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James V. Neel and Yuri E. Dubrova: Cold War Debates and the Genetic Effects of Low-Dose Radiation

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Abstract. This article traces disagreements about the genetic effects of low-dose radiation exposure as waged by James Neel (1915–2000), a central figure in radiation studies of Japanese populations after World War II, and Yuri Dubrova (1955–), who analyzed the 1986 Chernobyl nuclear power plant accident. In a 1996 article in *Nature*, Dubrova reported a statistically significant increase in the minisatellite (junk) DNA mutation rate in the children of parents who received a high dose of radiation from the Chernobyl accident, contradicting studies that found no significant inherited genetic effects among offspring of Japanese A-bomb survivors. Neel's subsequent defense of his large-scale longitudinal studies of the genetic effects of ionizing radiation consolidated current scientific understandings of low-dose ionizing radiation. The article seeks to explain how the Hiroshima/Nagasaki data remain hegemonic in radiation studies, contextualizing the debate with attention to the perceived inferiority of Soviet genetic science during the Cold War.

Keywords: Radiation studies, Low-dose radiation, Genetic effects, Cold War, James V. Neel, Yuri E. Dubrova

Introduction

Since the March 2011 nuclear disaster at Fukushima, a number of popular and scientific articles have addressed its potential health con-

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sequences. In a front-page discussion published in the *New York Times* on the relationship between low-dose radiation exposure and the risk of different forms of cancer, journalist Denise Grady suggests that "scientists disagree about the effects of very low doses of the sort that may have occurred in Japan" (Grady, 2011, p. 1). She bases this perception on an interview with Dr. Evan B. Douple, who studies atomic bomb survivors and is associate chief of research at the Radiation Effects Research Foundation in Hiroshima, who points out differences between the Hiroshima and Fukushima cases: "The [Hiroshima] survivors received their entire doses all at once to the full body, but exposure from the reactors may be gradual." The article mentions the survivor data collected by the Atomic Bomb Casualty Commission (ABCC) as central to interpreting the harm done by radiation exposure. It also offers a hyperlink to the Radiation Effects Research Foundation (RERF), the foundation that succeeded the ABCC, and that continues to produce research on the survivors of Hiroshima and Nagasaki as well as their offspring.

This *New York Times* article and many others like it share the assumption that what we know about Fukushima, we know only from what we understand about Hiroshima. Yet the genetic effects of low-dose radiation have vexed scientists since at least 1945 (Lindee, 1994), and a number of scientists (e.g., Richardson, 2012; Wing et al., 1999; Walker, 1994) have argued that the harm caused by radiation is underestimated in the Hiroshima and Nagasaki data. They cite a number of earlier scientists who have been critical of these studies, among them Gofman (1981) and Bertell (1985), who also disagree with the findings of the RERF. Gofman and Bertel concluded, essentially, that the multiple conflicts of interest involved in the professions of radiation health and health physics (also known as the physics of radiation protection) have contributed to this underestimation. At the other extreme from this position, there now exist a few scientists and professional associations who interpret the Japan data as having overestimated the risk from low-dose radiation, including Roger Clarke, President of the International Commission on Radiological Protection (Moore, 2002).

We approach these still contemporary debates by focusing on two key figures, James V. Neel and Yuri E. Dubrova, well-respected scientists trained on opposite sides of the Cold War. By exploring their biographical and historical commitments in terms of their generation, training, experience, and methodological predilections, we seek to explain how – and with what potential pitfalls – we have come to our

current privileging of certain knowledge about radiation risk. Petryna (2002, 2004) argues that Western scientists dismissed many scientific studies produced locally in the aftermath of the Chernobyl disaster because it was believed that these findings were compromised by the local political and economic situation encountered on the ground. Western nuclear experts were also concerned with how Chernobyl would affect future nuclear accidents. Finally, both Russian and Western authorities had parallel interests in denying the extent of radioactive pollution. What we are addressing here are different contested territories of purported scientific knowledge: the science produced locally by medical personnel requires differentiation from the expert science working at the level of populations and carried out by genetics researchers from the East and West. These latter are large-scale international expert studies and they are decidedly "not local." They are the studies that concern us here.

We argue that the elevation of the U.S.-constructed Japan data and research and the relative denigration of the Soviet-constructed Chernobyl data and research in genetic scientific circles during and after the Cold War have co-configured our contemporary understanding of the genetic effects of low-level ionizing radiation exposure. We further argue that the rise of the anti-Mendelian version of genetics known as Lysenkoism in the Soviet Union between the late 1920s and early 1960s, created a long-term effect on how American geneticists – including James Neel – perceived Soviet science. Western commentators have long used the Lysenko affair (discussed below) as a metaphor for the politicized and inept qualities of Soviet science. In the case of James Neel, we argue that the Lysenko period marked his formative years and ultimately his understanding of Soviet genetics science. As a key scholar in American genetics, Lysenkoism may have affected Neel's approach to Yuri E. Dubrova and the genetic claims he put forward after Chernobyl. The essay is divided as follows: first, we introduce biographical aspects of Neel's intellectual formation and research with the ABCC in Hiroshima and Nagasaki. Second, we introduce Dubrova's intellectual formation and his later research with post-Chernobyl populations. Third, we explore the disagreement between Neel et al. and Dubrova et al. Finally, we analyze their debate from both anthropological and historical perspectives, addressing the scientific and political differences that define these scholars and their divergent understandings of the genetic effects of radiation on human populations. Although Neel understood that the data obtained in Hiroshima and Nagasaki was imperfect, he defended it in a lifetime of influential genetics research

projects and publications. His eventual heated and sustained debate with the Chernobyl scholar Dubrova illuminates our understanding of Neel's approach to his own data and provides an additional window for understanding how the ABCC studies were understood in the context of the scholarship on radiation risk. Our aim is to illustrate an additional factor – the operative positioning of Soviet genetics as a tainted discipline – that both informed Neel and enabled his studies to maintain their privileged position in the radiation risk literature.

James Van Gundia Neel (1915–2000) and Population Genetics

James Van Gundia Neel is recognized as a primary founder of the field of human genetics in the United States, as well a good number of adjacent subfields. His 1994 memoir *Physician to the Gene Pool* provides a window into many of his career choices. Born in Hamilton, Ohio, in 1915, Neel studied general biology at the College of Wooster, Ohio. After working in the *Drosophila* (common fruit fly) laboratory of Warren P. Spencer, he later began graduate studies with geneticist Curt Stern (1902–1981), an expert on *Drosophila melanogaster*, the fruit fly remembered for its groundbreaking role in early genetic experiments. Stern had a profound influence on Neel. A German Jewish scientist who emigrated in 1933, Stern trained an entire generation of young geneticists in the United States. Known early in his career for his work with *Drosophila*, and building on Herman Muller's (1927) Nobel Prize–winning research that examined radiation-induced genetic effects on *Drosophila*, Stern (e.g., Spencer and Stern, 1948) concluded that there was no safe threshold below which radiation is not harmful. That is, he provided scientific support for what later came to be known as the linear no-threshold (LNT) model, which states that the relationship between dose and effect is linear: a lethal dose will produce a lethal effect, half of that dose will produce half of that effect, and so on, with no level being completely harmless. One important hypothesis of the LNT model suggests that the occurrence of cancer can be understood as directly proportional to the radiation dose received (National Research Council, 2006). The LNT model is in conflict with three other models: the threshold model (which proposes that low doses are harmless); the radiation hormesis model (which proposes that small doses can be beneficial); and the supralinear model (which proposes that ionizing radiation at very low doses is more harmful per unit dose than radiation at higher doses (Moore, 2002, p. 30; Tredici, 1987, p. 132).

Like many other German scientists, Stern was perceived in the American milieu as methodologically rigorous (Neel, 1987, p. 452), and his first Ph.D. student, James Neel, would come to build his own scientific reputation for methodological precision (Goldstein, 2012, p. 135). Neel's growing interest in human genetics lured him away from *Drosophila* studies, a move he knew would be difficult:

> Human geneticists of the current generation cannot imagine how I agonized over the decision to turn my back on the hard genetics of *Drosophila* and enter the soft and tainted field of human genetics…. The irony that I was simultaneously fumbling with thoughts of bringing *Drosophila*-type rigor into genetic studies of that most intractable of all organisms, man, is not lost on me. (Neel 1994, pp. 18–19)

In 1942, Neel decided to enter medical school as part of his scientific training, but he later returned to human genetics research. Appointed "interim director" of the Atomic Bomb Casualty Commission (ABCC), he was charged in 1947–1948 with determining "the nature and extent of the genetic effects caused by the atomic bombs detonated over Hiroshima and Nagasaki" (Neel and Schull, 1991a, p. 1). The publications – books, articles, essays, and reflections – that resulted from the ABCC were in the hundreds. Neel was co-author of a large proportion of those publications, usually as one of multiple co-authors, but occasionally he contributed as sole author, a point we return to later.

Between 1946 and 1997 the Atomic Energy Commission (AEC) funded numerous iterations and extensions of the ABCC study and other related genetics and radiation biology research (Neel, 1998). In 1991, a landmark comprehensive reprint and summary of the publications related to the original ABCC genetics study was published by the National Academy of Sciences-National Research Council, titled *The Children of Atomic Bomb Survivors: A Genetic Study*, edited by James V. Neel and William J. Schull. Neel had by that time become an icon in the field of human genetics, and the publication provided both he and Schull an additional prestigious academic venue to reflect on and circulate their earlier seminal research, which they reference in the introductory chapter as "the most extensive exercise in genetic epidemiology ever undertaken" (Neel and Schull, 1991a, p. 1). The arguments laid out in this chapter, titled "Orientation," remain relevant: they are based on Neel's original research and more than two decades later also remain the current position of the International Atomic Energy Agency (IAEA) and the United Nations Scientific Committee on the Effects of Atomic

Radiation (UNSCEAR). In this epic summary, Neel emphasizes that the Japanese survivors of the atomic bomb are the only population in the world that has been exposed to both high and low doses of ionizing radiation and the only population to be systematically studied. (He does not mention the studies on Marshall Islanders, who were not exposed to a direct bomb hit; much of that information was classified until 1994; see Johnston and Barker, 2008)

In order to understand the trajectory of Neel's work, one must understand the methodological and technological dynamism and thrill of discovery that human genetics experienced during his long career. Neel's original data set was first organized by his own efforts and those of his staff and colleagues in the early years of the ABCC in Japan, and over a lifetime he helped update it, applying new statistical techniques, partitioning various subsets of the data, and of course reconceptualizing some aspects of the original work. Neel was in agreement with the LNT model but also maintained that the probability of injury from low-dose exposure was so small that it appeared to be statistically insignificant. The original ABCC work created standards for all sorts of later understandings involving ionizing radiation, public health, community and worker safety, environmental litigation, etc. in the burgeoning nuclear industries associated with the post-World War II years – both war-related and energy-related. The scientific community, too, right through to the present recognizes the ABCC studies, and their follow-up studies, as foundational for understanding radiation effects on the human organism (National Research Council, 2006).

While working with ABCC data early on, Neel and his colleagues reported that they could find no overt evidence of significant variation in mortality in the cohorts of children born to parents who were exposed to radiation at Hiroshima and Nagasaki (Kato et al., 1966, p. 371 and reprinted in Neel and Schull, 1991c, p. 323). Neel and his colleagues continually reanalyzed the atomic bomb survivor data as part of their longitudinal project, finding that the life expectancy in the first generation of children (F1) of exposed parents looked to be, in his words, not demonstrable:

> This failure to demonstrate significant changes in F1 mortality as a function of parental exposure of course cannot be construed as evidence that no genetic effects resulted from exposure to these nuclear devices. Unless one is willing to argue that man differs from all other forms of life thus far studied, lethal and semilethal mutations of a type which might manifest themselves as mortality in the first decade or so of life were induced. But the present study

> provides no evidence for their existence. (Kato, Schull, & Neel 1966, p. 365)

In other words, Neel understood that mutations probably did occur in the first generation, but at the time of these particular studies, he understood that these changes in mortality were not perceivable with the scientific methods available at the time.

The scope and breadth of the studies conducted by Neel and his team over time are indeed impressive (Neel and Schull, 1956; Neel, 1958, 1998, 1999a, b; Schull and Neel, 1965; National Research Council, 2006). At each juncture, Neel and his colleagues applied the latest statistical techniques and the most recent conceptualizations of genetics to a subset of the original ABCC data. But the summary findings never changed: the atomic bomb exposures were understood as causing no serious genetic mutations in the F1 generation. As Neel himself recognized, the Japanese bomb survivors had experienced both high- and low-dose radiation exposure and thus differed from later comparisons drawn with scientists and workers accidentally exposed during the Manhattan Project and nuclear reactor workers who experienced a more constant low-level exposure. Nevertheless, the standard line of argument extending from Neel and the Japan studies led to the defense of the view that the potential harm to future generations from genetic mutation was small or not perceptible.

Opponents of the Hiroshima and Nagasaki data and findings took much longer to emerge within the framework of secrecy that characterized the Cold War era. When they did surface, they clustered in two general areas: research done in the aftermath of Chernobyl by Soviet and post-Soviet scientists, and research carried out on workers in the nuclear industries by American occupational epidemiologists. Wing et al. (1999) characterize three types of atomic bomb studies: inherited genetic effects among children of exposed parents, fetal irradiation, and Life Span Study (LSS) of cancer risk, all of which use different statistical techniques and all of which draw from the original ABCC/Neel studies. These authors suggest a constellation of reasons that help explain the scientific authority of the A-bomb survivor studies when multiple alternative studies suggested that radiation risk estimates from the original data could be underestimating the cancer risk from protracted low-level exposure to radiation (Mancuso et al., 1977; Beral et al., 1988; Kneale and Stewart, 1995; Morgenstern et al., 1997; Wing et al., 1999, p. 136).

> Despite the increasing availability of information about long-term follow-up of badge-monitored nuclear workers, standard-setting bodies continue to rely on the Life Span Study (LSS) of A-bomb survivors as the primary epidemiological basis for making judgments about hazards of low-level radiation. Additionally, faith in the internal and external validity of studies of A-bomb survivors has influenced decisions about the design, analysis, and interpretation of many worker studies. (Wing, Richardson, and Stewart 1999, p. 133)

Wing, Richardson, and Stewart meticulously explain the design issues that differentiated the LSS A-bomb survivor study from the epidemiological studies of nuclear industry workers. First, these authors concede that one of the strengths of the survivor studies is large sample size – somewhere between 75,991 (Wing et al. 1999) and 86,611 (Ozasa et al., 2012) LSS cohort survivors – an aspect of the Hiroshima studies that Neel continually emphasized. Second, these authors point to the fact that some survivors had experienced high doses of radiation and whole-body exposure to gamma and neutron radiation, an exposure quite different from longer term and lower dose exposure. Most critically, the LSS studies that were organized 5 years after the bombings were considered a sort of "natural experiment" in spite of the fact that those in the study were the ones who survived the bombing. This "healthy survivor" effect is a point that the physician and epidemiologist Alice Stewart (1906–2002) often pointed to in her critique of the ABCC studies. She argued that neither the LSS nor the worker studies were looking at representative populations (see Green, 1999, p. 283). Nevertheless, in part because of their longitudinal nature and statistical power, the A-bomb studies became the gold standard for understanding radiation safety.

As Wing et al. illustrate (Stewart was a co-author), whenever research appeared that seemed to challenge an important aspect of these original studies, the defense strategy was to compare certain aspects of the challenging study to the original studies and then find the challengers lacking in some fundamental manner. In summary,

> Neither the worker studies nor the A-bomb survivors studies are free of measurement problems and other biases. Nevertheless, studies of A-bomb survivors have continued to play a predominant role in radiation risk estimation despite their focus on an extreme exposure situation, evidence of selective survival, and unresolved questions about inaccuracy of dose estimates. Occupational stud-

> ies, which investigate low-level exposures similar to those of regulatory concern and have advantages of individual dose measurements, lack of reliance on interviews, and absence of selection related to surviving an atomic attack (Table 1), have been kept in the background. (Wing et al., 1999, p. 142)

In our own assessment of the later Neel–Dubrova debate, we agree with Wing, Richardson, & Stewart that research delegitimation also takes place. But in our own analysis of this debate, there is an added dimension. The historical stigmatization of Soviet science and genetics that had begun much earlier made Soviet-based scientific studies later challenging the A-bomb research more easily dismissible.

There is convincing archival evidence, for example, of Neel's low regard for Lysenkoism in his personal letters to friends and colleagues. In 1959, for example, Neel wrote to the esteemed Ukrainian *Drosophila* geneticist Theodosius Dobzhansky (an outspoken critic of Lysenkosim), concerned about the pirating of his textbook written with Jack Schull. Neel inquires about whether pirating is common in Russia, and shares with Dobzhansky that in spite of his mixed feeling about Russian pirating, he is otherwise happy to contribute to "Brother Lysenko's discomfiture." In a letter dated July 8, 1959, he writes:

> Dear Prof. Dobzhansky:
>
> I have just received a request from a Romanian to translate the textbook of Jack Schull and myself into his language; in the letter he makes casual reference to a Russian translation of our text, as well as Stern's. Needless to say, it came as a bit of a surprise to us to find that we might possibly in some small way contribute to Brother Lysenko's discomfiture. Be that as it may, knowing how you have kept in touch on things genetic in Russian, I wonder if you could give me any idea as to just how many of the American genetic texts have been pirated in this fashion. . . (Correspondence, James V. Neel to Th. Dobzhansky, July 8, 1959, Theodosius Dobzhansky Papers, The American Philosophical Society)

This letter provides a partial basis for our claim that Neel would later view Soviet science and the Chernobyl studies with a great deal of skepticism.

Wing et al. point out that at the time when the earliest A-bomb survivor studies were released, the U.S. government was concerned about insurance claims, labor relations, and adverse public sentiment toward the nuclear industry. The historian Walker (1994) describes this

period as an anxious time for the American public. This public sentiment had to be managed. In 1953, President Eisenhower ushered in the ''Atoms for Peace'' program both to placate negative public sentiment and to redirect nuclear research away from military pursuits and toward peaceful uses. This included unveiling the project of nuclear energy production. A vital component of the ''Atoms for Peace'' program was to reassure the public that the radiation risks posed by the nuclear industry were acceptable. The 1950s then, presented a historical moment where government pressure to report little evidence of genetic damage from radiation would have been necessary (Lindee, 1994), and Neel was not immune to this pressure. Nevertheless, Neel was reluctant to conclude that his science was in any way compromised by politics. Even in his later memoir, he wrote, ''it was science, not politics, that prevailed'' (Neel, 1994, p. 89; Goldstein, 2012, p. 134). Wing, Richardson, and Stewart understand this juncture from a different perspective. Their work suggests that the studies of A-bomb survivors had the momentum of the first half of the century behind them, and that this scientific work took place within an entrenched environment of secrecy, deference to authority, and even disdain for workers (Wing et al., 1999, p. 147). In turn, this perspective supports the idea – clearly articulated in the writings of the physician and epidemiologist Bertell (1985) – that international standards of that time had the goal of recommending an 'acceptable' trade-off rather than the goal of protecting worker and public health. Our argument explores an additional dimension and suggests that the ''momentum'' of the A-bomb survivor studies also benefited from the decrepit state of Soviet science in the 1950s, and that this historical understanding among scientists carried over into the later Neel–Dubrova debates that took place years later.

By 1996 the Cold War was over and the nuclear industry had successfully been established. The politics were different. Chernobyl had taken place and James Neel was experiencing a direct challenge from a genetic scientist based in the East. The Soviet (Ukrainian) scientist Yuri Dubrova and his colleagues had published studies on the genetic effects of Chernobyl, data that contradicted numerous aspects of Neel's updated genetic studies. In a rare single-authored paper titled ''Genetic Studies at the Atomic Bomb Casualty Commission – Radiation Effects Research Foundation: 1946–1997,'' Neel (1998) reaffirms the ultimate reliability of the ABCC studies and related research, and directly confronts problems he sees in Dubrova's Chernobyl work. Dubrova's research, focused on the aftermath of Chernobyl, studies populations of workers and civilians who received varying doses of radiation during the

meltdown and then continuous low-level ionizing radiation long beyond the initial exposure. Thus, similar to Wing et al.'s (1999) nuclear worker studies, the Chernobyl studies of Dubrova were also deemed inferior to the LSS work. We provide context for this denigration of the Chernobyl data in the following sections.

Yuri E. Dubrova (1955–) and Population Genetics

Yuri Evgenjevitch Dubrova grew up in Kiev and earned his undergraduate degree in biology at Kiev State University. In 1982 he finished his Ph.D. at the Institute of General Genetics in Moscow, where his mentor Yuri Petrovich Altukhov would eventually become director (1992–2006). Altukhov (1936–2006) was closer to Neel's generation than Dubrova and at one point had also exchanged intellectual fire with Neel. He had completed postgraduate studies in the Biological and Soil Science Faculty of Moscow State University and became a head of a new Laboratory of Population Genetics at the Institute of General Genetics in the Soviet Union. This coincided with the rebirth of Mendelian genetics after the Stalinist silencing (Salmenkova and Politov, 2011), a history we outline below. Interestingly, it was Altukhov's work on gene mutations in the context of environmental pollutants that brought him into conflict with Neel.

In 1979, Nikolay Petrovich Dubinin and Altuknov produced an article for the *Proceedings of the National Academy of Sciences* titled "Gene Mutations (de novo) Found in Electrophoretic Studies of Blood Protein of Infants with Anomalous Development." Throughout Dubinin's career as a leading Soviet radiation geneticist and a director of the Soviet Institute of Genetics, he argued that a dramatic increase in the percentage of children born with congenital defects was a direct result of exposure to mutation-causing pollutants in the environment, including radiation.[1] Although various approaches for studying biochemical mutations have been devised, determining the human biochemical mutation rate increases continues to be a formidable problem. Using a framework devised by the Soviet-American Cooperation Program in the interest of environmental protection, US and Soviet scientists set out to develop a way to estimate and more importantly, differentiate, the various types of genetic load in humans. Specifically, they aimed to

[1] We should note that even today, however, the extent to which germinal mutations are increased by exposure to a variety of pollutants and the possibility of their subsequent transmission to offspring remains a matter of debate.

develop a technique with which to identify correlations between new mutations in the offspring of exposed parents and mutagenic agents in the environment. The Soviet scientists suggested that children with congenital birth defects are especially important for monitoring studies because of their high gene mutation rate, presumably the outcome of a mutational event traceable to one of the parents. In contrast to the ABCC studies that relied on mass screening of newborns' blood samples for rare electrophoretic protein variants to detect genetic variation, Dubinin and Altukhov screened selectively. They targeted rare, non-polymorphic electrophoretic variants of proteins in premature and physically disabled infants. Dubinin and Altukhov preferred selective screening because it allowed them to reduce the size of samples needed for a statistically reliable estimate for the rate of genetic mutation. Whereas ABCC findings relied on extremely large study populations, the Soviet scientists demonstrated that by focusing on infants (with anomalous development) they were able to significantly reduce sample size "by more than two orders of magnitude" for statistically reliable estimations of mutation rates in humans (Dubinin and Altukhov, 1979, p. 5228).

Dubinin and Altukhov's findings implicitly challenged the ABCC studies because they showed that the search for new biochemical mutations by mass screening of mostly healthy children of exposed survivors was a futile effort. This is because, as the Soviet scientists argued, mass screening of mostly normal children reduced the appearance of mutations in the study sample (Altukhov, 2006, p. 363). In other words, unlike Soviet research that showed higher frequency of rare protein variants in unhealthy children, Neel's F1 generation study using mass screening of children of atomic bomb survivors showed that "in no instance is there a statistically significant effect of parental exposure" to radiation (Neel and Schull, 1956; Schull et al., 1981, p. 1220). Since the ABCC studies did not find any significant variation in protein structure that would indicate a genetic mutation, the mutagenic change found in the Japanese cohort was attributed to "natural stressors" and not due to radiological factors coming from the atomic bomb. When the respected epidemiologist Martin J. Gardner investigated the clusters of childhood leukemia at the Sellafield nuclear plant and designed a study to see if an external dose of radiation to fathers working at the plant may be explanatory (he posited a mutation in the sperm cells), he, too, received skeptical comments from Neel and Schull (Roberts, 1990, pp. 24–25). One could argue, then, that Neel and Schull would defend the Hiroshima and Nagasaki data against the challenge of *any* researcher,

Soviet, post-Soviet, or Western, that questioned the authoritative findings of the A-bomb studies, strengthened by a seemingly unassailable statistical and longitudinal solidity. Nevertheless, we find something more aggressive and personal in the debate with Dubrova about mutations that are worthy of further consideration.

The 1979 Soviet publication by Dubinin and Altukhov alarmed North American population biologists and geneticists (e.g., Coulthart et al., 1984). This group included Neel, who responded in a 1984 article co-authored with Harvey W. Mohrenweiser pugnaciously titled, ''Failure to Demonstrate Mutations Affecting the Protein Structure or Function in Children with Congenital Defects or Born Prematurely.'' Neel and Mohrenweiser argued that the Soviet team's implied message – that there is a cause-and-effect relationship linking parental biochemical exposure and congenital defects – had not been proven. The Soviet study was methodologically defective, they claimed, because Dubinin and Altukhov never described the full composition of their population, a necessary prerequisite to meeting the methodological rigor of population genetics that was already firmly in place. In his 1990 book titled, *Population Genetics: Diversity and Stability*, Altukhov once again attempted to diminish the hegemony of the ''gold standard'' ABCC studies by demonstrating a link between environmental factors (pollution) and the inheritance of genetic mutations. Similar to his previous findings on children born with congenital defects, Altukhov argued that experiments with the common fruit fly subjected to radiation show that ''the frequency of induced mutations found at several enzyme loci was approximately 10 times higher in larvae than it was in adult flies'' (Altukhov, 1990, p. 185).

Dubrova expanded on Altukhov's work to develop new approaches to monitoring mutations in the human germline. In many ways, Dubrova picked up the scientific debate initiated by Altukhov in 1979 and did so having already benefited from training and scientific experience gained in both the East and the West. As a research fellow at the Institute of General Genetics in Moscow (1981–1995), Dubrova continued working with Altukhov on population genetics and rare electrophoretic variants of proteins in humans. In the late 1980s, Dubrova learned about the pioneering work of Alec Jeffreys, a British geneticist working on minisatellites. Jeffreys used these genetic segments to develop a DNA profiling technique now widely used by forensic scientists to identify individuals. Influenced by Jeffreys, Dubrova used the minisatellite segments for mutation monitoring. He collected blood samples from Chernobyl populations, and in 1994, with funding from the

Wellcome Trust, travelled from Moscow to Jeffreys' laboratory at the University of Leicester where he showed that the mutation rate in the germline of irradiated parents was higher than in the control group. In the same year, following the exodus of many geneticists from the former Soviet Union, Dubrova took a visiting research fellow position at University of Leicester in England and ultimately a professorship. His current research interests include the analysis of germline mutation following exposure to ionizing radiation, chemical mutagens, and some anticancer drugs; he is also interested in whether the children of exposed parents also show a tendency to produce mutations (see Dubrova's website at: http://www2.le.ac.uk/departments/genetics/people/dubrova/research). Dubrova is one of the very few Soviet geneticists who have emerged from the shadows of Cold War politics and become successful in the West. This is because most of Soviet science was paralyzed with the fall of the Soviet Union and the failure of Russia's economy to get off the ground. Loss of funding meant that the majority of Soviet scientists were unable to conduct even the most rudimentary research and many laboratories closed down. Dubrova's success in the West is even more remarkable given that Soviet genetics took a strange political and intellectual turn in the early twentieth century, causing the entire field to stagnate in the Soviet Union – or at least, from a Western perspective.

The Mid-Twentieth-Century Context of Soviet and North American Genetics

As a discipline, genetics emerged from the International Genetics Congresses between the two world wars, which brought together geneticists from the Soviet Union, the United States, Germany, and many other nations. But the maintenance of disciplinary and scientific consensus across this broad group proved short-lived. By the mid-1930s, Soviet, German, and American governments subordinated scientific research to nationalist ideologies and propaganda (Bird and Sherwin, 2005; Johnston, 2007; Krementsov, 2005; Pollock, 2006; Wolfe, 2012). In the Soviet Union especially, the Lysenko affair (discussed below) and the politics of the Cold War separated scientists into opposing Eastern and Western ideological camps (Krementsov, 2005).

In Joseph Stalin's Soviet Union, the Lysenko affair had devastating effects on the development of science in general (with the exception of physics), and on genetics in particular. Lysenko, a Ukrainian peasant who rose quickly through the communist party ranks (Pollock, 2006),

declared that species evolution was based on Lamarckian processes – the inheritance of acquired characteristics – and that genes were imaginary (DeJong-Lambert, 2012). The vast majority of geneticists dismissed this view then as now; but by the early 1930s, Lysenko's fervent followers openly attacked Mendelian genetics as a ''bourgeois perversion of science'' (Soyfer, 2001). ''Mendelism,'' Lysenko argued, was ''developed in Western capitalist countries not for agricultural purposes but for the reactionary eugenics, racism and other purposes'' (quoted in Pollock, 2006, p. 47). Virulent attacks by Lysenko and his followers directed at non-subscribers of Stalinist genetics, led to the arrest, imprisonment, or execution of many prominent Soviet geneticists. By 1948, and with the personal endorsement of Stalin, all genetics research was officially forbidden in the Soviet Union and declared an illegal discipline (Kojevnikov, 2004). Nevertheless, some small-scale genetics research did continue. Alongside the official Soviet scientific dictum, a vast network of NKVD (The People's Commissariat for Internal Affairs) secret prison laboratories (known as *Sharashka*) was designed to carry out studies related to the nuclear project, including genetics research. Laboratory B in Sungul, for example, housed Soviet geneticist Nikolay Tomofeev-Ressovsky and German nuclear chemist Nikolaus Riehl, scientists who conducted studies on the genetic effects of radiation on plants and animals, as well as the handling of radioactive materials. Only in 1964 was this ban lifted when hundreds of Soviet scientists – biologists, geneticists, chemists, physicists, and others – united in their opposition to Nikita Khrushchev's support of Lysenko. Since the ban, Soviet scientists often challenged the government's imposed secrecy by sporadically publishing research on the effects of radiation exposure (e.g., Medvedev, 1976). Nevertheless, these studies were rarely published in the West because Soviet authorities, fearing the dissemination of state secrets, practiced broad censorship. Even though certain information still flowed between the Soviet Union and the United States and was facilitated by the numerous U.S.-Soviet Cultural Exchange Programs, it was not until the disintegration of the Soviet Union in 1991 that international scientific collaborations resumed on a large scale. In the West, the ''ideologization'' of Soviet genetics did not go unnoticed (Lubrano and Solomon, 1980).

In 1958, the United States and Soviet governments signed the first in a series of bilateral cooperation agreements aimed to foster mutual understanding of the cultural, technological, and scientific developments of both nations. The Cold War political tensions and underlying ideologies of both camps, however, interfered in the process of these

exchanges by producing distortions and misunderstandings of research in both countries, especially in the realm of the biological sciences (Geltzer, 2012). Many U.S. delegates for example, viewed the exchanges as having no use value for the American scientific community except as an altruistic endeavor to disseminate factual information to their Soviet colleagues (Geltzer, 2012). American scientists often described Soviet research as an isolated enterprise lacking in imagination, constrained by an intrusive state, and fueled by propaganda that placed Soviet biological sciences "outside of the channels of world medical developments" (quoted in Geltzer, 2012, p. 44). Still others found Soviet research methods archaic and obsolete, their laboratories unexciting. U.S. assessments of Soviet biological sciences were often harsh, dismissive, and derisive. A 1959 report from an American Radiobiology Mission to the Soviet Union for example, concluded that "some of the work seems very good, some very bad, and most of it quite pedestrian" (quoted in Geltzer, 2012, p. 46).

In contrast, American geneticists themselves enjoyed the full support of the United States government within the confines, for example, of the Manhattan Project, the AEC, the ABCC, and then the RERF, but we should assume that their work was also affected by Cold War contingencies. Anthropologist Barbara Rose Johnston argues that the Cold War, for example, "not only resulted in overt efforts to keep information from the public (and therefore to deceive and lie to the public), but also generated biases that skewed scientific research from inception to conclusions" (2007, p. 8; see, also, Welsome, 1999; Masco, 2002; Gusterson, 2007; Hecht, 2012). In short, the Cold War analytical framework fueled misunderstandings and stereotypes on both sides of the East-West divide, and the "conception of Soviet biomedical science informed by this framework endures in multiple contexts" (Geltzer, 2012, p. 43). With the fall of the Soviet Union in 1991, research laboratories and their infrastructure were completely dismantled and many former Soviet scientists lost funding to continue working. The resultant disarray of Soviet genetics research and the corresponding Western sense of scientific superiority helps explain how the findings of Neel and his colleagues have long remained privileged and unharmed – in a word, authoritative – in spite of serious challenges from other research scientists.

The Recent Controversy: Dubrova et al. Versus Neel et al.

On the eve of the tenth anniversary of the 1986 Chernobyl disaster, the journal *Nature* published "Human Minisatellite Mutation Rate after the

Chernobyl Accident,'' in which Dubrova et al. (1996) contradict the ABCC Hiroshima and Nagasaki findings, generating controversy for years to come. Dubrova later found the controversy surprising, in light of evidence that suggested:

> the views, currently accepted in radiation genetics, may significantly underestimate deleterious effects of mutagens on living organisms. I mean the phenomenon of radiation-induced genomic instability, in which high mutation rate is observed not only in the directly irradiated cells, but also during a long time in their unirradiated offspring [3–5]. (Dubrova, 2006, p. 1116)

These results appear because traditional studies like those conducted by the ABCC associate genetic risk of mutagenic effect ''only with transmission of de novo mutations to the offspring and their transgenerational manifestation'' (Dubrova, 2006, p. 1116). Newer, more updated studies suggest, however, that high mutation rate has also been observed in individuals who were not exposed to radiation.

Dubrova et al.'s 1996 *Nature* and 2002 *Science* publications (after Neel's death) have become new landmarks in ionizing radiation debates. The 1996 piece was the first study of its kind to provide experimental evidence for radiation-induced increases in human germ cell mutation rates, in both exposed parents and their unexposed offspring. Neel responded in two single authored articles published at the end of the decade (Neel, 1996, 1999a). Neel's critiques – and Dubrova's response – can be understood as revolving around four separate but interrelated issues: (1) the meaning and usefulness of minisatellite mutations and junk DNA as indicators; (2) the comparability of animal and human studies with regard to radiation; (3) the measurement of the doubling dose in humans; and (4) the choice of control groups in human population studies. In the following sections we highlight how these issues were formed by scientific actors and institutions still deeply connected to the Hiroshima/Nagasaki data, and how these debates continue to frame our contemporary conceptions of ionizing radiation's effects on human populations.

Issue 1: Minisatellite Mutations and Junk DNA as Indicators

In recent years, biomedical research focusing on the effects of low-dose ionizing radiation has concentrated on minisatellite mutations, also known as ''junk DNA.'' Neel and Dubrova disagree on what minisatellite mutations and junk DNA indicators reveal about genetic mutation

in the offspring of irradiated parents. In his 1996 *Nature* article, Dubrova and his colleagues compare germline mutations (at the minisatellite loci) between parents and their children, using populations living in the heavily polluted areas of Belarus following the Chernobyl accident. They claim that the rate of mutation in DNA minisatellites was twice as high in children (F1) whose parents had been exposed to fallout from Chernobyl as in the control group.

For Neel, Dubrova's use of the minisatellite indicators is problematic. This is because the spontaneous mutation rate in junk DNA is known to be high, and because when Neel was writing in the late 1990s, neither the mechanisms driving minisatellite mutations nor the exact function of this particular strand of DNA were understood. Neel essentially reasserts the superiority of the A-bomb survivor studies and also cites the contradictory findings in the studies of Chernobyl liquidators (Kodaira et al., 1995; Mohrenweiser unpublished, cited in Neel, 1998).

Part of the disagreement concerns what "counts" as a mutation. In an early response, Neel (1996) concludes that Dubrova and Jeffreys's "mutations" are not phenotypically detectable. Tellingly, in the article "Two Recent Radiation-Related Genetic False Alarms: Leukemia in West Cumbria, England, and Minisatellite Mutations in Belarus," Neel (1999a, p. 304) attacks Dubrova's Chernobyl study for bringing "unwarranted distress to an already badly stressed population," a point that Neel and Schull had also made in their well-known 1956 and 1991 publications. Here, as Susan Lindee observes, Neel is reasserting a definition of mutation that seems still influenced by the need to assuage Cold War anxieties:

> While Neel does not believe that political concerns played any role in the selection of what signs counted as mutations at Hiroshima and Nagasaki, he acknowledges that social impact did play a role. A trait that would not "upset the new mothers of Hiroshima," he has explained, should not count as a mutation. Such a formulation interprets mutation as a distressful change . . . A mutation in this data set was a dangerous, threatening, or socially disturbing trait with implications for future human survival. This was the chosen scientific understanding . . . partly because this was the concept prevalent in the larger political debate . . . In cases lacking any compelling reason to decide otherwise, traits that were socially dangerous were classified as major and those without social implications as minor – essentially, as not mutations. (Lindee 1994, pp. 191–192)

One reason for Dubrova's use of minisatellite data is that the low frequencies of spontaneous mutation in the majority of protein-coding genes, together with the massive amount of samples required, make it difficult to detect genetic instability. So although studies have shown that ionizing radiation exposure can lead to genomic instability in mammalian somatic cells, there is still very little experimental evidence for radiation-induced genomic instability in the germ cells (Dubrova et al., 1998; Wyrobek et al., 2007). But minisatellites have high rates of both spontaneous and induced mutations, which allow for "mutation induction to be evaluated at low doses of exposure in very small population samples" (Dubrova et al., 1998, p. 689). Even as recently as 2012, it is not clear what the correlation is between increased minisatellite mutation rate and disease (Jeffreys interview in Hodge and Wegener, 2006; Bauer, 2010). Yet recent findings by ENCODE scientists (who together published approximately thirty articles in September 2012 at http://www.encodeproject.org) suggest that junk DNA may be vital to understanding gene regulation and disease risk (Kolata, 2012). While a number of studies report increased minisatellite mutations in both human and animal germline cells exposed to ionizing radiation (Dubrova, 2005; Bouffler et al., 2006), there is no agreement as to the significance of these findings. This prompts a question: why, for example, do Hiroshima and Nagasaki survivors not show heightened minisatellite mutations (Kodaira et al. 1995)? Dubrova (2003) offers a few possible explanations: (1) the Japanese atomic bomb population received smaller doses of radiation than other exposed populations; (2) the total number of individuals studied may have been too small for detecting significant differences; and (3) the Japanese atomic bomb survivor studies did not distinguish between paternal and maternal exposure. Dubrova's investigation of minisatellite mutation rates among children of Chernobyl survivors and children of residents living near the Semipalatinsk nuclear test site in Kazakhstan showed "significant associations between paternal (but not maternal) exposure and increased mutation rates" (Wyrobek et al., 2007, p. 16).

In a recent summary of the minisatellite mutation or "junk DNA" controversy, Susanne Bauer, an environmental health scientist and a sociologist of science, argues that it has proven difficult to turn the junk DNA work into a biodosimetry tool (2010, p. 216). The function of the minisatellite mutations was poorly understood, and they proved to be an unsatisfactory marker of radiation damage. This was a point also made by Neel (1996, p. 5).

Issue 2: Extrapolating from Animal to Human Studies

Another subset of the Neel–Dubrova debate revolves around comparing animal and human studies. In the US, the 1942–1946 Manhattan Project spawned a new industry, one that would create a broad array of toxic man-made radioactive materials, and with them a need for safety standards. In order to develop the standards, large amounts of biomedical data had to be collected at national laboratories that were at the forefront of developing nuclear technologies – e.g., Oak Ridge, Tennessee and Brookhaven, New York. Despite 80 years of research in animal systems that has identified a number of germ-cell mutagens, however, neither ionizing radiation nor chemical exposure has been confirmed as a germ-cell mutagen in humans (Wyrobek et al., 2007). For decades, the accepted scientific explanation was that the discrepancy was rooted in the biological differences between humans and animals.

At the start of the Manhattan Project most of the information on the effects of radiation on the human body was derived from studies on *Drosophila*. By 1947, major experiments began on mice at the Mammalian Genetics and Genomics Program at Oak Ridge Laboratories. Under the direction of William and Lianne Russell, the ''mega-mouse'' study subjected hundreds of thousands of male mice to ionizing radiation in order to examine the mutagenic effect on their offspring and to assess human genetic consequences of low-dose exposure. Their results show that the frequency of genetic damage may be higher than previously thought (Jolly, 2003, p. 112 summary of letter from William Russell to Shields Warren, January 1951). But even though this research demonstrated that chemical agents and ionizing radiation could induce heritable mutations in the germ cells, radiation studies on humans have failed to show the same effect (Russell et al., 1981; Wyrobek et al., 2007). Nevertheless, mice data eventually laid the groundwork for the establishment of national standards for acceptable levels of radiation exposure for humans – even though extrapolating human risk from animal studies has proven to be difficult because of ''differences in absorption, metabolism, and excretion of drugs and chemicals'' (Brent, 2004, p. 988).

Neel was a key figure in comparing mouse data to human data. In a 1990 article in the *Annual Review of Genetics*, Neel and Susan Lewis argued, ''humans may not be as sensitive to the genetic effects of radiation as has been projected by various committees on the basis of data from the most commonly employed paradigm, the laboratory mouse''

(1990, p. 328). In Neel's 1996 article "The Genetic Effects of Ionizing Radiation on Humans," he suggests that *both* the mouse data and the A-bomb survivor data were "conservative" and that "a major revision downward in the previous estimates of the genetic risks of radiation for humans must be very seriously considered" (p. 4). Neel believed that great care must be taken in extrapolating from mice to humans (1998, p. 5434). The reason, he argued, is not only that mice seem to be more sensitive to radiation exposure, but also that extrapolation is difficult because of the nature of laboratory studies: for instance, the repeated use of a few irradiated males to impregnate many females, the reliance on a single inbred line reproduced in a controlled mating system, the relative immaturity of the mouse fetus at birth, and the use of a male-based model (Russell and Russell, 1996; Rader, 2004). In a 1998 solo article published in the *Proceedings of the National Academy of Sciences*, Neel reiterates, "yes, there are genetic risks in exposure to ionizing radiation, but current national and international recommendations regarding permissible exposures now can be seen as incorporating an even wider margin of safety than appeared to be the case when they were promulgated" (Neel, 1998, p. 5436). Given this positioning, then, when Dubrova's 1996 Chernobyl research challenged the ABCC human data by suggesting radiation sensitivities far greater than were previously thought to exist, Neel (1998, pp. 5435–5436) wondered whether parallel experiments involving mice should begin. In the end, Neel concluded that "at this level of genetic resolution, there is no need for animal experimentation," aside from studies that would lower the experimental radiation doses to better match and approximate the Japan data on humans. And yet Neel was not enthusiastic about this sort of work either.

Neel's rejection of Dubrova's work makes sense given Neel's own early involvement and training in *Drosophila* genetics; it also makes sense given Neel's interest in defending the ABCC work and in reassuring the public about genetic risk after Hiroshima. However, while a 2006 Report from the National Research Council (NRC) finds no direct evidence of harm to human offspring from exposure of parents to radiation, the authoring committee notes that such harm has been found in animal experiments and that there is "no reason to believe that humans would be immune to this sort of harm" (National Research Council, 2006, p. 10, also known as BEIR VII). This report thus admits that the animal studies research cannot be readily dismissed. This line of thinking began as early as the 1980s, when Bertell (1985, pp. 44–45) interpreted Muller's *Drosophila* research as foreshadowing a reduction

in the survival ability of the human species as several generations are exposed to ionizing radiation, a prediction that Bertell assumes was ignored by the AEC in its consistent mission to underplay the harms caused by atomic weaponry and atomic energy. In her book *No Immediate Danger*, Bertell (1985, p. 164) refers to a 1959 AEC report that radioactive fallout from atomic tests conducted in the single month of September 1958 would result in 100,000 major birth defects, 380,000 stillbirths or infant deaths, and 900,000 fetal deaths "in the thousands of years to come… ." She doesn't say how many of these estimated deaths could be due to genetic effects, but Bertell's perspective was that there was a willful mission of the AEC to ignore the dangers of atomic weaponry and of ionizing radiation.

Issue 3: Doubling Dose Estimates in Human Populations

To measure the genetic effects of ionizing radiation, scientists relied on a metric known as the doubling dose – the amount of radiation necessary to double the naturally occurring rate of mutation (Neel, 1996, p. 2). Neel recognized that the doubling dose was a statistical construct, or simply "a convenient metric," but he also recognized that "in an imperfect world, the doubling dose concept supplies a perspective, if blurred, difficult to obtain by any other approach" (1998, p. 5433). The conclusion he reached after approximately 50 years of research was that the doubling dose established through the original studies – an acute gonadal exposure of approximately 2.0 Sv equivalents – is a conservative estimate, meant to preserve the safety of future generations from genetic risk in the form of mutations. Neel outlines his reasoning in both his *Teratology* (1999a) and *Proceedings of the National Academy of* Sciences (1998). He argues, among other points, that the proximally exposed parents in the ABCC studies had lower socioeconomic status than the control population; cousin marriage (and with it, by implication, the mutation rate) is high in the bombed region of Japan, accounting for 6% of newborns in Hiroshima and 8% in Nagasaki; human and *Drosophila* data are not comparable; and, finally, there is a great deal of difficulty in comparing the experimental data on human and mouse doubling dose. All of this is to say that Neel stood by the ABCC estimate of the doubling dose and defended it until his death.

It is no surprise, then, that Neel responded sharply to Dubrova et al.'s (1996) minisatellite study published in *Nature*. Neel (1999a) read Dubrova as suggesting that the doubling dose for chronic radiation exposure produced two hundred times greater sensitivity in humans

than the literature suggested. And Dubrova (2006, p. 1116) has since claimed: "ample evidence, obtained in the last two decades, suggests that the views, currently accepted in radiation genetics, may significantly underestimate deleterious effects of mutagens on living organisms." But many scientists, such as those interviewed for *The New York Times* article on the Fukushima disaster cited at the outset of this essay and those cited by Wing et al. (1999) as defenders of the LSS studies, continue to stand by the ABCC data and studies.

Issue 4: Control Group Choice in Human Population Studies

Another key disagreement between Neel and Dubrova focuses on control group choice in human population studies. In the Chernobyl case, Dubrova used parents and children from Great Britain – not the Chernobyl or nearby area populations – as a control. Dubrova chose this population because no appropriate ethnic populations existed that had not been already irradiated. Neel labeled this "a violation of one of the first principles of genetic epidemiology, since these controls are neither matched by environment nor by ethnicity" (1999a, p. 304). In 2002 Dubrova repeated the minisatellite study among the offspring of parents who received high doses of radiation as a consequence of Soviet nuclear testing in Kazakhstan. The revised study design addressed all of Neel's critiques. In the 2002 study, Dubrova shows that exposure to radioactive fallout in the mid-1940s to early 1950s (the height of atmospheric nuclear testing) doubled the germline mutation rate in the affected populations. A scientist from England's National Radiological Protection Board observed that the Kazakhstan study "provides the most convincing evidence to date of heritable mutations induced in humans following parental exposure to ionising radiation" (Bouffler and Lloyd, 2002, p. 10).

Others Weigh In

More recently, the Neel–Dubrova debates drew additional scholarly attention. In 2009, the New York Academy of Sciences published *Chernobyl: Consequences of the Catastrophe for the People and the Environment*. In a chapter on radiation-induced mortality rates, Alexey Yablokov et al. from the former Soviet Union estimate that the number of worldwide deaths related to Chernobyl disaster radioactive fallout between 1986 and 2004 was 985,000 (2009, p. 210). This is a much

higher number than has been previously given. Citing studies in voles, they additionally suggest that in places where there was radioactive fallout, an increase in chromosomal aberrations will continue for as long as seven generations (Yablokov et al., 2009, p. 77). In contrast, The Chernobyl Forum (2006), which includes the United Nations organizations such as the IAEA, reports that in 1986, 28 emergency workers died as a result of acute radiation syndrome (ARS). The report also states that "among the more than 4000 thyroid cancer cases diagnosed in 1992–2002 in persons who were children or adolescents at the time of the accident, fifteen deaths related to the progression of the disease had been documented by 2002" (2006, p. 16). The Chernobyl Forum report also concluded that current environmental conditions inside the exclusion zone have a positive impact on plant and animal life, even though some scholars argue that these conclusions were based on limited information from Western peer-reviewed journals and generally, did not take into account scientific reports from Eastern European scholars (Mousseau and Møller, 2011). In sharp contrast to the Chernobyl Forum findings, the Ukrainian National Commission of Radiation Protection estimates that 500,000 people have already died (Vidal, 2010). In a similar vein, the International Physicians for the Prevention of Nuclear War (IPPNW) claims that the death toll among liquidators falls between 112,000 and 125,000 (IPPNW, 2011) thus supporting the institutions that count higher effects from Chernobyl than the United Nations. Whatever the case, Yablokov's study drastically deviates from mainstream scientific consensus – and even non-mainstream scientific consensus – on the effects of low-dose radiation.

The critiques of Yablokov and his colleagues have so far been harsh. Scholars have argued that Yablokov's findings in the Chernobyl volume are inconsistent, lack rigorous scientific methodology, and exclude scientific research that reports lesser or no negative impacts of the Chernobyl accident (Dreicer, 2010). For instance, Mikhail I. Balonov, a Soviet trained radiobiologist and a consultant for the IAEA, concludes in a review published by the New York Academy of Sciences that "intervention of incompetent people, although having academic titles, in this delicate process prevents adequate public information and decision making by authorities responsible for protecting the population" (Balonov, 2011, p. 3). It is clear that Balonov is referring here to Yablokov. Before becoming a consultant for the IAEA, Balonov was one the first Soviet scientists to begin work on the biological effects of Chernobyl fallout. Shortly after the Chernobyl disaster, Soviet authorities sent Balonov to confront a crowd of people in the con-

taminated zone who were anxious about radiation exposure and feared a government cover-up. To assuage their fears, Balonov appeared with ''a government scientist, who, along with his pregnant wife, would be living and working in the contaminated area'' (2006, http://iaea.org/newscenter/focus/chernobyl/pdfs/balonov.pdf).

Still more recently, in a letter to the editor published in *Mutation Research/Genetic Toxicology and Environmental Mutagenesis*, Jargin (2012), writing from his position at the People's Friendship University of Russia in Moscow, challenges the findings reached by Dubrova et al. throughout the 1990s, arguing strongly that the exposures of the irradiated families used in their studies were too low to explain the increases in minisatellite mutation rate. In a stern reply in the same journal, Dubrova (2012, p. 103) asks, ''Where has the author been before?'' suggesting that Jargin appears to be a neophyte in this debate.

Conclusions

In a recent issue of *Bulletin of the Atomic Scientists*, anthropologist Johnston (2011) articulates the unspoken fears of civilian populations everywhere. She notes that in the initial hours and days after the earthquake and tsunami, the Japanese government and Tokyo Electrical Power Company (TEPCO) reported only minor damage to the Fukushima nuclear power plant, declaring that the ''venting of hydrogen gas'' posed ''no threat to health.'' Johnston points out the similarity of the Fukushima assertions to the oft-repeated Cold War era assertion that ''low-level exposure to radiation represents no human threat,'' a statement that we recognize now as serving to ''meet government and industry needs'' (2011).

''Reassuring'' public discourses such as these have intellectual histories (Johnston, 2011; Button, 2010). But so too do scientific journal debates that replicate and embody the epistemological preferences for particular forms of method and experimentation established in the early twentieth century. The debate over ionizing radiation and genetic risk involves actors local and global, national and international, singular and collaborative, all of whom endured a series of wars and government interventions and shared in the trajectories of scientific cultures located in the United States, Europe, Japan, and the Soviet Union. Both James Neel and Yuri Dubrova are products of political and scientific milieux. The contours of their lives, collaborations, and research trajectories shed additional light on our understanding of scientific knowledge

about radiation. This article draws partial inspiration from Latour's (1987) implication that by "following scientists around," the social scientist can better comprehend the coproduction of facts, theories, machines, human actors, and social relations.

By focusing on the biographies and intellectual trajectories of two scientists on either side of the Cold War and post-Cold War, we have examined how contemporary understandings of radiation risk are linked to these figures, their training, to the development of methods and statistics in the field of genetics, and to the degraded state of Soviet genetics and how this translated into scientific privilege. In other words, the research on the genetic effects of low-level ionizing radiation exposure has been "co-configured" by scientific advances and socio-political context. Recent anthropological studies of science assume that social, historical, political, and economic conditions are already embedded in the fundamental directions of scientific research (e.g., Latour, 1987, 1988; Latour and Woolgar, 1986; Fujimura, 1998). Applying this reasoning to our understanding of genetics and ionizing radiation, we recognize that there is a set of interests at play in the determination of our current scientific knowledge.

James Neel's status and scientific reputation gave him the opportunity to help establish radiation safety standards for nuclear workers and the public. Through the interdependency of funding and prestige, they also enabled him and his colleagues to build the large-scale statistical models in population and epidemiological genetics that have become the gold standard for research, while other scientific methods and approaches were dismissed as substandard. Of course, the questionable status of Soviet genetics in the early part of the twentieth century and the rise of Lysenkoism had already helped that narrative take form (DeJong, 2010; Bauer, 2010). This meant that while the debates over radiation effects were not entirely resolved by the scientists working on them, their findings-in-process became consequential in other realms of power and knowledge, such as the courtroom. Neel and Schull (1991b, p. 492) make reference to a study on congenital defects related to the Hanford nuclear facility, observing that the authors of that study use the Japan data to dismiss possible associations of paternal employment at the plant and the defects. In the final chapter of their 1991 publication of the Japan research, Neel and Schull (1991b) defend the nuclear industry with respect to the incidence of leukemia in children of parents working at the Sellafield nuclear reprocessing plant in Seascale, West Cumbria, England. Clearly, Neel felt both equipped and compelled to

offer his scientific opinion to defend the standards already in place and to opine in cases involving workers' health and safety.

As the historian Brown (2012, p. 3; 2013) recently noted in her summary of the 1990s court cases brought by residents living downwind from the Hanford Nuclear Site in southeastern Washington against five corporations working for the U.S. government: "Scientists drawing on American-directed studies of Japanese bomb survivors narrowed the field of inquiry to a few carcinomas and thyroid disease. Downwinders, however, connected their sheep born without eyes to birth defects in their children." The thyroid study cited by Brown did not address genetic effects or many other health problems that Russian scientists have discussed in medical literature as 'chronic radiation syndrome,' a condition never documented in the U.S. studies. The Hanford cases point to how the scientific status of the Japan studies – what they found and what they failed to find – continues to play an important role not only in scientific debates about the genetic effects of ionizing radiation, but also dozens of other debates taking place in scientific journals and courtrooms that still rely on Hiroshima and Nagasaki atomic bomb data as the standard against which all other information is compared.

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