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Radiation, Cancer, and Mutation in the Atomic Age

ABSTRACT

Following World War II, the publication of accounts such as John Hersey's *Hiroshima* (1946) documented the devastating effects of atomic weaponry on inhabitants of the two Japanese cities targeted by atomic bombs. Yet the American government presented a positive image of the atom's benefits for its citizens in peacetime. In the late 1940s and 1950s, the U.S. Atomic Energy Commission sought to develop nuclear medicine and nuclear energy alongside its continued production and testing of atomic weapons. In both its civilian and military endeavors, the agency maintained that its safety guidelines were sufficient to protect workers and the general population from dangerous exposures to ionizing radiation. In the 1950s, mounting concerns about the hazards of low-level radiation exposure, particularly from atomic weapons fallout, raised the stakes of understanding and protecting against the health hazards of ionizing radiation. Radiological protection guidelines focused on preventing the somatic effects of radiation—leukemia, cancer, and life-shortening—for which experts postulated a safety threshold. By contrast, the genetic effects of ionizing radiation on an individual's fertility and gametes appeared to be dose-dependent even at the lowest levels. Geneticists challenged the perceived gap between somatic and genetic effects of radiation by arguing that radiation-induced mutations play a role in diseases, especially cancer. In doing so, they also contested the Commission's portrayal

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The following abbreviations are used: ABCC, Atomic Bomb Casualty Commission; ACBM, Advisory Committee on Biology and Medicine; AEC, U.S. Atomic Energy Commission; BEAR, Biological Effects of Atomic Radiation; DOE, Department of Energy; Evans Papers, Robley Duglinson Evans Papers, MC 80, Massachusetts Institute of Technology, Institute Archives and Special Collections, Cambridge, MA; *JHB*, *Journal of the History of Biology*; NARA, U.S. National Archives and Research Administration, College Park, MD; NAS, National Academy of Sciences; NCRP, National Committee on Radiation Protection and Measurements; NRC, National Research Council; OpenNet, DOE OpenNet database of declassified documents, identified here by accession number, available at https://www.osti.gov/opennet/; RG, Record Group.

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of a safe atomic future. This article examines how concerns over low-level radiation from fallout facilitated acceptance of the then-controversial somatic mutation theory of carcinogenesis, which became an enduring feature of cancer biology.

KEY WORDS: U.S. Atomic Energy Commission, Advisory Committee on Biology and Medicine, genetics, mutation, cancer, radioactive fallout

This article grows out of a question that puzzled me while doing research on the Atomic Energy Commission's postwar provision of radioisotopes to scientists and physicians: why was it that not until the fallout debates did the agency, and indeed many scientists, begin to take seriously the hazards of low-level radiation, although geneticists had been warning of them for decades? After all, H. J. Muller had demonstrated in 1927 that X-rays could induce mutations in fruit flies, and that the mutation rate was linear with dose.[1] This finding was rapidly extended to other forms of radiation as well as to other organisms.[2] Radiation had also been correlated with the appearance of cancer (especially leukemia) from the 1920s and 1930s, most tragically in the radium dial painters.[3] However, these two observations were rarely linked, as both scientists and safety officials treated "genetic" effects—mutations—as separate from "somatic" ones. In effect, this paper addresses the closing of that gap in the 1950s.

The conceptual distinction between the genetic and somatic effects of radiation had a pragmatic underpinning. In the 1920s, radiation safety guidelines were set by a body of radiologists, the International Commission on X-Ray and Radium Protection, and subsequently by an American body, the Advisory Committee on X-Ray and Radium Protection (later renamed the National Committee on Radiation Protection and Measurements). Before World War II, the "tolerance dose" set by these groups guided industry in controlling occupational radiation exposure, and subsequently provided the basis of radiological safety in the Manhattan Project.[4] These expert bodies

1. H. J. Muller, "Artificial Transmutation of the Gene," *Science* 66 (1927): 84–87.

2. Indeed, some of these studies of radiation-induced mutations preceded Muller's. Luis A. Campos, "Radium and the Secret of Life" (PhD dissertation, Harvard University, 2006).

3. Claudia Clark, *Radium Girls: Women and Industrial Health Reform, 1910–1935* (Chapel Hill: University of North Carolina Press, 1997).

4. Daniel Paul Serwer, "The Rise of Radiation Protection: Science, Medicine and Technology in Society, 1896–1935" (PhD dissertation, Princeton University, 1977); Barton C. Hacker, *The Dragon's Tail: Radiation Safety in the Manhattan Project, 1942–1946* (Berkeley: University of California Press, 1987); J. Samuel Walker, *Permissible Dose: A History of Radiation Protection in the*

approached radiation as a toxicological problem, assuming the existence of a threshold below which exposure would not be harmful. By contrast, geneticists did not find evidence of a threshold for the genetic effects of radiation—namely, mutations. Ionizing radiation seemed to induce mutations in proportion to dose, even at the lowest levels of exposure, although the population-wide effects of such mutations remained an area of debate.[5]

Until the 1950s, physicians and health physicists tended to hold key Atomic Energy Commission (AEC) positions and articulate its policy. However, over the course of that decade geneticists began to influence public opinion and scientific policy about radiation's hazards, shifting emphasis away from acute effects to long-term consequences. Muller had suggested in 1948 that radiation-induced mutations in somatic cells might be responsible for malignancies, and this idea, picked up by others, gained traction over the 1950s and 1960s.[6] This notion subverted the AEC's categorical distinction between somatic and genetic effects, and it also focused concern on the carcinogenic potential of radioactive fallout products from atomic weapons testing, such as strontium-90 and iodine-131.[7] If there were no threshold for damage to genes, then even minute amounts of these contaminants could induce cancer-causing mutations.

This article re-examines how geneticists successfully contested the AEC's official views concerning the relative safety of low-level radiation. As other scholars have noted, the National Academy of Sciences' Committee on the Biological Effects of Ionizing Radiation provided an especially important platform for prominent geneticists to advance their concerns, even as this required

Twentieth Century (Berkeley: University of California Press, 2000); Soraya Boudia, *Gouverner les risques, gouverner par le risque: Pour une histoire du risque de la société du risque* [Governing Risk, Governing Through Risk: Risk and Risk Society in Historical Perspective] (Habilitation à diriger des recherches, Université de Strasbourg, 2010).

5. John Beatty, "Weighing the Risks: Stalemate in the Classical/Balance Controversy," *JHB* 20 (1987): 289–319.

6. H. J. Muller, "Some Present Problems in the Genetic Effects of Radiation," *Symposium on Radiation Genetics*, *Journal of Cellular and Comparative Physiology* 35, suppl. 1 (1950): 9–70, on 56. This paper was presented at the Symposium on Radiation Genetics, sponsored by the Biology Division of Oak Ridge National Laboratory, Oak Ridge, Tennessee, 26–27 Mar 1948.

7. Robert A. Divine, *Blowing on the Wind: The Nuclear Test Ban Debate, 1954–1960* (New York: Oxford University Press, 1978), chap. 10; Carolyn Kopp, "The Origins of the American Scientific Debate over Fallout Hazards," *Social Studies of Science* 9 (1979): 403–22; J. Christopher Jolly, "Thresholds of Uncertainty: Radiation and Responsibility in the Fallout Controversy" (PhD dissertation, Oregon State University, 2003); Alison Kraft, "Scientists in Cold War Britain, the Nuclear Fallout Issue and the Origins of Pugwash, c. 1954–1957," *Journal of Cold War Studies* 17, no. 4 (2015).

them to set aside their strong disagreements about the population-wide consequences of an increased mutation rate.[8] Geneticists were effectively able to address "somatic effects" by expanding the potential consequences of radiation-induced mutation to include cancer, despite the fact that this theory of carcinogenesis was controversial among pathologists, oncologists, and health physicists.[9] Their linear, no-threshold model for radiation effects left no scope for viewing low-level exposures as benign. The somatic mutation theory of cancer also turned the politics of this dread disease on its head, as the fear of cancer had been exploited in the 1940s by the AEC in their plans to make available reactor-generated radioisotopes and new radiation sources to improve the health of citizens. By the 1970s, the growing scientific and public concerns about the health risks of exposure to low-level radiation, particularly from environmental contamination, threatened not only the acceptability of above-ground nuclear weapons testing (which was banned in the 1963 Test Ban Treaty), but also the major civilian benefit the AEC was then promoting, namely nuclear power.[10]

ATOMIC ENERGY AS A TOOL FOR FIGHTING CANCER

The U.S. government was surprisingly unprepared for the devastating effects of radiation from its use of atomic weapons in Japan. The military expected that, as Norman Ramsey put it, "any person with radiation damage would have been killed with a brick first."[11] The distinctive and disturbing effects of

8. Jolly, "Thresholds of Uncertainty" (ref. 7); James F. Crow, "Quarreling Geneticists and a Diplomat," *Genetics* 140 (1995): 421–26; John Beatty, "Masking Disagreement among Experts," *Episteme* 3 (2006): 52–67; Jacob Darwin Hamblin, "'A Dispassionate and Objective Effort': Negotiating the First Study on the Biological Effects of Atomic Radiation," *JHB* 40 (2007): 147–77. As the National Academy of Sciences began limiting access to archival files reflecting their committees' deliberations, I could not review the documents that these authors cite when I visited the archives in fall 2013.

9. Jolly, "Thresholds of Uncertainty" (ref. 7), chap. 12. For a broader perspective on debates over cancer causation, see Robert N. Proctor, *Cancer Wars: How Politics Shapes What We Know & Don't Know About Cancer* (New York: Basic Books, 1995).

10. J. Samuel Walker, *Containing the Atom: Nuclear Regulation in a Changing Environment, 1963–1971* (Berkeley: University of California Press, 1992).

11. Norman F. Ramsey, *The Reminiscences of Norman F. Ramsey*, 1962, *Columbia University Oral History Microfiche Collection* (Westport, CT: Meckler, 1985), 166–67, as quoted by Michael D. Gordin, *Five Days in August: How World War II Became a Nuclear War* (Princeton, NJ: Princeton University Press, 2007), 54. On the lack of communication through government channels about known hazards of radiation, see Sean Malloy, "'A Very Pleasant Way to Die':

atomic weaponry were documented by the military's Joint Commission for the Investigation of the Effects of the Atomic Bomb in Japan and subsequently by the Atomic Bomb Casualty Commission.[12] But the American government did not widely disseminate this information, and Japanese accounts were censored, though some journalists did document in the American press the peculiar suffering of atomic bomb survivors.[13]

It was not only a question of the suppression of information. Atomic energy had already developed a different, more promising image, one that the U.S. government reinforced after the war. Although the dangers of radiation had been documented since the earliest days of X-rays and radium use, so had its power to treat disease, especially cancer. Postwar optimism about the medical uses of radioisotopes and neutron sources drew on decades of efforts to harness the therapeutic promise of radioactivity, most notably by the Curies. In the 1930s, cyclotrons supplied the growing demand for artificial radioisotopes, especially phosphorus-32 and iodine-131, both of which were being used clinically.[14]

The nuclear reactors build for the bomb project, in comparison to cyclotrons, could produce radioactive materials on an industrial scale. In an attempt to cast a positive light on the terrible new technology of destruction, politicians tapped the popular expectation that after Americans achieved victory in the Pacific, atomic energy could vanquish cancer. (See fig. 1.) On May 15, 1947, during budget hearings, Representative Everett M. Dirksen argued that the newly founded AEC should have a $25,000,000 cancer program. Cancer killed "one person every 3 minutes," as he asserted; this amounted to "Seventy-Two Pearl Harbors Every Year."[15] It seemed especially fitting to Dirksen that the

Radiation Effects and the Decision to Use the Atomic Bomb against Japan," *Diplomatic History* 36 (2012): 515–45.

12. John Beatty, "Genetics in the Atomic Age: The Atomic Bomb Casualty Commission, 1947–1956," in *The Expansion of American Biology*, ed. Keith R. Benson, Jane Maienschein, and Ronald Rainger (New Brunswick, NJ: Rutgers University Press, 1991), 284–324; M. Susan Lindee, *Suffering Made Real: American Science and the Survivors at Hiroshima* (Chicago: University of Chicago Press, 1994).

13. Most notably John Hersey, *Hiroshima* (New York: A. A. Knopf, 1946).

14. J. L. Heilbron and Robert W. Seidel, *Lawrence and His Laboratory: A History of the Lawrence Berkeley Laboratory* (Berkeley: University of California Press, 1989). On the Curies and radiation therapy, see Soraya Boudia with Dominique Pestre, *Marie Curie et son laboratoire: Sciences et industrie de la radioactivité en France* (Paris: Éditions des archives contemporaines, 2001).

15. Statement of Everett M. Dirksen, 15 May 1947, in U.S. Congress, House, *Independent Offices Appropriation Bill for 1948, Hearings*, 80th Congress, 1st sess. (Washington, DC: U.S.

FIG. 1. Political cartoon from the *Dallas Morning News*, 12 Aug 1945, depicting the use of atomic energy to destroy cancer, only days after atomic weapons were detonated over Hiroshima and Nagasaki. Reprinted with permission of the *Dallas Morning News*.

successor to the Manhattan Project should take on the cancer problem: "If we are going to spend a few hundred million dollars in the atomic-energy field to perfect an instrumentality of death, then let us take a little of that money to develop an instrumentality to preserve life."[16] At the end of the appropriations

Government Printing Office, 1947), 1539. The AEC's proposed fiscal year 1948 budget was $500,000,000.

16. Ibid., 1540.

process, Congress earmarked $5 million for the AEC's efforts in cancer research in 1948.[17]

The AEC had not asked for this programmatic responsibility; indeed, the Commissioners had expressed outright skepticism about its wisdom. Yet as it turned out, the Congressional cancer appropriation offered the agency an immediate opportunity to demonstrate the medical benefits of atomic energy. The political value of this became evident as early as the summer of 1947, as the AEC's General Advisory Committee, led by J. Robert Oppenheimer, informed the Commission that their expectations for the quick inception of domestic atomic power were completely unrealistic.[18] To provide expert guidance on applications of atomic energy to health and life science, the Commission appointed an Advisory Committee on Biology and Medicine (ACBM), chaired by Alan Gregg of the Rockefeller Foundation.[19] In addition, the agency founded a Division of Biology and Medicine, whose director would report directly to the Commission and General Manager.[20] In October 1947, Shields Warren was appointed Director of this Division, which was responsible for radiological safety at AEC installations as well as the agency's biomedical research program. A professor of pathology at Harvard Medical School, Warren had previously served on the Interim Medical Advisory Committee and headed the Navy's medical survey team of Hiroshima and Nagasaki.[21]

Relying on the ACBM for input, the AEC began designing a cancer program in line with the Congressional appropriation, one that did not duplicate the efforts of other government agencies.[22] Formally accepted by the

17. Stuart M. Feffer, "Atoms, Cancer, and Politics: Supporting Atomic Science at the University of Chicago, 1944–1950," *HSPS* 22, no. 2 (1992): 233–61, on 258. See also Peter J. Westwick, *The National Labs: Science in an American System, 1947–1974* (Cambridge, MA: Harvard University Press, 2003), chap. 7.

18. A version of this report was published in AEC, *Recent Scientific and Technical Developments in the Atomic Energy Program of the United States* (Washington, DC: U.S. Government Printing Office, 1948), 43–46. See also Brian Balogh, *Chain Reaction: Expert Debate and Public Participation in American Commercial Nuclear Power, 1945–1975* (Cambridge: Cambridge University Press, 1991), 83.

19. The ACBM replaced a temporary Medical Review Board. Summary of the 68th AEC Meeting, 25 Jun 1947, NARA, RG 326, E67A, Box 23, Folder 7 Advisory Committee on Biology and Medicine.

20. Westwick, *National Labs* (ref. 17), 247.

21. AEC Press Release No. 64, "Dr. Shields Warren Appointed Interim Director of Biology and Medicine," 24 Oct 1947, NARA, RG 326, E67A, Box 23, Folder 8, Division of Biology and Medicine—Organization and Functions.

22. This was a stipulation of the legislation authorizing the cancer research budget. On the role of the ACBM in planning the cancer program, see AEC Press Release No. 55, "United States

Commission in January 1948, the AEC's cancer program involved three major activities, two of which committed funding to programs already in place.[23] First was supporting the work of the Atomic Bomb Casualty Commission (ABCC), whose studies of the medical consequences of the bombings of Hiroshima and Nagasaki included assessment of cancer incidence in survivors.[24] Second, the agency would make certain radioisotopes already available for sale from Oak Ridge free of charge for cancer research, diagnosis, and therapy. Beginning in April 1948, Oak Ridge offered radiosodium, radiophosphorus, and radioiodine without charge (save shipping) for uses related to cancer. Early in 1949, the program was expanded to include all radioisotopes; by August of that year, the cancer program accounted for over 2,000 shipments of isotopes.[25] The third part of the cancer program involved the AEC in experimental cancer therapy, which required new organization and infrastructure.[26] The agency set up a clinical cancer research unit at the Oak Ridge Institute of Nuclear Studies, and built a fifty-bed Argonne Cancer Hospital in Chicago.[27] The Commission anticipated that the hospital would be "primarily for research in which radioisotopes will be used to treat cancer patients."[28]

Atomic Energy Commission Names Advisory Committee for Biology and Medicine," 12 Sep 1947, NARA, RG 326, E67A, Box 23, Folder 7, Advisory Committee on Biology and Medicine.

23. A Plan for a Cancer Research Program for the Atomic Energy Commission, Report by the Director of the Division of Biology and Medicine, Atomic Energy Commission, 5 Jan 1948, OpenNet NV0702018; AEC 26, Cancer Research Program for the Atomic Energy Commission, 30 Jan 1948, NARA, RG 326, E67A, Box 64, Folder 5, Research in Biological and Medical Science.

24. The AEC was already funding the ABCC year to year. Beatty, "Genetics in the Atomic Age" (ref. 12); Lindee, *Suffering Made Real* (ref. 12). On the radioisotope distribution program, see Timothy Lenoir and Marguerite Hays, "The Manhattan Project for Biomedicine," in *Controlling Our Destinies: Historical, Philosophical, Ethical, and Theological Perspectives on the Human Genome Project*, ed. Philip R. Sloan (Notre Dame, IN: University of Notre Dame Press, 2000), 29–62; Angela N. H. Creager, *Life Atomic: A History of Radioisotopes in Science and Medicine* (Chicago: University of Chicago Press, 2013).

25. "AEC Distributes 8,363 Shipments of Radioactive and Stable Isotopes in Three Years," NARA, RG 326, E67A, Box 45, Folder 13, Distribution of Stable Isotopes Domestic.

26. David E. Lilienthal to Bourke B. Hickenlooper, 15 Dec 1947, Appendix D to AEC 26, Cancer Research Program for the Atomic Energy Commission, NARA, RG 326, E67A, Box 64, Folder 5, Research in Biological and Medical Science.

27. David E. Lilienthal to Alan Gregg, 9 Jul 1948, NARA, RG 326, E67A, Box 64, Folder 5, Research in Biological and Medical Science.

28. AEC, *Atomic Energy and the Life Sciences* (Washington, DC: U.S. Government Printing Office, 1949), 91. I offer more detail on the cancer research unit at Oak Ridge in Creager, *Life Atomic* (ref. 24), chap. 9.

Popular coverage of the AEC's cancer program focused on the expectation that radioisotopes administered to cancer patients would localize to specific tumors and deliver internal radiation. One sensational case, reported in 1947, reinforced this hope. A patient diagnosed with adenocarcinoma of the thyroid was treated with iodine-131. The radioactive iodine was localized to several metastatic tumors, both making the many tumors detectable but also, over time, shrinking their size.[29] As Alfred Maisel put it in *Collier's*: "the case of Mr. B is one of the most hopeful things that have hit the medical world in a long, long time. For it demonstrates two uses of atomic energy—as tracer detectives and as interbody medical bullets—that are finding increasing application in scores of ways in laboratories and hospitals all over the United States."[30] The article was illustrated by the striking composite image of a man in pajamas and robe arising from a wheelchair, encircled by a healing mushroom cloud. During the early years of the AEC under the leadership of David Lilienthal, when its status as a civilian agency was challenged by those who felt control of atomic energy should rest in the hands of the military, touting the humanitarian side of the atom was politically valuable.[31]

David Bradley, author of *No Place to Hide*, blamed faith in the therapeutic promise of the atom for the blasé attitude about radioactive contamination following the 1946 test explosions in the Pacific:

> We were surprised at first to find so little interest in the Bikini tests. But we really had no right to be. Atomic energy was an uncomfortable subject. Things like John Hersey's *Hiroshima* were rough. How much more pleasant to consider the coming miracles of healing, the prolongation of life, the days of sunny leisure which people were everywhere promising.[32]

Some did voice concern about the dangerous effects of radiation. In 1947, Paul Henshaw, an officer of the Supreme Command Allied Power in Japan, wrote a popular article, "Atomic Energy: Cancer Cure . . . or Cancer Cause?"[33]

29. S. M. Seidlin, L. D. Marinelli, and Eleanor Oshry, "Radioactive Iodine Therapy: Effect on Functioning Metastases of Adenocarcinoma of the Thyroid," *Journal of the American Medical Association* 132 (1946): 838–47.

30. Albert Q. Maisel, "Medical Dividend," *Collier's* 119 (3 May 1947): 14, 43–44, on 43.

31. Richard G. Hewlett and Francis Duncan, *Atomic Shield: A History of the United States Atomic Energy Commission, vol. 2, 1947–1952* (Berkeley: University of California Press, 1990). An excellent portrait of Lilienthal is offered by Alex Wellerstein, "Knowledge and the Bomb: Nuclear Secrecy in the United States, 1939–2008" (PhD dissertation, Harvard University, 2010), chap. 7.

32. David Bradley, *No Place to Hide* (Boston: Little, Brown & Company, 1948), 167–68.

33. Paul S. Henshaw, "Atomic Energy: Cancer Cure . . . or Cancer Cause?" *Scientific Illustrated* 2 (1947): 46–47, 84.

Strikingly, however, his main concern about cancer was in Japanese survivors (and future atomic warfare survivors), whose burns "may someday turn malignant."[34] A related concern was that "cell damage resulting from high-energy radiation" might cause cancer. Low-energy radiation was off the hook—as the article stated, "We know now that exposures less than one roentgen a day are safe."[35]

RADIATION SAFETY AND HEREDITY

At issue in assessments of the dangers of radioactive fallout from peacetime atomic tests—or, for that matter, from the prospect of nuclear war—was which specific health effects counted as serious hazards. Early radiation protection standards were designed to prevent any detectable short-term effects from exposure. As radiological physicist Gioacchino Failla put it in 1932, "We may now define the safe upper limit of the tolerance dose or simply the 'tolerance' dose as that dose of radiation which experience has shown to produce no permanent physiological changes in the average individual."[36] However, after World War II, in recognition of a growing consensus that exposure below a certain limit might not be harmless, the National Committee on Radiation Protection and Measurements (NCRP) renamed their recommended radiation exposure limit the "maximum permissible dose."[37]

The NCRP tended to focus on the so-called somatic effects of radiation—the direct consequences of exposure on bodily health. In addition to radiation burns and other acute effects of exposure, radiation had also been correlated with the appearance of tumors and leukemias (or blood dyscracias, as they were often called) from early observations of the consequences of radium and X-ray

34. This view reflects an inflammation theory of carcinogenesis going back to the nineteenth century; Axel Schmidt and O. F. Weber, "In Memoriam of Rudolf Virchow: A Historical Retrospective Including Aspects of Inflammation, Infection and Neoplasia," in *Infection and Inflammation: Impacts on Oncogenesis*, ed. T. Dittmar, K. S. Zaenker, and A. Schmidt (Basel: Karger, 2006), 1–15.

35. Henshaw, "Atomic Energy" (ref. 33), 47.

36. As quoted in Robert S. Stone, "The Concept of a Maximum Permissible Exposure," *Radiology* 58 (1952): 639–61, on 642.

37. Walker, *Permissible Dose* (ref. 4), 10–11. Gilbert F. Whittemore Jr., "The National Committee on Radiation Protection, 1928–1960: From Professional Guidelines to Government Regulation" (PhD dissertation, Harvard University, 1986).

exposure in the first three decades of the twentieth century.[38] But there was another class of effects, those on one's offspring, and setting a "permissible dose" for these genetic effects proved elusive. In contrast to the framework that guided radiation safety standards, geneticists saw no basis for recognizing a lower threshold for the mutational effects of radiation, which seemed roughly proportional to dose even at low levels.[39] Well into the 1950s, there remained a gap—partly disciplinary, partly conceptual—between this genetic perspective and that of the health physicists who formulated and implemented radiological protection for the AEC.

After World War II, the question arose of whether genetic effects should influence the setting of "permissible dose" levels for ionizing radiation.[40] In 1949, Robley Evans, a physicist known for his studies of the radium dial painters, published an article entitled "Quantitative Inferences Concerning the Genetic Effects of Radiation on Human Beings."[41] Evans's analysis suggested that exposure at or under the U.S. government's permissible dose level would not significantly increase the mutation rate beyond its spontaneous level.[42] A March 2, 1949, press release from the Science Service presented Evans's article as a challenge to Muller's public pronouncements that "that human beings are now in danger of acquiring harmful hereditary changes from peacetime exposures to atomic radiation and some kinds of X-rays."[43] The key point of dispute was whether radiation exposure from atomic energy installations (or, for that matter, clinical applications of radiomaterials) significantly

38. Similar observations were made with X-ray exposure; there were 94 cases of X-ray-induced skin cancer in the medical literature by 1911. On this and on early observations of radium-induced cancer, see Ronald L. Kathren, "Pathway to a Paradigm: The Linear Nonthreshold Dose-Response Model in Historical Context," *Health Physics* 70 (1996): 621–35.

39. L. C. Dunn, *A Short History of Genetics: The Development of Some of the Main Lines of Thought: 1864–1939* (Ames: Iowa State Press, 1991 [1965]), 169.

40. Jolly, "Thresholds of Uncertainty" (ref. 7), chaps. 3 and 4.

41. Robley D. Evans, "Quantitative Inferences Concerning the Genetic Effects of Radiation on Human Beings," *Science* 109 (1949): 299–304.

42. Evans' estimates of radiation-induced mutation rates appear to have been based on "Genetic Effects of Irradiation with Reference to Man," by D. G. Catcheside (2 Jun 1947), and "Tolerance Doses in Relation to Genetic Effects of Radiation" (28 Apr 1947), a memorandum by D. E. Lea; both of these were prepared for the Tolerance Dose Panel of the Protection Sub-Committee of the Medical Research Council, and manuscripts of them, along with Evans's notes, are in the Evans Papers, Box 1, Folder Genetics #1.

43. Frank Thone, Science Service Biology Editor, "Long-Range Debate Stated on Genetic Effects of Radiation, 3/2/49," Evans Papers, Box 1, Folder Genetics #1. For an example of media coverage of Muller's views, see "Radioactive Rays Held Peril to Race: Dr. H. J. Muller, Nobel Prize Winner, Warns of Exposure Changing Germ Cells," *New York Times*, 2 Apr 1947, 38.

increased the baseline mutation rate in humans. As Evans put it in a letter, "Everyone admits that a small amount of genetic change is always induced by radiation. However, one must remember that spontaneous genetic mutations are taking place all the time anyhow. I have felt that the important point to be emphasized is the ratio between induced and spontaneous mutations."[44]

Evans's figure for the spontaneous mutation rate in humans had been extrapolated from the incidence of rare genetic diseases. He calculated a "doubling dose," that is, the radiation level that would cause a doubling of the spontaneous mutation rate, of 300 roentgens per individual per generation.[45] The maximum permissible radiation dose used by the AEC was 0.1 roentgen per workday. A worker who was exposed to this level each day might accumulate 250 roentgens over a ten-year period, still below the doubling dose.[46] In reality, few workers were exposed to the maximum permissible levels, so Evans argued that the induced mutation rate from occupational radiation exposure would be low, practically negligible.

Evans circulated his manuscript to over thirty scientists, including geneticists as well as health physicists and radiologists. Among those who received it were many AEC-related scientists, including Gioacchino Failla, Simeon Cantril, Austin Brues, Robert Stone, John Lawrence, Joseph Hamilton, Shields Warren, Stafford Warren, Bertram Low-Beer, Lauriston Taylor, and Karl T. Compton, and geneticists D. G. Catcheside, Curt Stern, George Snell, and H. J. Muller.[47] The initial response to Evans's manuscript was largely positive. Alfred Marshak thought perhaps he should emphasize that mutation was not the only deleterious effect of ionizing radiation (pulling attention back to somatic effects), and Low-Beer wondered if Evans should have taken into account effects on plasmagenes as well as nuclear genes.[48] But neither disagreed with Evans's interpretation. Even so, not everyone was satisfied with existing safety regulations. Based on considerations of genetics and cancer risk, Merle Tuve and Paul Aebersold both expressed a preference for a tolerance

44. Robley D. Evans to E. E. Stanford, 3 Jun 1949, Evans Papers, Box 1, Folder Genetics #1.

45. Evans did not originate the doubling dose; underlying it was the widespread assumptions that a doubling of the natural mutation rate would not cause significant injurious effects in a population. Jolly, "Thresholds of Uncertainty" (ref. 7), 95.

46. Evans, "Quantitative Inferences" (ref. 41), 302.

47. The complete list is in the Evans Papers, Box 1, Folder Genetics #1.

48. Alfred Marshak to Robley D. Evans, 7 Mar 1949, and B.V.A. Low-Beer to Evans, 23 Feb 1949, Evans Papers, Box 1, Folder Genetics #1. For more on cytoplasmic heredity and plasmagenes, see Jan Sapp, *Beyond the Gene: Cytoplasmic Inheritance and the Struggle for Authority in Genetics* (New York: Oxford University Press, 1987).

dose of 0.01 roentgen per day instead of the current limit of 0.1 roentgen per day.[49]

Curt Stern liked the paper, as did Donald Charles and George Snell (though he felt the probability of radiation-induced chromosomal translocations should also be considered).[50] James Neel commended Evans for doing "a very real service" in tackling the problem, although he added that "we may not see eye to eye on certain of your basic assumptions."[51] But not all geneticists were so conciliatory. Muller sent a long letter full of criticisms—or as Evans put it, "a few points of scientific interest, and many matters regarding personalities and prejudices."[52] Muller discounted the reliability of the geneticists (particularly D. G. Catcheside) whose numbers Evans relied upon, and accused both persons working for the AEC and radiologists as having a vested interest in minimizing the genetic dangers of radiation in the eyes of the public.[53] His disagreement with Evans is not surprising, given the divergence in their perspectives on whether the increase in mutations from artificial radiation (from both military and civilian uses) was a cause for concern. That Evans sought Muller's views on the manuscript at all is more surprising. In the margins of the press release on the physicist's 1949 paper, next to a paragraph detailing Muller's criticisms of the preprint, Evans wrote, "an' that ain't all sister!"[54]

According to Evans, in a discussion with Sewall Wright at the University of Chicago late in 1948, Wright initially agreed with him that an exposure of 0.1 roentgen per day could be considered safe from a genetic standpoint.[55] Whereas Muller contended that a large segment of the population would suffer genetic damage from occupational and medical exposure to radiation, Wright was not worried about such population-wide effects.[56] But after the paper was

49. M. A. Tuve to Robley Evans, 2 Jan 1940, and Paul C. Aebersold to Robley D. Evans, 9 Feb 1940, both in Evans Papers, Box 1, Folder Genetics #2.

50. On Stern's appraisal, see Robley D. Evans to B.V.A. Low-Beer, 2 Mar 1949, Evans Papers, Box 1, Folder Genetics #1; Donald R. Charles to Robley D. Evans, 23 Sep 1948, Evans Papers, Box 1, Folder Genetics #1; George D. Snell to Robley D. Evans, 4 Feb 1949, Evans Papers, Box 1, Folder Genetics #2.

51. James V. Neel to Robley D. Evans, 6 Apr 1949, Evans Papers, Box 1, Folder Genetics #2.

52. Robley D. Evans to B.V.A. Low-Beer, 2 Mar 1949, Evans Papers, Box 1, Folder Genetics #1.

53. H. J. Muller to Robley D. Evans, 5 Feb 1949, Evans Papers, Box 1, Folder Genetics #2.

54. Written in margin of Thone, "Long-Range Debate" (ref. 43).

55. Robley D. Evans to J. O. Hirsenfelder, 22 Jul 1949, Evans Papers, Box 1, Folder Genetics #1.

56. Jolly, "Thresholds of Uncertainty" (ref. 7), 90–93; Muller, "Some Present Problems" (ref. 6). As Jolly notes, Muller's idea of "genetic death," introduced in his comment following Wright's paper, was developed more fully in H. J. Muller, "Our Load of Mutations," *American Journal of Human Genetics* 2 (1950): 111–76. The disagreement among geneticists over the consequences of

published, Wright challenged Evans's calculations as they pertained to effects on individuals.[57] As Wright noted, Evans's benchmark for the spontaneous generation rate in humans was the occurrence of two rare but devastating hereditary medical conditions—hemophilia and epiloia.[58] Their incidence was used to calculate a rate of 10^{-5} mutations per gene per generation. Experiments with *Drosophila*, however, had demonstrated that not all mutations were as deleterious as the two pathologies Evans selected, in which case his estimate was far too high. Extrapolating instead from the spontaneous mutation rate determined in experiments with *Drosophila* resulted in an estimate of 10^{-7}. As Wright observed, reducing the spontaneous mutation rate by two orders of magnitude also decreased the doubling dose Evans had calculated at 300 roentgens to a much more worrisome level of only 3 roentgens. If the lower spontaneous rate was correct for humans, Wright argued, then exposures within the permissible dose could, in fact, alter the incidence of mutations significantly in the offspring and descendants of the exposed person's children (but perhaps not detectably, as most mutations are recessive).[59]

This concern about radiation's mutational effects soon surfaced in discussions among military leaders about whether the government should publicize scientific views about genetic hazards as part of civil defense information. The Armed Forces Policy Council appointed an Ad Hoc Committee on Genetic Effects of Atomic Energy that met on March 12, 1950. The committee involved division leaders and scientists from the AEC and academic geneticists, all of whom also had some connection to the AEC. The members included Shields Warren, Paul B. Pearson, Max Zelle (the staff geneticist for the Division of Biology and Medicine), William Russell, George Beadle, Curt Stern, and James Neel. These geneticists agreed with Wright that Evans's spontaneous mutation rate of 10^{-5} in humans might be two orders of magnitude too high, a result that ramified through all of Evans's safety estimates. If the doubling dose were only 3 roentgens, personnel in atomic plants working near the

increased mutations in populations continued through the 1950s; see Beatty, "Weighing the Risks" (ref. 5).

57. The origin of Wright's paper was a presentation at Oak Ridge in March 1948 that discussed the population-level effects of radiation exposure. The published version included an addendum in which he specifically discussed Evans's 1949 paper and its problematic assumptions.

58. J.B.S. Haldane, "The Rate of Spontaneous Mutation of a Human Gene," *Journal of Genetics* 31 (1935), 317–26; M. Gunther and L. S. Penrose, "The Genetics of Epiloia," *Journal of Genetics* 31 (1935), 413–430; Dunn, *Short History of Genetics* (ref. 39), 175.

59. Sewall Wright, "Discussion on Population Genetics and Radiation," *Symposium on Radiation Genetics*, *Journal of Cellular and Comparative Physiology* 35, suppl. 1 (1950): 187–205.

permissible dose levels might accumulate this level of radiation exposure within a few months. This raised questions about whether the government's limits would protect exposed individuals against genetic damage. As the divergent viewpoints of Muller and Wright made clear, the consequences for the population as a whole remained uncertain. In the end, the Armed Forces Policy Council chose not to publicize information about atomic energy's genetic risk.[60]

Even if the government did not disclose this information, others were already communicating the hazards of radiation exposure for human heredity. Several popular books on atomic energy drew attention to worrisome aspects of the increasing civilian and military uses of atomic energy. For example, Arnold Grobman's 1951 book *Our Atomic Heritage* specifically criticized the health standards at the Commission's installations for allowing radiation exposures that could results in genetic damage.[61] The Commission decided to prepare a public statement for "on project" use only, "should publication of the book generate undue anxiety among the Commission and Commission contractor personnel."[62] The AEC's principal concern was to reassure workers at the atomic energy plants that the agency's precautions protected them. As Christopher Jolly comments, this approach—"to publicly ignore any issue regarding the hazards or criticism unless forced to by averse publicity—seemed to have become de facto AEC policy."[63] Interestingly, even Grobman's criticisms of the agency did not touch the agency's radioisotope distribution program. *Our Atomic Heritage* included a section on "Hot Atoms" that effused about isotopes as a new tool in medicine, and drew on the AEC's publications to describe many of the research results already achieved in medicine, agriculture, science, and industry.

60. Ibid.; Jolly, "Thresholds of Uncertainty" (ref. 7), 95–103.

61. Arnold B. Grobman, *Our Atomic Heritage* (Gainesville: University of Florida Press, 1951). Grobman had worked with Donald Charles at Rochester, in mouse experiments supported by the AEC to ascertain the genetic effects of various doses of ionizing radiation. Charles's 1950 papers on this work, published in *Radiology*, did not mention that the data showed a linear dose-response without a lower threshold. Grobman, by contrast, drew attention to the lack of evidence for a threshold for damage. Jolly, "Thresholds of Uncertainty" (ref. 7), 113–16.

62. Notes on 541st AEC meeting, 26 Mar 1951, Genetic Effects of Radiation on Human Beings, NARA, RG 326, E67A, Box 65, Folder 8, Radiation Hazards Unclassified.

63. AEC officials tried to dissuade Grobman (who had previously worked in an AEC-supported radiation genetics laboratory at University of Rochester) from publishing his book, on the grounds that such a popular account would not add to his scientific stature. Jolly, "Thresholds of Uncertainty" (ref. 7), 117 (quote), 119.

The ACBM conferred many times on the human genetic risks for low-level radiation exposure—which were highly uncertain—and what AEC policy should be. At one meeting at which the AEC's program of genetics research was reviewed, members "agreed that the knowledge of human mutations rate is extremely important." A review of progress in setting permissible dose levels for external radiation and radioisotopes followed. The committee was "gratified to learn that during the past two years there has not been a single case of radiation injury due to the activities of the AEC."[64] But this very wording evaded the issue of genetic damage. Were genetic mutations caused by exposure to artificial radioactivity to be considered "radiation injuries," particularly if the damage was to one's offspring? If so, how low would the permissible dose have to be set to assure radiological safety?

New research on mammalian genetics (much of it sponsored by the AEC) underlined the concerns raised by Muller about the genetic consequences of even "permissible" radiation doses. Alexander Hollaender, who headed radiobiology research at the AEC's Oak Ridge National Laboratory, was a major contributor to this development, supporting research programs and organizing conferences aimed at resolving these issues.[65] Under Hollaender's leadership, William and Lianne Russell launched their "mega-mouse" study to provide a reliable estimate of the spontaneous mammalian mutation rate to help assess the genetic dangers to people from low-level radiation exposure. Their early results suggested that the mammalian spontaneous mutation rate might be an order of magnitude lower than that for *Drosophila*.[66] This would render Evans's estimates of the doubling dose off by three orders of magnitude, making the prospect of safety from genetic damage even more remote.

Other experiments pointed to the *cumulative* effects of radiation exposure. In research at the University of California, Berkeley sponsored by the AEC, Delta Uphoff and Curt Stern found that low doses of X-rays administered to *Drosophila* sperm in small doses over a prolonged period resulted in the same effects as when the overall dosage was administered all at once. As the authors concluded, "There is no threshold below which radiation fails to induce

64. Minutes, 24th ACBM Meeting, 10–11 Nov 1950, Washington, DC, OpenNet NV07111806, 10 and 11.

65. Karen A. Rader, "Alexander Hollaender's Postwar Vision for Biology: Oak Ridge and Beyond," *JHB* 39 (2006): 685–706.

66. Jolly, "Thresholds of Uncertainty" (ref. 7), 112, summarizing a letter from William Russell to Shields Warren, Jan 1951; Karen A. Rader, *Making Mice: Standardizing Animals for American Biomedical Research, 1900–1955* (Princeton, NJ: Princeton University Press, 2004), chap. 6.

mutations."[67] Assuming that mammals responded similarly, then long-term exposures to ionizing radiation, no matter how low, would cause genetic damage. Geneticists advising the AEC were well aware of the conundrum this presented for finding a certifiably safe radiation exposure level. As Bentley Glass, who joined the ACBM in the fall of 1955, put it: "the [agency's] recommendations are on the basis of what seems to be practicable, not on the basis of what radiation is considered genetically harmless. There is no such amount of radiation. All geneticists, I am sure, pretty well agree on this."[68]

LINKING HEREDITY TO CANCER IN THE FALLOUT DEBATE

The increased pace of nuclear tests, and the "transient increases in the radioactivity level in many communities of the United States," as two AEC officials put it, catalyzed new concerns about the civilian health costs of nuclear peace.[69] In 1951 and 1952, the U.S. government detonated twenty nuclear devices at the Nevada Test Site, and eleven more during the first half of 1953.[70] One May 1952 test dispersed radioactive debris as far away as Salt Lake City.[71] In the spring of 1953, in a period of heavy nuclear testing, some sheep men reported unusually high losses of lambs and ewes. An investigation of their complaints, under the auspices of the Commission, absolved radiation of the livestock deaths, though questions persisted.[72] The AEC's *Thirteenth Semiannual Report* steadfastly denied that testing imperiled anyone: "No person has been exposed to a harmful amount of radiation from fall-out. In

67. Delta E. Uphoff and Curt Stern, "The Genetic Effects of Low Intensity Irradiation," *Science* 109 (1949): 609–10.

68. Minutes, 56th ACBM Meeting, 26–27 May 1956, Washington, DC, OpenNet NV04111749, p. 28. On Glass, see Audra J. Wolfe, "The Organization Man and the Archive: A Look at the Bentley Glass Papers," *JHB* 44 (2012): 147–51.

69. Merrill Eisenbud and John H. Harley, "Radioactive Dust from Nuclear Detonations," *Science* 117 (1953), 141–47, on 141.

70. Hewlett and Duncan, *Atomic Shield* (ref. 31), 672–73; Richard G. Hewlett and Jack M. Holl, *Atoms for Peace and War, 1953–1961: Eisenhower and the Atomic Energy Commission* (Berkeley: University of California Press, 1989), 146–47.

71. Barton C. Hacker, *Elements of Controversy: The Atomic Energy Commission and Radiation Safety in Nuclear Weapons Testing, 1947–1974* (Berkeley: University of California Press, 1994), 81.

72. Barton C. Hacker, "'Hotter Than a $2 Pistol': Fallout, Sheep, and the Atomic Energy Commission, 1953–1986," in *The Atomic West*, ed. Bruce W. Hevly and John M. Findlay (Seattle: University of Washington Press, 1998), 157–75.

general, radioactivity resulting from fall-out has been many times below levels which could cause any injury to human beings, animals, or crops."[73]

Behind the scenes, however, the AEC launched Project Sunshine, a secret worldwide data collection effort to assess the accumulation of radioactive fission products from nuclear weapon tests in soil, plants, animals, and humans. The Division of Biology and Medicine arranged for the covert collection of baby bones throughout the world, by cooperating with American philanthropies and medical missions with contacts in South Asia and Latin America.[74] Earlier AEC assessments of fallout had already pinpointed strontium-90 as the most worrisome fission product.[75] As a RAND report on Project Sunshine put it: "The risk is simply this: The bone-retentive and radioactive properties of Sr^{90} endow it with a high carcinogenic capability."[76] So-called internal emitters were especially dangerous because once embedded in the bone, they continued to irradiate the organs. The long half-life of strontium-90—nearly thirty years—created additional concerns, as the radioelement could persist in the environment, moving through the food chain.[77] Thus its ingestion by humans via meat or milk from contaminated pastures could result in long-term effects, particularly cancer.

Atomic weapons themselves were changing in ways that underlined concerns about fallout. So-called hydrogen bombs, first tested at the Pacific Proving Ground in 1952, released significantly higher levels of radioactivity and fission products than conventional bombs. In March 1954, the AEC's test at Enewetak of one such thermonuclear device, Bravo, spread radioactive ash widely across the Pacific, engulfing the "Lucky Dragon," an unfortunately named Japanese

73. AEC, *Thirteenth Semiannual Report* (Washington, DC: U.S. Government Printing Office, 1953), 78.

74. Most of the samples collected from 1953 to 1956 were from stillborn infants. Advisory Committee on Human Radiation Experiments, *The Human Radiation Experiments: The Final Report of the President's Advisory Committee* (New York: Oxford University Press, 1996), 402–7; Memorandum to the Advisory Committee on Human Radiation Experiments, 8 Feb 1995, OpenNet NV0750699; Jolly, "Thresholds of Uncertainty" (ref. 7), 152–61.

75. Project Gabriel, begun by the AEC beginning in 1949, estimated hazards from radioactive fallout from nuclear warfare. Its calculations were based on theoretical considerations, not assay samples, but it highlighted the biological effects of strontium-90 as a key danger. AEC Division of Biology and Medicine, Jul 1954, "Report on Project Gabriel," DOE Archives, RG 326 AEC, Box 3363, Folder 26, OpenNet 0720894; Hacker, *Elements of Controversy* (ref. 71), 180–82.

76. RAND Corporation, "Worldwide Effects of Atomic Weapons: Project Sunshine," 6 Aug 1953, R-251-AEC, OpenNet NV0717541, p. 4.

77. Ibid. On the ecological concerns, see Creager, *Life Atomic* (ref. 24), chap. 10.

fishing boat.[78] Nearly two dozen fishermen aboard suffered injuries from the radiation exposure and one died in September 1954; these casualties received extensive media coverage in the United States as well as in Japan.[79] In response, the AEC made no concessions to critics. Lewis Strauss, now Chair of the Commission, publicly denied that the fallout could have injured anyone.[80]

Geneticist Alfred H. Sturtevant rebutted Strauss's statements about fallout in his presidential address at a June 1954 meeting of the Pacific Division of the American Association for the Advancement of Science, and his address appeared subsequently in *Science*. In recounting the hazards of radiation exposure, Sturtevant connected mutagenicity with so-called somatic effects: "There is reason to suppose that gene mutations, induced in an exposed individual, also constitute a hazard to that individual—especially in an increase in the probability of the development of malignant growths, perhaps years after the exposure." In comparison with the kind of radiation injuries taken into account in setting a permissible dose for exposure, there was not a lower limit to the amount of radiation that might induce such somatic mutations. Sturtevant concluded that "there is, in fact, no clearly safe dosage."[81]

The initiation of an open debate between the AEC and dissenting scientists made for great media coverage, which in turn fueled public alarm.[82] Early in

78. When it was selected as a test site, the name of the atoll was spelled Eniwetok, but the U.S. government later altered the spelling in accordance with Marshallese preference to Enewetak. Hacker, *Elements of Controversy* (ref. 71), 14, 140; Ralph E. Lapp, *The Voyage of the Lucky Dragon* (New York: Harper, 1958); "The H-Bomb and World Opinion," *Bulletin of the Atomic Scientists* 10, no. 5 (1954): 163–65.

79. Aya Homei, "The Contentious Death of Mr. Kuboyama: Science as Politics in the 1954 Lucky Dragon Incident," *Japan Forum* 25 (2013): 212–32.

80. "Chairman Strauss's Statement on Pacific Tests," quoted in "The H-Bomb and World Opinion," (ref. 78); Kopp, "Origins of the American Scientific Debate" (ref. 7). Strauss's statement contained several patent untruths, such as the suggestion that the fishermen's skin lesions were due to "the chemical activity of the converted material in the coral, rather than to radioactivity."

81. A. H. Sturtevant, "Social Implications of the Genetics of Man," *Science* 120 (1954): 405–7, on 406. Sturtevant built on H. J. Muller's earlier suggestion that the "general biological effects of radiation" were attributable to damage to the genes in somatic cells; Muller, "Some Present Problems" (ref. 6), 44. Numerous scientific publications earlier in the twentieth century speculated on a mutational origin for cancer (including a 1937 paper by Muller). For examples, see those cited in Jolly, "Thresholds of Uncertainty" (ref. 7), 496; Howard D. Lipshitz, *Genes, Development and Cancer: The Life and Work of Edward B. Lewis* (Dordrecht, The Netherlands: Kluwer, 2004), 390; and Ilana Löwy, *Preventive Strikes: Women, Precancer, and Prophylactic Surgery* (Baltimore, MD: Johns Hopkins University Press, 2010), 169.

82. The media coverage and public debate is detailed in Divine, *Blowing on the Wind* (ref. 7).

the winter of 1955, the Eisenhower administration was discussing how it might defuse criticisms about the dangers of fallout by disclosing more information. The preparation of a press release stalled between the AEC and the State Department.[83] Just before it was released, Ralph Lapp, a longtime critic of the agency, published two pieces on the scale of fallout from Bravo, one in the *Bulletin of Atomic Scientists* and the other in the *New Republic*. Lapp asserted that a hydrogen bomb the size of Bravo would release a hot cloud of debris that could travel 200 miles in several hours, contaminating the whole area with serious-to-lethal radioactivity.[84] The AEC report, appearing on the heels of Lapp's articles, largely confirmed his grim assessment.

The press amplified these concerns. *Newsweek*, which described the AEC report as the "terrible truth," projected the horrific consequences that would result from a thermonuclear explosion over the Northeast: "Drifting down from the sky, the ash will poison everything it touches within a cigar-shaped area of 7,000 square miles. It can threaten all life in a state the size of New Jersey."[85] At a February 22 hearing, members of the Senate Subcommittee on Civil Defense of the Armed Services Committee asked AEC officials why an independent scientist publicly disclosed the radiological dangers of hydrogen bombs before the agency did.[86] For their part, the Joint Committee on Atomic Energy pursued these issues at a hearing on April 15, 1955, though this amounted to little more than an opportunity for AEC officials to reassure the public that radioactive fallout from weapons testing was being monitored and current levels would not cause any immediate or long-term health hazards.[87]

During the same period, a decidedly mixed picture emerged from the ABCC's long-term studies of Japanese survivors of the atomic blasts. On the one hand, as *Time* reported, the incidence of leukemia among those who had been within 1,000 meters of the blasts was "more than 600 times the normal incidence of leukemia in Japan."[88] A higher-than-normal incidence of leukemia was observed also among those nearly two miles away from the explosion,

83. Hewlett and Holl, *Atoms for Peace and War* (ref. 70), 279–87.

84. Ralph E. Lapp, "'Fall-Out': Another Dimension in Atomic Killing Power," *New Republic* 132, no. 7 (14 Feb 1955): 8–12; Divine, *Blowing on the Wind* (ref. 7), 36–38.

85. "To Live—or Die—with It," *Newsweek* 45 (1955): 19–21, on 19; Divine, *Blowing on the Wind* (ref. 7), 38.

86. Hewlett and Holl, *Atoms for Peace and War* (ref. 70), 287–88; Michael Straight, "The Ten-Month Silence," *New Republic* 132, no. 10 (7 Mar 1955): 8–11.

87. Divine, *Blowing on the Wind* (ref. 7), 44–47.

88. "The Nuclear Revolution," *Time*, 6 Feb 1956, 83–84.

suggesting the long-range health consequences of radiation exposure from atomic detonations.[89] One physician associated with the study extrapolated to the question of fallout: "Dr. Moloney expects other forms of cancer to appear later, and he suspects that the radioactive fall-out of hydrogen bombs will have even greater cancer-producing effect."[90] On the other hand, studies of genetic damage in the offspring of survivors were less alarming. In 1955, the ABCC geneticists announced their results, which were "negative"—or inconclusive.[91] Conservative-leaning publications seized on this conclusion to rebut alarmism about radiation exposure. *US News & World Report*, for example, published a story entitled "Thousands of Babies, No A-Bomb Effects."[92] The ABCC's genetic study had been both scientifically difficult and politically contentious from the outset, in part because defining which phenotypes qualified as mutations in survivors' children was not straightforward.[93] Moreover, most geneticists thought it unlikely that surveying congenital abnormalities in children born to survivors would reveal even those mutations that had occurred.

H. J. Muller continued to address publicly the hazards of radiation exposure, whether from atomic energy or clinical use of X-rays. Pointing out in a June 1955 article in *Science* that radiation damaged genes in linear relationship to dose, he observed: "no exposure is so tiny that is does not carry its corresponding mutational risk."[94] In the midst of the nuclear arms race and Eisenhower's Atoms for

89. By and large, the exposures from the detonations over Hiroshima and Nagasaki were not a result of radioactive fallout, but radiation from the blast itself. See Radiation Effects Research Foundation, "RERF's Views on Residual Radiation," 8 Dec 2012, published on their website, http://www.rerf.jp/news/pdf/residualrad_ps_e.pdf (accessed 18 Nov 2014).

90. "Nuclear Revolution" (ref. 88).

91. This announcement of results was to the American Academy of General Practice, 29 Mar 1955; see Gladwin Hill, "Effect of A-Bomb Is Found Limited," *New York Times*, 30 Mar 1955, 16. A monograph was published the following year: James V. Neel and William J. Schull, *The Effect of Exposure to the Atomic Bombs on Pregnancy Termination in Hiroshima and Nagasaki* (Washington, DC: NAS–NRC Publication 461, 1956).

92. "Report on Hiroshima: Thousands of Babies, No A-Bomb Effects," *US News & World Report*, 8 Apr 1955, 46–48. This story, an interview with ABCC director Robert Holmes, contained several inaccuracies.

93. As Lindee shows, the ABCC's guidelines reflected social realities and criteria as well as scientific ones: M. Susan Lindee, "What Is a Mutation? Identifying Heritable Change in the Offspring of Survivors at Hiroshima and Nagasaki," *JHB* 25 (1992): 231–55. The ABCC also studied effects on life span: Crispin R. C. Barker, "From Atom Bomb to the Genetic 'Time Bomb': Telomeres, Aging, and Cancer in the Era of Molecular Biology" (PhD diss., Yale University, 2008), chap. 1.

94. H. J. Muller, "Genetic Damage Produced by Radiation," *Science* 121 (1955): 837–40, on 837.

Peace campaign, which promoted the development of a nuclear power industry, such a declaration was politically inconvenient for the administration. (In turn, the AEC—namely, Lewis Strauss—prevented Muller from presenting a paper at the Atoms for Peace conference that summer.[95]) In reality, Muller conceded that the development of nuclear arms could be justified on the grounds of protecting democracy and intellectual freedom against totalitarianism. Yet he was clear about the quantifiable biological cost of peacetime weapons testing: "In order to decide whether a continuance of the tests is justified, it is necessary first to admit the damage and then to weigh our estimate of it against the potential benefits to be derived from the tests or, rather, against the probable damage that would follow from the alternative policy."[96]

THE BEAR REPORT AND THE SOMATIC MUTATION HYPOTHESIS

In part to provide an independent assessment of radiation hazards amidst public controversy, the U.S. National Academy of Sciences convened a panel to assess the Biological Effects of Atomic Radiation. The experts appointed to the panel were organized into six committees: genetics, pathology, agriculture and food supply, meteorology, oceanography and fisheries, and disposal of radioactive waste.[97] In June 1956, the academy issued the panel's analysis, known as the BEAR Report. The report recommended reducing the maximum cumulative radiation exposure to reproductive cells from 300 to 50 roentgens, and limiting the average exposure through age thirty in the population at large to 10 roentgens.[98]

95. Elof Axel Carlson, *Genes, Radiation, and Society: The Life and Work of H. J. Muller* (Ithaca, NY: Cornell University Press, 1981), 356–58; Hewlett and Holl, *Atoms for Peace and War* (ref. 70), 268.

96. Muller, "Genetic Damage Produced by Radiation" (ref. 94), 838. On Muller's engagement with the public, see Susan Lindee, "Performing Anger: H. J. Muller, James V. Neel and Radiation Risk," in *Human Heredity in the Twentieth Century*, ed. Bernd Gausemeier, Staffan Müller-Wille, and Edmund Ramsden (London: Pickering & Chatto, 2013), 205–16.

97. Beatty, "Genetics in the Atomic Age" (ref. 12); Lindee, *Suffering Made Real* (ref. 12); Jolly, "Thresholds of Uncertainty" (ref. 7), chap. 6; Toshihiro Higuchi, "Radioactive Fallout, the Politics of Risk, and the Making of a Global Environmental Crisis, 1954–1963" (PhD dissertation, Georgetown University, 2011).

98. "Summary Report," in National Academy of Sciences, *The Biological Effects of Atomic Radiation: A Report to the Public* (Washington, DC: NAS–NRC, 1956), 8. On the simultaneous publication of a non-coincidentally similar report in the United Kingdom: Hamblin, "'A Dispassionate and Objective Effort'" (ref. 8).

Both the genetics and the pathology committees addressed the health-related effects of radiation, but offered strikingly different assessments. Geneticists focused on low-level doses and emphasized that "any radiation is genetically undesirable."[99] Even small increases in radiation exposure would result in deleterious mutations, or "genetic defects." "Each one of these mutants must eventually be extinguished out of the population through tragedy."[100] The committee focused particular concern on the overuse of radiation in medicine, which amounted to as much radiation exposure for most Americans as background radiation (3–4 roentgens), and much more than the radiation from fallout (0.1 roentgens). The geneticists' recommendation was to "keep all of our expenditures of radiation as low as possible."[101] As John Beatty has shown, it was not easy for the committee to reach consensus on key issues, including whether radiation-induced mutations would be deleterious to human populations.[102] In fact, because the two most prominent geneticists on the panel (Muller and Wright) were barely on speaking terms, a non-geneticist, Warren Weaver, was brought in to serve as chair.[103]

A main task of the Committee on Pathologic Effects was to evaluate concerns about whether the level of contamination of strontium-90 from fallout might lead, through its presence in milk and food, to increased incidence of human cancer. The BEAR Committee on Pathologic Effects acknowledged a possible link between radiation from strontium-90 and certain cancers, but concluded that current levels posed no such threat.[104] Radiation under certain levels could be "harmless to individuals." In discussing "late" effects of radiation—namely leukemia—observed among Japanese atom bomb survivors as well as radiologists, they emphasized that these individuals received either

99. "Report of Committee on Genetic Effects of Atomic Radiation," in NAS, *Biological Effects* (ref. 98), 23.

100. Ibid., 25 and 26.

101. Ibid., 30.

102. Beatty, "Masking Disagreement among Experts" (ref. 8).

103. "During a conversation on October 18, 1974, Dr. Bronk mentioned that the two most prominent geneticists on the Committee on Genetic Effects of Atomic Radiation refused to speak to one another. Therefore, Dr. Bronk persuaded Dr. Weaver to act as chairman and mediate the situation, despite the fact that Dr. Weaver was trained as a statistician, not a geneticist." Note labeled "1955," NAS–NRC Archives: Committee on BEAR Records Group: ADM: ORG: NAS: Committees on BEAR: Genetic: Appointments: 1955.

104. NAS, *Biological Effects* (ref. 98), 21. The Committee on Pathologic Effects similarly acknowledged the existence of injuries from long-term, low-dose exposures, such as leukemia and skin cancer, but insisted that "among those who have adhered to present permissible dose levels, none of these effects have been detected" (34).

a nearly fatal single dose of radiation or, for those exposed occupationally, "higher than acceptable permissible dose rates."[105] At the exposure levels recommended by the geneticists "as desirable for large populations," the pathological panel claimed, "there would be no demonstrable somatic effect, although a theoretical minor shortening of life span could not be ruled out."[106] The pathology group also emphasized natural sources of radiation, which they viewed as innocuous:

> In developing an unequivocally safe amount, we can recall that a certain degree of radiation exposure has always been with us, even excluding X-rays, in the form of gamma radiation from minerals, cosmic rays, and radioelements normally in the body. These levels vary greatly from one location or altitude to another and are not considered to produce harmful effects.[107]

Clearly, part of the disparity between how members of the BEAR genetics panel and those of the pathology panel viewed low-dose radiation is attributable to disciplinary orientation. Most geneticists were comfortable extrapolating from linear dose-response curves in flies to humans, and from gametes to somatic cells. It is perhaps not surprising that physicians and health physicists resisted seeing the complexities of radiation-induced cancer reduced to mutations. But the two panels were also different in the nature and extensiveness of relationships between their members and the AEC. The Pathological Effects Panel was populated by current and former Manhattan Project and AEC employees. Warren Weaver, who headed the Genetics Panel though the publication of the 1956 report, saw conflict of interest behind this group's deliberations. As he confidentially asked Detlev Bronk (head of the Rockefeller Foundation), "do you feel entirely comfortable about the degree of commitment and (perhaps unconscious) loyalty to the AEC that exists in the pathology group? I do not."[108]

There was also very significant overlap between AEC employees and members of the various subcommittees of the National Committee on Radiation Protection and Measurements (NCRP), the organization that recommended exposure limits for various forms of ionizing radiation. This facilitated the flow of information between the organizations, but it also meant that those NCRP

105. "Report of Committee on Pathologic Effects of Atomic Radiation," NAS, *Biological Effects* (ref. 98), 36 and 39.

106. Ibid., 39.

107. Ibid.

108. Warren Weaver to Detlev W. Bronk, 12 Jul 1956, NAS–NRC Archives: Committee on BEAR Records Group: ADM: ORG: NAS: Committees on BEAR: Genetic: General: 1956.

panel members working for the AEC had access to classified data not available to other members.[109] At one point, the AEC pressed the NCRP for advance information on the permissible doses they would set for radiation workers.[110] These circumstances, in addition to the travel funding that the NCRP received from the AEC, threatened the independence of the expert body setting standards for radiation protection.[111] Thus the potential for conflicts of interest affected the entire radiological safety apparatus.

Following the publication of the BEAR Report, Weaver was critical of the AEC for being "too defensive" and not candid enough about the hazards of atomic energy.[112] He was particularly unhappy with AEC Chair Lewis Strauss. He felt the Academy should release an official statement in response to Strauss's public comment on July 20, 1956, that "mass hazard from fallout is not a necessary complement to the use of large nuclear weapons."[113] A year later he sent a blistering letter to AEC scientist Merrill Eisenbud critiquing the agency's use of the word "certainly" in describing their confidence that strontium-90 from atmospheric radioactive fallout was not deposited at hazardous levels in soil.[114]

For their part, officials at the AEC repeatedly pointed to the low level of radiation exposure resulting from tests as compared with natural exposure to radioactivity and with normal clinical uses of radiation (particularly X-rays). For example, on May 6, 1955, Willard F. Libby wrote Linus Pauling a letter explaining why the AEC was "justified in saying that although genetic effects are unknown, the test fallout is so small as compared to the natural background and, more important, to the variations in the natural background which are customarily accepted, that we really cannot say that testing is in any way likely to be dangerous."[115] The AEC's July 1956 semiannual report to Congress,

109. Lauriston S. Taylor, *Organization for Radiation Protection: The Operations of the ICRP and NCRP, 1928–1974* (Springfield, VA: National Technical Information Service, 1979), 7-016.

110. Ibid.

111. On the travel funding, see ibid., 7-032.

112. Weaver to Eisenbud, 2 Jun 1957, NAS–NRC Archives: Committee on BEAR Records Group: ADM: ORG: NAS: Committees on BEAR: Genetic: General 1957.

113. Quote from "Strauss Hints Way Found to Restrict the Spread of Radioactive 'Fallout'," *Amsterdam Evening Recorder*, 20 Jul 1956, p. 1; Warren Weaver to George W. Beadle, 24 Jul 1956, NAS–NRC Archives: Committee on BEAR Records Group: ADM: ORG: NAS: Committees on BEAR: Genetic: General 1956.

114. Weaver to Eisenbud, 2 Jun 1957 (ref. 112), 3.

115. W. F. Libby to Linus C. Pauling, 6 May 1955, in NARA, RG 326, E67B, Box 49, Folder 7, Medicine, Health & Safety 13 Genetics; W. F. Libby, "Radioactive Fallout and Radioactive Strontium," *Science* 123 (1956): 657–60.

reiterating Libby's analysis on the matter, asserted that "at the present level of weapons' testing, the present and potential contribution of strontium-90 to the world ecology is not a significant factor."[116]

More and more scientists, however, agreed with minority Commissioner Murray, who dissented on this issue from the view in the AEC's report. Ralph Lapp published criticisms of the official interpretation, and Linus Pauling cited the link between strontium-90 and cancer in a 1959 letter in the *New York Times*.[117]

Journalists focused public concern on radioactivity entering the food supply, particularly the possible contamination of milk with strontium-90 from weapons testing, due to the grazing of cattle on Western pastures in the vicinity of the Nevada testing site. Strontium-90 became a symbol of the "atomic poison" released by nuclear weapons.[118] An editorial in *America* published on June 15, 1957, was simply entitled "The Strontium-90 Debate," and referred to the radioisotope as a "menace to life and health."[119] The AEC underestimated the symbolic importance of contaminating food, especially milk. Throughout the twentieth century in the United States the purity of milk has been a potent icon; earlier worries about impure milk containing tuberculosis bacilli were transmuted into fears about milk contaminated with radioactivity.[120] Infants and children who assimilated strontium-90 from contaminated milk into their skeletons could suffer decades of radiation exposure. During the 1956 Presidential campaign, Democratic candidate Adlai Stevenson proposed a unilateral test ban of hydrogen bombs to protect Americans from the accumulating radioactive fallout.[121] Fear of fallout was not enough to prevent Eisenhower's reelection, but the issue had become central to American politics.

116. AEC, *Twentieth Semiannual Report* (Washington, DC: U.S. Government Printing Office, 1956), 106.

117. Ralph E. Lapp, "Strontium-90 in Man," *Science* 125 (1957): 933–34; Linus Pauling, "Effect of Strontium-90," *New York Times*, 28 Apr 1959, 34; J. Christopher Jolly, "Linus Pauling and the Scientific Debate over Fallout Hazards," *Endeavour* 26 (2002): 149–53.

118. Carl Larsen, "Midwest Center for Research on Radiation Effect," *Chicago Daily Sun-Times*, 17 Jan 1955, 1, 4.

119. "The Strontium-90 Debate," *America* 97 (1957): 318.

120. Kendra D. Smith-Howard, "Perfecting Nature's Food: A Cultural and Environmental History of Milk in the United States, 1900–1970" (PhD dissertation, University of Wisconsin, Madison, 2007), chap. 5.

121. See Adlai E. Stevenson, "Why I Raised the H-Bomb Question," *Look* 21, no. 3 (1957): 23–25. The Public Health Service began monitoring strontium-90 levels in milk in 1958, and the increases they reported, though levels were still below the maximum permissible dose, reinforced public alarm. See Divine, *Blowing on the Wind* (ref. 7), 263–64.

Source of estimate	Type of radiation	Region irradiated	Types of leukemia produced	Probability of leukemia of specified type per individual per rad (or rem) to region irradiated per year		
				Estimated range		"Best" estimate
				Lower limit	Upper limit	
Atom-bomb survivors	Gamma rays plus neutrons	Whole body	All	0.7×10^{-4}	3×10^{-4}	2×10^{-4}
Ankylosing spondylitis patients	X-rays	Spine	Granulocytic (only?)	0.6×10^{-4}	2×10^{-4}	1×10^{-4}
Thymic enlargement patients	X-rays	Chest	Lymphocytic (only?)	0.4×10^{-4}	6×10^{-4}	1×10^{-4}
Radiologists	X-rays, radium, etc.	Partial to whole body	All (?)	0.4×10^{-4}	11×10^{-4}	2×10^{-4}
Spontaneous incidence of leukemia (Brooklyn, N.Y.)	All natural background sources	Whole body	All (?)		10×10^{-4}	2×10^{-4}

FIG. 2. Table comparing rates of leukemia among four populations exposed to ionizing radiation and a control population. *Source*: E. B. Lewis, "Leukemia and Ionizing Radiation," *Science* 125 (1957): 965–72, on 971. Reprinted with permission from American Association for the Advancement of Science.

EDWARD LEWIS AND LOW-LEVEL RADIATION RISKS

A 1957 publication by Caltech biology professor Edward B. Lewis in *Science* underlined the urgency of health risks from radioactive fallout. He compared studies of leukemia in four populations exposed to ionizing radiation: "(i) survivors of atomic bomb radiation in Japan, (ii) patients irradiated for ankylosing spondylitis, (iii) children irradiated as infants for thymic enlargement, and (iv) radiologists."[122] (See Fig. 2.) In each case, the dose-effect response appeared linear, and there was no evidence of a threshold below which exposure posed no hazard.[123] The risk of leukemia caused by various kinds of exposure was comparable, leading Lewis to postulate a minimum estimate of induced leukemia as 2×10^{-6} per individual per rem per year, the newer dosage designation of rem being equivalent to the older roentgen.

The leukemia incidence among exposed and unexposed Japanese populations was crucial to Lewis's analysis, as it strongly suggested there was no lower threshold for risk from ionizing radiation. The Japanese atomic bomb survivors were problematic surrogates insofar as the fallout debates hinged on the detrimental effects of radiation at low levels. Many Japanese survivors had been

122. E. B. Lewis, "Leukemia and Ionizing Radiation," *Science* 125 (1957), 965–72, on 965.

123. As Jolly points out, Lewis did not claim to have proven linearity, but argued that the evidence pointed that way and that his estimates were valid within a factor of three. "Thresholds of Uncertainty" (ref. 7), 494. See also Edward J. Calabrese, "Origin of the Linearity No Threshold (LNT) Dose-Response Concept."

exposed at relatively high levels, whether they had exhibited radiation sickness or not, as the AEC was quick to point out. However, the number of cases of leukemia diagnosed in Japanese survivors, even those over 1,000 meters from the blast epicenter, provided compelling evidence that ionizing radiation induced leukemia in linear, dose-dependent fashion.

Lewis attributed the linear dose-dependence of leukemia incidence on radiation exposure to the somatic mutation hypothesis for carcinogenesis. On the basis of his analysis, Lewis argued that the growing concentration of strontium-90 from fallout could be sufficient to raise the incidence of leukemia in the United States by as much as 5 to 10 percent. This estimate relied on many assumptions; the concentration of strontium-90 in the human skeleton necessary to induce leukemia was not known.[124]

Even before it appeared in print, Lewis's paper was taken up into the political debates over nuclear weapons testing. Albert Schweitzer, Nobel Peace Prize Laureate in 1952, drew on Lewis's data in his "Declaration of Conscience," challenging nuclear warfare and weapons testing, which was published by the Nobel Prize Committee on April 24, 1957.[125] On NBC's "Meet the Press," Lewis's findings were disputed by Strauss himself. Strauss attacked Lewis's credentials, stating "that he is not a specialist in leukemia, [and] that it is too early to say what acceptance his hypothesis will receive among scientists who are students of leukemia."[126]

The Hearings on the Nature of Radioactive Fallout and Its Effects on Man, convened by Congress in 1957, provided an opportunity for the AEC's critics—and even its supporters—to question the agency's intransigence. Congressman Chet Holifield, who chaired the Joint Committee on Atomic Energy, berated the Commission for not disclosing more information earlier on the nature and extent of radioactive fallout from weapons testing, and for establishing a "party line" of downplaying its dangers.[127] Strauss misjudged the tenacity and political connections of the agency's scientific critics, even as he dismissed their concerns as hysteria. As observed in the minutes of an ACBM meeting, "The AEC was not prepared for the emotionalism within segments of the scientific community but felt that the results of the current hearings on fallout should be beneficial."[128] Although the agency continued to extol the

124. Minutes, 52nd ACBM Meeting, 9–10 Sep 1955, Washington, DC, OpenNet NV04111746, 13.
125. Lipshitz, *Genes, Development and Cancer* (ref. 81), 395.
126. As quoted in ibid., 396–97.
127. Hewlett and Holl, *Atoms for Peace and War* (ref. 70), 454–55.
128. Minutes, 63rd ACBM Meeting, 18 Jun 1957, Washington, DC, OpenNet NV0712175, 6.

scientific benefits of radioisotopes (and atomic energy generally), there were also references to "radioisotopes as possible contaminants in food products," as well as to the hazardous genetic and somatic consequences of exposure to all forms of ionizing radiation.[129]

Edward Lewis was among the many scientists and officials giving testimony at the 1957 Congressional Hearings. He presented his recently published analysis of leukemia incidence among A-bomb survivors, radiologists, and patients treated with X-rays, emphasizing the apparent linearity of the dose-response curve, and the lack of evidence for a threshold. Several others at the hearings cited his results, many favorably, but others critically.[130] Despite scientific uncertainties, the views of Lewis, Sturtevant, and Muller were influential. A report from the United Nations Committee on the Effects of Atomic Radiation published on August 10, 1958, predicted a rise in additional deaths from leukemia worldwide due to radioactive fallout, as Lewis had. The increase in radioactivity, they pointed out, was slight—only 5 percent of total radiation received from natural sources. However, even small increases in radiation could lead to increases in cancer.[131] Strikingly, the BEAR Committee on Pathology issued a rebuttal, in the form of Commentary on the United Nations Report. They were the only group from the NRC committee structure to do so. The panel remarked:

> The question of induction of leukemia or other types of cancer in man by very small doses of radiation has been treated in the United Nations report to suggest that the hypothesis of linearity and threshold effects as applied to the behavior of somatic cells have equal livelihood of validity. Our committee inclines to the view that many forms of cancer, including leukemia, arise through a more or less complex series of responses. While somatic mutations may be included among these, it seems doubtful that a strict linearity analogous to that seen in the genetic effects of radiation is as likely to hold in the case of these conditions. We note also that there is a considerable body of experimental evidence favoring non-linearity in specific instances.[132]

129. U.S. Congress, Joint Committee on Atomic Energy, *The Nature of Radioactive Fallout and Its Effect on Man*, Hearings before the Special Subcommittee on Radiation, 85th Congress, 1st sess., 2 pts. (Washington, DC: U.S. Government Printing Office, 1957), on 1960.

130. Lipshitz, *Genes, Development and Cancer* (ref. 81), 398.

131. Divine, *Blowing on the Wind* (ref. 7), 222.

132. NAS–NRC, Committee on Pathologic Effects of Atomic Radiation, Report II, *A Commentary on the Report of the United Nations Scientific Committee on the Effects of Atomic Radiation* (Washington DC: NAS–NRC, Publication 647, 1959).

In sympathy with these views, AEC officials and many of its researchers resisted giving up the threshold concept.[133] A 1958 paper published by Miriam Finkel, a researcher at Argonne National Laboratory, presented results of a study in which mice were exposed to various doses of strontium-90. The expected effects of life-shortening and leukemia were observed, but mice receiving the lowest dose did not show any of these effects.[134] Finkel argued that a threshold for strontium-90 existed that was well below human exposures due to fallout—an interpretation soon challenged by Linus Pauling and others. In response, Argonne's director Austin Brues asserted that the theory of linearity remained unproven.[135] Merrill Eisenbud, who worked as Director of the AEC's Health & Safety Laboratory and Manager of the New York Operations Office, dates the decline of this threshold view to 1963, although one 1966 agency publication, while stating that there is no threshold for genetic damage from radiation, says there is a threshold for somatic symptoms.[136]

In addition, many physicians remained unpersuaded by the somatic mutation theory. Shields Warren bluntly noted, "This theory is not accepted by many oncologists."[137] For other putative cancer-causing agents, such as viruses and hormones, there was a long history of research and a possible mechanism.[138] How mutations caused cancer (and whether they were chromosomal aberrations or point mutations) was not clear to anyone, least of all the geneticists who advocated the theory. As Peyton Rous (discoverer of the first animal tumor virus) commented in 1959, "A hypothesis is best known by its fruits. What have been those of the somatic mutation hypothesis? It has

133. See discussion under "Radiation and Cancer Rates" in *Human Radiation Studies: Remembering the Early Years, Oral History of Merrill Eisenbud*. Conducted January 26, 1995, through the DOE by Thomas J. Fisher, Jr. and David S. Harrell, published at https://ehss.energy.gov/ohre/roadmap/histories/0456/0456toc.html (accessed 5 Dec 2014).

134. Miriam P. Finkel, "Mice, Men and Fallout," *Science* 128 (1958): 637–41.

135. Austin M. Brues, "Critique of the Linear Theory of Carcinogenesis," *Science* 128 (1958): 693–98; Divine, *Blowing on the Wind* (ref. 7), 223–25.

136. Isaac Asimov and Theodosius Dobzhansky, *The Genetics Effects of Radiation* (Oak Ridge, TN: U.S. AEC Division of Technical Information, 1966), 35–36; *Human Radiation Studies* (ref. 133).

137. Shields Warren, "You, Your Patients and Radioactive Fallout," *New England Journal of Medicine* 266 (1962): 1125. On the reception of the somatic mutation in the medical community, see Jolly, "Thresholds of Uncertainty" (ref. 7), chap. 12.

138. In acknowledgement of these etiologies, Lewis mentioned that a somatic mutation might cause a cell "to release, or to respond to, viruslike or hormonal agents." Lewis, "Leukemia and Ionizing Radiation" (ref. 122), 970.

resulted in no good thing as concerns the cancer problem, but in much that is bad. . . . Most serious of all the results of the somatic mutation hypothesis has been its effect on research workers. It acts as a tranquilizer on those who believe in it."[139] However, other non-geneticists prominent in radiation research were open to seeing somatic mutations as the cause of radiation-induced aging and cancer. Paul Henshaw, a biophysicist who worked for the AEC's Division of Biology and Medicine, and Gioacchino Failla, an influential radiological physicist, were persuaded by the statistical evidence implicating gene mutations in radiation-induced life-shortening and carcinogenesis.[140] After the BEAR report, the National Academy of Sciences sponsored several scientific meetings involving geneticists, radiologists, and radiobiologists to encourage discussion of radiation effects and safe exposure levels. This was especially important given that a 1957 report by the BEAR Genetics Panel about medical uses of X-rays prompted concern from the American College of Radiology (who felt their expertise concerning safe use of radiologic equipment was being challenged) as well as Harvey Picker, President of the Picker X-ray Company.[141]

Over the 1960s, the somatic mutation of cancer causation steadily gained ground. Geneticists seeking empirical evidence of somatic mutation associated with cancer had tended to look to cytogenetics. The spread of human karyotyping techniques made possible the identification of many chromosomal abnormalities in tumor cells or cells exposed to carcinogens.[142] The nuclear chemist and medical researcher John Gofman, who became a prominent critic

139. Peyton Rous, "Surmise and Fact on the Nature of Cancer," *Nature* 183 (1959): 1357–61.

140. Failla, however, did not abandon his belief in a threshold for radiation effects. Paul S. Henshaw, "Genetic Transition as a Determinant of Physiologic and Radiologic Aging and Other Conditions," *Radiology* 69 (1957): 23–29; G. Failla, "Considerations Bearing on Permissible Accumulated Radiation Doses for Occupational Exposure," *Radiology* 69 (1957): 18–22; Jolly, "Thresholds of Uncertainty" (ref. 7), 434–49. This excellent dissertation offers a much more thorough account of the controversy over the somatic mutation theory of carcinogenesis.

141. See George Wilson to D. W. Bronk, 30 Jul 1957, NAS–NRC Archives: Committee on BEAR Records Group: ADM: ORG: NAS: Committees on BEAR: Laughlin-Pullman Report; as well as documents in adjacent folders, Committees on BEAR: Laughlin-Pullman Report: Review by Professional Organizations 1957; and Committees on BEAR: Laughlin-Pullman Report: General 1957.

142. See Soraya de Chadarevian, "Putting Human Genetics on a Solid Basis: Human Chromosome Research, 1950s–1970s," in *Human Heredity in the Twentieth Century*, ed. Bernd Gausemeier, Staffan Müller-Wille, and Edmund Ramsden (London: Pickering & Chatto, 2013), 141–52, esp. 142; María Jesús Santesmases, "Human Chromosomes and Cancer: Tumors and the Geographies of Cytogenetic Practices, 1951–1956," this Issue.

of the AEC (his employer) for denying the somatic risks of low-level radiation, based his analysis of cancer risks on chromosomal abnormalities in irradiated cell lines well into the 1970s.[143] Studies of DNA structure and mutagenesis, however, provided another compelling way to document such changes—at the level of molecules, not chromosome morphology. It is worth emphasizing that the initial emphasis on the somatic effects to mutations by geneticists such as Muller and Sturtevant was unrelated to the 1953 double-helical structure of DNA proposed by Watson and Crick. But as damage from ionizing radiation became conceptualized and detected in terms of chemical changes to DNA, this provided a way to screen for other potential mutagens, particularly synthetic chemicals.[144] Research into DNA damage at the molecular level led in surprising directions, particularly the discovery of DNA repair, which revealed that not all mutations are irreversible.[145] In general, however, research on mutagens and their mechanisms of action reinforced the conviction that environmental carcinogens worked by damaging DNA, strengthening the grounds for the somatic mutation theory—even as "mutation" also shifted its referent from chromosome to base pair.[146]

Concomitant with greater acceptance of the somatic mutation theory of carcinogenesis was an adoption of the linear, no-threshold model for low-dose radiation effects. In the face of continuing scientific uncertainties about the shape dose-response curve for low ionizing radiation levels, assuming a linear relationship to zero seemed a reasonable precaution.[147] In drafting the 1960

143. Ioanna Semendeferi, "Legitimating a Nuclear Critic: John Gofman, Radiation Safety, and Cancer Risks," *HSNS* 38, no. 2 (2008): 259–302.

144. Angela N. H. Creager, "The Political Life of Mutagens: A History of the Ames Test," in *Powerless Science? Science and Politics in a Toxic World*, ed. Soraya Boudia and Nathalie Jas (New York: Berghahn, 2014), 46–64.

145. Doogab Yi, "The Coming of Reversibility: The Discovery of DNA Repair between the Atomic Age and the Information Age," *HSPS* 37, suppl. (2007): 35–72.

146. On the many such shifts in the history of mutation, see Luis Campos and Alexander von Schwerin, eds., *Making Mutations: Objects, Practices, Contexts* (Berlin: Max Planck Institute for the History of Science Preprint 393, 2010); Elof Axel Carlson, *Mutation: The History of an Idea from Darwin to Genomics* (Cold Spring Harbor Laboratory, NY: Cold Spring Harbor Laboratory Press, 2011).

147. At least one critic views the evidence in the 1950s for the adoption of the linear, no-threshold model as less than compelling. See Edward Calabrese, "The Genetics Panel of the NAS BEAR I Committee (1956): Epistolary Evidence Suggests Self-Interest May Have Prompted an Exaggeration of Radiation Risks that Led to the Adoption of the LNT Cancer Risk Assessment Model," *Archives of Toxicology* 88, no. 9 (2014): 1631–34. On the politics of dose-response curves, see Robert N. Proctor, *Cancer Wars: How Politics Shapes What We Know and Don't Know About Cancer* (New York: Basic Books, 1995).

BEAR Report, the Pathology Panel's Subcommittee on Hematology recommended adopting a "no threshold" position for the sake of policy, even though some of its members believed a threshold must actually exist.[148] By the time of the subsequent 1972 Report of the Advisor Committee on the Biological Effects of Ionizing Radiation (BEIR I, successor to BEAR), the linear no-threshold model for somatic effects had become the default position.

> Until recently, it has been taken for granted that genetic risks from exposure of populations to ionizing radiation near background levels were of much greater import than were somatic risks. However, this assumption can no longer be made if linear non-threshold relationships are accepted as a basis for estimating cancer risks.[149]

This is not to say that the matter was uncontested. Members of BEIR III were famously unable to reach consensus for their 1980 report over whether a strictly linear model accounted for the effects of low-dose exposure to ionizing radiation.[150] Nonetheless, the linear, no-threshold model remains a default hypothesis for the hazards of ionizing radiation below the range for which there is not conclusive epidemiological data, even as researchers develop more sophisticated mathematical models for cancer risk as a function of radiation dose.[151]

CONCLUSIONS

In the late 1940s, the widely heralded use of atomic energy to conquer cancer initially gave radioactivity a positive valence—radioisotopes in particular were expected to be the AEC's major weapon in this war. This, in conjunction with the confidence of physicians and health physicists in the safety of the "permissible dose" adopted by the government led to complacency about, and perhaps even neglect of, longstanding genetic views of the mutagenicity of radiation. But the fallout debates changed public perception of the relationship

148. Jolly, "Thresholds of Uncertainty" (ref. 7), 5, 21.

149. NRC Advisory Committee on the Biological Effects of Ionizing Radiations, *The Effects on Populations of Exposure to Low Levels of Ionizing Radiation* (Washington, DC: NAS–NRC, 1972), 2.

150. Edward P. Radford, "Human Health Effects of Low Doses of Ionizing Radiation: The BEIR III Controversy," *Radiation Research* 84 (1980): 369–94.

151. Jan Beyea, "Special Issue on the Risks of Exposure to Low-Level Radiation," *Bulletin of the Atomic Scientists* 68 (2012): 10–12.

of atomic energy and cancer; radioisotopes began to be understood as threats to health (especially strontium-90, accumulating in milk) rather than "medical bullets," as they had been termed in 1947.

Geneticists such as H. J. Muller and Alfred Sturtevant challenged confidence in the adequacy of the U.S. government's radiological protection policies by contesting the existence of an inherent distinction between the effects of ionizing radiation on reproductive cells versus that on the rest of the body. This essay has shown that well before the fallout debates of the 1950s there had emerged a debate among researchers and government officials over whether permissible dose levels for radiation should be set to prevent genetic as well as somatic effects, and if so, what limits would ensure such protection. Health physicists such as Robley Evans contended that the existing radiation limits, set to prevent somatic effects (especially cancer) in atomic energy workers, were also sufficient to protect their gametes. Over the course of the 1950s, prominent geneticists shifted the discussion about the hazards of mutations from occupational exposure, radioactive fallout, and clinical uses of X-rays to encompass the health of exposed individuals, not only of their offspring. Edward Lewis's deft analysis of epidemiological evidence from the ABCC and the medical literature made the somatic mutation theory compelling and plausible, even as the details of how mutations induced cancer remained shadowy.

The connections between radiation, mutation, and cancer that were forged through these debates in the 1950s endured, shaping the public perception of emerging information about other environmental contaminants, even after the Test Ban Treaty had put anxieties about radioactive fallout to rest. The 1962 publication of Rachel Carson's *Silent Spring* expanded the scope of concern to include synthetic chemicals, "the sinister and little-recognized partners of radiation in changing the very nature of the world—the very nature of life."[152] This had two striking effects on the growth of such research in the 1960s and the 1970s. First, the AEC itself became a major supporter of environmental science, including that related to health.[153] During the 1960s, the agency began conducting research into chemical mutagenesis alongside its work on radiobiology, leading to the establishment of a computer registry for chemical

152. Rachel Carson, *Silent Spring* (Boston: Houghton Mifflin, 1962), 6.

153. Stephen Bocking, *Ecologists and Environmental Politics: A History of Contemporary Ecology* (New Haven, CT: Yale University Press, 1997); Judith Johns Schloegel and Karen A. Rader, *Ecology, Environment and "Big Science": An Annotated Bibliography of Sources on Environmental Research at Argonne National Laboratory, 1955–1985*, ANL/HIST-4 (Chicago: Argonne National Laboratory, 2005).

carcinogens was at Oak Ridge National Laboratory.[154] Second, research into DNA structure and molecular genetics provided a new arena for understanding the health effects of mutagens and carcinogens. The somatic mutation theory has become, as one book on cancer biology puts it, "the prevalent theory of carcinogenesis."[155] As this article has tried to show, this tenet of cancer biology is part of the complex scientific and political legacy of atomic energy in the mid-twentieth century.

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154. Scott Frickel, *Chemical Consequences: Environmental Mutagens, Scientist Activism, and the Rise of Genetic Toxicology* (New Brunswick, NJ: Rutgers University Press, 2004), 56–59.

155. Carlos Sonneschein and Anna M. Soto, *The Society of Cells: Cancer and Control of Cell Proliferation* (Oxford: BIOS Scientific Publishers, 1999), xi.