



On the origins of the linear no-threshold (LNT) dogma by means of untruths, artful dodges and blind faith



Edward J. Calabrese*

Department of Environmental Health Sciences, School of Public Health and Health Sciences, University of Massachusetts, Amherst, MA 01003, USA

ARTICLE INFO

Article history:

Received 2 June 2015
Received in revised form
16 July 2015
Accepted 17 July 2015

Keywords:

Risk assessment
Dose–response
Linear dose response
Cancer
Mutation
LNT
Ionizing radiation

ABSTRACT

This paper is an historical assessment of how prominent radiation geneticists in the United States during the 1940s and 1950s successfully worked to build acceptance for the linear no-threshold (LNT) dose–response model in risk assessment, significantly impacting environmental, occupational and medical exposure standards and practices to the present time. Detailed documentation indicates that actions taken in support of this policy revolution were ideologically driven and deliberately and deceptively misleading; that scientific records were artfully misrepresented; and that people and organizations in positions of public trust failed to perform the duties expected of them. Key activities are described and the roles of specific individuals are documented. These actions culminated in a 1956 report by a Genetics Panel of the U.S. National Academy of Sciences (NAS) on Biological Effects of Atomic Radiation (BEAR). In this report the Genetics Panel recommended that a linear dose response model be adopted for the purpose of risk assessment, a recommendation that was rapidly and widely promulgated. The paper argues that current international cancer risk assessment policies are based on fraudulent actions of the U. S. NAS BEAR I Committee, Genetics Panel and on the uncritical, unquestioning and blind-faith acceptance by regulatory agencies and the scientific community.

© 2015 Elsevier Inc. All rights reserved.

1. Introduction

In the course of recent assessments of the historical and scientific foundations of dose responses models, it was learned that the linear dose response model was deliberately promoted to advance ideological agendas of some of the world's most prestigious radiation geneticists (Calabrese, 2008; Calabrese, 2013a, 2015a, 2015b). These individuals intentionally misled/deceived the scientific and world communities at the highest possible levels, including in a 1946 Nobel Prize Lecture (Calabrese, 2011a; Calabrese, 2012), in their scientific publications (Calabrese, 2011b; Calabrese, 2013b; Caspari and Stern, 1948; Muller, 1950a, 1954; Uphoff and Stern, 1949), in their role as members of the U.S. NAS (Calabrese, 2013a; Calabrese, 2015b, 2015a) and in publications of the NAS [BEAR Committee, Genetics Panel – (Anonymous, 1956a; National Academy of Sciences NAS)/National Research Council NRC, 1956). Collectively, these deceptive actions became highly significant when they facilitated an unchallenged and blind-faith adoption of the Linear Dose Response (LDR) model for cancer risk assessment of ionizing radiation and later of chemical carcinogens (Calabrese, 2011b, 2013b, 2009a). The adoption of the LDR model

affected the magnitude of financial resources involved in regulatory actions, toxic tort decisions and medical practices; it also affected risk communication messages to the general public, educational practices, governmental research funding priorities, as well as decisions related to lifestyle and child rearing.

The impact of these deceptions has been substantial and, to this day, they significantly affect and dominate regulatory policies and risk assessment practices. Since these disturbing findings were published as a series of separate papers in diverse scientific journals, (e.g. mutation, radiation and toxicology journals) (Calabrese, 2015b, 2015a, 2011c, 2012, 2011b, 2013b, 2009a, 2014a, Calabrese, 014b), it has become necessary to develop an integrated and holistic version of this complex story. In addition, newly unearthed materials on key individuals have been discovered and incorporated herein to clarify previous historical frameworks. Finally, critical feedback recently received from reviewers, editors and others in the research community has proven invaluable in tempering the perspective and improving the content and context of this assessment.

This paper follows an historical timeline, starting with the professional/scientific relationship between Hermann Muller and Curt Stern and their subsequent collaborations on ionizing radiation during the Manhattan Project. The many, and, at times, bizarre ways in which Stern tried to prevent acceptance of the threshold model supportive findings of Ernst Caspari, a member of

* Fax: +1 413 545 4692.

E-mail address: edwardc@schoolph.umass.edu

the Manhattan Project team, in order to promote the LNT model, are detailed. Muller's Nobel Prize Lecture with emphasis on his assessment of the nature of the dose response in the low dose range, especially in light of the Caspari findings, is critiqued, leading to an assessment of how he and Stern acted to cover up Muller's Nobel Prize Lecture deceit via obfuscation of the Manhattan Project findings and the strikingly false subsequent statements of Muller in the scientific literature. The paper then assessed how the leadership of Muller and Stern profoundly affected beliefs on dose response within the genetics community during the 1950s, especially seen through the actions of the NAS BEAR I Genetics Panel in 1956 which assured the acceptance of the LNT by falsifying and fabricating the research record, thereby constituting scientific misconduct at the highest possible level.

2. The Curt Stern–Hermann J. Muller connections

Previously, this author had extensively researched the history of the non-linear (hormetic) dose–response model, its scientific foundations and its failure to thrive and out-compete the linear no-threshold (LNT) dose–response model during the first half of the 20th century (Calabrese, 2011b, 2005, 2009b; Calabrese and Baldwin, 2000a, 2000b, 2000c, 2000d, 2000e). As a continuation of this research activity, efforts have been exerted to assess in detail the historical and scientific origins that have resulted in the validation and acceptance of today's LNT model. During this investigation, it became evident that the role of Hermann J. Muller was essential to the adoption of the LNT model and needed greater clarification.

During this assessment of Muller, interest grew in the research activities of the Manhattan Project at the University of Rochester, especially those under the direction of Curt Stern who employed the fruit fly to investigate the nature of the dose response in the low dose range. Stern was of particular interest because he had a long personal and professional relationship with Muller that would markedly impact the LNT deception story. Stern had helped to organize the Fifth International Genetics Congress in Berlin during the fall of 1927 (Carlson, 1981). It was at this meeting that Muller first presented his landmark findings on X-ray-induced mutations in fruit flies (Muller, 1927, 1928), research that would eventually lead to his Nobel Prize in 1946 (Muller, 1946a). Later, Muller and Stern would have a conflict over Muller's deliberate failure to acknowledge a prior discovery by Stern that provided proof for the linear arrangement of genes, an issue that was then a very significant question in biology. Stern would challenge Muller on this point directly via a carefully documented letter dated August 8, 1929 [American Philosophical Society (APS) (American Philosophical Society, 1929a)]. Stern informed Muller that his earlier publication in *Biologischen Zentrablatt* (September, 1926) addressed the “theory of the linear arrangement and have specifically stated it in the title of the paper”. Stern concluded his letter to Muller with the statement that his manuscript “had been written before your [Muller's] first papers about them appeared.” Nearly six weeks later, in a letter dated October 3, 1929, Muller would respond “I am very sorry to have omitted mention of your work in my discussion of translocation and not to have given you credit for having made the first cytological demonstration of a genetically demonstrated translocation and pointed out its significance for the theory of linear arrangement”. He then indicated that he had enclosed a “carbon copy of a note I am sending in on the subject to the *American Naturalist*, which I hope you will consider as rectifying this mistake” (American Philosophical Society, 1929b). While Stern caught Muller in a significant professional indiscretion, he let Muller “control” the narrative by not objecting to Muller's version of the correction. Nonetheless, this

arrangement proved to be acceptable to Stern as seen in an October 23, 1929, letter from Stern to Muller, restoring a positive tone to their relationship (American Philosophical Society, 1929c). One could speculate what might have happened to the LNT story if Muller and Stern had not reconciled, possibly preventing Muller's involvement in the Manhattan Project as described below.

3. The Manhattan Project: Curt Stern and LNT

After Stern¹ initiated research on the Manhattan Project in 1943, he contacted Muller, then a professor of biology at Amherst College (1940–1945), to serve as a consultant to the project. Under normal circumstances this might have been routine, but Muller had a questionable past, abandoning the US to live and research in the Soviet Union from about 1934–1938 (Carlson, 1981). Stern nonetheless obtained approval by the U.S. government for Muller's participation in the radiation genetics project. Muller's involvement proved to be extensive, involving detailed technical written communications with Stern and other team members, visits to the University of Rochester, and a donation of his Muller-5 strain of *Drosophila* (Calabrese, 2011c).

The Manhattan Project of Stern was designed to expand the study of high dose ionizing radiation on genomic mutations to include the area of chronic, lifetime exposures at relatively low doses and very low dose rates. The first experiment under Stern's direction was an acute (i.e., short duration) exposure study over a broad dose range. It was conducted by Warren Spencer, a professor at the College of Wooster with a PhD from Ohio State University in the area of *Drosophila* biology. Previous research by several of Muller's students (Hanson and Heys, 1929, 1932; Oliver, 1930, 1931), at very high doses and over a limited dose range, provided support for the hypothesis that the nature of the dose response for X-ray-induced mutation was linear.

In the Spencer study, the effects of X-rays were assessed on sex-linked recessive lethality using *Drosophila* with acute/short term (2–40 min) exposures and a dose-rate ranging from 10 to 96 r/h. This resulted in a range of cumulative doses from 4000 r down to 25 r (i.e., lowest cumulative dose yet tested). Following a data collection period from December, 1944 to June, 1955, Spencer reported that X-rays induced gonadal mutations in a manner that were linear across the dose response continuum, just as Stern and Muller had predicted (Calabrese, 2011c).

Ernst Caspari, a Ph.D. in insect behavior, directed the next study. From October 1945, to August 1946, Caspari assessed the effects of gamma rays on *Drosophila* sex-linked recessive lethality. In Caspari's study the females were first mated, placed on an egg laying suppression diet, and then exposed to the gamma radiation (2.5 r/day) for 21 days with sperm stored in the female's spermatheca. In the Caspari study, there was an aging component to the sperm that was not in the Spencer study. The dose rate used in Caspari's study was much lower (13,200 times lower) than that used in Spencer's acute study at the same cumulative dose (Calabrese, 2011c).

The data from the chronic exposure study of Caspari supported a threshold dose–response model. Stern initially rejected the

¹ In the case of the University of Rochester mammalian radiation geneticist Donald Charles, despite the use of over 400,000 mice, his research was largely unproductive, with no methodologically-based technical publications during the time of the Manhattan Project which ended in 1946 (see Charles (1950) for a brief descriptive paper). An additional summary paper (Charles et al., 1961) was published [after Charles's death (Anonymous, 1955a)] that tried to salvage the research effort with no obvious success. The failure of Charles to deliver a scientifically significant product for the Manhattan Project, given the level of resources directed to it, represented a substantial failing.

interpretation of Caspari as seen in written correspondence ([American Philosophical Society, 1947a](#)). Stern thought the findings were aberrant due to an unexpectedly high mutation rate of the “controls” that obscured a linear dose response, yielding only the appearance of a threshold response. Despite this rejection by his mentor, Caspari dug into the published literature and found convincing support for his rather than Stern’s interpretation ([Kaufmann, 1947](#); [Muller, 1945, 1946b](#); [Rajewsky and Timofeef-Ressovsky, 1939](#)). To his credit, Stern accepted the data-based argument of Caspari.

Caspari’s data were unexpected and somewhat troubling to him because they challenged the linear paradigm of the radiation genetics community. Therefore, Caspari decided to send his findings to another leading researcher, Milisav Demerec, head of genetics at Cold Spring Harbor, for review and comment. Caspari was looking for a way around this problem (i.e., alternative interpretation) and hoping that the influential Demerec might offer a solution. Reflecting the bias of the radiation genetics research community at this time, Demerec wrote back to Caspari, acknowledging the problematic nature of the data, and rather than himself providing the hoped for insight, asked Caspari what could be done to “save the hit theory” ([American Philosophical Society, 1947b](#)). There was little question that the Caspari data had created a problem and, in fact, it would be referred to by Stern as a “problem” in future correspondence [Letter of Stern to Noviski – ([American Philosophical Society, 1948](#))]. Demerec would later become a member of the BEAR I Committee, Genetics Panel that recommended the acceptance of the linear dose–response model.

While Stern seemed to accept Caspari’s findings that supported the validity of his control data, he nonetheless challenged the authenticity of the data in other ways. The manuscript that Stern and Caspari developed in the late summer/early fall of 1946 contained a six-page discussion, mostly arguing that Caspari’s (rather than Spencer’s) findings should not be accepted until it could be shown why his threshold-supporting data differed from the earlier linear dose–response findings of Spencer. This position, in and of itself, was problematic in that the two papers had several dozen important methodological differences (e.g., temperature of 18 °C vs. 24 °C, egg-laying suppression vs. enhancement diets, irradiation by X-rays vs. gamma rays, young vs aged sperm, male vs female exposures and numerous other differences – [see [Table 2, \(Calabrese, 2011c\)](#), making it virtually impossible (if not impractical) to resolve the differences.

Even though the Caspari study adopted technical and methodological improvements over the Spencer study and had avoided serious operational errors of the Spencer study (e.g., Spencer’s failure to control temperature, his combining of treatment groups with the same cumulative exposure but with dose rates that differed by up to 2.5 fold, his failure to match control and treatment groups over the same time periods, and his inconsistent calibration of the X-ray machine, etc.) and errors in the modeling of low dose responses (see detailed criticisms – ([Bonnier and Lünig, 1949](#); [Bonnier et al., 1949](#)), it was strangely the Spencer study with its linear dose response that became the gold standard and not the Caspari study.

Discussion in the Caspari paper, as noted above, made it clear that the findings in support of a threshold should not be accepted until the differences between the two papers could be resolved. As untenable as this position was, Stern’s actions were even more inexplicable as he would not place a similar constraint upon the flawed Spencer paper that supported linearity. It is bizarre, if not unheard of, for investigators to ask the scientific community not to accept the validity of their findings until it could be reliably determined why their findings differed from a study of considerably lesser quality and reliability. Moreover, not placing at least the same constraints on the weaker study, for which Stern was also a

co-author, calls into question the investigator’s non-biased and objective approach to research. As a very accomplished scientist, Stern should have known that resolving differences between these two studies was not realistically possible.

Stern’s unusual behavior makes sense when viewed as an attempt to blunt any challenge to the linear dose–response model (i.e., by demanding that the data of Caspari not be accepted). Stern ensured the success of this strategy by sending the Spencer and Caspari manuscripts to his own journal, *Genetics*, and by fully controlling their publication, including the Caspari discussion. There is no evidence that he submitted either of the papers for an independent peer review as the papers were submitted to the journal on November 25, 1947, and published less than five weeks later in January 1948 ([Caspari and Stern, 1948](#); [Spencer and Stern, 1948](#)).

At this point it was not clear whether Muller had seen the Caspari data prior to his Nobel Prize Lecture on December 12, 1946. During the Lecture he disavowed any possibility that a threshold dose response could occur in the induction of mutations by ionizing radiation. He demanded a switch to the linear dose–response model, stating, “there is no escape from the conclusion that there is no threshold” ([Muller, 1946a](#)). Not knowing whether Muller had seen Caspari’s data in support of a threshold model prior to his Nobel Prize Lecture, several science historians with considerable knowledge of Muller and that era were then contacted. Yet, none of these attempts answered the question. Fortunately, substantial correspondence between Muller and Stern, Caspari, Spencer and others was obtained from archival libraries. The archived records revealed that Stern wrote to Muller on September 24, 1946, to request his services in reviewing the Caspari manuscript in preparation for journal submission. A follow-up letter from Muller on September 27, 1946, accepted this invitation and on November 6, 1946, Stern sent the manuscript to Muller at the University of Indiana. On November 12, 1946, Muller acknowledged receipt of both the letter and the manuscript. He also indicated that he had briefly read the manuscript and recognized that the findings supported a threshold dose response, seriously challenging the linear model. Muller strongly encouraged Stern to find the means to undertake a replication study and indicated that he would try to provide a detailed evaluation prior to his Nobel Prize trip to Europe in early December. Clearly, this November 12th letter acknowledged that Muller had seen Caspari’s data, understood the challenge to the linearity model, was not dismissive of the findings and acknowledged Caspari’s competence and the need to repeat the findings (see [Table 1](#) for the series of Stern/Muller correspondence statements).

Muller’s evaluation of the Caspari manuscript occurred five weeks after his Nobel Prize Lecture in the form of a detailed letter to Stern dated January 14, 1947 ([American Philosophical Society, 1947c](#)). Based on this analysis, Muller had not changed his opinion. He unequivocally stated that he could not find any meaningful criticism of Caspari’s work (i.e., “I have so little to suggest in regard to the manuscript.”) and he restated the need to replicate the findings (i.e., “Unfortunately, therefore a replication seems to be imperative.”). Thus, the statements written in private by Muller to Stern were those of a scientist, while his unequivocal public rejection of the threshold model at the Nobel Prize Lecture was deceptive and not without ideological underpinnings. Knowing that uncertainty existed in the low dose zone and that further study was needed, Muller could have acted more forthrightly by pronouncing his conditional rather than categorical support of the LNT model in Stockholm. Even four months later he remained steadfast and continued to advocate his unqualified support for the linear dose–response model. In a presentation to the New York Academy of Medicine in 1947, he stated that “there is then absolutely no threshold dose...and even the most minute dose carries a

Table 1

Letter correspondence demonstrating that Muller had seen and considered Caspari's threshold supportive findings prior to his Nobel Prize lecture on December 12, 1946 (American Philosophical Society, 1946/1947; Calabrese, 2011c).

September 24, 1946 – Stern to Muller:

"Dr. Caspari's report on his work is now being typed and I wonder whether we could bother you with sending you a copy for your new comments."

September 27, 1946 – Muller to Stern:

"Also, I'd be glad to see Caspari's paper too."

November 6, 1946 – Stern to Muller:

"Caspari's manuscript has finally been typed and we would appreciate very much your critical reading of it."

November 12, 1946 – Muller to Stern:

"I have just arrived from an absence of over 2 weeks and find the Caspari manuscript here waiting for me. Unfortunately, it catches me again when I am in a tremendous pressure of work, trying to make up both the trip just passed and for another one to come in a few weeks. However, I see that it is very important and shall do all I can to go through it in a reasonable time, surely before I leave again early in December. I hope that Caspari can wait that long if necessary. In the meantime I wonder whether you are having any steps taken to have the question tested again, with variations in technique. It is of such paramount importance, and the results seem so diametrically opposed to those which you and the others have obtained, that I should think funds would be fourth coming for a test of the matter. It is not, of course, that I doubt Caspari's reliability at all, but only that I naturally share the same doubts which he himself expressed. Of course, I am only judging by the summary and a quick glance through the paper, and have not had the opportunity to read the details."

definite chance of producing a change exactly proportional to the size of the dose" (Muller, 1948).

Muller's statement in a letter to Stern (American Philosophical Society, 1947c) about having "so little to suggest in regard to the [i.e., Caspari] manuscript" may not have been quite truthful, as Muller himself was most likely responsible for the only two changes introduced to the paper prior to its submission to the journal *Genetics*. With the exception of these two changes, the published study in *Genetics* was identical in every way to that paper which was sent to both Muller for his pre-submission review and to the Atomic Energy Commission (AEC) in 1947. In the journal version, the first and most significant change was the deletion of a key sentence in the Conclusion of the 1947 AEC version (Caspari and Stern, 1947). The deleted sentence is as follows: "From the practical viewpoint, the results presented open up the possibility that a tolerance dose for radiation may be found, as far as the production of mutation is concerned" (page 15). This statement indicated support for the threshold dose–response model. The second change was significant in that it added the name of Hermann J. Muller to the Acknowledgments of the published paper. It seems more than just coincidence that the only two changes imparted to the journal version consisted of (1) the deletion of a concluding statement in support of a threshold dose–response model and (2) the simultaneous addition of Muller's name to the acknowledgment section. There should be little doubt that removing the threshold conclusion statement was of profound benefit to Muller as it would help him sustain the ideological dominance of his favored LNT model. Muller clearly had the means, motive and opportunity to mitigate the threat imposed by Caspari's paper on the LNT model. So, was Muller responsible for deleting the key concluding sentence in support of a threshold model? Well, we may never know for sure, but strong circumstantial evidence seems to point in that direction.

In the aftermath of the Nobel Lecture, Stern followed Muller's suggestion to repeat the findings of Caspari. However, his two experienced doctoral researchers, Spencer and Caspari, had left for the College of Wooster and Wesleyan University in Middleton, Connecticut, respectively. Consequently, Stern tapped a new Master's student, Delta Uphoff, a recent graduate of Russell Sage

College of Albany, New York, to replicate the Caspari research (Calabrese, 2011c). Data from her first experiment piqued Stern because her control values for mutation rates were about 40% below those found in the literature, including Caspari's study. Stern expressed his concern to Muller and also asked Muller to share his largely unpublished data with him on variation among controls for the mutation rates of aging sperm in the fruit fly. In a series of letters between Muller and Stern, Muller confirmed that the findings of Uphoff were not reliable and that the unpublished (and published) data were supportive of the Caspari control results. Muller's data led to an acknowledgment in the discussion section of the Uphoff and Stern manuscript (Uphoff and Stern, 1947) that the control group data were not interpretable and that the low control group value was most likely due to investigator bias. Thus, in a rather unprecedented move, Stern was quick to place blame on the inexperienced Uphoff. This manuscript, which importantly acknowledged the assistance of Muller, was sent to the Manhattan Project/AEC where it became classified and publicly unavailable. Thus, the acknowledgment by Stern of Uphoff's unreliable control data, together with the letter exchanges between Muller and Stern regarding the reliability of Caspari's control data, clearly indicated that Muller had strong confidence in the Caspari and not the Uphoff control data (Calabrese, 2011b).

Stern then had Uphoff undertake a follow up replication study. She again reported a similar unacceptably low control group response. As in the first case, the findings were again not interpretable. Finally, in a third experiment that was undertaken, another problem arose. This time it was not the control group, which seemed to respond as expected, but the treatment group whose response far exceeded that predicted by a linear dose–response model. At this point, Uphoff had finished her degree and eventually joined the National Institutes of Health (NIH) as a staff researcher. However, the damage was done to the Stern initiative regarding the Manhattan Project/AEC. Each attempt to replicate the Caspari findings had significant problems. Could anything be salvaged?

In January of 1949, Stern decided to submit a technical note to the journal *Science*, integrating the five major experiments conducted under his direction for the Manhattan Project/AEC. These involved the studies of Spencer and Caspari and the three Uphoff replications. In this *Science* paper, Stern attempted to rescue the first two Uphoff experiments that he already knew had aberrant control groups (Uphoff and Stern, 1947) and, according to multiple letter exchanges (Table 2), Muller also knew. Stern also chose to ignore certain data that were not in support of the linear model (Caspari and Stern, 1947) and, again attacked the Caspari study as aberrant even though nothing had changed except for the occurrence of even more data supporting the reliability of Caspari's

Table 2

Stern–Muller temporal letter exchange concerning the aged-stored sperm control mutation rate [see (Calabrese, 2015a) – supplement for a more complete letter exchange].

Curt Stern wrote a letter to Hermann J. Muller on January 22, 1947 (American Philosophical Society, 1947d) informing him that "At the present time it looks as if our new control data [probably the results of the first three months of the first Uphoff experiment; note that her first month's reading was an especially low mutation rate of 0.005%] for aged sperm are considerably below those of Caspari's." He then asked Muller to "send me your figures on rate of sex-linked lethal in sperm aged several weeks, (most desirably, if you have them, data on three weeks), in comparison to control data from non-aged sperm?"

On February 3, 1947 (Lilly Library, 1947) Muller answered by stating that "... sperm of males which are about a week old and have been copulating freely [as in Caspari's experiment] during that period have only about 0.07 or 0.08 per cent of lethal. Thus the latter sperm, after three weeks, should contain something like 0.28 per cent of lethal."

control group. These multiple flip-flops by Stern were befuddling and surely required explanation, yet none were provided. The inferior Spencer study continued to receive strong support from both Stern and Muller even though, as noted above, it had very significant problems, none of which was noted by Muller in his letters to Stern regarding the research of Spencer, September 13, 1946 (American Philosophical Society, 1946) and Caspari on January 14, 1947 (American Philosophical Society, 1947c).

The *Science* paper of Uphoff and Stern (1949) was beneficial both to the LNT model and to Muller himself as its chief advocate. Stern was successful in artfully molding the interpretations of experimental data to fit the LNT mantra. He achieved this goal while the scientific community remained unaware that he and Uphoff (with Muller's support) had acknowledged just a year earlier that their own findings were not interpretable. Now, in the absence of any new data, these same findings were not only acceptable but also argued in support of the LNT model. And Caspari, who had successfully challenged Stern earlier, now remained silent as his findings in support of a threshold model were being undercut in favor of Muller's LNT model. As for Muller, he must have surely felt relief as he was spared the trouble of having to defend his highly deceptive comments at the Nobel Prize Lecture. Since the *Science* paper (Uphoff and Stern, 1949) was only a short one-page note, consisting mostly of a single table, Stern and Uphoff promised the science community a more detailed follow-up paper that would provide important methodological information and other relevant data. However, Stern and Uphoff never did publish the promised follow-up study and there exists no evidence that their colleagues in radiation genetics ever requested them to do so.

The strategy of Muller and Stern to deceive and obfuscate on the nature of the dose response in the low dose zone was successful. This is evidenced by the fact that the Spencer and Stern paper (Spencer and Stern, 1948) and the *Science* technical note by Uphoff and Stern became the highly influential and commonly cited papers. These "flawed" papers provided the scientific foundations upon which the linear dose response model was justified to the science community and, nearly a decade later, to the U.S. Congress at hearings (Congressional Hearings of 1957) partially inspired by the NAS report of the BEAR Genetics Panel (Calabrese, 2013a; Crow, 1957; Glass, 1957; Joint Committee on Atomic Energy, 1957; Muller, 1957). On the other hand, the technically superior and more relevant paper by Caspari in support of a threshold interpretation received virtually no attention; it was, in essence, unfairly but successfully marginalized. Various leaders in the field repeated false limitations of the Caspari study (Higgins, 1951; Jolly, 2004; Singleton, 1954) that were inspired by the deceptive comments of Stern and Muller e.g., (Muller, 1950b, 1954; Uphoff and Stern, 1949). For example Singleton (1954) echoed that Caspari's study could not be accepted because it had an aberrantly high control group. Ironically, this was Stern's original challenge that already had been so effectively rebutted by Caspari and Muller's own data (see Table 2 for letter exchange between Stern and Muller).

After the *Science* paper, Muller published several papers that repeatedly criticized Caspari's study as being too unreliable because of its high control group data. For example, in his 1950 article entitled "Some present problems in the genetic effects of radiation" in the *Journal of Cellular and Comparative Physiology*, Muller (1950a) provided an explicit characterization of the findings produced by Caspari and Stern (1948). Muller states on page 10 "A recent paper by Spencer and Stern...extends the principle (i.e., one-hit principle) down to total doses of 50 r and 25 r". In the next paragraph, he states: "It is true, in a parallel paper...Caspari and Stern have reported results somewhat deviating from the above." In footnote 1 on page 10 of the article cited above, Muller

adds "Uphoff and Stern have published a report of further work, with doses as low as 50 r, given an intensity as low as 0.0165 r per minute. The results obtained are entirely in conformity with the one-hit principle. A consideration of these results, together with the early work, leads to the conclusion that the deviation first referred to (the Caspari and Stern (1948) findings) was caused by a value for spontaneous mutation rate that happened to be unusually high." Although this repeatedly false criticism by Muller was indeed highly disconcerting, other geneticists seemed too willing and ready to accept it, more or less on 'blind faith' and without proper review and verification. If they had chosen to follow the data originating from Muller himself (Muller, 1945) and his own graduate students (Byers, 1954; Byers and Muller, 1952) as well as others (Graf, 1972; Rinehart, 1969) then perhaps the findings of Caspari, and not of Uphoff, would have received public attention and support. Thus, Muller continued to perpetuate a false view that was discredited by his own statements/data. Shamefully, there is no evidence that anyone challenged Muller on these contradictions. Furthermore, Muller claimed that the research of Delta Uphoff and Curt Stern was "entirely in conformity with the one-hit principle" (Timoféeff-Ressovsky et al., 1935). What Muller neglected to state was that Uphoff's first two experiments displayed an aberrantly low control group responses based on Muller's own extensive data involving some 200,000 fruit flies (Muller, 1946). A letter from Curt Stern to Ernst Caspari (fall 1947) (American Philosophical Society, 1947a) addressed the control group issue. It states: "The radiation data continues to be puzzling. Delta's difference between control and exper[imental group] appears to be due mainly to a much lower control group value than yours. However, Muller informs me that this data give an aged control value close to yours. Thus, my first idea that your results could be "explained away" by assuming that your control value happened to be unusually high, seems unlikely. Rather does Delta's control appear too low". Muller's false and self-contradictory statements about Caspari's findings may be understood within the context of his ideological focus on establishing the LNT model for risk assessment and in the preservation of his legacy – a legacy that would have been severely tarnished if the deceptive remarks he made during his Nobel Prize Lecture had been discovered.

A further example of Muller's duplicity in promoting the LNT concept was his inaccurate characterization of the dose-rate used in the Uphoff experiments (Uphoff and Stern, 1949), which was 0.00165 r/min, i.e., 50 r in 30,240 min or in 21 days) (Uphoff and Stern, 1949). In his paper entitled "Radiation Damage to the Genetic Material" in the *American Scientist*, Muller (1950b) indicated that their research extended "the principle of proportionality of mutation to doses down to doses of 50 r and 25 r and of less than 0.001 r/min with a time-intensity relation differing by over 400,000 times from that of our high intensity dose." By using the incorrect dose-rate of < 0.001 r/min (instead of 0.00165 r/min) Muller (1950b) extended the linear extrapolation over 400,000-fold, some 150,000-fold greater than what the correct dose-rate would have predicted. Just as in the case of validating the Uphoff control groups (discussed above), no one challenged Muller on this point. It is doubtful that Muller's actions was a simple editorial-typo as it involved two discrete changes, removing a 65 and adding a < sign. Furthermore, Muller (1950b) had correctly cited the value as 0.00165 r/min in a previous paper.

4. The NAS BEAR I Committee Genetics Panel

The actions of Muller and Stern (cited above) were critical in persuading the radiation genetics community to adopt the LNT perspective, which was reinforced at multiple levels. By the early 1950s, according to Crow (1995), LNT had become the dominant

view of this group, despite having little support elsewhere. This timing is important as it set the stage for the actions of the NAS Genetics Panel on the Biological Effects of Atomic Radiation, which issued its landmark report on June 12, 1956, and published its technical report in the journal *Science* (Anonymous, 1956a) later that month.

Since the nature of the dose response in the low dose range was a critical issue, it would be important to know how the Genetics Panel debated this issue, what the nature of the debate was, what votes were taken on the general dose response issues, and who were the leading participants in the discussions. The Genetics Panel formally met on November 20 and 21, 1955, at Princeton University and on February 5 and 6, 1956, in Chicago. Transcripts were obtained for both of these meetings. The Panel had a follow up meeting March 1, 1956, with partial attendance and only a meeting summary (i.e., no transcript was taken). Intermeeting communications among Panel members were encouraged via the exchange of working documents and draft materials. These communications were typically preserved in the historical record, and it was generally possible to obtain copies of papers and correspondences of the Panel members on BEAR I from their respective institutional libraries. Although that which was archived varied according to each person, an effort was made to obtain complete sets of information on all Panel members. As a result, copious files on Panel members were obtained, enabling the reconstruction of Panel activity to a high degree.

The transcripts of the Genetics Panel indicate that the members debated neither the nature of the dose response at low doses, the expectations of a linear or a threshold dose response nor any other dosimetric issue. Dr. Tracy Sonneborn from the University of Indiana, a Panel member and colleague of Hermann Muller, wrote a general guiding statement of principles for the Panel to follow; see (Calabrese, 2015a) – Supplementary material. The basic framework consisted of four principles, i.e., that all doses of ionizing radiation were (1) harmful, (2) irreversible, (3) cumulative, and (4) displayed a linear dose–response relationship. No member of the Panel challenged these perspectives. In fact, at the Princeton meeting of the Genetics Panel, Professor Alfred H. Sturtevant from California Tech asserted his disdain for the medical profession that still adhered to an anachronistic belief in the threshold dose response model. Sturtevant stated that he had "no doubt about the correctness of the linear dose response" and that any effort to further document support for it would only be for the "propaganda value" needed to educate and convince the non-geneticists; see (National Academy of Sciences (NAS), 1955) – Transcription, November 21, 1955.

The Panel's single-minded uniformity of belief regarding the nature of the low-dose response was profoundly significant as it tended not only to limit discussion and preclude debate but also to ensure adoption of their preconceived notions. Due to this lack of discussion and absence of debate, the Panel was challenged to identify other activities that could productively fill its meeting times. The Panel Chair, Dr. Warren Weaver of the Rockefeller Foundation, forged ahead and challenged the 13 geneticists on the 17-member Panel to provide estimates of genetic damage to the U.S. adult population given a specific exposure to the gonads. The purpose of this exercise was to see how closely individual estimates of damage might converge among a blended mix of high level expert geneticists who had collective experiences studying an array of diverse populations, including fruit flies, bacteria, paramecia, yeast, human populations and clinical patients, among others. Weaver argued that a greater convergence (i.e., agreement) among individual damage estimates would tend to yield a greater confidence by society in the Panel's scientific conclusions and recommendations. Although one geneticist resigned from the Panel due to overriding academic commitments, the remaining 12

considered the challenge and the need to independently complete the assignment within about one month following the meeting of February 5–6, 1956. Of the 12 geneticists three (Tracy Sonneborn, Clarence C. Little and James V. Neel) eventually decided that there was too much uncertainty for the question to be quantitatively addressed with any degree of accuracy or reliability and that any population-based estimates would simply be misleading. For example, Neel stated that the scientific foundations needed to make such estimates of genetic damage were so uncertain that providing them would be a violation of his obligation to society as a scientist; see the April 6, 1956 letter from Neel to Weaver, cited in Jolly (2004). After the refusal of these three Panel members to participate in the exercise and provide estimates, the nine remaining geneticists may have had similar misgivings, at least to some extent, but nonetheless provided quantitative estimates of genetic damage within the prescribed time; see (Calabrese, 2015a) – Supplementary material.

When the Panel finally published its paper in *Science*, it indicated erroneously that six (instead of nine) geneticists took up the challenge and provided such estimates (i.e., "Six of the geneticists on this committee considered theproblem."). This apparent discrepancy triggered a more extensive assessment of communications among panel members and related information regarding the estimates of damage. Chairman Weaver gave James Crow the task of organizing the submitted material and integrating tables listing the damage estimates of each participating geneticist. As a result of this process, it quickly emerged that there was considerable disagreement among Panel members concerning the identity and appropriate use of methods and assumptions in conducting the assignment. Thus, as one can imagine, confusion about the assignment and the lack of a clear protocol yielded estimates of extreme variability. Panel members were highly uncertain of their own estimates, which often radically disagreed with the estimates of fellow Panel members. In spite of the fact that each geneticist employed the linear dose–response assumption, the results of this exercise led to anything but a convergence. A close reading of all the contributions reveals that some of the "experts" had little idea how to approach the problem. This can be highlighted in the case of James Crow, the last surviving member of the Panel, who died in 2012. For example, on March 29, 1956, Crow stated (Crow, 1956): "I shall use as a minimum estimate a direct extrapolation from *Drosophila* and as a maximum some calculation from the sex-ratio in the Japanese cities. An estimate from mouse data turns out to be just about half way between these, so I shall use it as the most probably estimate." The non-sequiturs inherent in such biological reasoning demonstrate how poorly some of the leading experts addressed this issue. As the other geneticists expressed similar levels of uncertainty and disagreement, it is not surprising then that the Panel would share their documentation with neither external reviewers nor the interested public.

A major problem arose as a result of the extreme variability among the individual estimates. That is, the uncertainty of these estimates would erode public confidence in the Panel's pronouncements. Crow perceived the problem and memorialized his concern in a letter to Chairman Weaver of March 29, 1956: "The limits presented on our estimates of genetic damage are so wide that the reader will, I believe, not have any confidence in them at all." Thus, Crow believed that if the Panel shared its uncertainty with the public then the likelihood of winning their acceptance of any scientific and policy guidance would be seriously threatened. Crow then made a unilateral decision to exclude the estimates of three of the geneticists (i.e., Kaufmann, Wright and Demerec), the three with the lowest estimated damage values; see (Calabrese, 2015a) – Supplementary material for a detailed assessment for each of these three excluded values. Although Crow's decision

markedly reduced the amount of variation within the group, this initial "adjustment" was simply not enough to solve the variability problem. Crow then strongly urged the Panel not to share the six remaining and highly variable assessments with the scientific community and public. The Panel eventually voted on Crow's recommendation, and the majority decided in favor of it, thus essentially eliminating anyone from the interested public or the science community from critically examining the data or the process by which these estimates were derived. While a copy of the voting tally was obtained, specific information on votes of individual members was discovered for four members. Based on their preserved correspondence, (Calabrese, 2015a) – Supplementary material, Crow, Glass, Muller and Sonneborn all voted not to share the data.

The aforementioned analysis reveals that the Genetics Panel deliberately falsified the research record in the *Science* article by reporting that only six geneticists provided estimates of radiation induced genetic damage. This was patently false as nine geneticists provided detailed estimates within the prescribed period of time. There was no expectation and no established protocol for the exclusion of estimates as each geneticist on the Panel was considered an independent world-class expert in his own area of genetics. The person who excluded the three estimates was Crow, who lacked the authority to do so. In fact, the exercise on estimating risk of genetic damage was designed to develop a gage of expert agreement or lack thereof. Removing the three estimates was a deliberate act to obscure and mitigate the magnitude of disagreement and uncertainty that existed among the experts. Furthermore, the report did not even acknowledge that three other Panelists refused to participate in the exercise because too much uncertainty precluded the possibility of making any reliable estimates, (Calabrese, 2015a) – Supplementary material. Finally, the *Science* article contained an inaccurate estimate of response variability in the range of plus or minus ten-fold on either side of the mean. More specifically, the *Science* paper states, "These six geneticists concluded, moreover, that the uncertainty in their estimation of the most probable value was about a factor of 10. That is to say, their minimum estimates were about 1/10, and their maximum estimates about 10 times the most probable estimate". This 100-fold uncertainty markedly misrepresented the range of uncertainty of the six remaining Panel geneticists for estimating the next generation, which had a mean uncertainty value of 756 (312.5 median). See Table 1 of identified individual values in Calabrese (2015a) – Supplementary material.

The Genetics Panel of the NAS, as a group, therefore deliberately sought to misrepresent the research record in their landmark *Science* publication on three distinct aspects. These included: the incorrect statement that only six geneticists provided genetic damage estimates when nine did; the failure to report that three other geneticists refused to provide any estimates at all because of the high level of uncertainty of this exercise; and, finally, the uncertainty range for the six geneticists was given as 100 fold when the mean value was actually 756 fold. These actions of fabrication and falsification by the Genetics Panel were undertaken to ensure that governmental agencies, legislative bodies and the general public would be more likely to accept the Panel's LNT-derived policy recommendations for assessing the risk of ionizing radiation.

5. BEAR I Genetics Panel report – fallout

Following its acts of falsification and fabrication of the research record, the Genetics Panel continued to show its arrogance in the aftermath of the BEAR I Panel and at the start of BEAR II (fall, 1956). In this case, several leading biologists had requested that

the Genetics Panel provide documentation that would explain/support its decision to recommend the adoption of the linear dose–response model for risk assessment purposes, (Calabrese, 2015b) – Supplementary material and Glass (1956). The biologists noted that the BEAR I Panel had proclaimed the correctness of the LNT model, but it failed to provide any written scientific basis for its decision. Since providing documentation to support major decisions is the main mission of any NAS Committee, the BEAR I Genetics Panel, by this standard, clearly failed to perform its mission. However, in a decision that may be difficult to understand, the Panel actually refused to do so, deciding instead to redirect its efforts to identifying research areas for future funding. Furthermore, it is highly unusual, if not astonishing, that the Panel actually informed the President of the NAS, Detlev Bronk that it had decided not to provide documentation to support the LNT recommendation. In fact, no documentation in support of the LNT decision ever existed at the time of the BEAR I Genetics Panel report on June 12, 1956, and now it would have to be written well after the fact – a serious problem in and of itself. Secondly, the Panel members openly noted that they preferred to spend their time identifying research priorities for funding opportunities, some of which would be of interest to their own research laboratories. No evidence has been found to suggest that President Bronk ever objected to the Panel's no documentation decision, which was shared with him in a letter from George Beadle, Chair of the BEAR II, Genetics Panel (Beadle, 1957) on September 11, 1957. Thus, the President of the NAS was complicit in the decision not to require the BEAR Genetics Panel to document its support of the LNT model.

The BEAR I and II Panels consisted of essentially the same individuals except for two changes. The Chair (i.e., Warren Weaver) stepped down so he could award grants from the Rockefeller Foundation to Panel members without an obvious conflict of interest, and one new person (TG Dobzhansky) who had been invited for BEAR I, but was unavailable at the time.

The BEAR I, Genetics Panel released their report amongst a flurry of media attention with front page stories in the *New York Times* (Leviero, 1956) and *Washington Post* (Haseltine, 1956). Other leading venues, including *US New and World Report* (Anonymous, 1956b), *The Saturday Review* (Muller, 1956), *Time Magazine* (Anonymous, 1956c, 1956a), *Science* journal (Anonymous, 1956c), *The Lancet* (Anonymous, 1956f, 1956g) and others, also had articles on the BEAR I Genetics Panel report. The *New York Times* called it the most extensive study ever conducted by such a leading group of experts. Yet, in retrospect the evidence shows that the effort failed in critical ways, especially in not even debating the key question concerning the nature of the low dose zone in the dose–response paradigm. The Panel proclaimed the validity of the linear model at the start and never felt the need to justify this fundamental decision, even following a subsequent challenge by leading biologists. Such inappropriate actions of the Panel continued, as it even deemed it necessary to fabricate and falsify the record in their key *Science* publication to ensure that their views would be accepted. All this was clearly expressed in newly unearthed records of the Panel's correspondence. The dishonesty of the Panel was nothing new as it was simply carrying on a tradition seeded a decade earlier by Hermann J. Muller at his Nobel Prize Lecture.

The explicit deceptions of some Panel members continued even some 35 years after the fact. For example, Panel member and geneticist Bentley Glass (Glass, 1991), in a book review about the Rockefeller Foundation, retold the BEAR I, Genetics Panel story reported in the 1956 *Science* article concerning how the Panel obtained its estimates of genetic damage in the U.S. population. Glass wrote that Chairman Weaver sought to overcome vast disagreements among Panelists by instructing them to return to their hotel rooms and work out their damage calculations individually.

The following day, Glass reports, the disagreements were profoundly diminished and a strong consensus emerged. The story by Glass may well be how he remembered the event but his memory is strongly contradicted by the factual record. The fabrications of Glass started with his "authoritative" quote from Weaver that inspired the geneticists to return to their rooms. The quote does not exist in the meeting transcripts. The story of Weaver sending Panelists to their hotel rooms to work on their estimates and of their returning the next day in triumphal consensus likewise never occurred. In fact, Weaver charged them to return to their respective homes and gave them about a month to work on the estimates. Thus, once again, based on the transcripts and substantial subsequent written communications, Glass bears false witness. Glass's most significant fabrication is that the Panelists actually reached a strong quantitative agreement. The consensus story was not real but faked by Weaver and the Panel as discussed above and detailed elsewhere, (Calabrese, 2015a) – Supplementary material.

The highly regarded Glass, among whose honors included being a President of the AAAS and Phi Beta Kappa, amongst numerous other honors, repeated, therefore, the long established false narrative, reinforcing the LNT mantra well into the modern era of risk assessment and doing so with great appeal to his authority. This is therefore the story of not only how the U.S. and world governments came to adopt the linear dose response for risk assessment but also how its origins were forged by deception, artful dodges and blind faith to become established, preserved, protected and reinforced by those very people (e.g. Genetics Panelists) and organizations (e.g. NAS) that society is supposed to trust.

6. The Rockefeller Foundation and the LNT

In 1954, the Board of Trustees of the Rockefeller Foundation (RF) developed the proposition that it was necessary for the United States (U.S.) to undertake a major assessment of ionizing radiation on humans and the environment. One of their Board members was Dr. Detlev Bronk, who was also serving at that time as the President of the Rockefeller Institute for Medical Research (which would become Rockefeller University in 1965) and President of the U.S. National Academy of Sciences (NAS). Prior to this time, Dr. Bronk had also been the President of Johns Hopkins University and the President of the American Association for the Advancement of Science (AAAS) in 1952. Bronk took the proposal of the RF Board of Trustees to the NAS and received permission to undertake this project as an official NAS activity (Hamblin, 2007). This new project was called the NAS Biological Effects of Atomic Radiation (BEAR) Committee. The project involved six independent technical panels for different areas of concern (e.g., genetics, pathology, oceanography and fisheries, agriculture, meteorology, and waste disposal and dispersal). The panels were created by Dr. Bronk and administratively overseen by the RF.

All six BEAR Committee expert panels were chaired by renowned experts in their respective fields except for the Genetics Panel, which was chaired by Warren Weaver, a mathematician and long-time administrator at the RF (Rees, 1987). Interestingly, Bronk selected Weaver to chair the Genetics Panel and, as such, this selection represented a striking deviation in panel construction and leadership. Although multiple individuals with considerable relevant scientific expertise and strong leadership skills were already on the Genetics Panel, none of them would be selected as Chair. Overlooked in the selection process were: George Beadle, the future President of the University of Chicago (and 1958 Nobel Prize winner); Alexander Hollender, the highly regarded scientific administrator at Oak Ridge; Clarence C. Little, the past President of

the Universities of Maine and Michigan; and Milislav Demerec, Head of Genetics at Cold Spring Harbor.

In the selection of panel members, one suspects that Bronk and Weaver may have intended to "stack the deck" with radiation geneticists who supported the LNT. For example, Ralph Singleton was a radiation geneticist at the Brookhaven National Laboratory who at the time, questioned the linearity hypothesis and reported a non-linear relationship between mutation rate and dose rate, with disproportional increases at higher doses (Singleton, 1954; Richter and Singleton, 1955; Sparrow and Singleton, 1953). In an April 17, 1955 article in the *New York Times*, (Anonymous, 1955b) Singleton challenged the linearity concept for genetic damage stating "there probably is a safe level of radiation, below which no genetic changes occur." Singleton's expertise and the timing and topic of his publications would seem to have easily qualified him for membership on the Genetics Panel, assuming of course that the key objective was to form a panel representing diverse viewpoints to encourage discussion and thoughtful consideration. As it turns out, Singleton was not appointed to the Genetics Panel but to the Agriculture Panel of BEAR I.

The BEAR Panels were the creation of the RF, fully funded by the RF, administered by RF staff and directed by a member of the RF Board of Trustees, who was also President of the NAS. Not only did Dr. Bronk help to conceptualize the project, but he was also part of the organization that funded the project and led the organization that received the funding and oversaw the project, including guiding the selection of panel chairs and their members.

For a long time, the RF was a major funding organization for radiation geneticists, including members of the Genetics Panel. The funding of such members extended over three decades, much of which was during the employment of Weaver and also under his direction. As noted in Wynchank (2011) and prior to the creation of the Genetics Panel, the RF had funded nearly four million dollars to the University of Indiana for research in the area of radiation genetics alone. Such funding supported the research activities of Professors Sonneborn and Muller, both members of the BEAR Genetics Panel.

Weaver was clearly aware of the importance of RF funding to radiation geneticists and showed no reluctance in connecting the Panel's success to opportunities of lavish funding for its members. Weaver specifically stated at the February 5, 1956 meeting of the Genetics Panel that he would "try to get a very substantial amount of free support for genetics if at the end of this thing we have a real case for it. I am not talking about a few thousand dollars, gentlemen. I am talking about a substantial amount of flexible and free support to geneticists", (National Academy of Sciences (NAS)/National Research Council (NRC), 1956) – NAS transcripts, February 5, page 35. As part of his interaction with the Genetics Panel, he prefaced his funding remarks with the statement that "There may be some very practical results – and here is the dangerous remark – don't misunderstand me. We are just all conspirators here together." The remarks of Weaver were blunt and remarkably focused linking the project outcome to the funding interests of the geneticists on the Panel. Such a blatant coupling of funds and outcome were highly manipulative.

Could such an inducement, as grant support, really be persuasive enough to affect the performance, judgment or integrity of esteemed scientists on an NAS Panel? In his 2007 dissertation (Seltzer, 2007), Seltzer sheds some light on this question. He concluded that members of the Genetics Panel saw themselves as funding advocates for radiation genetics (p. 285 footnote 208). Furthermore, it was hoped that the Genetics Panel, which would continue into the foreseeable future, would affect the directions and priorities of funded research in genetics. Seltzer (2007) also further showed that such expectations were in fact evidenced in correspondence between members of the Genetics Panel, i.e.,

Beadle, Dobzhansky, Muller and Demerec. In a letter to Beadle, Demerec ([American Philosophical Society, 1957a](#)) offered a funding plan that could be achieved by “setting aside a fund (let us say, one hundred million dollars), to be administered by some competent organization (such as the National Academy of Sciences) and used during a period of 20 or 25 years to fund already functioning research centers so as to attract and train first rate scientists”. Dobzhansky ([American Philosophical Society, 1957b](#)) responded to this proposal by stating that he would “needless to say, be all in favor (of) \$100,000,000 for research in general genetics.... but I would find it hard to keep a straight face arguing that they (general genetics) must be studied to evaluate the genetic effects of radiation on human populations”. This evoked from Demerec ([American Philosophical Society, 1957c](#)) the statement that “I, myself, have a hard time keeping a straight face when the talk is about genetic deaths and the tremendous dangers of irradiation. I know that a number of very prominent geneticists, and people whose opinions you value highly, agree with me”. Finally, Dobzhansky ([American Philosophical Society, 1957d](#)) responded by saying “Let us be honest with ourselves – we are both interested in genetics research, and for the sake of it, we are willing to stretch a point when necessary. But let us not stretch it to the breaking point! Overstatements are sometimes dangerous since they result in their opposites when they approach the levels of absurdity. Now, the business of genetic effects of atomic energy has produced a public scare, and a consequent interest in and recognition of (the) importance of genetics. This is to the good, since it will make some people read up on genetics who would not have done so otherwise, and it can lead to the powers-that-be giving money for genetic research which they would not give otherwise” ([American Philosophical Society, 1957d](#)).

These shared comments by key members of the Genetics Panel provide previously unknown insights into motivations of the leading radiation geneticists of that era and the group that legitimized LNT for use by society. According to [Seltzer \(2007\)](#), these letters made two points: (1) that the geneticists were quite focused on the viability of their discipline and (2) that they were cognizant of and acted upon opportunities to manipulate the current situation (e.g., to stretch a point) for the purpose of increasing the likelihood of greater funding. It seems as though the persuasiveness of grant funding is more powerful than one could have imagined, even for esteemed scientists.

When viewed from a grander perspective, the RF displayed an undue and unheard of influence over the course of cancer risk assessment within the United States and throughout the world. The RF directed and funded the entire process that resulted in the adoption of the LNT, all hidden within the prestige of the U.S. NAS due to the multiplicity of roles played by Bronk. Weaver used his long-honed knowledge and skills concerning the vulnerability of academics for external grant funding and lured Panel members with funding possibilities on the basis that their area would be seen as important to society. Such manipulations raise serious ethical issues. In fact they paved the way for the very activities that occurred within the Genetics Panel, that is, misrepresenting the research record to enhance its policy recommendations. To ensure a “proper” narrative, Weaver the mathematician, and not one of the geneticists, drafted the final report of the Genetics Panel ([Glass, 1991](#)). At an organizational level, the RF manifested hegemony over the BEAR Genetics Panel, warping and corrupting a risk assessment process that had lasting, social and economic public policy consequences. At an individual level, Bronk’s failure to require the panel to document the scientific basis for the LNT recommendation and the Panel members’ self-serving decision to identify funding opportunities instead of writing the report, together represent unscrupulous behaviors that enabled them to establish the legitimacy of the LNT model without having to

defend their position and, at the same time, optimizing their future funding options.

7. Conclusions

- The recommendation by the U.S. NAS in 1956 to adopt the LNT model was rapidly accepted by governments worldwide and provided the basis for estimating cancer risks from ionizing radiation and chemical carcinogens over the past six decades.
- The recommendations of the U.S. NAS BEAR I Committee, Genetics Panel were ideologically-driven with no written scientific basis provided by the Panel. The Genetics Panel explicitly refused to provide a written documentation when formally challenged to explain their recommendations. Moreover, the President of the NAS became complicit in the Panel’s questionable and irregular actions by taking no corrective action, even after receiving notification by letter of the Panel’s refusal to provide such a report.
- Studies under the direction of Curt Stern at the University of Rochester/University of California-Berkley using *Drosophila* provided the scientific basis for the LNT of the BEAR I Genetics Panel. Detailed re-analyses of these studies has revealed serious flaws in the acute study by Warren Spencer and in key follow up chronic exposure experiments by Delta Uphoff. Curt Stern intentionally concealed critical limitations of the Uphoff findings which had Stern and Uphoff characterize these findings as “uninterpretable”. Stern, in cooperation with Hermann Muller, deliberately misrepresented and marginalized the findings of Ernst Caspari which supported a threshold model.
- The NAS Genetics Panel committed scientific misconduct by falsifying, fabricating and then publishing in the journal *Science* its doctored estimates of human genetic risk to radiation exposures. The Panel’s deceptions were designed to prevent the scientific community and the general public from knowing the profound uncertainties entailed in its genetic risk estimates, thereby insuring the ready acceptance of its policy recommendations.
- Current cancer risk assessment policy and practices are based on fraud and deception by key leaders of the radiation geneticist community and by the U.S. NAS, BEAR I, Genetics Panel. Their deceptions were uncritically adopted by regulatory agencies and the scientific community worldwide and provide the foundation of cancer risk assessment and risk communication messages. The implications of such fraudulent actions are profound and likely to affect: human health risk assessment, adoption and use of new technologies, cost benefit assessments at multiple societal levels, toxic tort actions/decisions, and in the education of the public on vast areas of environmental health and medical treatment practices.

Funding sources

Research activities in the area of dose response have been funded by the United States Air Force and ExxonMobil Foundation over a number of years. However, such funding support has not been used for the present manuscript.

References

- American Philosophical Society, 1929a. Stern letter to Muller, August 8, 1929.
 American Philosophical Society, 1929b. Muller letter to Stern, October 3, 1929.
 American Philosophical Society, 1929c. Stern letter to Muller, October 23, 1929.

- American Philosophical Society, 1946. Muller Letter to Spencer and Stern, September 13, 1946.
- American Philosophical Society, 1946/1947. Curt Stern Papers, Hermann J. Muller File, Philadelphia. (<http://amphilosoc.org>).
- American Philosophical Society, 1947a. Stern Letter to Caspari. Stern Papers, Caspari File, Fall 1947, undated.
- American Philosophical Society, 1947b. Caspari Letter to Stern. Stern Papers, Caspari File, September 25, 1947.
- American Philosophical Society, 1947c. Muller Letter to Stern. Stern Papers, Muller File, January 14, 1947.
- American Philosophical Society, 1947d. Stern letter to Muller, January 22, 1947.
- American Philosophical Society, 1948. Stern Letter to Edward Noviski. Stern Papers, Noviski File, March 19, 1948.
- American Philosophical Society, 1957a. Demerec Letter to Beadle. Milislav Demerec Papers, August 1, 1957.
- American Philosophical Society, 1957b. Dobzhansky Letter to Demerec. Milislav Demerec Papers, August 3, 1957.
- American Philosophical Society, 1957c. Demerec Letter to Dobzhansky. Milislav Demerec Papers, August 9, 1957.
- American Philosophical Society, 1957d. Dobzhansky Letter to Demerec. Milislav Demerec Papers, August 13, 1957.
- Anonymous, 1955a. Dr. Donald R. Charles mourned by Educators. Rochester Democrat and Chronicle. Wednesday, November 30, 1955.
- Anonymous, 1955b. Experts Explode Fall-out Myths, New York Times, 42.
- Anonymous, 1956a. Genetics Panel and W. Weaver, Chair). National Academy of Sciences (NAS), Biological Effects of Atomic Radiation (BEAR). Genetic effects of atomic radiation **123–124**, 1157–1164.
- Anonymous, 1956b. What You Should Know About Danger from X-rays. US News and World Report, June 29, 1956, pp. 44–48.
- Anonymous, 1956c. X-ray Danger. Time Magazine – Medicine, October 10, 1956, p. 67.
- Anonymous, 1956d. Atomic Radiation: The rs are Coming. Time Magazine–Science, June 25, 1956, 64–65.
- Anonymous, 1956e. Biological effects of atomic radiation Science – *News Sci.* **123**, 1110–1111.
- Anonymous, 1956f. Biological effects of atomic radiation. *Lancet* **167**, 1007.
- Anonymous, 1956g. Radiation hazards. *Lancet* **167**, 999–1000.
- Beadle, G.W., 1957. Letter to Detlev Bronk. American Philosophical Society, Philadelphia, PA.
- Bonnier, G., Lünig, H.C., 1949. Studies of x-ray mutations on the white and forked loci of *Drosophila melanogaster*. I. A statistical analysis of mutation frequencies. *Hereditas* **35**, 116–189.
- Bonnier, G., Lünig, H.C., Perje, A.M., 1949. Studies of x-ray mutations on the white and forked loci of *Drosophila melanogaster*. II. A study of the formation of Gynandromorphs and other kinds of mosaics. *Hereditas* **35**, 301–336.
- Byers, L., 1954. Thermal effects on the spontaneous mutation rate in mature spermatozoa of *Drosophila melanogaster*. *Caryologia* **6** (Suppl), 694–696.
- Byers, L., Muller, H.J., 1952. Influence of ageing at two different temperatures on the spontaneous mutation rate in mature spermatozoa of *Drosophila melanogaster*. *Genetics* **37** (5), 570–571.
- Calabrese, E.J., 2005. Historical blunders: how toxicology got the dose–response relationship half right. *Cell. Mol. Biol.* **51**, 643–654.
- Calabrese, E.J., 2008. Why it is important to toxicology and toxicologists. *Environ. Toxicol. Chem* **27**, 1451–1474.
- Calabrese, E.J., 2009a. The road to linearity: why linearity at low doses became the basis for carcinogen risk assessment. *Arch. Toxicol.* **83**, 203–225.
- Calabrese, E.J., 2009b. Getting the dose response wrong. Why hormesis became marginalized and the threshold model accepted. *Arch. Toxicol.* **83**, 227–247.
- Calabrese, E.J., 2011a. Muller's Nobel lecture on dose–response for ionizing radiation: ideology or science? *Arch. Toxicol* **85** (12), 1495–1498.
- Calabrese, E.J., 2011b. Toxicology rewrites its history and rethinks its future: giving equal focus to both harmful and beneficial effects. *Environ. Toxicol. Chem* **30** (12), 2658–2673.
- Calabrese, E.J., 2011c. Key studies used to support cancer risk assessment questioned. *Environ. Mol. Mutagen* **52** (2011), 595–606.
- Calabrese, E.J., 2012. Muller's nobel prize lecture: when ideology prevailed over science. *Toxicol. Sci.* **126** (1), 1–4.
- Calabrese, E.J., 2013a. How the US National Academy of Sciences misled the world community on cancer risk assessment: new findings challenge historical foundations of the linear dose response. *Arch. Toxicol.* **87**, 2063–2081.
- Calabrese, E.J., 2013b. Origin of the linearity no threshold (LNT) dose–response concept. *Arch. Toxicol.* **87**, 1621–1633.
- Calabrese, E.J., 2014a. The Genetics Panel of the NAS BEAR I Committee (1956): epistolar evidence suggests self-interest may have prompted an exaggeration of radiation risks that led to the adoption of the LNT cancer risk assessment model. *Arch. Toxicol.* **88** (9), 1631–1634.
- Calabrese, E.J., 2014b. Response to letter of R.J. Cicerone and K. Crowley regarding “How the US National Academy of Sciences misled the world community on cancer risk assessment: new findings challenge historical foundations of the linear dose response. *Arch. Toxicol.* **88** (1), 173–177.
- Calabrese, E.J., 2015a. Cancer risk assessment foundation unraveling: new historical evidence reveals that the US National Academy of Sciences (US NAS), Biological Effects of Atomic Radiation (BEAR) Committee Genetics Panel falsified the research record to promote acceptance of the LNT. *Arch. Toxicol.* **89** (4), 649–650, and supplementary material.
- Calabrese, E.J., 2015b. An abuse of risk assessment: how regulatory agencies improperly adopted LNT for cancer risk assessment. *Arch. Toxicol.* **89** (4), 547–648, and supplementary material.
- Calabrese, E.J., Baldwin, L.A., 2000a. The marginalization of hormesis. *Hum. Exp. Toxicol.* **19**, 32–40.
- Calabrese, E.J., Baldwin, L.A., 2000b. Radiation hormesis: its historical foundations as a biological hypothesis. *Hum. Exp. Toxicol.* **19**, 41–75.
- Calabrese, E.J., Baldwin, L.A., 2000c. Radiation hormesis: the demise of a legitimate hypothesis. *Hum. Exp. Toxicol.* **19**, 76–84.
- Calabrese, E.J., Baldwin, L.A., 2000d. Tales of two similar hypotheses: the rise and fall of chemical and radiation hormesis. *Hum. Exp. Toxicol.* **19**, 85–97.
- Calabrese, E.J., Baldwin, L.A., 2000e. Chemical hormesis: its historical foundations as a biological hypothesis. *Hum. Exp. Toxicol.* **19**, 2–31.
- Carlson, E.A., 1981. *Genes, Radiation and Society: The Life and Work of H.J. Muller*. Cornell University Press, Ithaca, NY.
- Caspari, E., Stern, C., 1947. The influence of chronic irradiation with gamma-rays at low dosages on the mutation rate in *Drosophila melanogaster*. MDDC-1200. U.S. Atomic Energy Commission, Hathi Trust Digital Library, pp. 1–18. Available at: (<http://www.hathitrust.org>).
- Caspari, E., Stern, C., 1948. The influence of chronic irradiation with gamma-rays at low dosages on the mutation rate in *Drosophila melanogaster*. *Genetics* **33**, 75–95.
- Charles, D.R., 1950. Radiation-induced mutations in mammals. *Radiology* **55** (4), 579–581.
- Charles, D.R., 1961. Tihen J.A., Otis E.M. and Grobman A., Genetic effects of chronic X-irradiation exposure in mice. *Genetics* **46**, 5–8.
- Crow, J.F., 1956. Letter to Weaver. Lilly Library (Sonnenborn manuscripts), Manuscript Department 1956. Indiana University, Bloomington, IN.
- Crow, J.F., 1957. Testimony – Statement of Dr. James F. Crow, Professor of Genetics and Zoology, University of Wisconsin. Hearings before the Special Subcommittee on Radiation of the Joint Committee on Atomic Energy, In: Congress of the United States, 85th Congress, 1st Session, Part 1. United States Government Printing Office, Washington DC.
- Crow, J.F., 1995. Quarreling geneticists and a diplomat. *Genetics* **140**, 421–426.
- Glass, B., 1956. Memo to the Committee on Genetic Effects of Atomic Radiation. American Philosophical Society, Philadelphia, PA.
- Glass, B., 1957. Testimony – Statement of Dr. Bentley Glass, Professor of Biology, the Johns Hopkins University. United States Government Printing Office, Washington, DC.
- Glass, B., 1991. The Rockefeller Foundation: Warren Weaver and the launching of molecular biology. *Q. Rev. Biol.* **66** (3), 303–308.
- Graf, U., 1972. Spontaneous mutations in *Drosophila melanogaster*. *Humangen Hum. Genet* **16** (1), 27–32.
- Hamblin, J.D., 2007. A dispassionate and objective effort: negotiating the first study on the biological effects of atomic radiation. *J. Hist. Biol* **40** (1), 147–177.
- Hanson, F.B., Heys, F., 1929. An analysis of the effect of the different rays of radium in producing lethal mutations in *Drosophila*. *Am. Nat.* **63**, 201–213.
- Hanson, F.B., Heys, F., 1932. Radium and lethal mutations in *Drosophila* further evidence of the proportionality rule from a study of the effects of equivalent doses differently applied. *Am. Nat.* **66**, 335–345.
- Haseltine, N., 1956. Nation's top scientists call for atomic radiation control; fear shorter life expectancy and mentally deficient babies. *The Washington Post*, 14, June 13.
- Higgins, E., 1951. Atomic radiation hazards for fish. *J. Wildl. Manag* **15** (1), 1–12.
- Joint Committee on Atomic Energy, 1957. 85th Congress, 1st Session. Summary-analysis of Hearings 27–29 May, and 3–7 June, 1957 on the Nature of Radioactive Fallout and its Effect on Man. United States Government Printing Office, Washington, DC.
- Jolly, J.C., 2004. Thresholds of Uncertainty: Radiation and Responsibility in the Fallout Controversy (Dissertation). Oregon State University, Oregon, p. 591.
- Kaufmann, B.P., 1947. Spontaneous mutations rate in *Drosophila*. *Am. Nat.* **81**, 77–80.
- Leviero, A., 1956. Scientists term radiation a peril to future of man: even small dose can prove harmful to descendant of victim, report states. *The New York Times*, 1, June 13, 1956.
- Lilly Library, 1947. Muller Letter to Stern. Indiana University, Bloomington, IN, February 3, 1947.
- Muller, H.J., 1927. Artificial transmutation of the gene. *Science* **66**, 84–87.
- Muller, H.J., 1928. The problem of genic modification. *Verhandlungen des V. Internationalen Kongresses für Vererbungswissenschaft (Berlin, 1927)*. *Z. Induct. Abstamm. Vererb. Suppl. Band 1*, 234–260.
- Muller, H.J., 1945. Age in relation to the frequency of spontaneous mutations in *Drosophila*. *Yearb. Am. Philos. Soc.*, 150–153.
- Muller, H.J., 1946a. The production of mutations. Nobel Lecture. *Nobelprize.org*. (<http://www.nobelprize.org/nobel-prizes/medicine/laureates/1946>).
- Muller, H.J., 1946b. Age in relation to the frequency of spontaneous mutations in *Drosophila*. American Philosophical Society, Philadelphia, PA, pp. 150–153.
- Muller, H.J., 1948. Mutational prophylaxis. *Bull. N. Y. Acad. Med.* **24** (7), 447–469.
- Muller, H.J., 1950a. Some present problems in the genetic effects of radiation. *J. Cell. Comp. Physiol.* **35** (Suppl. 2), 9–70.
- Muller, H.J., 1950b. Radiation damage to the genetic material. *Am. Sci* **38** (1), 32–59.
- Muller, H.J., 1954. The nature of the genetic effects produced by radiation. In: Hollaender, A. (Ed.), 1954. McGraw-Hill Book Company, New York, pp. 351–473.
- Muller, H.J., 1956. Race Poisoning by Radiation. *The Saturday Review* 37–39, 9–11, June 9, 1956.
- Muller, H.J., 1957. Potential Hazards of Radiation– Congressional Testimony, Hearings before the Special Subcommittee on Radiation of the Joint Committee on

- Atomic Energy. In: Congress of the United States. 85th Congress, 1st Session, Part 1. United States Government Printing Office, Washington, DC.
- National Academy of Sciences (NAS). 1955. Transcript of the Genetics Panel. National Academy of Sciences Committee to Study the Biological effects of Atomic Energy. First Meeting, Princeton NJ, November 20–21, 1955.
- National Academy of Sciences (NAS)/National Research Council (NRC). 1956. The Biological Effects of Atomic Radiation (BEAR): A Report to the Public, NAS/NRC; Washington DC.
- Oliver, C.P., 1931. An Analysis of the Effect of Varying the Duration of X-ray Treatment upon the Frequency of Mutations (Doctor of Philosophy thesis). University of Texas, Austin.
- Rajewsky, B.N., Timofeef-Ressovsky, N.W., 1939. Hohen-stahlung und die Mutationsrate von *Drosophila melanogaster*. Zeitscher. Indukt. Abstam. Vererb. **77**, 488–500.
- Rees, M., 1987. Warren Weaver 1894–1978 – A Biographical Memoir, 1987. National Academy of Sciences, Washington, DC.
- Richter, A., Singleton, W.R., 1955. The effect of chronic gamma radiation on the production of somatic mutations in carnations. Proc. Natl. Acad. Sci **41** (5), 295–300.
- Rinehart, R.R., 1969. Spontaneous sex-linked recessive lethal frequencies from aged and non-aged spermatozoa of *Drosophila melanogaster*. Mutat. Res. **7**, 417–423.
- Seltzer, M.W., 2007. The Technological Infrastructure of Science (Dissertation). Virginia Polytechnic Institute and State University, Blacksburg, VA.
- Singleton, W.R., 1954. The effect of chronic gamma radiation on endosperm mutations in maize. Genetics **39**, 587–603.
- Sparrow, A.H., Singleton, W.R., 1953. The use of radiocobalt as a source of gamma rays and some effects of chronic irradiation on growing plants. Am. Nat. **87** (832), 29–48.
- Spencer, W.P., Stern, C., 1948. Experiments to test the validity of the linear R-dose/mutation at low dosage. Genetics **33**, 43–47.
- Timofeef-Ressovsky, N.W., Zimmer, K.G., Delbruck, M., 1935. Über die Natur der Genmutation und der Genstruktur. Nachrichten von der Gesellschaft der Wissenschaften zu Göttingen: Mathematische-Physikalische Klasse, Fachgruppe VI. Biologie **1** (13), 189–245.
- Uphoff, D.E., Stern, C., 1947. Influence of 24-hour gamma-ray irradiation at low dosage on the mutation rate in *Drosophila*. MDDC-1492, U.S. Atomic Energy Commission, Hathi Trust Digital Library, pp. 1–6. Available at: (<http://www.hathitrust.org>).
- Uphoff, D.E., Stern, C., 1949. The genetic effects of low intensity irradiation. Science **109** (2842), 609–610.
- Wynchank, S., 2011. The Rockefeller Foundation and its Support of Radiobiology up to the 1970s. Rockefeller Archive Center, Research Reports, Online. (<http://www.rockarch.org/publications/resrep/rroonlinealpha.php>).