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Review Article

More fraudulent history of cancer risk assessment: The US National Academy of Sciences Biological Effects of Atomic Radiation (BEAR) I Genetics Panel used falsified data greatly exaggerating hereditary/cancer risks

Edward J. Calabrese [a,*], Paul B. Selby [b,1]

[a] School of Public Health and Health Sciences, Department of Environmental Health, Morrill I-N344, University of Massachusetts, Amherst, MA, 01003, USA
[b] Retired from Oak Ridge National Laboratory, Oak Ridge, TN, USA

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ABSTRACT

This paper reports that data used by the US National Academy of Sciences (NAS) Biological Effects of Atomic Radiation (BEAR) I Genetics Panel (1956) to estimate risks of hereditary damage in the US population were falsified, greatly exaggerating the risks. These risk estimates were mostly based on the first of many mouse specific-locus experiments of William and Liane Russell, Oak Ridge National Laboratory, which were determined in 1996 to be erroneous by a US Department of Energy (DOE) investigation of scientific misconduct. The basis of the falsification is that William Russell removed data on a large mutation cluster from the control group resulting in a falsely elevated estimate of the induced frequency of radiation-induced gene mutations. While DOE subsequently compelled the Russells to correct the record, these corrections were never retrospectively applied to the Genetics Panel (1956) report, which used the falsified Russell data. Thus, no corrections have been made by the NAS or regulatory agencies, such as the EPA, whose national risk assessment policies/practices for cancer risk assessment were significantly corrupted and overstated by these errors. Based on the discovery reported herein that the Genetics Panel's policy recommendations considerably overestimated hereditary risks based upon Russell-inspired falsified publication, it seems imperative that the Genetics Panel report (1956) published in *Science* be retracted due to inherent falsification-based inaccuracies that continue to impact governmental regulatory agencies, such as the EPA, and the global community that often rely upon the US NAS and regulatory agencies for guidance, as well as the broader scientific community and general public.

1. Introduction

This paper reports a recent "discovery" that the US NAS BEAR I Genetics Panel in 1956 used deliberately manipulated data from the radiation geneticist, William L. Russell, also a Panel member, to affect greatly exaggerated estimates of radiation-induced hereditary risk. The story herein is about why Russell acted in such a manner, how he manipulated a highly prestigious team of US radiation geneticists who also had their own serious version of improbity [1,2,3], how these actions were discovered, how the NAS and EPA institutions have stone-walled any restorative actions to the scientific record, and why the flawed/contaminated but highly influential NAS BEAR I Genetics Panel publication in *Science* [4] requires an immediate retraction.

2. The Selby-Russell dispute: A turning point in risk assessment history

Thirty-years ago, in 1995, key features of William Russell's research opus on the mouse specific-locus test (SLT) were threatened with a claim of research falsification and scientific misconduct by Paul B. Selby, a long-time close associate of Russell and his equally prestigious wife, Liane. The previous year, Selby had inadvertently discovered a series of data irregularities in the Russell research files as he was in the process of assisting William and Liane Russell in the electronic data storage of their five decades of research within Oak Ridge National Laboratory (ORNL). What Selby [5] discovered was a pattern of the Russells' data interpretations that excluded clusters of spontaneous mutations from their

* Corresponding author.
E-mail addresses: edwardc@umass.edu (E.J. Calabrese), pbs@mac.com (P.B. Selby).
[1] Home Address: 4088 Nottinghill Gate Road, Upper Arlington, OH 43220, USA.

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publications and computer databases. Selby became suspicious of this process while transferring scores of massive databases from a mainframe computer (for which the data management software was being discontinued) into more efficient form for use on personal computers. Selby had access to substantial additional information about the experiments in their highly restricted research files, which his suspicions caused him to investigate.

Selby [5] reported that the exclusion of the cluster mutations in the control group started with the first major publication of Russell [6], that is, about 45 years earlier than Selby's troubling discovery. The Russell research plan was an ambitious scientific gamble by Alexander Hollaender, Russell's Oak Ridge National Laboratory (ORNL) supervisor, since the research would be quite expensive, involving millions of mice in a unique approach designed to permit the unequivocal phenotypic detection of recessive mutations in the first generation after parental exposures to ionizing radiation. (Decades later these "mega-mouse" experiments were also applied extensively to numerous chemicals.) In Russell's 1951 professional/career make or break paper, not only did his experimental system work but his mouse model, with no mention of the complication of a huge cluster of spontaneous mutations in his concurrent control, was 15–20 times more susceptible to induction by X-rays of transgenerational/reproductive cell gene mutations than the long time Gold Standard, the fruit fly model [6]. Thus, building upon this deception from 1951 onward–and with additional such clusters occurring in his subsequent experiments including at least one cluster (an especially large one) being found in 1955–the progress of Russell and his team was one of considerable growth, with some very notable contributions.

Prior to the Russells' major new advance, the field had been dominated by *Drosophila* geneticists, led by Hermann J. Muller, who had received the Nobel Prize only five years before in 1946 for being the first to induce gene mutations with X-rays. Now it appeared that the field would have a unique mammalian model with far greater extrapolative potential for human biology and quantitative risk assessment. The Russell research represented a type of "sea change" for radiation genetics, a true changing of the guard with mammalian geneticists led by the Russells likely to take on a far more prominent role in research and governmental advisory activities than previous *Drosophila* studies.

This was the research environment that Paul Selby entered as a graduate student working under the direction of William Russell in the late 1960s. After about thirty years of collaborating directly with William and Liane Russell, Selby thought that he was familiar with the entire research enterprise of the Russells, but he was mistaken. The accidental discovery by Selby of the well-hidden cluster mutations in various studies, and their exclusion from publications, was extremely puzzling and unsettling to a now very experienced Selby, leading him to suspect a possible serious violation of research ethics. Without confronting William and Liane Russell, Selby spent many months carefully reading the past publications of the Russells and doing computer simulations of their experiments to reach a decision as to whether this matter was so serious that people at a high level in DOE must be contacted. In June of 1995, Selby contacted leadership within the DOE with his substantial accusatory findings, leading to a formal evaluation by the DOE with four external international genotoxicity experts.

In his 2020 recounting of the Selby-Russell controversy, Selby [5] summarized the key elements, or what would become the basis, of the BEAR I Genetics Panel's use of the Russell mouse mutation data. Here are the essential elements summarized therein: male mice were administered a dose of 600 Roentgens (R) of X-rays at a dose rate of about 90 R/min in Russell's initial specific-locus experiment. Russell ensured that any induced mutation took place in only stem cell spermatogonia by only observing offspring sired after the long sterile period. According to Russell's [6] first published preliminary results, he had found "53–54" specific locus mutations in 48,007 offspring in the radiation exposed experimental group. In contrast, he reported only two specific-locus mutations in the 37,868 offspring in the concurrent control. From those data Russell [6] calculated "a mean induced, or irradiated minus control, mutation rate of (25.0 ± 3.7) x 10^{-8} per roentgen, per locus".[2] Even though the experiment was completed within a few years, the final results were not published until 1958 [7]. At that time Russell indicated that the mutation frequencies were 111/119, 326 (0.00093) (9.3 X 10^{-4}) and 6/106,408 (0.000056) (5.6 X 10^{-5}) in the treatment and control groups, respectively. In his write up, Selby explicitly stated that "these results were of great interest to committees that made early attempts to estimate hereditary risk of radiation in humans." This was certainly the case with the US NAS BEAR I Genetics Panel. In fact, the data that Russell shared with the BEAR I Genetics Panel regarding the radiation-induced mutation frequency were obviously the data published in his 1951 paper [6]. Indeed, in his own risk calculation made for that committee, which was dated February 24, 1956 [8], Russell applied his induced mutation frequency of 25 x 10^{-8}. Selby [5] in 1995 discovered that the actual results from Russell's first experiment, which had major influence on the BEAR I Report, were vastly different, with the final experiment having 90 additional mutations in the control group in addition to the 6 reported in 106,408 offspring. From the breeding records showing this large cluster and the date of the meeting at Cold Spring Harbor at which Russell [6] presented his preliminary findings, it is known that part of that cluster was discovered before the meeting. Also, long before the BEAR I Genetics Panel met, all data from that first experiment had been collected and at least one other extremely large spontaneous cluster had occurred—and not been reported—in another experiment (in 1955, this time in the experimental group). It is obvious that Russell chose not to make the committee or the scientific community aware of this unforeseen complication, and that as more clusters occurred in experiments on males—each one eventually known to be produced by a "masked mosaic" parent [9]—the Russells followed their procedure of not reporting them. Selby in late 1994 [5] realized that the ORNL historical control of 28/531,500 that he and others had used for comparisons for decades was fraudulent. That obviously meant that hereditary risks of radiation had been overestimated—possibly to an extreme degree.

Thus, one can see how the BEAR I Genetics Panel would have proceeded with an assumed strong radiation treatment effect, as well as the consternation of Selby regarding his inadvertent discovery of the additional 90 mutations. The complicated situation that ensued, following Selby's discovery of the unreported event and other similar events by the Russells and their potential risk assessment significance, is what is referred to as the Selby-Russell Dispute in this paper. The present paper focuses on the fact that the BEAR I Genetics Panel from February to May 1956 utilized the control group with the now recognized incorrect 2/37,868 control mutation rate as a basis for their risk estimates, which would profoundly affect the subsequent policies and practices of the US EPA. This error was corrected some 45 years later following a demand of the DOE that resulted from a formal evaluation of the Russells' research methods and data as occurred in the Selby-Russell Dispute as described below.

The Selby-Russell controversy has been summarized in considerable detail by Selby [5]. In brief, as a result of the investigation by the DOE, William and Liane Russell acknowledged committing serious scientific

[2] Procedure for deriving Russell's mutation rate.
Experimental Group for 7 loci and 600 r: 53/48,077
Control Group for 7 loci: 2/37,868

Experimental mutation frequency per locus = 53/(48,077 x 7) = 0.000157
Control mutation frequency per locus = 2/(37,868 x 7) = 0.00000755
Induced mutation frequency per locus = 0.000157–0.00000755 = 0.000150
Induced mutation frequency per locus per R = 0000150/600 = 0.000000248 = 25 x 10-8.

errors and were compelled by the DOE to correct their long-standing misrepresentations [9,10]. The Russells therefore admitted that their male and female control groups were in error by 120 % for males and females combined. By suppressing the control group values by 120 %, the Russells had falsely elevated radiation induced mutation risks—with this distortion being carried over to Muller's claims about genetic deaths and cancer risks. A parallel analysis by Selby [11,12] indicated that their errors were far greater than they admitted, being in the 5-7-fold range.

In practical terms, the Russell action paved the way to obtain misleading, that is, false, significant mutational effects at lower doses, a factor that can markedly affect risk assessment and public policy decisions. When mouse studies of Russell using 37.5 and 86.0 R were adjusted for the more accurate control group data, no statistically significant treatment effects were observed [13]. These manufactured changes permitted the conclusion of a theoretical decrease of the dose of radiation estimated to double the background mutation rate from over 100 R to about 40 R using the Russell adjustment factor. If a compromise of the Russell and Selby figures were used it would decrease the radiation-induced mutation effects per rad by 50–75 % even assuming a linear dose response model, increasing the doubling dose to the 150-200 R range. However, in practical experimental terms the correction of the Russell data suggests that the radiation exposure at low doses exhibits a practical threshold with hormesis being a viable hypothesis. The actions of Russell, therefore, created the means to change the rules of the research and the evaluation scheme, making it far easier to claim statistical significance when it had not been achieved [14], giving greater plausibility to linear modeling when it was not warranted, even raising it to a default status, and creating public policies based on a "precautionary principle" philosophy in which the data supporting such a decision were unethically compromised.

The mistakes and the corrections, regardless of which corrections were used—those by the Russells or Selby—both show that the guidelines of EPA for cancer risk assessment policies which were born from the recommendations of the BEAR I Genetics Panel reports [4,15] as published in *Science* need to be corrected. One would have thought that this should have significantly impacted the toxicology and risk assessment communities and regulatory agencies, especially in the areas of hereditary and cancer risk assessment. However, it failed to do so, falling mostly on deaf ears. This was principally due to the fact that the Russells' research was unique, massive and could only be conducted within a major governmental research facility, such as ORNL. Therefore, most genotoxicity researchers, while appreciating the scope and range of the Russell findings and their general significance, did not do research in this significant but narrow research area with its own version of uniquely technical features. In other words, the Russells had few, if any, peers in the US genotoxicity community since no other laboratory was doing similar research.

The DOE-mandated Russell corrections likewise had the potential to challenge the central dogma of regulatory agency toxicology, that is, its acceptance of the Linear-Non-Threshold (LNT) model. The Russell data were unique and powerful, being based on vast sample sizes, getting into the several millions of mice. By using a mammalian model, the Russell approach was expected to guide regulatory agencies for hereditary and cancer risk assessment based on its scientific value and mutational uniqueness, improved human relevance and massive statistical power. This was the professional dream of Hollaender, and the Russell research was a major key to his career success and the image of ORNL in the scientific world, and in the governmental funding area. It was at the core of the massive investment by the DOE into the Russell research plan.

The DOE-mandated corrections by the Russells [9,10] clearly and unknowingly slipped past the regulatory and scientific communities, having shown no impact on key policy areas that they could have affected. The incorrect findings, analyses and interpretations of the Russells were never highlighted, clarified and/or corrected in the peer-reviewed scientific literature. In retrospect, a strong case could be made that the NAS and EPA were successful in their orchestration of a modern day administrative driven bureaucratic cover up, working side-by-side in a malignant-like and highly self-serving manner. Likewise, the once scientifically self-righteous DOE, having forced the Russells to correct the research record, was now happy to rid itself of this terrible "Selby" problem without doing the necessary follow up corrective and educational activities. In fact, the groups that were affected most, by far, were the US NAS that was actually responsible for the disastrous *Science* publication and the US EPA that embraced it as their longstanding institutional "Bible", with each never making an effort to correct the record. These organizations simply let the strikingly false information continue to resonate and contaminate the scientific record, including the journal *Science*, where the NAS BEAR Panel had published their findings [4]. Given the highly prestigious nature of the NAS and *Science,* their reputations would have, in fact, enhanced the longstanding acceptance of the unreliable Russell findings.

3. Re-discovering the Selby-Russell dispute and attempts to correct the record

Some 20 years after Selby challenged his former academic mentor (William L. Russell) and ORNL research supervisor (Liane Russell), Calabrese [13] analyzed the Russell-Selby conflict in considerable technical depth and determined that correction of the Russell [9,10] errors based on their PNAS papers yielded a threshold dose response for ionizing radiation induced gene mutation for male mice and a possible hormetic effect for female mice. This Calabrese [13] directed Russell-based corrected version had the potential to reverse the LNT conclusion of nearly 40 years before. In fact, had Russell correctly reported his findings from the start, going back to 1951, his data would have supported the adoption of a threshold dose response model by the 1956 BEAR I Panel and carried through to the 1972 BEIR I Committee [16] for mutational/cancer responses, rather than the LNT model which was adopted.

4. The Russell deceptions: means, motive and opportunity

At the core of the Russell deception was his recognition that his early claim—based on the initial experiment with the hidden massive complication of the cluster in the control—that his mouse model was far more sensitive to the induction of mutations than the competitive fruit fly model of Muller gave him a huge advantage. By removing the control group cluster mutations, Russell found his future, promoting a model that falsely appeared to be 15–20 times more sensitive than its rival fruit fly [17].

Thus did the hidden and far-reaching secret of William Russell start. He surely had the continuing means, motive and opportunity to execute and achieve his professional goals of scientific prominence. The scientific hegemony-like data control strategy of the Russells would continue without discovery until Selby revealed it some 45 years later, to the shock of all the key players: Selby, William and Liane Russell, and the DOE. This disaster crystallized as a so-called "perfect scientific storm". We see a uniquely talented husband and wife, who could have been successful anywhere, leading a project that no one else in the entire US was working on and therefore had no serious critics or direct rivals, with a supervisor in Hollaender who wanted great personal success and would be inclined to overlook the failings of his superstar Russell team. (It seems unlikely that the Russells ever informed him of the big problem.) In fact, Calabrese and Selby [18] have also reported that Russell and Arthur Upton, future Director of the US National Cancer Institute, covered up a massive negative mouse radiation cancer study in the late 1950s. Thus, Russell had a high degree of expertise when it came to using scientific deception to advance his career or ideological interests. William and Liane Russell also worked at a government laboratory with a history of enriching the U^{235} used in the first nuclear bomb and thus where scientific secrets were tightly guarded. William and Liane Russell, thus, were perfectly insulated. That is, until their long-time trusted

colleague, Paul Selby, was permitted to see into their secret world in his effort to preserve their scientific legacy. Then the secret started to unravel, with Selby now becoming the target of their defense, as he would soon lose his research position in a uniquely flawed scientific whistle-blower action that caught the DOE administration by surprise, not knowing how to proceed.

While I (EJC) had heard some unverified aspects of the Russell story in the late 1990s from several individuals, it never led to any follow up as I placed it more in the realm of scientific speculation and acute interpersonal conflicts, which sometimes occur, both being zones I was strongly inclined to avoid. However, nearly 20 years later, I accidently came across a one-page unpublished document in my files from a very well-known but recently deceased nuclear physicist, Ted Rockwell, relating to the topic, highlighting Selby, the Russells and their festering dispute along with enough accompanying substance that made me curious. I knew Rockwell reasonably well, his history, achievements and reputation, and had multiple conversations with him in the past about radiation hormesis. He was a person of considerable substance and objectivity. So, I (EJC) then decided to learn more. As a start I contacted Selby and Liane Russell, as William Russell had been long deceased. While Liane Russell politely, but firmly, declined to discuss the incident with me, I ended up having a 12-h telephone conversation/interview with Selby over several days with me taking copious notes, addressing questions about his life story, his career and the Selby-Russell dispute. Thus, I (EJC) decided that I needed to learn as much as possible about the life and science of William Russell, the history of radiation genetics research at ORNL, the eventual Selby-Russell dispute, as well as make a detailed attempt to understand and untangle the science underlying their dispute, not having any sense of what would be learned and where it would lead.

My (EJC) focus on the Russell-Selby issue at that time was directed toward its risk assessment implications and the BEIR I (1972) and subsequent BEIR Committee assessments. I (EJC) was also interested in other significant findings of Russell such as the occurrence of genetic damage repair, a discovery that resulted in William Russell being nominated several times for the Nobel Prize [13]. Even though I had backtracked the Russell corrections only as far as BEIR I [16], I (EJC) knew that Russell failed to report the gene cluster mutational findings as early as 1951. At that time, I (EJC) had not thought to link the Selby-Russell debate to the 1956 BEAR report.

My (EJC) interest in the BEAR I Genetics Panel activities at that time targeted the Panel's decision to report the estimates of ionizing radiation damage from only six geneticists on the Panel when estimates were provided by nine [19–24]. My (EJC) considerable historical reconstruction of Panel activities via meeting transcripts and hundreds of letters and memos indicated that the Panel, as led by Warren Weaver and James Crow, deliberately withheld legitimate mutation risk estimates that revealed the occurrence of massive uncertainty amongst the Panel. Their quick removal of the two most variable and divergent mutational estimates (i.e., Demerec [25] and Wright [26]) would ensure that recommendations of the Panel would look much more reliable, thus more likely to be seriously considered and not dismissed. These behaviors led me to assert that the entire complicit BEAR I Genetics Panel committed scientific misconduct via research falsification and that their publication in *Science* should be retracted. This created a scientific storm of controversy [27]. In the end, the then editor of *Science* (and now President of the NAS), Marcia McNutt, declined to act, on the basis that all the principals were dead and could not explain their actions and defend themselves. She made this decision even though I was able to show that there was historical precedent for such activities, with a prominent example being the Nobel Physics Prize research of Robert Millikan. At that time McNutt was a finalist for the presidency of the NAS with her name posted on the NAS website. Yet, she inexplicably did not recuse herself from this decision, as it seemed to create an obvious and serious conflict of interest. The decision of McNutt not to recuse herself should have been disqualifying for the position of NAS president unless appropriately clarified [27].

5. The new "discovery"

Now nearly a decade later, while finalizing a new paper on the BEAR I Genetics Panel's historical impact, I made the "new' discovery that revealed a direct connection between the early flawed mouse specific-locus research of Russell and the LNT risk estimates of the BEAR I Genetics Panel, something that I had simply not recognized until 2025.

This discovery occurred as I was critically reviewing the precise methods used by each of the nine BEAR I Genetics Panel members who provided estimates of US population genetic damage due to a presumed exposure to 10 R. It soon became clear that the falsified estimate of the Russells was the basis for many of the derived risk estimates. The present paper shows that the DOE-based discredited Russell estimates were used by the NAS BEAR I Genetics Panel and were central to their estimates of risk. In fact, the estimates of leaders, such as George Beadle [28], Bentley Glass [29], James Crow [30,31], Hermann J. Muller [32], and others, relied heavily upon the incorrect estimate of the induced mutation frequency per roentgen per locus by Russell [6], thereby inflating their risk estimates by some 120–500 % for children (F1 generation) of exposed parents for the US population as well as their two other endpoints (i.e., genetic damage at ten generations and for all descendants – termed total genetic damage) [31]. This procedure employed an induced mutation rate expressed per locus per R from the mouse data of Russell and use of *Drosophila* ratios of overall lethals plus sublethals to deleterious mutations as well as other data and assumptions. These data were employed to estimate the total number of mutations/genetic deaths (i.e., Muller's concept and terminology), using a type of mouse-fruit fly hybrid model for risk estimation. In the case of Russell [8], he used an alternative method for estimating hereditary risks, not following the rather well-worn path of the above noted panelists. However, one of the terms used in his risk calculation was his induced mutation frequency of 25×10^{-8} per roentgen per locus. Russell used only the mouse—that is, no data from flies—to predict the risk. The only estimate of first-generation damage in mice that he relied upon was from his third specific-locus experiment with a 300 R acute exposure in which the mean litter size of the irradiated group at weaning was only 96 % that of the control—suggesting that 4 % of the offspring had died between conception and weaning. He argued that all serious phenotypic effects caused by induced recessive lethal mutations would be within the range of one order of magnitude below 4 % and one order of magnitude above 4 %.

The question may be raised as to why most of these geneticists independently adopted an interspecies hybrid model, that is, a mouse-fruit fly combination risk predictor. The most likely reason was that the mouse data were considered approximately 15 to 20-fold more sensitive than the fruit fly for radiation-induced mutation [17] based on the early Russell deception [6]. The use of the more sensitive mouse model would yield far greater population risks based on their LNT application while also likely enhancing the opportunity for greater academic research funding [19]. In addition, the mouse data was inadequate concerning understanding the relationship of lethal and semi-lethal mutations to the production of deleterious mutations. These two factors led panel members to utilize data from fruit flies concerning such things as the ratio of "semi-lethal" mutations to "deleterious" mutations while combining it with the estimate of Russell for the induced mutation rate in mice [33]. In the case of the Panelists, they used essentially the same estimates from the Russell data. However, there was potential interindividual Panelist variability in their selection of fruit fly mutation type ratios. They also introduced different choices such as the number of assumed gene loci, which ranged considerably from as low as 5000 to 100,000. Russell's estimates, which were based only on mammals, assumed 20,000 gene loci. There were other different parameter estimates amongst the panelists, such as whether and how to account for deaths of affected offspring over generations. All parameter

selections were at the computational mercy of the most fundamental assumption, which was that the Russell radiation-induced mutation rate, which became a multiplier affecting other parameter assumptions, was based on his actual data. Surely it never crossed anyone's mind that there might have been a horrendous complication in his control group data that was swept under the rug.

6. Action needed

The NAS BEAR I Genetics Panel estimates therefore have been in error since 1956. Its final assessment of the risk, and that there was reasonably good agreement within the Panel, was widely promoted by many of the Panelists who published separate subsequent accounts of its policy recommendations and risk assessment implications. Many of the Panelists also testified to the US Congress within a year of the 1956 BEAR publication, further metastasizing the uncorrected errors and their widespread implications.

As noted, when the Russell errors were attempted to be corrected based upon the Russell (1996 and 1997) and Selby (1998a,b) papers, no subsequent corrections or clarifications were made for any of the three NAS reports [i.e., technical report in *Science* [4], "THE BIOLOGICAL EFFECTS OF ATOMIC RADIATION—SUMMARY REPORTS" [15] and "THE BIOLOGICAL EFFECTS OF ATOMIC RADIATION–A Report To The Public" [34]. There was just the reporting in various non-governmental papers that there were errors and corrections offered by the Russells as demanded by the DOE, with Selby publishing his own version of what he thought was the more correct interpretation. The first known attempt to make a corrective adjustment that affected the critical risk assessment implications of the Russell corrections was published by Calabrese [22] with the focus being on the 1972 BEIR Committee report with no consideration given to the BEAR I Genetics Panel 1956 report. The Selby-Calabrese [14] publication showed that there is no longer support from specific-locus data for the LNT and that there appears to be a threshold for specific-locus mutations at about 100 R for dose rates of 0.0007 R/min through 0.8 R/min, and that that threshold might be considerably higher.

The present paper reports that the BEAR I Genetics Panel adopted the fraudulent Russell mouse specific-locus mutational data. Those Genetics Panel estimates therefore were based on the Russells' highly distorted data. In the present situation, William and Liane Russell were challenged on those matters by Selby and the DOE when they were alive. The Russells participated in the DOE procedures that have been preserved. The prior argument of McNutt with respect to my (EJC) earlier retraction request that related to investigators not having the opportunity to defend themselves therefore isn't relevant because the Russells were publicly charged, defended themselves and were forced to admit their errors in a publication. The expert panel investigating them inexplicably did not accuse the Russells of ethical and research misconduct, possibly because the Russells committed to correcting the errors in the scientific literature and perhaps the Committee felt sorry for the now elderly (85-year-old) William Russell, who may have presented a sympathetic figure, and whose long career and other accomplishments they respected (as do we). The issue of ethical and research misconduct by the Russells has been addressed by Calabrese and Selby [18].

The NAS BEAR I Genetics Panel unknowingly adopted the falsified estimates of William Russell, "one of their own", without demanding or even requesting to see the original data. Given the dishonesty of the entire Panel with respect to the degree of scientific uncertainty concerning its estimates, why would they think it was necessary to scrutinize the data of Russell prior to accepting it for US policy? They did not.

The present paper asserts that the DOE expert panel that required corrections by the Russells should have demanded that corrections be made in the key NAS documents that were affected by the falsified Russell estimates. Alternatively, perhaps that should have been the responsibility of the DOE after reading the official report of the investigation. Since the DOE, the Russells, NAS, EPA and editors at *Science* have not acted to make such changes, it is necessary to retract the 1956 NAS BEAR I Genetics Panel *Science* publication [4] due to its major historical and continuing significant influence on global regulatory agencies and the broader scientific communities. This unresolved issue affects many deeply ingrained scientific and public policy areas.

CRediT authorship contribution statement

Edward J. Calabrese: Writing – review & editing, Writing – original draft, Conceptualization. **Paul B. Selby:** Writing – review & editing, Conceptualization.

Use of generative AI and AI-assisted technologies

No AI software was used in the preparation of this manuscript.

Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Edward Calabrese reports financial support was provided by US Air Force. Edward Calabrese reports financial support was provided by ExxonMobil Foundation. If there are other authors, they declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Data availability

No data was used for the research described in the article.

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