perceived; is it voluntarily accepted or involuntarily accepted? In a classic paper, Chauncey Starr[49] suggests that the public accept risks voluntarily 100 times as dangerous as the involuntary risks. One of the cheapest ways of reducing a risk is to buy a good seat belt for the car and to use it; buying and using a seat belt corresponds to an expenditure of $5,000 per life saved.[50] People find seat belts inconvenient to use so that only 20% of Americans use them; the mandatory installation of airbags is suggested. An airbag costs about $100 when installed initially. The total expenditure in the U.S. is then about $1 billion per year assuming 10 million new cars per year. They would probably save 10,000 lives a year, leading to a cost of $100,000 per life saved. This still seems cheap, but some people still object to their use. For involuntary risks, costs of $1,000,000 and more are suggested.

In the 1977 OSHA hearing on the proposed emergency standard for benzene, Professor Richard Zeckhauser, using OSHA's own study pointed out that it was a $1 billion decision, and yet OSHA had not written down a single number about the benefits of the proposed

[49] Chauncey Starr, Science, 165, 1232 (1969).

[50] Richard Wilson, "Examples in Risk Benefit Analysis," Chemtech October 1975.

standard.

For the same hearing I made a risk calculation for benzene and showed that on a conservative basis, OSHA was proposing that society spend $300 million to save one hypothetical life. The budget of the National Institutes of Health is only three times this and even the gross national product is only two trillion dollars so that we can only afford to save 5,000 or 10,000 lives on this basis. Many more occasions than this arise in which lives can be saved and we cannot afford to spend $300 million for each one. For this reason, I prefer a number closer to $1 million spent for every hypothetical life saved.

For example, in an industry employing 10,000 people subject to a risk of 10^{-3}/year, 10 people a year would lose their lives and at least $10 million a year should be spent to reduce this figure.

The Nuclear Regulatory Commission was probably the first regulatory commission in the U.S. to face up to this problem. In a decision after a long three-year hearing[51] the NRC suggested that if exposure to radiation can be reduced at a cost of $1,000 per man rem it should be. The risk of radiation corresponds according to the numbers in the appendix (and quoted in many other places) to 10^{-4} per man rem. This is calculated on a linear, non-threshold basis. One thousand dollars per man rem corresponds to $10,000,000 per life saved. The NRC considered this to be a temporary figure and suggested a large, long public hearing probably with other

[51] Decision of the Commission in the "As Low As Practicable" hearing RM-50-1.

agencies involved, to decide on this number. Meanwhile they chose $1,000 as being a round number larger than any other presented in testimony at the hearing.

The cost of reducing a risk has also been addressed recently by the International Commission on Radiological Protection.[52] In their publication ICRP 22 they discuss the cost for saving a life in terms of their own new unit, the Sv or sievert. Translated into older units, they quote numbers from $10 to $250 per man rem, or with the risk factors I (and they) assume (10^{-2}/Sv = 10^{-4}/man rem) between $100,000 and $2,500,000 per life saved (conservatively calculated).

My figure of $1,000,000 falls near the top of this.

Another way of looking at the same problem is to realize that if money must be spent on control equipment, lives will be lost in the process. These are secondary effects of the decision process. It is a well known feature of a decision process that if the primary effects are small, the secondary effects must be carefully examined however hard that may be.

Thus, about half of any expenditure on reducing occupational exposure might be expected to be on capital equipment—often construction equipment. In construction work, people die in all sorts of accidents from bulldozer accidents to falling off roofs. The oft quoted example is that three people died in building the Brooklyn Bridge. The total number of workers killed in construction work the U.S. was 2,200 in 1975.[53]

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[52] International Commission on Radiological Protection, (ICRP) Reports ICRP 22, ICRP 26, Pergamon Press, London and New York.

[53] Accident Facts, 1976 ed., p. 22 published by the National Safety Council.

The total receipts of the construction industry were $164 billion in 1972.[54] But this contains a great deal of duplication, due to subcontracts, etc. If we assume that this represents $80 billion of primary construction contracts, I derive a number that for every $36 million spent in construction one life will be lost.

Thus, for this secondary effect alone, no expenditure more than $72 million total ($36 million capital) should be made merely to save one hypothetical life, or $72 million a year to save more than 1 life per year, because it will result in a net loss of life in society as a whole, and even in the subset of working men.

My figure of $1,000,000 to save a life may be low; but other distinguished men think it high. Thus, Nobel laureate Joshua Lederberg says:

> "We might be willing to double our health expenditures for 20% improvement in health; this would imply a willingness to invest $400,000 to prevent a death, which is on the high side of present day political judgments."[55]

Indeed, there are many cases in medicine where lives can be saved for $100,000 or less. Artificial kidney cost $30,000, and an intensive care unit often costs only $20,000 per life saved. An average cost of cancer treatment is about $50,000 (in 1977) and saves perhaps 30% of all cases, corresponding to $150,000 per life saved.

It is also useful to try to imagine how we would best spend

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[54] Statistical Abstract of the U.S., Table 1248.

[55] "How Safe is Safe?—The Design of Policy on Drugs and Food Additives," National Academy of Sciences, 1974, p. 68.

money to save lives. One might well spend $1,000,000 on 20 full time police to reduce automobile accidents or a more strict regulation of automobile speed limits, imprisonment or those with a high concentration of alcohol in the blood, and so on. Thus, expenditure of $1 million in Massachusetts alone could probably save 10 lives a year.

As Trevor Kletz pointed out:

> "There is nothing humanitarian in spending lavishly to reduce a hazard because it hit the headlines last week and ignoring the other (hazards)."[56]

[56] T.A. Kletz, "The Risk Equations: What Risks Should We Run," New Scientist, p. 320, 12 May 1977.

I now go through several examples showing how this procedure would apply to cases past and in process.

14. Example: Benzene

A recent OSHA hearing on a proposed rule for benzene exposure brought forth testimony on its possible carcinogenicity and testimony on comparisons of risks and costs to reduce these risks.

There is doubt whether benzene properly belongs in group I(a) or in group I(b). Carcinogenicity (leukemogenicity) in humans has, it seems, only been related to aplasia of the bone marrow, and therefore could be due entirely to the toxic nature of benzene at high doses, in much the same way way the correlations between alcohol consumption and cancer have been described. Animals exposed to benzene have not developed leukemia except at doses (200 ppm) where the toxic effects are evident.[57] Nonetheless an estimate of a risk can be found, using a conservative linear procedure.

At the exposure level of 10 ppm, previously established as a maximum to avoid the toxic effects, the risk is 3.3×10^{-5}, including possible leukemias, possible other cancers, and even possible heart trouble in analogy with cigarette smoking as suggested earlier in the specific suggestions on calculating cancer risks. This would put it in class I(b) if cartinogenicity is accepted. According to my proposed categorization, this is greater than 1×10^{-5} and thus it would be incumbent on industry to discuss measures to reduce the

[57] See testimony of myself, Richard Wilson, in the OSHA hearing on the proposed emergency temporary standard for benzene, July 1977.

exposure if these can be done at the cost of less than $10 per 10^{-5} risk. In fact, the cost according to OSHA's study, is closer to $300 per 10^{-6} risk ($300 million for each hypothetical life saved). I assume that OSHA will, in its aim of protecting the worker's interest, tend to produce a smaller number for this than industry would. I will show in later examples how such a large figure would be absurd in other situations.

However, according to my proposed procedure, the OSHA proposed standard of 1 ppm exposure would reduce the risk to below 10^{-5} and no reporting and detailed discussion of costs with OSHA would be necessary, although obviously industry would reduce the risk if it could be done without great expense.

II. Example: Drinking Water

Workers in areas with heavily chlorinated water are exposed to a lot of chloroform, up to 200 ug per liter in Miami and New Orleans.[58] This, through water in drinking fountains, etc., gives in high areas risks of 3 x 10^{-7}/year.[59] This would be an acceptable occupational exposure, and therefore not of concern to OSHA; but as noted below, it should be of concern to EPA and FDA. But if OSHA sets the level of risk at which a risk benefit analysis is demanded at 10^{-7}, it would be necessary to evaluate the risk of any drinking fountain in the workplace.

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[58] "Preliminary Assessment of Suspected Carcinogens in Drinking Water," Report to the U.S. Congress by U.S. Environmental Protection Agency, December 1975.

[59] "Health Effects of Drinking Water," National Academy of Sciences/National Research Council, May 1977.

15. Example: Vinyl Chloride

In the 1960's workers were exposed to levels of vinyl chloride monomer in the air of 1 part in 100 and in some cases up to 10% for short times. Ten percent is the level of anesthesia and some workers passed out.

At levels of 1 part in 1000 the risk of cancer per year is over 1%. Once the carcinogenicity of vinyl chloride was discovered, there was rightly an outcry and the exposure was reduced--by a factor of 1000. The occupational exposure is now 1 ppm in the air for an 8-hour working day and gives a risk just about 10^{-5}. Reduction this far is justified, but further reduction would, according to my proposed rules, not be warranted, both because the expense would be too great and the risk is already low. To reduce the occupational level to 1 ppm, and to reduce the environmental exposure to present levels the cost is $80 M/year and $200 M fixed cost, which, amortized at 20% per annum comes to $40 M/yr for a total of $120 M/yr. To reduce this still further will cost more than this $120 M/yr or more than $100 million per life saved.[60]

[60] Data from Society of the Plastic Industry. A critic might regard these numbers as high, but not 100 times too high.

17. Example: Asbestos

There is a lot of data on occupational exposure to asbestos. I refer to a summary here.[61] For example, I append Table I from this review here. I note that asbestos exposure increased the lung cancer risk a factor of 11 compared with the average risk of smokers and nonsmokers. Thus we have the number of cancers in a group of N asbestos workers (half smoker and half nonsmokers) was 11 times the risk averaged over smokers and nonsmokers times N or 11 times half the risk for a smoker times N or 5 times the risk of a smoker alone. The historical risk due to asbestos was then $5 \times (1.2 \times 10^{-3}/yr)$ for lung cancer alone or $2.5 \times 10^{-2}/yr$ assuming other cancers and heart disease are also important consequences of asbestos exposure. Therefore, according to my suggested criterion, immediate action would be warranted—as was indeed the case. Unfortunately a reduction of exposure by a factor of 1000—as was possible for vinyl chloride may not be possible for asbestos, and a detailed calculation of cost to reduce the risk is necessary.

It has been suggested that we replace asbestos by fiberglass to eliminate cancers. It has also been suggested that the carcinogenic nature of asbestos is due to the long fibers and fiberglass will have long fibers also. Indeed, when fibers from fiberglass

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61 Irving J. Selikoff and E. Cuyler Hammond, "Multiple Risk Factors in Environmental Cancer," Ch. 28 in Persons at High Risk of Cancer: An Approach to Cancer Etiology and Control, Ed. J.F. Fraumeni. New York: Academic Press, 1975.

TABLE I

Expected and observed deaths among 370 New York-New Jersey asbestos insulation workers, January 1, 1963-December 31, 1973, by smoking habits

	Number of men	Person-years of observation	Cause of death: Lung cancer			Pleural mesothelioma	Peritoneal mesothelioma	Asbestosis
			Expected*	Observed	Ratio			
History of cigarette smoking	283	2,195	4.07	45	11.06	7	14	19
Current smokers	181	1,443	2.48	32	12.09	6	7	12
Ex smokers	102	752	1.59	13	8.18	1	7	7
No history of cigarette smoking	87	708	1.58	2	1.27	0	7	6
Never smoked	48	409	0.84	0	–	0	5	3
Pipe/cigar only	39	299	0.74	2	2.70	0	2	3

*Expected deaths are based upon age specific white male death rate data of the U.S. National Office of Vital Statistics from 1963-71, disregarding smoking habits. Rates were extrapolated from 1972-73 from rates for 1967-71.

were injected subcutaneously to rats, cancers were caused which were similar to asbestos cancers.[62]

Therefore replacement of one chemical by another must be done with caution and only after it has been established that the net risk will be reduced. The proposed OSHA regulations seem inadequate in this regard; if one chemical is found to be carcinogenic, it must be replaced by another which may not have been studied as carefully.

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62 W.F. Stanton and C. Wrench, "Mechanism of Mesothelioma Induction With Asbestos and Fibrous Glass, Journal of the National Cancer Institute, 48, 797 1972.
F. Pott, F. Huth, and K-H. Fredricks, "Tumorigenic Effects of Fibrous Dusts in Experimental Animals," Environmental Health Per-
[illegible]
[illegible] Wagner, A. Timbrell, E. Miller, M. May and E. [illegible] Relation to Fiber Dimension," Journ. Nat. Cancer Inst., 40 [illegible].

18. Example: Urban or Rural Job Location

The risk of air pollution in the eastern U.S. is normally attributed to high sulphur and particulate levels. The evidence is based on laboratory and epedimiological data.

As shown in the Appendix, I deduce an average risk of death from all causes of air pollution in eastern U.S. of 1.5×10^{-4}/year this is higher than my suggested occupational limit of risk of 10^{-5}/year.

Most of the air pollution risk is not a cancer risk and may not be strictly comparable. But the section on benzo (a) pyrene suggests that in major U.S. cities the hazard can be appreciable. Data from the National Air Surveillance network exist in a few selected cities up to 1970 (apparently discontinued since) gives concentration of 1 to 2 nanograms/m^3 (Worcester, Mass.; Baltimore, Maryland, etc.)[63] and half this indoors. There is then a risk from cancer alone of 10^{-6}/year.

This is not a large occupational risk, but if there is no discrimination among risks we would have to reduce the exposure to the lowest feasible level. What should be considered feasible in such a case? Should industry in Baltimore move to a location with a lower air pollution—such as Hawaii or Maine?

Alternatively industry can help the community to enforce tough air pollution controls.

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[63] "Preferred Standards Path," Report for Polycyclic Organic Matter, U.S. Environmental Protection Agency, Oct. 1974, Table C-1.

Nor would such criteria apply only to industry. It might be ruled, for some reason other than financial that it is unfeasible for an industry to move far from its sources of iron or coal. But this need not apply to our major universities whose functions might be carried out even better in a rural setting.

The variations among employees are so great that it would seem that only a clear cut financial criterion for feasibility of reducing the risk, as I propose, is a workable one.

19. Example: Airline Pilot

As noted, an airline pilot has an occupational risk (death by accident) of 3×10^{-4}. It might be argued it is not a cancer risk and therefore acceptable. But the cancer risk (cosmic rays) alone is 6×10^{-5}/year (see Table IV and Appendix).

Therefore, a reduction, if feasible, of occupational cancer risks below a level of 10^{-5} would lead to changes in the airline industry unless "feasible" includes sensible financial criteria. It is feasible for airplanes to fly at 10,000 feet and reduce the cancer risk (although the accident risk might rise). A stewardess performs a job (high level waitress) which could be considered unnecessary and it is feasible for passengers to serve their own sandwiches or to eat when they land. It is not feasible to add enough shielding (20 feet of lead) to reduce the cosmic ray level, because most airplanes would not then fly.

The number of U.S. air crew at risk is 50,000 persons full-time equivalent. Therefore at least $15 million per year ($3 \times 10^{-4}$ x 50,000 x $1,000,000) should be spent to reduce accidents if they can be reduced to zero. Probably more than this is spent so the airline industry meets my criterion. But if we were to demand $300 million per life saved (as is the effective demand even for a hypothetical life in the OSHA benzene hearings), the airline industry should spend $4.5 billion a year to reduce accidents. Probably with expenditures of this magnitude, on air traffic control, blind landing equipment, etc., accidents could indeed be reduced; but it seems obviously an excessive amount.

20. Example: Teacher in Massachusetts

The laws of the Commonwealth of Massachusetts insist that every teacher, in a school or college, be tested for tuberculosis every 3 years. For those who have a positive reaction to a skin test this must be by a chest x-ray. The purpose is not to protect the individual, but to protect society by making sure that the tuberculosis is not transmitted to students. If the chest x-ray is carried out with a reasonably good x-ray set and with reasonably good medical technicians the equivalent whole body dose is about 7 milliroentgens.[64] (Mobile x-ray units used to give doses as high as 1000 millroentgens.) This dose every 3 years becomes 2 millironentgens/year and the risk is half the cancer risk of a transcontinental flight per year or 3×10^{-7} per year.

This is an involuntary risk forced upon me, and one in which I even have to take time and trouble to expose myself. If OSHA were to insist on reduction of all occupational risks greater than 10^{-7}, their regulation would be in immediate conflict with state law compelling me to take this x-ray.

This is clearly a case where society reasonably asks a worker to undertake a risk—albeit a small one—so that society may benefit. That society asks this of me seems reasonable. That society should ask workers to undertake small risks also seems reasonable.

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[64] Measured at Harvard University Health Services by Dr. J. Shapiro at my instigation.

21. Workers in certain dyestuff industries

One of the most important occupational problems has been cancer of the bladder among certain dyestuff workers. The first of these noted was among β napthylamine workers.[64A] As interpreted by Pochin in Table III of reference 10 there was an incidence of 24,000 per million per year or a yearly risk of 2.4×10^{-2}. Benzidine is also a potent carcinogen (carcinogenic potency in animal tests = 0.05 as defined earlier and discussed in reference 34) and in one plant 20/25 developed bladder cancer.[64B]

The lifetime risk is close to unity and the yearly risk =0.05. A study of the plant showed no easy way of reducing the risk. The risk was greater than 1%, so according to my prescription the plant should have been shut down and it was and in the U.K. benzidine production is banned.

Although the animal tests reviewed in reference 24 showed liver cancers, bladder cancer showed up in man. The bladder cancers may be attributed to the body excreting the poison. This effect may be an effect of high doses only. At lower doses, the incidence of bladder cancers may fall below the linear relationship.

64A R.A. Case, M.E. Hosker, D.B. McDonald and J.T. Pearson, Brit. J. Ind. Med., 11 75 (1954).

65B M.R. Zavon, C. Hoegge and Dula Bingham, "Benzidine Exposure as a Cause of Bladder Cancer in Man," Archives of Environmental Health, 27 1, 27 July 1973.

22. Modifications of Procedure for FDA and EPA

The same classification of carcinogens can also be used for the FDA and EPA. However, it is important to realize that the regulatory procedures should be quite different, particularly when it is assumed that there is no absolute threshold below which there is no risk.

It is reasonable, and it has historically been accepted, that workers in their occupation can undertake certain distinct risks which are larger than risks to the general population. However, if every member of the population were to be exposed to each and every occupational risk at the same level as the exposed workers, the total risk would be excessive. Accordingly, society can, and does, aim to reduce the risks to the public to a smaller value.

For example, the International Committee on Radiological Protection (ICRP) set up in 1927 to regulate the burgeoning x-ray industry, sets a standard for occupational exposure (5 R/year), 30 times what they set as acceptable for the general public (170 mr/year). If we take this same factor of 30 between an occupational risk and a public risk an individual risk of 3×10^{-7} and below might be regarded as acceptable for FDA and EPA. I do not want, at this time, to claim that this same factor of 30 between an occupational risk and a public risk should always apply. But some factor seems appropriate.

In a discussion of chemicals—especially di-ethylstilbestrol (DES)—can appear as accidental additives in foodstuffs, the

the Commissioner of FDA (in February 1977) recommended a lifetime risk of 10^{-6} as acceptable--which is a yearly risk of 1.5×10^{-8}. At first sight, this proposal seems inconsistent with mine. However, his procedure for calculating the risk, the Mantel-Bryan extrapolation procedure, is less conservative in most cases than the simpler, and more easily justified, procedure here. The extra factor of conservatism is about the factor of difference. My proposal would give the same result as that of FDA's commissioner in all applications of interest.

11. Suspect Carcinogens: OSHA Category II

It is also possible to use risk analysis techniques when carcinogenisis is only suspected. As cited above, it is not clear, for example, whether benzene should be considered a carcinogen in itself or not; on the assumption that it is, however, I evaluated a conservative risk which suggests that previous regulatory levels for benzene were adequate and conservative even if benzene proves to be a carcinogen.

The crucial feature that enabled me to do this for benzene was the existence of a body of data on persons exposed to benzene at dose levels 10-40 times the previous limits. Animal date at high dose levels also became available after the hearing and gives a conservative upper limit in agreement with the risk from human data.

Therefore, I suggest that upper limits to risk be calculated, wherever possible, on all suspect carcinogens in OSHA's category II. In many of these cases, animal data may not be available, and the cheap and fast mutagenesis data may have to be used to establish a reasonable upper limit.

It seems reasonable to insist that no new occupational exposure to even a suspect carcinogen be allowed unless the upper limit of risk is less than 10^{-2} and not this large if it can easily be reduced, although one would not have such a strict criterion as when a definite threshold level of carcinogenesis is known.

Increased use of a new chemical can clearly be allowed as more precise data establish a lower upper limit of risk.

My proposal on suspect carcinogens would avoid a ridiculous situation that can occur with OSHA's proposed rules. If industry propose to manufacture and use a new chemical, it is not allowed unless it is proven to be toxic or carcinogenic. Although the regulatory authorities do insist on some carcinogenesis tests, the regulations provide no incentive to make these tests as sensitive as possible. Indeed, as pointed out by Schneiderman,[65] there is an incentive to make experiments less accurate than possible, because then there is less likelihood of the chemical being proven carcinogenic. Under the proposed procedures, exposure to a proven carcinogen must be reduced to the lowest feasible level--much more severe than for suspect carcinogens. My proposed procedure leads to a much smaller difference between actions for suspect carcinogens and proven carcinogens and indeed provides incentives for good (sensitive) experimentation since upper limits to the risk can then be reduced, and increased exposure allowed, without the fear that a proof of carcinogenesis will cause unnecessarily expensive restrictions.

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[65] M.A. Schneiderman and N. Mantel, "The Delaney Clause and a Scheme for Rewarding Good Experimentation," Preventive Medicine, 2, 165 (1973).
M.A. Schneiderman and N. Mantel, "Estimating Safe Levels--A Hazardous Undertaking," Cancer Research, 35, 1374 (1975).

15. Conclusions

The aim of OSHA in classifying carcinogens is good, although I believe the distinction between categories I and II is not correctly drawn. More important, however, is the discussion of regulatory action. The aim of simplicity and definitiveness is nullified by the use of the nebulous phrase "to the lowest feasible level." To the extent this phrase is undefined, we are as much up in the air as before. If the phrase is defined to mean zero, or close to zero, the whole OSHA proposal is unworkable. A definite meaning can be defined by means of risk analysis. As soon as risk analysis is accepted the sharp distinction in regulatory action between carcinogens, suspect carcinogens and other hazards, real and potential, vanishes.

I propose that a risk analysis be carried out for all carcinogens and for most suspect carcinogens. If the risk, at the estimated exposure, is less than 10^{-5}, OSHA should take no action. I have shown that if OSHA acts on risks less than this, inconsistencies and absurdities creep in.

APPENDIX

APPENDIX I

Explanation of and Sources for Risk Calculations

I.1. Introduction

It is hard to be as consistent as one would wish about the calculations of risk. In some cases below I have followed my own prescription (aflatoxin) in others I have preferred to take the estimate of a government report or committee where it did not differ widely from my own. This leads to a small inconsistency, and discrepancies between the numbers presented here and numbers I presented in earlier hearings and in publications. The detail below will, I hope, make the reliability of the numbers and the small inconsistencies clear.

I also do not make here detailed least squares fits to the data, but only approximate fits--often graphical. Others can take the data and improve on the risk estimates, but my estimates will not be off by more than 25% from this cause.

I.2. Radiation

Radiation cancers have been studied in humans and animals. I take the numbers here from the Report of the Committee on Biological Effects of Ionizing Radiation (BEIR) of the National Academy of Sciences, November 1972. This uses a linear interpolation for low doses also. Radiation works directly on the cell and avoids problems with toxicity at high dose levels.

There have been tests with over one million mice at the Oak Ridge National Laboratory. These suggest that the effect at low dose rates is 1/4 of that calculated from this linear curve. This reduced the number of cancer cases from background radiation, but probably not the number of cases from diagnostic x-rays or jet airplane flights where the dose is rapid. It is often stated that chemical carcinogens give a smaller insult to each cell than radiation. To this extent we might expect a low dose rate effect to occur for chemicals. The numbers here remain conservative upper limits.

Cross-country airplanes travel at an altitude of 35,000 feet (10 kilometers). At this height there is an appreciable dose due to cosmic radiation, including neutrons. To estimate this, I use the UNSCEAR report.[2] The dose at a latitude of 55°N is 2000

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[1] "The Effects on Populations of Exposure to Low Levels of Ionizing Radiation," Report of the Advisory Committee on the Biological Effects of Ionizing Radiation. (BEIR) National Academy of Sciences, National Research Council, November 1972.

[2] Report of the United National Scientific Committee on the Effects of Atomic Radiation, UN, NY, 1962, page 201, figure 1. It might be thought that the neutrons would be absorbed by the airplane and the body. Since they are in equilibrium with the surrounding air, this can be allowed for by taking the dose at a lower altitude from the figure. This is a small reduction.

millirems/year of ionizing radiation and 3600 millirems/year of neutrons. This will increase markedly during a period of a solar flare.

Using 365 days a year, a 5 hour flight at 10 km gives a dose between 3 and 4 millirems. Now, using the relationship between cancer incidence and whole body dose in the BEIR report,[59] I find a cancer risk of 5×10^{-7} per transcontinental flight. This is smaller than (1/6 of) the risk of accident and is normally neglected in any discussion of the risks of airplane travel. But it is derived on a comparable basis to the cancer risks discussed here and helps to put them in perspective.

The doses from diagnostic x-rays, from buildings and so on, are obtainable from various places. One of them is the UNSCEAR reports[2] and the BEIR report[1] referred to above. I have also prepared a convenient list.[3] The risks are obtained from the dose list by simple multiplication of the dose and the risk per unit dose in the previous paragraph and reference 1.

I note here that many studies have been made of the radiation levels in buildings and how to reduce them. A lot of the radiation dose comes from inhaling the radioactive gas, radon, which can produce other radium daughters in the lungs. This could be stopped by painting the bricks with epoxy resin. Although obviously

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[3] Chapter IX, R. Wilson and W. Jones, Energy, Ecology, and the Environment, Academic Press, 1974.

TABLE 9-6

Some Typical Radiation Doses*

Source	Dose (mrem/yr)	Radiation cancers/yr if all U.S. population so exposed
Potassium 40 naturally occurring in body	20	1000
Potassium 40 naturally occurring in neighboring body	2	100
Gamma rays from neighboring soil and rocks (av.)	50	2500
Gamma rays inside brick or stone buildings	30-500	1500-24,000
Cosmic rays at Vernon, Vermont	30	2000
Background dose at sea level (av.)	100	5000
Background dose at sea level in Kerala, India (av.)	500-2000	25,000-100,000
Cosmic rays at Denver, Colorado	67	3000
3-hr jet-plane flight	2	100
60 hr/month of jet-plane flight (pilot)	500	24,000
Medical diagnostic X rays in U.K. (av.)	14	1000
Medical diagnostic X rays in U.S. (av.)		
1964	55	2600
1970	95	5000
Weapons tests "fall-out"	3	150
AEC "design criteria" for reactor boundary (upper limits for actual use)	5	250
Within 20-mile boundary of BWR with 1-day hold-up but leaky fuel (gaseous emission) (av.)	0.1	250
Within 20-mile boundary of PWR with leaky fuel (av.)	0.002	0.02
Within 20-mile boundary of coal plant (av.)	0.01	0.1

* Lists such as this can be combined from various general sources: [1, 2, 42, 43, 44, 45, 46, 47].

feasible, it is not cost effective according to my criteria.[3A]

3A. "Final Report on Study of the Effects of Building Materials on Population Dose Equivalents," Dade W. Moeller, D.W. Underhill, Report by Harvard School of Public Health for U.S. Environmental Protection Agency, December 1976.

I.J. Saccharin

The risk stated in Table IV comes from animal data at high doses and an extrapolation to the known total consumption of saccharin in the U.S. The number here comes from the testimony Dr. Marvin Schneiderman to the Rogers Committee of the U.S. House of Representatives.[4] I have checked his numbers and agree with them. Dr. Schneiderman took the data from a second generation of rats exposed to saccharin. To be wholly consistent with the other data here, we should take first generation data only--then the number should be divided by 3. Dr. Sidney Wolfe, Director of the Health Research Unit, in a press interview, suggested a death rate which corresponds to twice the figure taken here.

[4]See also Federal Register, April 15, 1977, page 20001.

I.4. Drinking Water

The principal carcinogen in drinking water is chloroform--found to be carcinogenic in both rats and mice.[5] this carcinogen is not produced by chemical industry, but by action of chlorine in water purification systems on organic matter.[6]

A survey of concentrations has been done by EPA[7]. Concentrations of 200 ppm have been found in drinking water of Miami and New Orleans and are taken here. The risk can be calculated directly from the rat and mice data or can be gotten from a National Academy Report.[8] The National Academy of Sciences calculate the risk as 3.7×10^{-7} lifetime risk (upper limit, 95% confidence) for 1 µg/liter concentration in water. Although I originally derived a slightly higher figure I will take their upper limit as a best estimate. This leads to 6×10^{-9}/year for 1 µg/liter or 1.2×10^{-6} for a yearly risk with a level of drinking water of 200 µg/liter, such as we find in Miami and New Orleans. I assume that the NAS calculations refer to ordinary intake of water. The risk is larger in many cases when bromodi-

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[5]"Report on Carcinogenesis Bioassay of Chloroform," National Cancer Institute, 1976.

[6]J. Carroll Morris, Harvard University, "Formation of Halogenated Hydrocarbons by Chlorination--A Review," Report to the Environmental Protection Agency, PB 241511, March 1975.

[7]National Organics Reconaissance Survey Quality Water Data. U.S. Environmental Protection Agency; sheets enclosed.

[8]Drinking Water and Health, Report of the Committee on Safe Drinking Water. Advisory Committee on Toxicology, National Academy of Sciences, National Research Council, May 1977. (reference 58).

clorethane and dibromochloromethane are included; it is probable that they are more carcinogenic than chloroform. I assume that the NAS report has already allowed for cancers at other than at the primary site.

I.5. Benzo (a) pyrene (BaP)

Polluted air contains many carcinogens. One that has been identified and its carcinogenicity noted is benzo (a) pyrene. It is also frequently monitored. There are other polycyclic organic compounds that are probably carcinogenic, but they have not all been monitored.

If we make the assumption--reasonable for a rough calculation and in any case all we can do--that all burning processes produce the carcinogenic polycyclic hydrocarbons in equal proportions, then a monitoring of benzo (a) pyrene can give us a relative hazard index. This can be made into an absolute hazard index by epidemiological studies of which I here quote two: a study of lung cancer in British gas workers[9] and a comparison of lung cancer in industrial Liverpool and rural North Wales.[10] In each case the increase of lung cancer (among non-smokers) is tentatively attributed to polycyclic hydrocarbons with benzo (a) pyrene as an index. These are compared, for example, in a review[11] and it is stated that it is prudent to assume that breathing air with an average concentration of 10 ng/m^3 is equivalent to smoking one cigarette a day.

[9] P.J. Lawther, B.T. Commins, R.E. Waller, "A Study of the Concentration of Aromatic Hydrocarbons in Gas Workers' Retort Houses." Br. J. In. Med., 22, 13 (1965).

[10] P. Stocks, "Cancer in N. Wales and Liverpool Region," Supplement to British Empire Cancer Campaign Annual Report, 1951.

[11] Malcolm. C. Pike, et. Al., "Air Pollution," Chap. 14 in Persons at High Risk of Cancer, Ed. J.F. Fraumeni. Academic Press (NY) 1975.

TABLE 2

Age-standardized lung cancer mortality rates (per 100,000 per year) for men aged 35-74 by amount of cigarettes smoked in Liverpool and rural North Wales [26] [see Reference 10]

Packs/day (approx.)	Mortality Rates	
	Rural area	Liverpool
Nonsmokers	11	50
½	69	168
1	147	248
1½	232	389
2	344	527

TABLE 3

Estimated increase in male lung cancer death rate (per 100,000 per year) per ng/m³ BP content of air and per cigarette smoked per day

Data source		Increase per ng/m³ BP in air	Increase per cigarette smoked per day	Estimated U.K. equivalence: ng/m³ BP =1 cigarette/day	Estimated U.S. equivalence: ng/m³ BP =1 cigarette/day
British carbonization workers [21, 22] [44 9]		0.4	9	23	11
Liverpool and rural North Wales [26] [44 10]	Non-smokers	0.4	7	17	9
	Cigarette smokers	1.4	7	5	2.5

this "match" was made for lung cancer; but because of the similarities between cigarette smoking and air pollution, I will assume that the other problems of cigarette smoking—such as other cancers, including bladder cancer, and heart disease, also occur with polycyclic organic matter in the same proportion. The total average risk from cigarette smoking in the U.K. is 0.7×10^{-6}/cigarette[12] or about 2×10^{-4} for 1 cigarette/day, and about half this in the U.S.

Even as late at 1970, many American cities had ambient concentrations out of doors of benzo (a) pyrene of 1.5 ng/m^3, giving a yearly risk of 1.5×10^{-5}. Indoors the concentration is less, leading to an overall risk of half this amount.

Benzo (a) pyrene is also a known animal carcinogen of some potency. Data on Chinese hamsters where benzo (a) pyrene is ingested daily for a lifetime gives a potency such that there is 50% tumor induction for 100 µg ingested per kg body weight per day.[13] This potency agrees with the mutagenic potency of 5×10^{-2} µg/100 salmonella revertants in an Ames test, as shown by Meselson.[14]

Benzo (a) pyrene also gives cancers when injected into rats and mice subcutaneously. It is a constituent of cigarette smoke and may be the most active carcinogenic agent in cigarettes.

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[12] R. Doll and A.B. Hill, Brit. Med. J., 1, 1399 (1964).

[13] E. Chu and R. Malmgren, Cancer Res., 25, 884 (1965).

[14] loc. cit. (reference 34 in main text).

Benzo (a) pyrene and many other polycyclic hydrocarbons are produced in various incomplete combustion processes—open wood and coal burning, broiling of fish, chicken and meat, and automobile exhausts which I discuss in the next section.

I note, however, that the carcinogenic risk of benzo (a) pyrene ingested is less than calculated above for benzo (a) pyrene as an index of polluted air. Thus, about 1 ng/m^3 of benzo (a) pyrene polluted air gives a yearly risk of 10^{-5} and a lifetime risk of 6×10^{-4}. Thus a 60% tumor incidence is reached at 1000 times this concentration or 1 µg/m^3.

Man breathes in 10-20 (average 15) cubic meters of air per day,[15] so a man breathing this polluted air will breathe in 5 µg of benzopyrene per day or 0.2 µg ingested per kg body weight. Assuming this is all absorbed, this can then be compared to the dose for which Chinese hamsters have 50% tumor induction (which I read from the figure presented earlier from Meselson's paper (reference 34). This is 500 times greater than the air pollution figure of 0.2 µg (micrograms) indicating a smaller carcinogenic effect.

Part of this difference is, no doubt, because benzo (a) pyrene is only an indicator of many other carcinogens in smoke from incomplete combustion of carboniferous products. Also, perhaps, the mode of intake through the lung emphasizes lung cancers.

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[15] National Academy of Sciences, Particulate Polycyclics Organic Matter, Washington, DC, 1972, p. 29.

In this case, although at first sight it appears that one can have a direct comparison of human and animal data, the fact that benzo (a) pyrene is merely an indicator for other carcinogens, upsets the comparison and the human effect is larger than a naive use of the animal data would suggest.

I.6. Charcoal Broiling Steaks

It has been noted that many carcinogens are produced in detectable amounts when steaks are charcoal broiled. Char-broiled chicken and broiled fish are also covered with carcinogens. These can come from the charcoal--it is well known that burning coal produces many carcinogens, and the first cases of environmental carcinogenesis noted by Sir Percival Potts 200 years ago came from burning coal--or they can come from the high temperature.

For example, Lijinsky noted over a dozen potential carcinogens[16] from broiling steaks. Of these, benzo (a) pyrene is the best known, and I use only this for this calculation which is therefore a lower limit. Nine micrograms is present in a 1 kg (2 lb) steak or 9 ppb or 2.2 micrograms for a 1/2 lb. steak. Benzo (a) pyrene may be the principal active agent in tobacco smoke and this corresponds to 900 cigarettes. Of course, lung cancer caused by cigarette smoking is caused by inhalation; data shown earlier in Figure 2 show that 50% of that 5 develop tumors when ingested with 0.1 mg/kg body weight. This leads to a carcinogenic potency potency of 5.5 for 1 mg/kg body weight ingested per year and leads to a risk for 1/2 lb. steak per week of 4×10^{-7}. The FDA has not yet banned charcoal broiling in restaurants even though it involves the processing the food and adding of carcinogenic substances.

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[16] W. Lijinsky and P. Shubik, Science, 145, 53 (1974)

The huge variety of ways benzo (a) pyrene can add itself to the food chain in concentrations of 10 parts per billion is illustrated by the section from the monograph of IARC.[17] In many countries smoked fish is believed to be a cause of stomach cancer.[18]

[17] The International Association for Research on Cancer Monograph, pp. 94-103, 1973.

[18] Occupational variations in mortality from gastric cancer in relation to dietary differences. J. Sigurjonsson, Brit. J. Cancer, 21, 65 (1967).

I.7. Aflatoxin

Aflatoxin is one of the most potent carcinogens. It appears in nature extensively. It grows on molds of various sorts.

Although it grows naturally it is important to realize that it can be controlled by society. The storage of grains can be careful or careless and the concentration of aflatoxin low or high accordingly. For peanuts the mold grows on the case, and if care is taken to throw out the nuts with cracked cases, the concentration can be further reduced.

A summary of the data on aflatoxins was made by the FDA.[19] At that time, the action level for concentration of aflatoxins in peanut butter was 20 microgram/kg (20 ng/kg = 2 x 10^{-8} = 20 parts per billion) and FDA proposed to reduce it to 15 ng/kg. Although they do not state it clearly in this document, a suggestion is made that the average concentration in American peanut butter is 3 parts per billion (compared to 200 parts per billion in sections of Thailand!) I will take this for the risk estimate.

The epidemiological data on man[20] suggests a lifetime risk for liver cancer only of 2 x 10^{-4} to 10^{-3} for 0.1 ng daily intake in a

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[19] 39 Federal Register, 42748, Dec. 6, 1974.

[20] T. Peers and C. Linsell, "Dietary Aflatoxins and Liver Cancer: A Population Based Study in Kenya," Brit. J. Cancer, 27, 473 (1973). R. Shank, J. Gordon, E. Wogan, A. Nondasuta, and B. Subhamani, "Dietary Aflotoxins and Human Liver Cancer III: Field Survey of Rural Thai Families for Ingested Aflatoxins," Fd. Cosmet. Toxicol. 10, 71 (1972b).
S. Van Rensberg, J. Van der Watt, I. Purchase, L. Pereira Coutinko, L. Markham, "Primary Liver Cancer Rate and Aflatoxin Intake in a High Cancer Area," S. Afr. Med. J., 38 2808a (1974).

man (average weight 70 kg).

This agrees with a rough average of 3 animal experiments,[21] for the same relative intake (expressed in mg/kg body weight for a lifetime) and mutagenesis tests[22] as interpreted by Meselson.[23] I note in passing that this is an example where animal and human carcinogenesis and mutagenesis data simultaneously exist.

This then gives a lifetime risk (read from Meselson's graph) or calculated direct from the data of

$$\frac{\log 2}{3 \times 10^{-3}} \text{ (Dose in mg/kg body weight/day)}$$

or 3×10^{-4} for 0.1 µg human intake/day.

For 3×10^{-9} concentration of aflatoxin in peanut butter, I find one tablespoonful (16 grams) gives 48×10^{-9} gms/aflatoxin day; 4 tablespoonsful peanut butter a day gives a lifetime liver cancer risk of 6×10^{-4}; and a yearly liver cancer risk of 10^{-5}. To obtain a total risk, I assume other cancers are produced in numbers equal to the liver cancers, and as many heart disease

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[21] W. Butler and J. Barnes, "Carcinogenic Action of Ground Nutmeal Containing Aflatoxin in Rats," Fd. Cosmet. Toxicol., 6, 135 (1968). W. Butler, M. Greenblatt and W. Lijinsky, "Carcinogenesis in Rats By Aflatoxins B1, G1, and B2, Cancer Res., 29, 220 6 (1969). G. Wogan, S. Paplicolunga and P. Newbreme, "Carcinogenic Effects of Low Dietary Levels of Aflatoxin B1 in Rats., Fd. Cosmet. Toxicol. 12, 681 (1974).

[22] J. McCann, E. Choi, E. Yamasaki, and B. Ames, "Detection of Carcinogens in the Salmonell/Microsource Test: Assay of 300 Chemicals," National Academy of Sciences: USA, 72, 5135, 1975.

[23] M. Meselson and K. Russell, loc. cit. (reference 34 in main text).

as cancers; this is the case for cigarette smoking and probably for vinyl chloride as noted earlier in my suggestions for risk estimation.

This is lower than earlier estimates of mine. The reductions come from three causes. Firstly, there is a reduction from 15 x 10^{-9} to 3 x 10^{-9} in the estimate of average aflatoxin concentration. But concentrations of 15 x 10^{-9} are allowed by present regulations. I also took the most pessimistic of animal studies. In my first estimate, moreover, I related animal to man at the same intake as a fraction of food intake (rather than as a fraction of body weight). This overstates the risk. I believe the value here is more reasonable.

We can also calculate the danger of aflatoxin in milk. According to recent surveys[24] levels of 0.1 ppb and above were found in 177 out of 302 samples—or roughly half. An average level might then be 0.1 ppb. One pint of milk (= 1/2 liter = 500 gm) contains as much aflatoxin as one tablespoonful (15 grams) of peanut butter with 3 ppbillion.

[24] Food Chemical News, p. 22, November 7, 1977; and p. 38, Nov. 28, 1977.

I.3. Alcohol

The cases of cirrhosis of the liver are assumed to be entirely due to alcohol consumption, the rate I assume to be proportional to the consumption. This is suggested by the WHO study referred to earlier where data for 14 countries, with average annual per capita intake varying from 4 to 25 litres were compared. In the U.S. the rate in 1974 was 2.1×10^{-4} for men and 1.1×10^{-4} for women[25] (the difference being consistent with the relative consumption of men and women) or an average of 1.6×10^{-4}.

The evidence for carcinogenicity of alcohol is confusing. It is unclear whether alcohol functions as a carcinogen, cocarcinogen, or through an indirect mechanism such as alteration of bacterial fl through the gastro-intestinal tract. Moreover, some or all may be due to impurities in the beer or wine. But for our purposes, these caveats don't matter. People drink beer or wine impurities and all and the intake is large enough that we are almost certainly above any threshold. But there is, for oral cancer at least, a very str synergism between smoking and alcohol. There have been correlatio between cancer and alcohol intake noted for the mouth and phary x larynx, esophagus, liver and possibly rectum.

Rothman[26] estimates the overall risk as follows. In 1968 14.4 cancers occurred in sites where alcohol has been associated, out o

[25] Statistical Abstract of the U.S.A., Tables 89, 93, 1317.

[26] K.J. Rothman, Ch. 9 in Persons at high risk of cancer. An appro to cancer etiology and control. Ed. J.F. Fraumeni. Academ Press 1977.

173, 665 total cancers in the U.S.A. This gives a risk of 7×10^{-5}/yr. These are probably concentrated in the 20% of heavy drinkers (who are usually also smokers) to give a risk of 2×10^{-4}/yr.

The average beer consumption in the U.S. is 2/3 pint/day[27] with approximately double the amount of alcohol consumed in wine and spirits. A light drinker (1 pint beer per day only) still has a risk of cirrhosis of 7×10^{-5}/yr.

[27]Statistical Abstract of the U.S. Table 1317.

I.9. Egg Yolk

Mice have been fed an extract from the yolk and the whites of hens' eggs and cancer has been induced.[28] The mice were not only fed for a lifetime, but to the offspring also for four generations.

The mice were fed 175 mg daily of extract. Assuming 25 grams per mouse, this is 7 grams/kg body weight. Sixty percent of the mice got tumors, corresponding to potency x dose = 1 for this dose since [1 -exp (-1)] = 0.6 (60%).

Man weighs 70 kg so if fed 500 grams per day he should also develop tumors at this rate. One egg weights 80 grams, so that according to this calculation anyone eating one egg per day has a 16% risk of cancer. I assume this is a lifetime risk, and discount the effect of feeding over several generations. The yearly risk becomes 3×10^{-3} for one egg/day which is a common dose.

This is a test at one laboratory only; so it would not warrant regulatory action and I do not include it as a risk in my tables. Nonetheless, risk is so much higher than the other risks in the table that further work seems necessary.

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[28] J. Szepsenwol, Proc. Soc. Exp. Biol. and Med., 112, 1073 (1963); 116, 1136 (1964).

I.10. Air Pollution--Sulphates, etc.

The National Air Quality Standards were set at a time when a threshold concept was dominant. The threshold concept suggests that if the concentration of sulphur dioxide, for example, in the air can be brought below a threshold, then there is no adverse health effect.

The threshold was chosen to be a level where no health effects had been observed, with a suitable safety margin. This was as a result of a very careful survey of data on a small number of people.[29]

The effects of sulphur oxides on people are mostly irritation of the bronchial tract. It transpires that careful measurements on animals by Dr. Amdur[30] and co-workers show that the resistance to bronchial flow in guinea pigs is in direct proportion to the sulphate concentration but the sulphates (sulphuric acide, zinc ammonium sulphate, which are prevalent in power plant plumes) are more important than others which come from natural causes (sodium sulphate or sulphur dioxide).

For a given mass of pollutants, the resistance is worse for small particles--just the size that escape the electrostatic precipators of a power plant. The sodium sulphate particles that form naturally in the environment come in larger particulates which get filtered in the nasal passages.

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[29] "Air Quality Control Criteria for Sulphur Oxides," AP-50, Environmental Protection Agency Report, Washington, D.C.

[30] M.O. Amdur, "Animal Studies," for Conference on Health Effects of Air Pollution, National Academy of Science, Oct. 3-5, 1973; M.O. Amdur, "The Long Road from Donora," Memorial Lecture, 1974; M.O. Amdur, Arch. Environ. Health, 23, 459 (1971); M.O. Amdur, Journal of the Air Pollution Control Association, 14, 638, (1968).

There are also some large scale epidemiological surveys. A study shows that incidence of bronchitis in 7 Japanese cities[31] is proportional to the sulphate level and Lave and Seskin[32] show the same effect for the mortality rate in the U.S. In Norway, the death rate in 156 winter weeks shows a linear relationship with the SO_2 concentration.[33] The numerical calculation has been confirmed by a recalculation of the same data.[34] Finally, the CHESS studies from the EPA[35] show health effect at sulphate levels as low as 10 $\mu g/m^3$, whereas sulphate levels at eastern cities are 20 $\mu g/m^3$.

These data are consistent with a linear relationship between mortality and sulphate concentrations with no threshold above the ambient levels and for public policy purposes this linear relationship should probably be used.

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[31] Y. Nishiwaki, et al., "Atmospheric Contamination of Industrial Ar as including Fossil Fuel Stations and the Method of Evaluating Possible Effects on Inhabitants." Report to the Conference on Environmental Effects of Nuclear Power Stations IAE-SM-145/16, International Atomi Energy Agency, Vienna.

[32] L. Lave and E. Seskin, "Air Pollution and Human Health," Science, 169, 723 (1970).

[33] W. Lindbergh, "General Air Pollution in Norway" Report from Smoke Damage Council, Oslo, 1968. Data plotted in ref. 30, main text.

[34] Report from Biomedical and Environmental Assessment Group, BNL 2053 30 July 1974, edited by L.D. Hamilton, Brookhaven National Laboratory, Upton, NY.

[35] J. Finklea, et al., "Health Effect of Sulphur Oxides", U.S. Environmental Protection Agency, referred to in Air Quality and Stationary Source Emission Control, prepared for the Committee on Public Wor s U.S. Senate, Serial 94-4, March 1975.

The differences between the threshold approach and the linear approach is considerable. Most cities in the U.S. are now in compliance with the sulphate and particulate levels of the Clean Air Act and if the levels were correctly set below the threshold there would be no mortality. With the recent data and the linear relationship, we calculate that about 20,000 persons per year east of the Mississippi have their lives shortened by up to 20 years by air pollution.[36]

Although these data suggest that sulphates are the cause of this mortality, this is not proven. Those cities which have high sulphate levels usually have high nitrate levels and particulate levels as well and the distinction is not clear. Moreover, the gases and particulates contain large quantities of known carcinogens and trace quantities of mercury and other heavy elements.[37]

The procedures for mitigation of the air pollution effects differ depending upon the existance or not of a threshold. If there is a threshold, supplementary control systems can be used to reduce sulphur emissions during unfavorable weather conditions so that the concentrations stay below the threshold.

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[36] See press release from BNL, July 1977; also Wilson and Jones, Energy, Ecology and the Environment, Academic Press, New York, 1974. Ch. VIII.

[37] Wilson and Jones, Ibid., Chap. VIII.

On the other hand, associated with any theory of the linear relationship is that the health effect is proportional to a long term average. Then supplementary control systems are of little use. This has led EPA to insist that sulphur be removed by stackgas scrubbers. If, however, the health effect is due to nitrates or particulates, this might be a useless waste of money. A far better mitigation procedure would be to insist that power plants be located hundreds of miles downwind of major population centers--such as on the northeastern seaboard of Maine--the health effects are reduced. In a report for ERDA,[38] Chang and Wilson show that mortality reduction factors of 10 or more can be obtained independently of which of the effluents actually cause the health effect.

I note that this is not a cancer risk. But there is probably a cancer risk due to polycyclic aromatic hydrocarbons of which benzo (a) pyrene is the usual indicator. These seem to concentrate in cities, and do not seem to have the long range effects of sulphur and nitrogen oxides. The cancer effect, for urban populations only, is calculated in the section on benzo (a) pyrene.

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[38] B. Chang and R. Wilson, "Mitigation of the Effects of Sulphur Pollution," Energy and Environmental Policy Center Report, Harvard University, Cambridge, MA. 02138, July 5, 1976.

I.11. Accident Risks

The accident risks are calculated a little differently in each case. The total number of deaths are divided by the number at risk. The sources of these numbers is listed in the tables.

The calculation sounds simple, but they must be made with circumspection. For example, I noted 46,000 deaths due to motor vehicle accidents, and it might be thought that the risk is a risk to the motor vehicle driver. But 8,600 are in collisions with pedestrians. I assume here (though the source quoted does not say) that most of these deaths are the death of the pedestrian usually due to no action on the pedestrian's part. Over 1/4 of the pedestrians killed are children, so that it may be reasonably assumed that alcohol consumption of the pedestrian is not a large factor.

In many cases, sources and calculations of other authors give different numbers. I have worked at these, and chosen the most plausible. In any case, the risk is good to better than a factor of 2 which is as good as the cancer risks are known.

Again in calculating the risk to truckers, and tractor drivers, I use data on accidents (for the numerator) and total number of trucks or tractors in use. I assume there is one driver per truck or tractor. Likewise for fishing accident risks, I use as the denominator in the risk equation the total number of fishing licenses, although in many of these cases the fishermen are firmly on shore and not subject to appreciable risk of drowning.

We are used to the ordinary risk of air travel which is the risk of accident. At the present time, air travel is fairly safe: one death for 1000 million miles of travel on a scheduled carrier.[38] The risk is, of couse, concentrated at take off and landing. Nonetheless, I use this to get a figure for the risk of accident for one 3000 miles (cross-country) flight; it is 3000/(1000 million) or 3×10^{-6}. One such flight per year gives a risk of 3×10^{-6} per year. A pilot, flying the FAA maximum of 50 hours/month has an accident risk of 3×10^{-4}.

[39] Bureau of Aviation, National Transportation Safety Board; see also: Accident Facts, 1976 Edition.

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Estimate of risk from environmental exposure to radon-222 and its decay products

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Consideration of the epidemiological evidence on radiogenic lung cancer in uranium miners and of the incidence of the disease generally leads to an upper estimate for the lifetime risk of 10^{-4} cases per working level month for members of the general population.

IN radiological protection, the emphasis is usually on limiting exposure to artificial radiation. Thus, there is a well-established approach to the protection of staff in hospitals from X rays and to the protection of members of the public from radioactive consumer goods or from effluent from nuclear power plants. How one should deal with exposure to natural radiation is less clear.

Human beings have always been exposed to natural radiation both from within and outside the body. The view adopted by the International Commission on Radiological Protection (ICRP) is that exposure to normal levels of natural radiation cannot usually be subject to control, but it is acknowledged that there may be unusual circumstances in which control is desirable[1]. The Commission has in mind circumstances in which there is a considerable increase in exposure as a result of human practices or choices of environment.

One of the principal ways in which members of the public receive natural irradiation is by breathing the radon-222 decay products in air. Radon gas is emitted by soil, rocks and building materials, all of which contain the parent radium to some extent. Radon can also be present in water, from which it is readily desorbed. Out of doors, the gas is dispersed in the atmosphere, but it concentrates indoors to a degree dictated primarily by the ventilation rate of the dwelling and the rate of radon input. The solid short-lived decay products of radon grow in rapidly[2] and, if attached to an aerosol rather than to walls or furniture, are inhaled and irradiate the lung tissues. Lung cancer may result.

Even in normal circumstances, human exposure to radon decay products is substantial compared with other components of the overall exposure to natural radiation. In abnormal circumstances, radon exposure may be dominant and perhaps also unacceptably high. Such abnormal circumstances stem from the use of building materials with relatively high radium content, for example, or the use of domestic water with exceptionally high radon content.

The basis of dose limitation in radiological protection is risk limitation. For example, the dose equivalent limit for workers is set so that the average risk likely to be incurred is comparable with the average prevailing for other workers in industries regarded as having high standards of safety. For members of the public, the level of acceptability of risk is probably an order of magnitude lower. It is important, therefore, to have some information on risk factors for environmental exposure to radon decay products if a system of control is to be considered.

Direct risk information does not exist for environmental levels of radon decay products. It is therefore necessary either to extrapolate from data for occupational exposure or to infer the risk from the incidence of lung cancer in the wider population. Both approaches are followed here. We have taken care neither to exaggerate the risk nor to overextend the evidence.

Uranium miner data

The only occupational data that can be used for obtaining a risk estimate for lung cancer caused by radon decay products are those from the exposure of underground uranium miners. Although the BEIR report[3] does give an estimate of the risk of lung cancer from whole-body external irradiation, this is insufficient for the present purpose. Of the four sets of data on miners, those from Sweden and Canada are considered to be less useful, and we have therefore concentrated on the data from Czechoslovakia and the United States. Information on exposure is given in terms of working level month (WLM), the unit commonly employed in mining. One WLM is defined as exposure for 1 working month of 170 h to a concentration of one working level (WL), where one WL is any combination of short-lived decay products of radon-222 per litre of air that will result in the emission of 1.3×10^5 MeV of α energy during complete decay[2,4,5].

Several factors cause the data on miners to be more difficult to use than might at first appear. The average concentrations throughout US mines in the 1940s and 1950s were high (7–15 WL) and mean exposures accumulated over the 1950s and 1960s were about 800 WLM (ref. 6); in the last decade, however, levels were lower by one to two orders of magnitude[7,8]. Most of the early information on exposure was obtained by the operators, frequently in areas where ventilation problems were known to exist: on the other hand, there was sometimes a tendency for higher exposures to be under-reported. All the measurements on aerosol characteristics, such as particle size and unattached fraction, have been made under modern mining conditions. These conditions include improved ventilation, the presence of diesel smoke (which probably overwhelms other particulates) and the virtual disappearance of the one-man mine. The evaluation of risk is also complicated by the presence of other carcinogens in mine air as well as by the miners' smoking history and their previous experience of hard-rock mining.

The estimates of lifetime risk that emerge from the data available on miners range from one estimate of 21–54 deaths from lung cancer per 10^6 WLM of collective exposure to one of 1,000 or so. Stewart and Simpson (unpublished) and Myers and

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Stewart[9] have evaluated the American and Czech data using various statistical techniques. Their work indicates that the incidence of lung cancer can be accounted for by a linear relationship with exposure, allowing a constant factor for non-radiogenic lung cancers; their estimate is 21–54 lung cancers per 10^6 WLM from the US data[10], but the Czech data imply a risk about three times as great[11]. The discrepancy is not readily explained. Stewart and Simpson also found a real or apparent threshold in some of the analyses, which suggests that the estimate of risk for low-level exposure may even include zero as a lower bound. (In such cases, Stewart and Simpson used the slopes of the response curves to derive risk factors.) Jacobi[12] also noted the discrepancy between the US and Czech data and proposed a range of 100–500 lung cancers per 10^6 WLM, which is virtually identical with an earlier estimate by UNSCEAR[13]: in neither case is a threshold effect posited. The highest estimate of risk of about 1,000 lung cancers per 10^6 WLM comes from Archer's recent proposal[14].

Whereas it is not possible completely to rule out any of these estimates of the lifetime risk from occupational exposure, our objective is to estimate risk to the general population from exposure to radon decay products in the general environment. In using the data for uranium miners to derive the lifetime probability of fatal lung cancer for members of the public, one must note that there are significant environmental and physiological differences between the two situations. These differences result from the possible contribution to lung cancer induction in mining environments of other dusts and gases, from differences in breathing rates, from differences in equilibrium ratios of the radon decay products, from high as opposed to low concentrations, from differences in smoking habits and from population distributions that differ in age and sex. Although some of these factors may tend in opposite directions, on balance, they suggest that members of the general public may be at a lower risk per unit exposure than miners. For example, breathing rates for a man resting indoors could be two to three times lower than for a man mining, with the concomitant decrease in exposure counteracted to some degree by an increase in the probability of depositing radon decay products in the lung.

We judge from the epidemiological evidence that the most defensible upper bound of the lifetime risk to the general population is 100 deaths from lung cancer for a collective exposure of 10^6 WLM, or 10^{-4} per WLM. This particular factor, although severely rounded to avoid the impression of unwarranted accuracy, can be supported by other indirect epidemiological evidence, which is detailed below, and by an analysis (W. Jacobi, personal communication) of the dosimetry of radon decay products in the manner of Jacobi and Eisfeld[15].

Lung cancer incidence

Most calculations of the exposure of the general population were initially made on the basis of outdoor radon levels. Indoor levels were known to be higher, but it is only recently that an appreciable number of measurements has become available. UNSCEAR[13] summarized the literature appearing before 1976, and more detailed measurements have since been published[16–19]. Information on living habits in the Northern Hemisphere indicates that more than 90% of the day is spent indoors, so that indoor exposure is used here to represent total exposure.

The activity concentration of radon indoors in the Northern Hemisphere lies, on the average, in the range of 0.5–1 pCi per l. If a value of 0.8 pCi l^{-1} is assumed, some measurements in the United States[16] indicate that this can be converted to a decay product concentration of ~0.004 WL. For 168 h per week of indoor occupancy, the exposure would be 0.2 WLM in 1 yr or 12 WLM in 60 yr. The latter period might be shortened somewhat, but this would not change our general conclusion.

Given this mean lifetime exposure of 12 WLM and the risk factor of 10^{-4} per WLM given above, a maximum lifetime risk of 0.12% is obtained by extrapolating linearly and by neglecting latency and accumulation interval. This is a relatively small fraction of the present lifetime risk of lung cancer (about 4% in the United States), which is largely attributable to cigarette smoking.

Making the reasonable assumption that exposure of the general population to radon decay products has been constant over several decades, one can compare the estimate of 0.12% with the incidence of lung cancer before the effects of smoking became pronounced. There are relatively good data[20] on incidence in the United States in 1930 which yield a lifetime risk of 0.1%, roughly 0.15% for males and 0.05% for females. Since not all lung cancers at that time would have been caused by radon, this indirect evidence supports the view that a value of 10^{-4} per WLM is a reasonable upper bound of risk to members of the public.

Quite independently, Cliff and others[17,18] have studied the relationship between the observed exposure to radon decay products and the observed incidence of lung cancer in British women. Taking an average exposure rate of 0.15 WLM per yr, and assuming a risk factor of 2×10^{-4} per WLM (ref. 6) as well as a 20-yr latency period, they predict a higher incidence of lung cancer than is observed from all causes in women below 40 years of age. This supports the evidence from the United States, and indeed an upper bound of 10^{-4} per WLM for the lifetime risk would not be incompatible with the British epidemiological evidence.

The overall evidence therefore points to an upper estimate for the lifetime risk of 10^{-4} cases per WLM for members of the general population.

Risk estimates compared

It is of interest to compare the risk of lung cancer from exposure to radon decay products with that from external penetrating radiation. On the basis of a linear non-threshold model, and purely for protection purposes, ICRP estimates that the risk factor for external irradiation of the lung alone is 2×10^{-3} per Sv or 2×10^{-5} per rem, where Sv and rem are the new and old units of dose equivalent, respectively[1]. Thus, the lifetime risk from an exposure to radon decay products of 1 WLM, namely 10^{-4}, would not exceed that from an external irradiation dose equivalent of 50 mSv (or 5 rem) to the lung alone.

It is difficult, however, to imagine circumstances in the general environment in which the lung alone would be exposed to external radiation: whole-body irradiation is much more likely. The ICRP takes the view that most, if not all, forms of fatal malignant disease and hereditary damage can be induced by exposure to ionizing radiation and that, when all the organs of the body are uniformly exposed, cancer of the lung in particular will account for about 10% of all harmful effects. Thus, the lifetime risk associated with an exposure to radon decay products of 1 WLM would not exceed the overall risk associated with a dose equivalent of about 5 mSv (or 0.5 rem) to the whole body from external radiation.

The annual dose equivalent limit recommended by the ICRP for individual members of the public is in fact 5 mSv.

This review has its origins in an international workshop on radiation protection principles for naturally occurring radionuclides held at Arlington, Virginia, in May 1978 by the Nuclear Energy Agency of the OECD. The authors, who were members of a group considering radon problems, have had a number of subsequent exchanges and discussions on the estimation of risk from this source. Thus the present paper should be attributed to them, even though they had the advantage of the initial group deliberations which included M. Kinoshita. The authors also acknowledge the assistance they received from M. C. O'Riordan.

FAILURES AND CRITIQUE OF THE BEIR-III LUNG CANCER RISK ESTIMATES

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Abstract

It is shown that the BEIR-III prediction of lung cancer incidence due to environmental radon is more than twice the actual incidence of all lung cancers among non-smokers. Histological evidence from autopsy studies of radiation victims and of non-smokers is reviewed, and it is shown that no more than 10% of all lung cancers in non-smokers can be due to radon. When alternative causes of lung cancer are considered, it is concluded that the BEIR-III estimates of lung cancer risk due to environmental radon are too high for non-smokers by at least a factor of about 40. The evidence for synergism between smoking and radiation induced lung cancer is reviewed, and it is concluded that the BEIR-III estimates for smokers are too high by at least a factor of about 10.

The bases for the BEIR-III model are reviewed, and it is concluded that it must be based almost entirely on the Czechoslovakian uranium miners; its estimated risks are higher than would be obtained from essentially all other studies. The possibility that there are other causes than radon for lung cancer among miners is discussed, and it is shown that the Czechoslovakian data are at least as consistent with the hypothesis that all excess cancers are due to other causes (proportional to length of employment) as with the hypothesis used in BEIR-III that they are all due to radon. It is concluded that other causes can easily explain all cases where lung cancer rates are high, whereas it is very difficult to explain the much lower rates for U.S. uranium miners (5 x lower than BEIR-III estimates) and other similar situations. It is pointed out that the BEIR-III lung cancer model is strongly supra-linear (i.e. concave downward), contrary to the bulk of experimental and theoretical evidence on that question, and contrary to other statements in the BEIR-III Report. It is concluded that the BEIR-III lung cancer risk estimates are probably too high by a factor of 2 to 5 for miners, and because of different smoking habits, the risk to the public may be another factor of 2 lower. The remainder of the over-estimate for effects of environmental radon must be due to non-linearity in the dose-response relationship.

I. Introduction

The health effect of radon exposure is a scientific problem of immense social significance. According to usual estimates (NAS-72, UN-77, NC-75), it is responsible for far more fatalities than all other environmental radiation combined, it is by far the most important contributor to environmental impacts of the nuclear energy industry, and it is responsible for far more occupational fatalities than all other occupational exposure to radiation combined, a number exceeding the total excess fatalities among the Japanese A-bomb survivors since 1950. The above mentioned estimates were based on the BEIR-I Report (NAS-72), but the recent BEIR-III Report (NAS-80) introduces a large increase into the estimates, thus making the problem several times more serious. It has been shown (Co-80) that it makes insulation of buildings for purposes of energy conservation far more damaging to human health than any other measure being taken to ease our energy problems.

The purpose of this paper is to offer a critique of these estimates. In Sec. II we use it to calculate the risk to non-smokers from environmental radon, and show that it predicts more than twice the total lung cancer rate actually experienced by non-smokers. In Sec. III we review the histological evidence which shows that no more than about 10% of the lung cancers among non-smokers can be due to radiation. In Sec. IV, we discuss alternative causes of lung cancer, which further reduces the fraction that can be caused by radiation, and in Sec. V we summarize and conclude that the BEIR-III model over-estimates the lung cancer rate in non-smokers due to environmental radon by at least a factor of 40. In Sec. VI we review the evidence on risk of radon exposure to smokers, and conclude that it is probably not more than four times the risk to non-smokers; this means that the BEIR-III model over-estimates the risk of low level radon exposure to smokers by at least a factor of 10. In Sec. VII, we consider the reasons for the large over-estimates in the BEIR-III Report.

II. BEIR-III lung cancer rates due to environmental radon and comparison with total lung cancer rates among non-smokers

The BEIR-III Report (NAS-80) gives the following estimates of the lung cancer risk from low-level radon exposure in terms of working-level-months (WLM):

age 35-49, risk = 10 X 10^{-6}/yr-WLM

50-64, 20

>65, 50

where ages refer to age at death. For latent periods between exposure and onset of these risks it gives

age 0-14, latent period = 25 years

15-34, 15-20 years (we use 17 yr)

>35, 10 years

where ages refer to age at exposure. This is a clear and unambiguous model which is readily usable for deriving numerical estimates. We begin by using it to calculate lung cancer rates due to environmental radon.

The first step in this process is to estimate the environmental exposures. Outdoor exposures (Co-80) are about 1 mWL (milli-working-level), but it is generally recognized that indoor exposures are much higher. A compilation of results from U. S. studies of indoor radon levels (Co-80) is given in Table 1. The 1977 UNSCEAR Report reviews the situation world wide and concludes that the average indoor exposure rate is 5 mWL. This value is in reasonable accord with the data in Table 1.

A working-level-month, WLM, is defined as the exposure received in 170 hours from a 1WL exposure rate. Since there are 8760 hours in a year, full time exposure to 1mWL gives (.001 X 8760/170=).052 WLM per year; this would be the average exposure if people spent all of their time outdoors. It is generally estimated that the average American spends 80% of his time indoors where radon levels are 5 times higher; this gives an additional exposure of (0.8 X 4 X .052=).166 WLM, bringing the total average exposure to 0.22 WLM/yr due to environmental radon.

In Table 2, we use this exposure rate, 0.22 WLM/yr, with the BEIR-III model, to calculate the expected lung cancer incidence for various age groups. Col.(2) gives the number of years of effective exposure to environmental radon, i.e. the number of years before the latent period; for older ages, this is 10 years less than the age because exposure during the last ten years contributes no risk in view of the ten year latent period. For younger ages, the more complicated pattern of latent periods in the BEIR-III model give entries in Col.(2) more than 10 years less than the age. The exposures in Col.(3) are just 0.22 WLM/yr times the number of effective years of exposure from Col.(2). Col.(4) lists the applicable risk factors from the BEIR-III model for each age group, and these are multiplied by the exposure to give the risk, or the expected rate for lung cancer mortality, in Col.(5). These may then be compared with observed age-specific rates.

In addition, Table 1 gives calculations of total rates in the U.S. population. The U.S. population (HEW-79) in each age group based on 1976 statistics is listed in Col.(6), and this is multiplied by the rates in Col.(5) to give the total

number of expected cases in the U.S. each year, in Col.(7). The total number of cases may be obtained as the sum of the number of deaths in each age group, and this sum divided by the total population (including ages 0-34) gives the expected lung cancer rate in the whole U.S. population. The result for this is 114 X 10^{-6}/year. Analogous procedures can be used to calculate the expected rate for all those above some specific age. For example, the rate for age >40, is the sum of the figures in Col.(7) for ages >40, which is 24,080, divided by the sum of the figures in Col.(6) for ages >40, which is 77.8 X 10^{6}. The result is 309 X 10^{-6}/year.

The comparison between the age-specific expected rates from Col.(5) of Table 1 and observed rates among non-smokers is shown in Table 3. The recent paper by Garfinkel (Ga-80) presents the results of a 12 year follow-up on one million Americans in a study by the American Cancer Society. The paper by Hammond (Ha-66) gave the results of the first four years of that study. Age groupings in that study were not the same as those used in Table 3, so the values listed there were derived on the assumption that rates were constant over each 5 year age group. For example, rates were given as age 62-66, 139; age 67-71, 147; age 72-76, 161; the rate listed in Table 3 for age 65-74 is then (0.2 X 139) + (0.5 X 147) + (0.3 X 161) = 149. The results in Table 3, given only for males and females combined, indicate little difference between the 4 year and 12 year follow-up.

The paper by Kahn (Ka-66) is based on the so-called "Dorn Study" of 293,000 U.S. veterans of World War II who carry government health insurance. It represents 8$\frac{1}{2}$ years of follow-up. A recent update on that study by Rogot (Ro-80) does not give absolute lung cancer rates, but the age-standardized ratio between smokers and non-smokers has remained the same which indicates that there has probably not been an important change in the rates for either.

The paper by Hammond and Horn (Ha-58) was an early study by American Cancer Society of 188,000 men involving only 15 lung cancer deaths among non-smokers (as compared with 150 deaths in Ga-80), and only 6 of these 15 were considered to be well-established as lung cancer whereas 3/4 of all cases in the study (mostly smokers) were considered to be "well established". Only a few age groupings were given and they did not coincide with those in Table 2, so the method described above for (Ha-66) was used to derive the values entered.

It is immediately evident from Table 3 that the BEIR-III estimates for lung cancer induced by environmental radon exceed the total lung cancer rates due to all causes among non-smokers by about a factor of two at every age. It is only fair to point out that this does not represent a direct discrepancy with the BEIR-III Report since the latter states that its estimates for non-smokers may be too high by a factor ranging from 1 to 6, favoring a factor intermediate between these.

Comparisons can also be made with total lung cancer incidence for all ages. A paper by Hammond and Seidman (Ha-80) gives the rate for ages above 40 to be 177 X 10^{-6}/year for men and 124 X 10^{-6}/year for women, whereas the rate calculated in Table 2 from BEIR-III for ages above 40 was 309 X 10^{-6}, a factor of two higher. For all ages, the rate among women was reported as 36 X 10^{-6}/year (Ha-58a) as compared with 114 X 10^{-6}/year calculated from BEIR-III in Table 2, a discrepancy of well over a factor of two.

All of the data we have presented are basically from three study groups, but in all three cases the BEIR-III estimates for lung cancer induced by environmental radon alone are a factor of two higher than actual total lung cancer rates among non-smokers. Harley and Pasternack (Har-80) have also reported difficulty in reconciling some of the recent estimates of radon-induced lung cancer risk with effects of environmental radon; they suggest a decrease in the risk with time after exposure due to repair mechanisms, but this is not used in BEIR-III.

Another approach to comparing the BEIR-III prediction with rates for non-smokers is to use national statistics from the early years of this century which is before cigarette smoking became popular. Data on lung cancer rates in various countries are listed in Table 4. We see that the rates in all but one of the countries included are less than 10 x 10^{-6}, and in Sweden the rate was only

1.3×10^{-6}. A calculation of the rate predicted by BEIR-III is shown in the last two columns of Table 2; the result based on U.S. population statistics for that time is 59×10^{-6}/year, an order of magnitude larger than several entries in Table 4.

In considering statistics from so long ago, consideration must be given to the efficiency of diagnosis. This question was discussed in extensive detail in a previous paper (Co-80a) and it was concluded that the numbers in Table 4 should be increased by a factor of 2 to 5 to account for missed diagnoses. Another consideration is that environmental radon exposures may have been lower at that time because buildings were less tight and hence did not retain radon as well. This would perhaps reduce the BEIR-III prediction by a factor of 2. In spite of these correction factors, it still seems that the BEIR-III model predicts total lung cancer rates due to environmental radon considerably larger than the total lung cancer rates in many countries early in this century.

III. Histological Evidence

The basic histological types of lung cancer are:

1. epidermoid (squamous cell)
2. small cell undifferentiated (SCU)
3. adenocarcinoma
4. large cell
5. mixed epidermoid + adenocarcinoma
6. bronchiolar or alveolar

All but No. 6 correspond to the World Health Organization classification; No. 6 was included here because it was included in some of the data, and because it is clearly not associated with radon induced lung cancer since radon daughters deposit in the upper part of the bronchial tree long before they can reach the bronchiolar or alveolar regions.

There is a great deal of evidence that lung cancers due to radon exposure are predominantly of type 2, small cell undifferentiated (SCU). The first such evidence was on the 19th century Erz Mountain miners (Lu-71) in which the predominant cell type was found to be SCU. The best and most quantitative evidence is the pathological study of autopsy tissue from U.S. uranium miners who have died of lung cancer (Ar-74). The principal results are shown in Table 5 which indicates that 69% of all excess cases were Type 2 (SCU). We see that this conclusion is roughly independent of dose. In the more recent data, the rate of occurrence of SCU has gone down (Sa-80) so that present indications are that perhaps 50% of all excess U.S. uranium miner cancers are of this type.

The data on Czechoslovakian uranium miners (Ho-77) are given in Table 6 where it is again evident that Type 2 is strongly increased. The calculation in the last three lines of the Table and explained in the caption indicate that 57% of excess lung cancers among these miners were Type 2 (SCU). As may be seen from Table 6, this predominance of Type 2 is similar in all dose ranges, and more recent data (Ku-79) extends the validity of this observation from <99 WLM to >400 WLM. The data on Japanese A-bomb survivors (Ci-74) is much poorer because of limited statistics. The frequency ratios between >200 rad and <1 rad exposures were 1.7 (3 cases) for Type 1, 3.9 (5 cases) for Type 2, and 2.0 (4 cases) for Type 3. Roughly, then, the extra cases were 1.2 of Type 1, 3.7 of Type 2, and 2 of Type 3, so (3.7/6.9=)54% were Type 2.

One method for determining histological types of lung cancer is sputum cytology which uses cells that are sloughed off in coughing rather than autopsy tissue. There is good evidence (Sa-76) that sputum cytology sometimes indicates epidermoid (Type 1) while autopsy tissue indicates SCU (Type 2); in many of these cases there were double tumors. It may be that this is due to the different phases of the disease being studied (Ra-80), or that epidermoid cells are more easily slougned off, or that SCU

develops later but progresses much more rapidly (Sa-76). This problem can be avoided if only data obtained from autopsy are considered. This means ignoring the sputum cytology study of Newfoundland fluorspar miners (Wr-27) in which only 2 of 29 cases were Type 2 while 26 were Type 1.

Our conclusion, then, is that for histological classification based on autopsy material, about 55% of all lung cancers induced by radiation are Type 2, SCU.

We now turn to the question of frequency of various histological types of lung cancer among non-smoking members of the general public. The data obtained from a literature search on this are summarized in Table 7. In cases where two different readings disagree, one case is recorded with a question mark for each type, and in summing, each such case is counted as 0.5. While these procedures are quite crude, they are expedient and alternative reasonable procedures would not appreciably change the results.

The over-all conclusion from Table 7 is that only about 5% of all lung cancers among non-smokers are SCU (Type 2). This conclusion is in essential agreement with the findings of each of the individual studies. If we assign half of the cases listed only as "anaplastic", or "undifferentiated" as Type 2, this would change the final percentage only from 4.6 to 6.3.

Nearly all of the data in Table 5 are from studies of tissue obtained in autopsy. The principal exception is the data of Vincent et al (Vi-77) in which only 42% were from autopsy, but the percentage of Type 2 in that study is not less than in the others, and there are relatively few Type 1 so our worries about Type 2 being improperly classified as Type 1 do not seem to be applicable here. There were a few cases in the work of Wynder (Wy-67) based on sputum cytology, but none of them were classified as Type 1.

It therefore seems valid to compare these results with those for radiation induced lung cancer discussed above, and it is immediately evident that the percentage of all lung cancers that are SCU among the miners is a factor of ten higher than among non-smokers in the general population, roughly 55% vs 5-6%. One possible objection to this conclusion is that it is based on histological information for all uranium miners and there might be a difference between smokers and non-smokers in this regard; but among the non-smoking miners for whom histological information is available, 4 of the 6 whites had SCU (and one of the others was a heavy cigar smoker), and both of the 2 American Indian non-smokers had SCU (Ar-76). Another possible objection is that the pathologists who studied the U.S. uranium miners were biased toward SCU, but three independent pathologists were involved and for the general population (i.e. non-miners) their percentage of all lung cancers that are SCU was only 14% (Sa-71), whereas for the groups whose work is included in Table 7, it was 30% for Wynder et al (Wy-76), 19% for Vincent et al (Vi-77), and 22% for Yesner et al (Ye-73). The Czechoslovakian miner and Japanese A-bomb survivor data were, of course, classified by entirely different groups of pathologists but their results are the same.

There is, of course, a possibility that the dose-response curve is different for different histological types of lung cancer, and that SCU decreases much more rapidly at low dose than other types. However, there is no evidence or precedent for such a hypothesis, and the dose independence of the fraction that are SCU at high doses argues against it. If we exclude this possibility, it seems difficult to avoid the conclusion

that about 50% of radiation induced lung cancers are SCU whereas only about 5% of all lung cancers among non-smokers are SCU. This means that no more than 10% of all lung cancers among non-smokers can be due to radon exposure (Appendix A). Since we have shown in Sec. II that the BEIR-III model over-predicts total lung cancer among non-smokers by a factor of 2, this means that the over-prediction is actually by a factor of

$2 \times 10 = 20$.

Even this degree of over-prediction is based on the supposition that all SCU lung cancers among non-smokers are due to radon exposure. We examine that question in the next section.

IV. Alternative Causes of Lung Cancer

There is no other type of cancer for which nearly so many causative chemical agents have been found as for lung cancer. A recent review (CoT-78) lists the following as causes of lung cancer: aromatic hydrocarbons including coal soot, coal tar, other products of coal combustion, petroleum, petroleum coke, wax, creosote, anthracene, paraffin, shale oils, and mineral oils; alkylating agents such as mustard gas, vinyl chloride, bis (chloromethyl) ether, chloromethyl methyl ether, and chloroprene; metal dusts such as arsenic, beryllium, cadmium, chromium, hematite (iron ore), lead salts, nickel, and asbestos. There are at least 1400 other chemicals on which there is carcinogenic information (Ch-74), although not necessarily related to lung cancer.

It has been suggested (CoE-72) that air pollution may be an important cause of lung cancer. It includes many of the aromatic hydrocarbons mentioned above, benzo-a-pyrene which is believed to be the principal cancer causing agent in cigarettes, and many compounds that are well known to irritate the lungs. There are probably many other contributing environmental factors as evidenced by large variations in lung cancer rates with geography. Based on 1950-1970 statistics (Ma-74), rates varied from state to state between 20×10^{-5} (ND) and 52×10^{-5} (LA) for white males with a strong tendency toward higher rates in industrialized states, and there is a strong tendency toward higher rates in cities than in rural areas. Some of these differences are probably due to variations in smoking habits, but it would be difficult to explain them all in that way.

There are large variations in lung cancer rates among men in different occupations. In a recent British study (RS-78), they were higher than average after correcting for age and social class by a factor of 1.8 for foundry moulders, 1.7 for electroplaters, 1.6 for plasterers and boiler firemen, and 1.5

for riveters, welders and cutters, bricklayers, and drivers. A study in Los Angeles County (Me-76) found 16 occupational categories with over twice the average lung cancer rates; the 24 occupations with the highest rates include 29% of all the lung cancers but only 15% of the workers; the steel manufacturing industry had 59 cases vs 21 expected. Since over 50% of all men were smokers when these statistics were gathered, even 100% smoking in individual occupations could not explain some of these high rates.

Exposure to tobacco smoke by non-smokers may also be an important cause of lung cancer among the latter. It is also widely believed that chronic bronchial disease can develop into lung cancer. One might consider the logical possibility that most alternative causes do not induce Type 2 lung cancer. However, Type 2 is one of the principal types induced by cigarette smoking; in the general population which includes smokers, 30% of all lung cancers are Type 2 (Arm-75), whereas among non-smokers we have seen that the percentage is only about 5%. It thus seems most unlikely that other chemical carcinogens do not cause Type. 2.

With all this evidence for other causes of lung cancer, it is very difficult to believe that nearly <u>all</u> Type 2 lung cancers among non-smokers are due to radon. An upper limit on the fraction caused by radon might be 50%, and 10% is probably a more reasonable estimate.

V. Conclusions For Non-smokers

In summary of the situation among non-smokers:

(1) The BEIR-III model predicts 2 times as many lung cancers due to radon exposure as the total actually observed.

(2) Histological evidence indicates that no more than about 1/10 of the observed lung cancers among non-smokers can be due to radon exposure.

(3) At least 1/2 of these cases are very probably due to other causes.

We thus conclude that the BEIR-III prediction of lung cancer incidence due to

This statement should not be interpreted to mean that the model is in error by that factor. The BEIR-III Report states that its estimates may be high by as much as a factor of 6 for non-smokers, but gives equal consideration to the possibility that it may not be too high at all, and concludes that "it is probable that the truth is somewhere in between." In view of the tendency of regulatory agencies to adopt a "prudent" position by using pessimistic estimates, they would almost surely assume that the BEIR-III estimates applied to non-smokers would not be high by more than a factor of two. In doing so, the evidence developed here indicates that they would be over-estimating the effects of radon exposure by at least a factor of 20!

The principal link in our reasoning which might be questionable is in assuming that radon levels in buildings have remained constant over time. If the rate of air exchange (ventilation rate) were more rapid in earlier times due to less efficient insulation, lifetime averaged radon exposures to those who have died in recent years would have been lower than we have assumed. Since present indoor levels are about five times outdoor levels, the maximum possible error from this source is a factor of five.

This possible variation with time of ventilation rates was discussed with three experts in the field; in each case an off-the-cuff estimate was requested, and was given only with considerable reluctance accompanied by caveats, but we present them here. A University of Pittsburgh engineering professor specializing in energy conservation research guessed that there was little change in average ventilation rates from 1930 until 1975. A research scientist in the National Association of Home Builders Research Laboratory (Rockville, MD) estimates that new homes built in 1940 had about 1.7 times higher ventilation rates than those built in 1970 (1-1.5 vs 0.5-1.0 air changes/hour), but he had no experience with pre-1940 construction; most of

this improvement occurred after 1965, whereas nearly all homes included in Table 1 were built prior to that time. He also points out that measurements of ventilation rates do not include opening doors (.06 air change for a typical open-close), open windows (0.25 air change/hour for a 4-inch opening), and furnaces (0.1-0.2 air change/hour); radon measurements in houses were generally carried out under normal lived-in conditions.

A research scientist at Lawrence Berkeley Laboratory with extensive experience in measuring ventilation rates in Berkeley (CA)area houses has found a roughly linear decrease by a factor of 3 in ventilation rates with construction date from the post-earthquake era (ca 1910) to the present; he emphasizes that it is not clear what portion of this is simply due to aging rather than to construction practice. Since the average age of houses in use has probably not changed appreciably over time, only the second factor is relevant. When all of these expert opinions are considered, it seems likely that past radon levels in homes were somewhat lower than present levels, but probably the lifetime average levels experienced by those who have died in recent years are not more than a factor of two lower than those reported in the measurements in Table 1.

Another source of uncertainty in our reasoning is that a WLM of exposure to environmental radon is not necessarily equivalent to a WLM exposure to miners, on which the BEIR-III model is based. Differences arise due to reduced breathing rates under normal environmental conditions, lung morphometry, and an increased percentage of radon daughters as free ions in the environment — these attach most easily to bronchial surfaces, but they also

attach to dust particles and hence are less prevalent in mines than in the environment. These matters were taken into account in a calculation by Harley and Pasternack (Har-80) who found that the rad/WLM were 0.5 for uranium miners, and 0.71, 0.64, 1.2, and 0.64 for an adult male, an adult female, a 10 year old child, and a 1 year old infant, respectively. Thus, environmental radon is more effective in causing lung cancer per WLM than radon in mines by a factor of about 1.3. When this is combined with the effects of variation of ventilation rates with time discussed in the previous three paragraphs, the total correction is perhaps a factor of 1.5. These corrections therefore have little bearing on the order of magnitude discrepancy that is basically the matter under discussion.

VI. The Radon Risk to Smokers

The rates predicted by BEIR-III from environmental radon exposure are far less than the actual rates for smokers, so other methods must be used to study their risk from radon. The question is whether there is a synergism between smoking and radiation in causing lung cancer, for if there were no such synergism, the rates for non-smokers would also apply to smokers.

There is strong evidence both for and against such a synergism. The evidence in favor is based on the data for U.S. and Czechoslovakian uranium miners. The data on U.S. miners are listed in Table 8 (Ar-76). Unfortunately, corresponding rates for non-miners are not given nor is the age distribution from which it can be calculated, but crude estimates indicate that the 1-359 WLM data can be used for these in analyzing the higher exposure data for non-smokers and 1 pack/day smokers. If this is done, the >1800 WLM data gives the lung cancer incidence rates due to radon as 1.2 for non-smokers and 8.1 for 1 pack/day smokers, a ratio of 6.8; the 360-1799 WLM data gives 0.66 for non-smokers and 2.2 for 1 pack/day smokers, a ratio of 3.3 (for the >1 pack/day smokers, these ratios are 8.9 and 3.0, and for the <1 pack/day smokers they are 5.6 and negative). An alternative procedure would be to assume that the "expected" number is negligible, in which case the ratio is 9.4/1.39 = 6.8 for the >1800 WLM group, and 4.1 for the 360-1799 WLM group. Roughly then, the data on U.S. uranium miners indicates that 1 pack/day smokers are about 5 times more susceptible to radon induced lung cancer than are non-smokers; this ratio seems to decrease with decreasing dose. However, there are at least two facts that indicate that this factor of five is too large. First, the latent period between exposure and development of lung cancer is known to be longer for non-smokers (NAS-80), which means that the smoker/non-smoker risk can be expected to decrease with time. Second, there is some indication that the non-smoker group started mining at a later age than

The evidence on Czechoslovakian miners mentioned in BEIR-III is that 30% of the miners were non-cigarette smokers, but only 8 of 115 miners (7%) whose smoking habits were reported in a histological study were non-cigarette smokers. There is no assurance that the 115 were representative of the entire group of victims, but if they were, the risk to smokers was [(93/70+(7/30)=] 5.7 times the risk to non-smokers. Again, this ratio is expected to decrease as follow-up continues because of the longer latent period for non-smokers.

While the studies of U.S. and Czechoslovakian miners indicates that there is a smoking-radiation synergism, there are several sources of evidence that there is not (Co-80a). The most spectacular of the latter is the close similarity in lung cancer rates between the nineteenth century Erz Mountain miners and modern U.S. miners who received the same exposure. The comparison may be seen in Fig. 1. Cigarettes did not come into common use until the second decade of the twentieth century, so the difference between the smoking habits of the two groups is dramatic.

A second evidence for no synergism between smoking and radiation exposure is the data on Japanese A-bomb survivors (Is-75) which gives the following relative risks:

non-smokers, <1 rad - 1.0

, >200 rad - 3.0

smokers, <1 rad - 6.2

, >200 rad - 8.6

We see that the risk from radiation is (3.0-1.0=)2.0 for non-smokers vs (8.6-6.2=)2.4 for smokers. It may be noted that the A-bomb survivors were followed almost 30 years after irradiation which is much longer than the follow-up on the miner groups.

The male/female ratio for excess lung cancer due to radiation among

non-exposed population, the male/female lung cancer ratio is about 3.0, presumably due to much more cigarette smoking among males. If there were a strong synergism, this ratio would be the same for excess cancers due to radiation. The observed value of unity is what is expected if there is no synergism.

In a nearly lifetime study of Swedish iron miners, Radford and Kinard (Ra-80a) found that the combined effects of smoking and radon exposure are nearly additive.

All of the available evidence summarized above is consistent with a model in which the risks are not very much higher for smokers than for non-smokers but the latent period is considerably longer for the latter. This would explain the observation that the risk for smokers appears to be about five times higher in the early years of follow-up but that after long follow-up the risks for smokers and non-smokers approach equality. It seems unlikely that the long term risk is more than four times as high for smokers than for non-smokers, and probably 2-3 times as high would be a better estimate.

At this point we refer to our previous conclusion that the BEIR-III model over-estimates the risk of environmental radon exposure to <u>non-smokers</u> by at least a factor of 40. The results developed in this Section then imply that the BEIR-III model over-estimates this risk for <u>smokers</u> by at least a factor of 10.

VII. Critique of BEIR-III procedures in estimating lung cancer risks

The available human data on lung cancer risks from radiation are shown in Fig. 1. The ordinate there is in risk/yr-WLM and the groups considered are mostly miners. Since BEIR-III risk estimates are given in age-dependent terms, an age distribution typical of these miner groups was used to determine the BEIR-III point in Fig.1. For similar reasons, the data on Japanese A-bomb survivors was normalized to the number over 20 years old in 1945; the BEIR-III conversion from rad-kerma to WLM was used, including RBE=5 for neutrons; with RBE=10, the point would be lowered by a factor of 1.27. For the ankylosing spondylitis patients and the Newfoundland fluorspar miners, the points given in BEIR-I as well as in BEIR-III are shown; in each of these cases, the doses have been lowered substantially in BEIR-III with no explanation, moving the points upward and to the left as indicated by dashed lines.

BEIR-III increases the values from Se-76 for the Czechoslovakian uranium miners by a factor of 23/13 under the assumption that essentially all of the excess cancers occurred over the last 13 years rather than in the full 23 years of follow-up; these increases* are indicated by vertical dashed lines in Fig. 1 leading to corrected points for exposures below 300 WLM (only these lower exposures were considered in BEIR-III).

One striking observation from Fig. 1 is that the BEIR-III model gives a lung cancer risk more than three times higher than that given by BEIR-I. In this section we look into the reason for this discrepancy and offer evidence that it is part of the explanation for the failures of the model pointed out in earlier sections. In subsection (a) we show that the BEIR-III estimate is

*Since the Czechoslovakian miner data in Fig. 1 were followed for an average of 21.3 years after beginning mining, the increases in Fig. 1 are by a factor 21.3/11.3.

derived almost solely from the data on Czechoslovakian uranium miners. Subsection (b) then shows that these Czechoslovakian data are highly suspect, and subsection (c) offers several other critiques of the data weighting used to derive the BEIR-III model. Subsection (d) then summarizes and draws conclusions.

(a) Source of the BEIR-III estimates

There is no detailed explanation for the derivation of the BEIR-III model given above in Sec. II, but the only applicable age dependence is in the Czechoslovakian miner and A-bomb survivor data. The A-bomb survivor data give values more than a factor of two lower than those used in the BEIR-III model; those aged 50 and above in 1945 were mostly over age 65 during the period of study (1950-74) but their risk was 19 x 10^{-6}/yr-kLM vs 50 x 10^{-6} in the BEIR-III model, and those aged 35-49 in 1945 were nearly all above 50 (and many were above 65) during the study period but their risk was 8.5 x 10^{-6}/yr-kLM vs >20 x 10^{-6} in the BEIR-III model. As noted previously, these discrepancies would be further increased by a factor of 1.27 if the more usual RBE=10 for neutrons had been used.

Clearly then, the BEIR-III model is based almost wholly on the Czechoslovakian miner data of Se-76; indeed it seems to be roughly in agreement with Table 2 of that paper which gives the age dependent results. Note that even the data from Se-76 shown in Fig. 1 lies well below the BEIR-III estimates derived from that same paper. This is explainable by the fact that the data in Fig. 1 are the age-averaged data of Se-76 which appear in Table 1 of that paper, whereas the BEIR-III model is based on Table 2. The difference is that Table 1 is data for the entire group of Czechoslovakian miners, whereas Table 2 is for the sub-group of these miners who began working in 1948-52.

(b) Suspicious aspects of the Czechoslovakian miner data

One suspicious aspect of Table 2 of Se-76 is the remarkable accidental agreements. In the four lowest dose categories of the youngest age group, the rates/WLM are .12±.42, .10±.16, .11±.14, and .13±.05 where ± represent one standard deviation. Each of these four values is far closer to the average of .115 than can reasonably be expected from the large standard deviations. The same thing happens with the other age groups: for the middle age group, the numbers are .10±.77, .18±.30, .22±.24, and .19±.11 giving an average of .17; and for the oldest age group they are .58±1.17, .54±.54, .72±.21, and .59±.12 for an average of .61. In these three sets of four numbers, the average deviation from the average is 0.17 standard deviations, which is about five times smaller than can reasonably be expected. The probability for such small average deviations to occur in all three sets is exceedingly small. It is these three sets of four numbers on which the BEIR-III risk estimate seems to be entirely based.

There are many minor irritants in trying to understand Se-76. They do not give the number of miners in any group or sub-group, they use an unconventional statistical treatment (although they use the conventional one in Ku-79), they do not give age distributions or time distributions for lung cancer, and they do not explain their methodology very completely.

But the most suspicious aspect of the Czechoslovakian data (as well as with some of the others) is that no consideration is given to other possible causes of lung cancer in the Czech mines. There are several carcinogenic agents known to be there (Lo-74, Co-62). These mines were at one time used for extraction of copper, silver, iron, cobalt, arsenic, bismuth, and nickel ores, and three of these — arsenic, nickel, and iron

ores — are believed to cause lung cancer (Col-78). There is a considerable amount of lead sulfide mixed with the uranium ore, and lead salts are also suspected of causing lung cancer. These mines are cool and damp, and wet in some seasons, so that chronic respiratory diseases are very common; there is some evidence that these can develop into lung cancer. The whole region, as well as the mines, are exceptionally dusty, with pneumoconiosis from silica inhalation common; it is possible that this may also lead to lung cancer. There are thus many potential alternative causes of the increased lung cancer among the Czechoslovakian miners. In fact the original reason for studying the U.S. uranium miners was to separate out effects of radon from some of these other carcinogenic agents.

In order to assess the relative roles of radon and other agents in the Czechoslovakian mines, we turn to a detailed examination of the data. The data available on lung cancer incidence as a function of dose and length of employment in the mines are shown in Table 9 as the upper entry in each box, with the central value in the center and 95% confidence limits before and after. These data are also shown in Fig. 2 with separate plots for each duration of employment. The lines on these plots are fits to a linear dose-effect relationship as would be expected if radon were the only cause of lung cancer, and to a dose-independent relationship (horizontal lines) in which the lines in the three plots are constrained to have ordinates in proportion to duration of employment. In spite of this additional constraint — the slopes in the linear dose-effect case were not constrained to be equal in the three plots in deference to the possibility of a dose rate dependence — the fits are as good for the dose independent hypothesis as for the linear dose-effect hypothesis. The 5.6

year data appear to fit the horizontal line better; the 14.0 year data appear to fit the slant line better, but the horizontal line does not come close to the error limit on any point; in the 9.5 year data, the horizontal line approaches the error limit on one point while the slant line approaches it on two points. The fact that the fits are about equally good means that there is as much evidence that radon causes none of the lung cancer as there is that it causes all of the lung cancer as assumed in BEIR-III.

As a further study of this matter, the data were fitted to

$$R = aD + bt \qquad (1)$$

where R is the lung cancer rate listed in Table 9, D is the dose in WLM, t is the length of employment in years (representing length of exposure to some other carcinogen), and a and b are constants to be fit to the data. The optimum fit was found to be

$$a = .075 \text{ cases/WLM } (\times 10^{-3}) \qquad (2)$$

$$b = 6.2 \text{ cases/year of employment } (\times 10^{-3})$$

Since these data are based on 25 years of follow-up, the BEIR-III procedure is to divide by 15 to get cases/year. Thus, the value of a corresponds to $(.075 \times 10^{-3}/15) = 5 \times 10^{-6}$ cases/yr-WLM which is four times lower than the BEIR-III estimate in Fig. 1. This analysis clearly indicates that the disregard of alternative causes of lung cancer is not justified for the Czechoslovakian miners.

(c) Other critiques of data weighting in the BEIR-III model

The only other data in Fig. 1 that give risks comparable to those derived from the Czechoslovakian data are the data from the Swedish Zinkgruvan mines. The reason the risk/WLM for that group is so high is that the radon levels in

those mines are quite low, 0.3-1 WL. Nevertheless, it is assumed that all of the excess lung cancers are due to radon. These mines are known to contain scarn, cummingtonite, and grunerite, fibrous materials akin to asbestos. There are essentially no dose-response data for those miners. It is simply assumed that all excess lung cancers are due to radon.

On the other hand, BEIR-III gives essentially no weight to the data on U.S. uranium miners, although they are probably the largest and most carefully studied group. They are the only group for which there is good information on smoking habits, and they encompass a very wide dose range with generally good statistics. BEIR-III makes a feeble attempt to explain the discrepancy between the U.S. and Czechoslovakian miners as a dose-rate effect, since dose rates were somewhat higher in the U.S. mines. This attempt is based on the fact that protracted doses of high LET radiation have been shown to give higher risks than the same dose administered over a short time period (Ma-78). However, from our understanding of this observation, we would expect the relevant time periods for protraction to be comparable to the period for cell mitosis, and indeed the experimental evidence for increased effectiveness from protraction in humans and in animals is observed to saturate when protraction reaches a few months. The protraction in the case of miners is over many years, so one would not expect a dose rate effect.

The discrepancy between the U.S. and Czechoslovakian miners leads us to the more general question of whether one should tend to accept the higher or lower values when two sets of data are in disagreement. Most of the data in Fig. 1 are based on the assumption that there are no aspects of mining that can cause lung cancer other than radon. This is clearly not the case. As pointed out in Sec. IV, there are many chemical agents that are known to cause lung cancer and some of these like metal ore dusts and diesel engine fumes are very strongly present in most mines. Somewhat elevated lung cancer risks have been reported in various other miner groups (Co-80b). There are many ways in which the mining environment differs from that of the general public (which is used as controls in these studies). It would thus be very easy to explain higher lung cancer rates than those produced by radon. On the other hand, it is very difficult to explain lower rates. It therefore seems appropriate when confronted with two sets of data which do not agree (e.g. U.S. vs Czechoslovakian miners) to give preference to the one showing the lower lung cancer risks. The BEIR-III Report does exactly the opposite; as is evident from Fig. 1, they select data to obtain the highest possible risks consistent with any data.

An entirely different objectionable aspect of the BEIR-III lung cancer model is that it is the only example in BEIR-III of a supra-linear model. As may be seen from Fig. 1, the risks at high doses are well established and clearly far below the predictions of the BEIR-III model (this includes the Czechoslovakian miner data). This cannot be explained as due to cell killing or sterilization because those processes are exponential with dose; for example, if a point at 700 WLM in Fig. 1 is reduced by a factor of 2 due to these processes, points at 2800 WLM and 7000 WLM would be reduced by factors of 2^4=16 and 2^{10}=1024, which is definitely contrary to observation. Thus the BEIR-III model is definitely supra-linear, implying that a linear dose response curve grossly <u>under-estimates</u> effects of low doses. Such supra-linear behavior of a dose-response curve is contrary to the bulk of evidence from other animal and

human data, is contrary to expectations from generally accepted theories, and is contrary to statements in other parts of the BEIR-III Report. On the other hand it is just what is expected if alternative causes of lung cancer among miners are contributing cases — they would not be important at high dose, but they would be important at low dose and greatly increase the number of cases.

One final criticism of the BEIR-III lung cancer estimate is that it uses the results from miners and concludes that they apply to the public with "characteristic smoking experience". However, 70% of both the U.S. and Czechoslovakian miners were smokers, whereas only about 33% of the general population are smokers — 40% of men and about 25% of women. If there is an important difference in the radon risk for smokers and non-smokers, this would make the BEIR-III estimates too high by about a factor of two when applied to the general population.

(d) Conclusions and discussion

Our conclusion from Fig. 1 and the above discussion is that BEIR-III uses a highly questionable data selection procedure and thereby probably overestimates the lung cancer risk to miners from radon by a factor between 2 and 5. In terms of Fig. 1, this risk is therefore probably in the range 5-10 x 10^{-6}/yr-WLM. In addition, the difference in smoking habits between miners and the public may reduce the risk to the public by another factor of two. However this is still more than the upper limit risks due to environmental radon as obtained in previous sections of this paper which are 40 x less than the BEIR-III estimate, or 0.5 x 10^{-6}/yr-WLM for non-smokers, and four times higher than that or 2 x 10^{-6}/yr-WLM for smokers.

This remaining discrepancy would suggest a break-down of the linear dose-response relationship at low doses (Co-60a); i.e., the linear assumption based on observed risks at high doses over-estimates the risks of low doses. Such a deviation from linearity is widely accepted for low LET radiation (NAS-80, NC-80), but has not ordinarily been accepted for alpha particles. However, it is suggested in BEIR-III for alpha particle induced bone cancer based on evidence from the radium dial painters.

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Table 1: Radon levels (in milli-working levels, mWL) in buildings from various U.S. studies (Co-80). Values are averaged over time of day and seasons of the year.

Location	Units	Av. Rn(mWL)	Comments
Florida	16	7	
Tennessee	15	8	
Boston	14	4	basements
	3	0.7	first floor
New York + New Jersey	21	9.8	basements
		5.2	first floor
Chicago	-	7	
	22	30	unpaved crawl space
Grand Jct.,CO	-	7	no U tailings
Florida	-	4	no high U content

Table 2: Lung cancer deaths expected from BEIR-III model for exposure of 0.22 WLM/y

Col.(2) is the number of years before the latent period at the average age of the group. For example at age 37, age 0-12 is before the 25 year latent period for those ages, and age 15-20 is before the 17 year latent period for those ages; hence, the entry is 12 + 5 = 17.
Col.(3) = 0.22 WLM/yr x Col.(2).
Col.(4) is from the BEIR-III Report.
Col.(5) = Col.(3) x Col.(4).
Col.(6) is from U.S. Statistics (HEW-79).
Col.(7) = Col.(5) x Col.(6).
Col.(8) is from U.S. Statistics (Ce-75).
Col.(9) = Col.(5) x Col.(8).

(1) age at death	(2) effective yr of exposure	(3) exposure (WLM)	(4) risk factor	(5) rate ($x10^{-6}$/yr)	(6) population 1976 ($x10^6$)	(7) expected cases 1976	(8) popul. 1910 ($x10^6$)	(9) expected cases 1910
35-39	17	3.7	10	37	11.9	440	6.40	237
40-44	25	5.5	10	55	11.1	610	5.26	289
45-49	30	6.6	10	66	11.7	770	4.47	295
50-54	42	9.2	20	184	12.0	2210	3.90	718
55-64	50	11.0	20	220	20.1	4420	5.06	1113
65-74	60	13.2	50	660	14.2	9370	2.5	1650
>75	70	15.4	50	770	8.7	6700	1.45	1117
					214.6	24520	92.0	5419
					(im.age 0-34)			

1976 Rate (all ages) = $24,520/214.6x10^6 = 114 \times 10^{-6}$/yr

1976 Rate (age >40) = $24,080/77.8x10^6 = 309 \times 10^{-6}$/yr

1910 Rate (all ages) = $5419/92.0x10^{-6} = 59 \times 10^{-6}$/yr

Table 3: Comparison of BEIR-III estimate of lung cancer rates from environmental radon (Col.(5) of Table 1) with total lung cancer rates among non-smokers from various studies.

		Ga-80					
Age	BEIR-III	M	F	M+F	Ha-66	Ka-66	Ha-58
35-44	46	40	10	25	7	0	
45-54	125	50	40	45	42	0	<30
55-64	220	140	90	115	105	100	72
65-74	660	270	170	220	149	300	>340
>75	770	600	380	490	430	460	

Table 4: Lung cancer mortality rates in various countries in the early twentieth century (Co-80a)

Country	years	Rate($\times 10^6$)
England & Wales	1901-10	8.8
Scotland	1906-10	11.6
Ireland	1901-10	3.3
Sweden	1905	1.3
Bavaria	1905-10	7.5
Switzerland	1901-10	6.7
Australia	1908-12	7.0
U.S.	1914	6.0
U.S. - East-North-Central	1914	3.9

Table 5: Percentage distribution of excess lung cancer among U.S. miners by histological type (Ar-74)

dose(WLM)	Type 1	Type 2	Type 3	Other	Total cases
1-359	8.7	76.6	12.2	2.5	10.8
360-1799	31.2	68.1	0.4	0.3	32.4
>1800	21.5	67.7	9.4	1.4	49.8
combined	23.4	68.9	6.6	1.1	92.9

Table 6: Observed/expected ratio for various histological types of lung cancer among Czechoslovakian uranium miners (Ho-77). The number of cases per 100 controls is shown in Line 5. Line 6 shows the additional cases among miners in a population size that would give 100 cases among controls; it is calculated as [(obs/exp)-1]xLine 5. The total additional cases of all types is found to be 370. The last line is the percentage of the 370 which are of each type.

dose (WLM)	Type 1	Type 2	Type 3	other	All types
<200 WLM	2.9	3.6	1.8	3.1	3.0
200-399	3.6	7.7	-	4.3	5.0
>400	9.1	9.2	1.3	3.3	7.5
combined	4.7	6.1	1.2	3.5	4.7
controls per 100 total	34.9	41.6	13.1	10.4	100
additional in miners	129	212	3	26	370
percent of additional	35	57	1	7	100

Table 7: Histological classification of lung cancers among non-smokers in various studies. Cases where different pathologists disagree are included in both types with question marks, and in summing these are multiplied by 1/2.

Ref.	Sex	Total	Type 1	Type 2	Type 3	Type 4	Type 6	anapl. Undif.	other	% Type 2
Wy-54	M	19	10	-	6	-	-	3	-	0
Kr-67	M	16	3	1	5	-	7	-	-	6.3
	F	73	3	3	42	-	25	-	-	4.1
Wy-67	M	8	2	1?	5	-	-	1?	-	6.3
	F	25	3+3?	1	11+5?	1+1?	2+3?	2+1?	-	3.8
Yi-77	M	24	8	3	8	0	3	-	2	13
	F	38	6	1	22	2	3	-	4	2.6
Ye-73	M	7	1	0	4+1?		-	1?	-	0
	F	25	4	2	14+1?	3+1?	-	2	-	7.7
Au-75	M	6	0	0	6	0	0	0	-	0
De-70	F	6	0	0	6	0	0	0	-	0
SUM		249	41.5	11.5	132.5	8	41.5	8.5	6	4.6

Table 8: Lung cancer incidence among U.S. uranium miners as a function of smoking frequency and radon exposure. Entries are cases/1000 person-years with total number of cases in parentheses. From (Ar-76)

cigarette smoking frequency	radon exposure		
	1-359 WLM	360-1799 WLM	>1800 WLM
non-smokers	.20(1)	.86(3)	1.39(2)
<1 pack/day	1.58(5)	1.14(3)	8.3(6)
1 pack/day	1.30(9)	3.52(29)	9.4(30)
>1 pack/day	2.7(8)	4.68(17)	13.3(15)
ex-smokers	2.97(3)	3.26(6)	10.1(9)

Table 9: Lung cancer incidence per 1000 miners (Ku-79) in the group of Czechoslovakian miners who began employment in 1948-52 vs radon dose and length of employment in mines. Upper lines represent the best value, with 95% confidence limits before and after. Lower lines are the two terms calculated from (1) with the least square fit values of a and b given in (2); the first term is the contribution from dose dependence, and the second term is due to dependence on length of employment in mines. Note that their sum is always well within the 95% confidence limits of the observed value.

dose (WLM)	years of employment in mines		
	4-7.9(=6)	8-11.9(=10)	>12(=16)
<100	-10, 12.6, 58 5 + 34 = 39	-21, 22.7, 232 5 + 57 = 62	-
100-199	21, 42.5, 70 11 + 34 = 45	19, 53.6, 106 11 + 57 = 68	-10, 66.0, 293 11 + 91 = 102
200-399	18, 43.0, 78 22 + 34 = 56	74, 107.1, (144) 22 + 57 = 79	67, 112.1, 172 22 + 91 = 113
400-599	-5, 36.4, 126 38 + 34 = 72	65, 110.4, 172 38 + 57 = 95	99, 155.6, 230 38 + 91 = 129
>600	-11, 44.1, 207 54 + 34 = 88	26, 93.0, 207 54 + 57 = 111	115, 189, 290 54 + 91 = 135

Captions for Figures

Fig. 1: Data on radiation induced lung cancer from various sources, and estimates by BEIR-I and BEIR-III. Error bars are one standard deviation. Dashed lines and points connected by them are explained in text.

Fig. 2: Additional lung cancer frequency among Czechoslovakian miners vs cumulative radiation exposure for groups employed 4-8 years (top), 8-12 years (middle), and >12 years. Data are from Ku-79. Horizontal lines represent fit to assumption that agents in the mines other than radon were responsible for all excess cases under the constraint that their effects are proportional to length of employment. Sloped lines represent fits to assumption that all effects were due to radon, with no constraint that they are independent of dose rate. If it were assumed that effects of radiation are independent of dose rate or increase with dose rate, the latter fits would be much poorer.

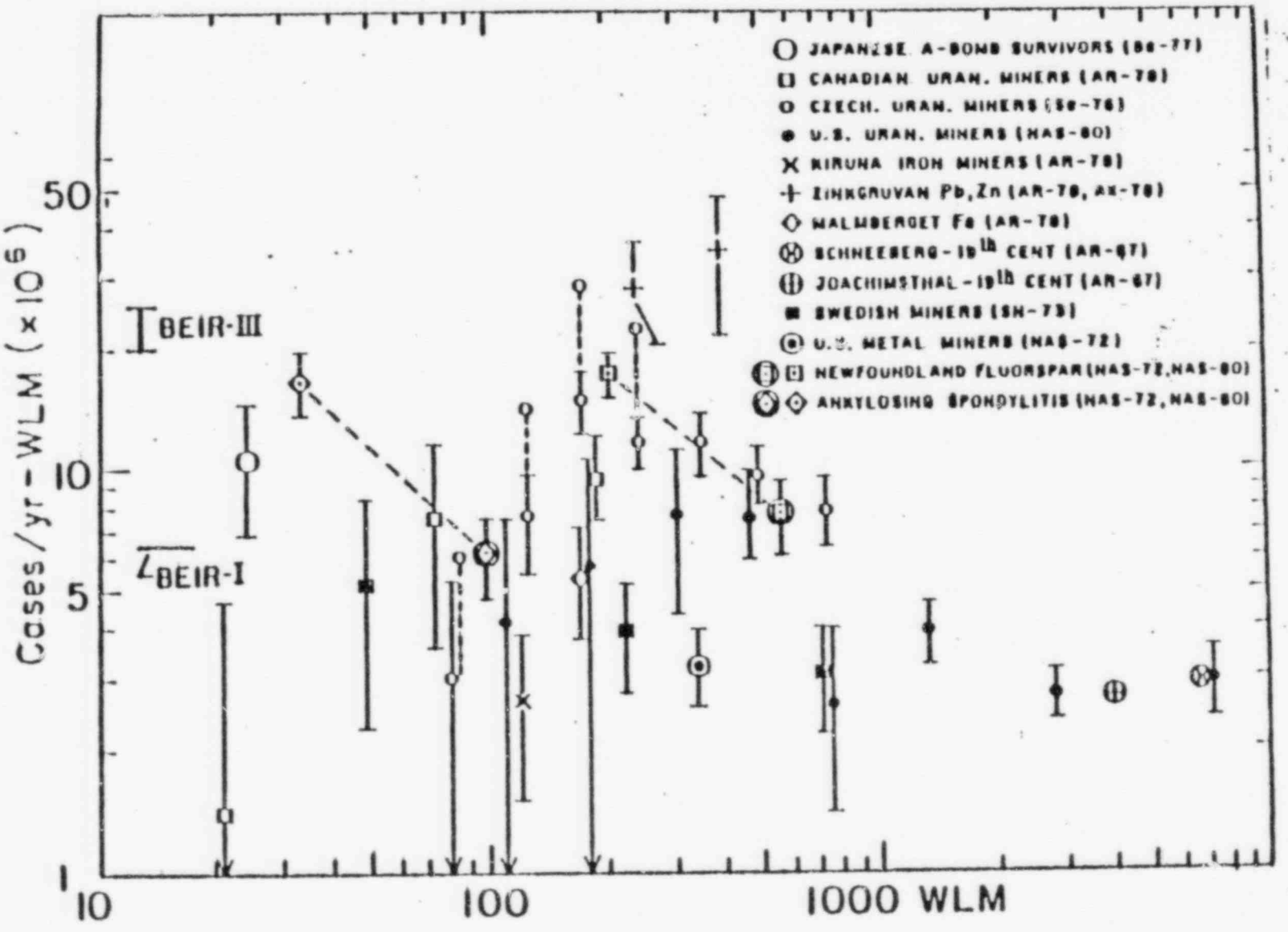
JAPANESE A-BOMB SURVIVORS (58-77)
CANADIAN URAN. MINERS (AR-78)
CZECH. URAN. MINERS (58-76)
U.S. URAN. MINERS (NAS-80)
KIRUNA IRON MINERS (AR-78)
ZINKGRUVAN Pb,Zn (AR-78, AX-78)
MALMBERGET Fe (AR-78)
SCHNEEBERG - 19th CENT (AR-67)
JOACHIMSTHAL - 19th CENT (AR-67)
SWEDISH MINERS (SH-75)
U.S. METAL MINERS (NAS-72)
NEWFOUNDLAND FLUORSPAR (NAS-72, NAS-80)
ANKYLOSING SPONDYLITIS (NAS-72, NAS-80)
BEIR-III
BEIR-I
Cases/yr-WLM (x10⁶)
50
10
5
1
10
100
1000 WLM

Fig. 1

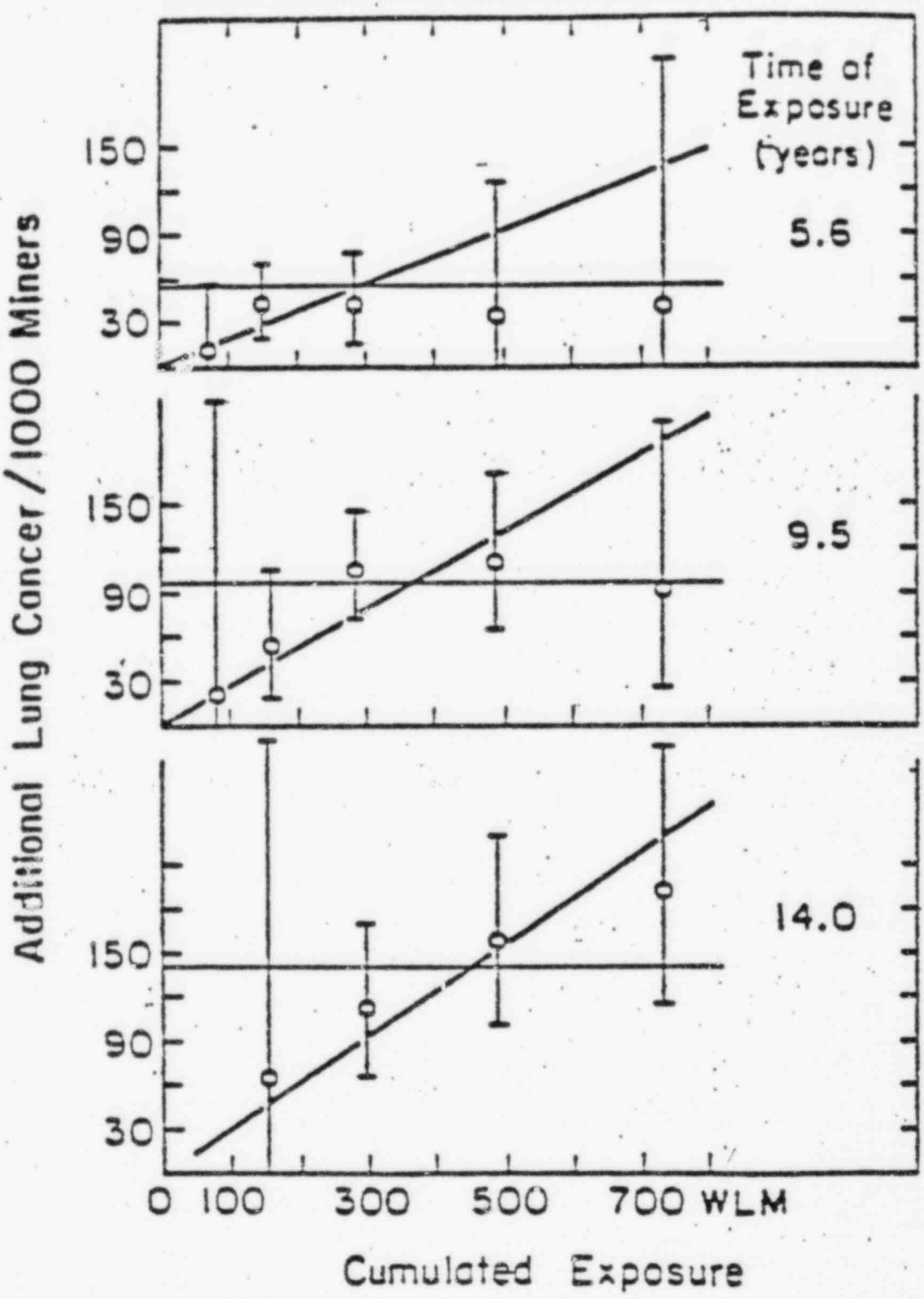
Time of Exposure (years)
5.6
9.5
14.0
150
90
30
Additional Lung Cancer / 1000 Miners
0 100 300 500 700 WLM
Cumulated Exposure

Fig. 2