

**FUKUSHIMA UPDATE: RADIOACTIVE FALLOUT
AND MORTALITY INCREASES IN THE UNITED STATES:
IS THERE A CORRELATION?**

Joseph J. Mangano and Janette D. Sherman

Our publication of “An Unexpected Mortality Increase in the United States Follows Arrival of the Radioactive Plume from Fukushima: Is There a Correlation?” in the *International Journal of Health Services* 42(1) reported an unusually elevated number of excess deaths in 122 U.S. cities during the 14 weeks following the mid-March 2011 arrival of airborne radioactive fallout from Japan. The publication has received considerable attention from scientists, media, and the public, since it is the first peer-reviewed publication to examine population-based data before and after the meltdowns at Fukushima. Most of the responses have been objective or supportive, with a minority offering criticisms of the methods and results. In this paper, we respond to several of the critiques, offering supportive evidence from our research and supportive research in the medical literature. We hope our comments are constructive and we view our research as a correlation, and *potential* evidence of a causal link, between radioactive exposures from Fukushima and increased health risk, underscoring the need for additional and prompt reports on the topic.

Our December 2011 article in the *International Journal of Health Services* 42(1) discussed potential health hazards of the multiple meltdowns at the Fukushima Daiichi nuclear power plant in Japan (1). After the arrival of airborne radioactive fallout, reported deaths in 122 U.S. cities rose unexpectedly compared with the same 14 weeks in 2010. Assuming the 122 cities represent the United States as a whole, the analysis calculated the number of excess U.S. deaths during this period to be 13,983, a figure relatively close to that found in the first four months after the Chernobyl meltdown in 1986 (2). Deaths for

infants under age 1 had a greater percentage excess than that for all ages (similar to the post-Chernobyl period).

The report received considerable interest, evoking questions and comments from scientific professionals, citizens, and the media. We shall address four comments here, using citations from the scientific and medical literature and updated information. We appreciate the opportunity to address these comments in a collegial manner. We recognize that this topic—that is, population-based studies of potential health consequences from Fukushima fallout—has never been published and thus is more likely to be the recipient of critical comments. This will be only one of many studies concerning the effects of Fukushima on health, research on which will be a decades-long process, and we sincerely hope that our efforts lead to an improved understanding of the topic, just one year after the nuclear meltdowns in Japan.

COMMENTS FROM REVIEWER #1:

ALEX WOLF

Wolf stated that “cause of death has not been analyzed” in our article. The source we used for deaths was the *Morbidity and Mortality Weekly Report* (MMWR), a publication of the U.S. Centers for Disease Control and Prevention. While deaths for all ages and for five age groups are given for 122 U.S. cities, no information on cause of death is available, except for pneumonia and influenza (3). Thus, our initial article began with data for all causes combined, for all ages and for infants under age 1. Updated analysis from the MMWR shows the all-age 2010-2011 increase in reported pneumonia and influenza deaths in the 14 weeks following Fukushima (March 20–June 25) was 11.49 percent versus 1.55 percent for the other 38 weeks, a much greater difference than for all causes combined (3.99% vs. 0.73%). Ultimately, all causes should be analyzed, but cause-specific data will be available only when final 2011 death statistics are made public, probably in late 2014.

Wolf also stated that there is “no known mechanism for low-dose radiation to cause acute death in infants or adults.” Our previous article addressed the heightened ability of relatively low doses of radioisotopes to harm the radio-sensitive fetus and infant within a short period after exposure (4). The large doses relative to small body weight, immature immune system, undeveloped liver less able to detoxify, and rapid cell division rate (of radiation-damaged cells) have been cited in many studies as a cause of harm to the very young. As stated in our previous paper, such harm can manifest as spontaneous abortion, premature birth, low birth weight, stillbirth, infant death, congenital malformation, under-active thyroid gland function in newborns, and brain damage—all within weeks or months after exposure.

Evidence supporting this relatively short latency appeared in the 1950s and the early 1960s, the era when hundreds of large-scale atomic bombs were

detonated in the atmosphere by the United States and Soviet Union. Sites of the detonations were remote, but wind and rain precipitated radionuclides—the same emitted by Fukushima—into the environment across the United States, where they were ingested and incorporated into humans through the diet.

The 13 percent decline in U.S. infant mortality rates (11% for neonatal mortality) from 1951–1965 easily represented the slowest decline in the 20th century; the 14-year periods before and after reveal about a 50 percent decline. The U.S. rate of low-weight births (<2,500 grams) rose 2 percent for whites and 35 percent for non-whites from 1950–1966, only to be followed by steady, sharp declines for nearly two decades (5, 6). In addition, reports from Greece (7), Germany (8), Wales/Scotland (9), and the United States (10) showed unusually large increases in leukemia diagnosed before the first birthday immediately after relatively low-dose exposures from the 1986 Chernobyl meltdown.

Aside from the very young, elderly persons are likely to bear the greatest risk from exposure to anthropogenic radiation. Immune system declines in later years and many elderly people suffer disproportionately larger adverse effects from existing medical conditions and/or the effects of pharmaceuticals, chemotherapy, radiation tests and treatments, or other medical procedures. The data cited in this response on pneumonia/influenza deaths, with 90 percent are persons over 65. These data after Fukushima may be one piece of evidence suggesting low-dose radiation can cause relatively rapid deaths in the elderly. The idea of a physically weakened, elderly person exposed to radiation from Japan, whose condition proves fatal within months of exposure, is a thesis not proved or disproved, but one that merits further examination.

Wolf claims that our article “has not considered” the dose-dependency in the assessment of radiation exposure from Fukushima and mortality risk. On the contrary, pages 49–51 present the environmental radioactivity measurements by the U.S. Environmental Protection Agency (EPA) immediately after the meltdowns began. We made clear that the EPA’s ability to detect radioactivity levels in late March and April 2011 for only a small proportion of samples of air (13.3%), precipitation (6.2%), milk (2.4%), and drinking water (2.4%) meant that “no meaningful temporal trends and spatial patterns can be discerned” in the United States from these few samples. The EPA also switched its program of weekly measurements to quarterly ones on May 3, fewer than seven weeks after airborne Fukushima fallout arrived in the United States, making analysis of dose data even more difficult.

Rather than “not considering” dose data, we used surrogate EPA findings of iodine-131 concentrations in precipitation 100 to 200 times above normal in late March 2011—knowing that better dose data will be needed. It may be possible to judge relative doses in various parts of the country by using EPA samples on gross beta in air, taken weekly in at least 55 U.S. cities in 2010 and 2011 (11).

Wolf asserts that “California cities would have received larger doses” from Fukushima relative to the United States as a whole. Not only does he not cite a

source for this statement, but as just mentioned, it is currently not possible to conclude which cities or states received more or less exposure using official EPA measurements, a statement we made in the article (p. 54). We also cautioned against examining data from individual cities, since changes in voluntary reporting practices and in random variations are more likely to alter results from individual cities than the United States as a whole.

Following his statement that California was relatively hard hit by radioactive fallout from Japan, Wolf cites data from the California cities of “Los Angeles, San Diego, and San Antonio.” The city of San Antonio is located in the state of Texas, not California.

Wolf concludes that “there are innumerable factors other than radiation that can affect these numbers, and indeed are likely responsible for the bulk of the measured effect.” He stated none, because no information on solutions has been made available.

By using the most recent data, the projected “excess” of 13,983 United States deaths for the 14-week period March 20–June 25, 2011, has increased to 21,851. Efforts to analyze potential factors must begin promptly.

COMMENTS FROM REVIEWER #2:

DR. ALFRED KORBLEIN

Körblein claimed that the increase in infant deaths in our previous report was a result of using 119 cities with 99 percent+ reporting in weeks 12 to 25 for 2010 and 2011 and there being only 104 cities that met these criteria in the previous 14 weeks. He states that a trend analysis shows a decrease in infant deaths and thus “the excess infant deaths come from the 15 additional cities.” This is a hypothesis, but one that cannot be proved because of substantial missing data in the other 15 cities, making the use of 119 cities in each period an “apples to oranges” comparison. A more prudent approach would be to question—not conclude—whether the missing 15 weeks was the cause of excess deaths.

We can test whether excluding the 15 cities is the sole reason for finding excess infant deaths. Comparing 2010–2011 changes in weeks 12 to 25 versus all other 38 weeks can be done by only including the weeks/cities in which deaths were reported for both 2010 and 2011. For example, infant deaths for Washington, D.C., in weeks 5, 8, 9, 10, and 12, 2010, were “unavailable” so the five infant deaths reported in these weeks in 2011 were removed from the analysis. Thus, 95.82 percent (6,079 of 6,344) of weeks/cities were included in the report for 2010 and 2011. The 2010–2011 increase in infant deaths for weeks 12 to 25 (+1.04%) was greater than the decrease during the other 38 weeks (–3.67%), suggesting that there was an actual excess.

Körblein also commented on whether the 2010–2011 change in reported infant deaths in weeks 12 to 25 (post-Fukushima) was significantly different than the change for the previous 14 weeks. Our method used a chi-square test and found

the difference (+1.80% vs. -8.37%) to be highly significant at 0.0002. In our analysis, the observed change in deaths was 1.0180 (change in deaths in weeks 12 to 25); the expected was 0.9163 (change in deaths in the previous 14 weeks); and the number of deaths was 2,722 (deaths in weeks 12 to 25, 2011).

Körblein, however, used several tests, concluding that the change was not significant:

- He first employed a t-test with 52 degrees of freedom to find the difference in infant death changes to not be significant at $p < 0.101$.
- When shown updated figures, comparing 2010–2011 infant death changes in weeks 12 to 25 with the other 38 weeks of the year, his test showed the difference to be highly insignificant at $p < 0.997$; our test found a difference of borderline significance at $p < 0.08$.
- Again using updated figures, Körblein even stated that the difference in changes for deaths in all ages was not significant at $p < 0.181$. We found the difference in changes to be highly significant at $p < 0.000001$ —not surprising since the sample size of deaths for all ages in weeks 12 to 25, 2011, was extremely large (154, 162). In addition, we found differences in deaths for ages 45 to 64, for ages 65-and-older, and for pneumonia and influenza all to be highly significant, while differences in deaths for ages 1 to 24 and 25 to 44 were not significant (see updated information below).

Table

Reported deaths, 2010 versus 2011, 122 U.S. cities by age at death and pneumonia/influenza (all ages): Weeks 12 to 25 versus other 38 weeks

Age	Weeks 12–25		Other 38 weeks		% Ch. 2010–2011		
	2010	2011	2010	2011	12–25	Other	Difference
<1	2680	2708	7660	7379	+1.04	-3.67	+4.71 $p < 0.08$
1–24	3191	3258	8800	8714	+2.10	-0.98	+3.08 $p < 0.22$
25–44	8458	8858	23248	23891	+4.73	+2.77	+1.96 $p < 0.21$
45–64	34512	36134	95475	96683	+4.70	+1.27	+3.43 $p < 0.00001$
65+	99328	103124	275185	276817	+3.82	+0.59	+3.23 $p < 0.000001$
All	148254	154162	410742	413728	+3.99	+0.73	+3.26 $p < 0.000001$
P+I	10317	11502	28062	28497	+11.49	+1.55	+9.94 $p < 0.000001$

Source: U.S. Centers for Disease Control and Prevention, Morbidity and Mortality Weekly Report, Volumes 59 and 60 (www.cdc.gov/mmwr) that uses 6,079 of 6,344 cities/weeks with reported deaths.

Statistical significance testing uses chi-square tests for each age group, with Observed = percent change in weeks 12 to 25; Expected = percent change in other 38 weeks; and N = number of deaths in weeks 12 to 25, 2011.

We recognize that experts sometimes choose different statistical methods to use the same set of data and there will be disagreements over the results. We respect Körblein's contentions, but believe we have selected the test that is more appropriately applied to our report, that is, to begin to explore whether U.S. deaths increased in a defined period after Fukushima, as had been found after Chernobyl fallout entered North America. Ours is a descriptive study of mortality change, which typically requires only a basic test of significance such as chi-square. We have discussed the matter with biostatistics experts in the United States and the United Kingdom, and both agree that while other tests can be used, our choice of a chi-square test is entirely appropriate for this paper—due not in small part to the enormous data base used (568,000 U.S. deaths reported annually to the MMWR).

We believe that Körblein's choice of a regression analysis using the dispersion of weekly deaths is not the most appropriate one for this analysis. Weekly changes in deaths from year to year vary slightly, for a variety of reasons. It is not imperative at this point to demonstrate consistency in weekly changes.

Körblein's choice of regression modeling should not discount a large and still-unexplained elevation in reported deaths for the period March 20–June 25—comparable to the still-unexplained increase that followed the Chernobyl disaster in 1986 (2). We believe that his choice of test may be more appropriate in future analyses. Perhaps the most critical aspect of a study such as this is to establish “red flags” suggesting potential consequences, to help guide future research.

COMMENTS FROM REVIEWER #3:
DR. ROBERT GALE

Gale claims that our article implicates “radiation released from these accidents and arriving in the United States as the likely cause” of unusual increases in U.S. deaths in the months following Chernobyl and Fukushima. We never specified “likely cause.” Rather, we presented these data as unusual occurrences that merit more detailed examination; and that while there are multiple potential contributing factors, exposure to radioactive isotope fallout should be considered as one of these.

Gale repeats Wolf's contention that no biological plausibility exists to support short-term death risk from relatively low doses of radioactivity. We have described our response to this contention in this and other reports. In addition, Gale states “there have been no radiation-related deaths in emergency or recovery personnel exposed to doses thousands of times higher.” This statement is misleading in that no reports on worker exposure and health have been released to date by either the Japanese government or the utility that operates the Fukushima plant. Moreover, a recent report found that 573 deaths in 13 municipalities in the evacuation zone have been attributed by officials to radiation exposure from

the meltdowns, with dozens more deaths under review (12). As with Chernobyl, there are reasons why the release of exposure, morbidity, and mortality data by the Japanese government and the Tokyo Electric Power Company has been intermittent, confusing, and slow.

Gale contends that “radiation doses received by Americans from Fukushima and Chernobyl are extremely low.” This statement failed to note that while such doses were lower than those received by local residents near Chernobyl, for example, they are higher than usual doses to Americans. We already have stated that EPA samples of iodine-131 in precipitation detected levels up to 200 times higher than typical levels. In addition, a recent report found levels of radioactive xenon-133 in Richland, Washington, up to 40,000 times higher than normal in the weeks following Fukushima (13).

Gale also states that “there is uncertainty whether such low doses of ionizing radiations (sic) are as likely to be harmful to humans as higher doses, even when adjusting for dose differences.” Most agree that the linear no-dose threshold model between radiation exposure and health risk is most likely to be accurate (14). Such a model means that *all* doses of radiation carry some risk to humans, even at the lowest levels, and all exposures are cumulative. Historically, assumptions that relatively low doses of radiation exposure posed no harm to humans were found to be false. Studies have documented an increased risk of childhood cancer mortality after pelvic Xrays to pregnant women (15); thyroid cancer increase after fallout from atmospheric atom bomb tests in Nevada (16); and increased cancer incidence and mortality for nuclear weapons plant workers occupationally exposed to radiation (17).

Gale says that “epidemiologic studies like that of Mangano and Sherman deal with correlations, not cause and effect.” He gives an example that persons with high blood pressure often awake with a headache—a correlation, but not a cause and effect between sunrise and hypertension. We agree that our previous article demonstrates a correlation and not a cause and effect. However, the potential cause we are presenting is not an everyday event such as sunrise. It is exposure to elevated levels of radioactive fission products, perhaps the most toxic of chemicals, from an event acknowledged to be one of the two most catastrophic nuclear meltdowns in history (and one that is still not controlled, one year later). While more study is needed, again, the potential that exposure to Fukushima fallout played a role in increased deaths in the months following the meltdowns is not an illogical assumption and merits more research.

CONCLUSION

It took nearly 12 years after the March 1979 meltdown at the Three Mile Island (USA) nuclear power plant before the first journal article examining changes in local morbidity and mortality appeared (19). Quickly, a second article was published by the same team from Columbia University, stating there was no link

between exposure from the meltdown and increased local cancer incidence—suggesting instead psychological stress as the cause (20). A re-examination of the same data by researchers from the University of North Carolina concluded there was a link between exposure levels and local increases in cancer incidence (21). Comments then followed from these two sets of researchers, which sometimes were harshly critical and disparaging of the others' motives (22–24). For example, a member of the Columbia research team accused the University of North Carolina researchers of causing a “brouhaha” by employing “poor science” and “advocacy parading as science” (22). Unfortunately, the issue of whether Three Mile Island emissions are linked with excess local cancer risk remains contentious, 33 years after the meltdown. We hope that attempts at understanding Fukushima health consequences do not encounter a similar fate.

Problems in determining the number of casualties from the catastrophic Chernobyl meltdown in 1986 also have been widespread. For several decades, some officials used a small number of liquidators who died shortly after extinguishing the fire at the stricken reactor as the total number of casualties; as late as early 2011, the World Nuclear Association still used the figure of 40 such deaths (25). In 2005, a Vienna forum led by the World Health Organization and the International Atomic Energy Agency resulted in an updated calculation of 9,000 persons with Chernobyl-related cancer worldwide (26). In 2009, in a new report that used more than 5,000 reports, many in Slavic languages never before translated into English, scientists estimated that Chernobyl caused 985,000 deaths worldwide in the first 20 years after the meltdowns (27).

We hope that published reports in the literature on potential Fukushima health consequences are conducted and reported promptly. While disagreements in methods and findings are inevitable and welcomed, opinions must be presented in an evidence-based, open-minded, and constructive manner. Based upon historic knowledge and the understanding of the risks of radioactive fallout, we ask that our findings correlating the Japanese fallout as one potential cause of excess U.S. deaths not be dismissed until further research is conducted. We look forward to working collegially with others interested in the topic.

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Direct reprint requests to:

Joseph J. Mangano
716 Simpson Avenue
Ocean City, NJ 08226

odiejoe@aol.com