INTERNATIONAL JOURNAL OF
HEALTH SERVICES
health and social policy
political economy and sociology
history and philosophy
ethics and law

editor-in-chief: vicente navarro

Volume 29, Number 2—1999

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A RISE IN THE INCIDENCE OF CHILDHOOD CANCER IN THE UNITED STATES

Joseph J. Mangano

From the early 1980s to the early 1990s, the incidence of cancer in American children under 10 years of age rose 37 percent, or 3 percent annually. There is an inverse correlation between increases in cancer rates and age at diagnosis; the largest rise (54 percent) occurred in children diagnosed before their first birthday. Rates rose for all 11 states and cities included in the analysis. A jump in cancer rates for children born in 1982–83 was followed by a drop; but another abrupt rise for the 1986–87 birth cohort has been sustained thereafter. Results indicate that the rising childhood cancer rate represents a far more serious problem in the United States than previous reports have suggested. The methodology used here adds three additional states and cities, analyzes children under 10 rather than under 15, begins the analysis in 1980 rather than in 1973, and extends the study to 1993, which may partially account for the new findings. There are no apparent explanations for these trends, suggesting that researchers should analyze the data more fully and propose hypotheses on potential causes. One possible factor, fetal and infant exposure to low-dose radioactivity, is explored here.

In recent years, published reports have measured the change in U.S. childhood cancer incidence (1, 2). While each has documented a modest rise in incidence for age group 0–14 for all cancers of about 1 percent a year, none has described the increase as unusual. These reports generally cover the period 1973 to 1991. Other reports have described similar trends in industrialized countries such as Australia, Japan, and Italy (3–5).

Most first world countries have centralized national tumor registries that aggregate data based on uniform rules and methods. The United States leaves the decision to collect cancer incidence data to each state or municipality. Only in 1973, as part of President Nixon's War on Cancer, did established registries from five states and four cities, representing just under 10 percent of the U.S.

International Journal of Health Services, Volume 29, Number 2, Pages 393–408, 1999

population, combine to form the Surveillance, Epidemiology, and End Results Program (SEER) of the National Cancer Institute (6). Many analyses of U.S. cancer incidence patterns rely on SEER data. However, while other states and cities operated established registries by the 1970s, they were not part of the SEER system. This article analyzes childhood cancer trends not just in the SEER areas, but in other localities with registries established in the 1970s. It will also subdivide the 0–14 year old population into more detailed age groups, and will examine specific incidence patterns heretofore unexplored.

METHODS

Data were collected from all nine SEER registries except Atlanta, which declined to participate. In addition to the SEER areas, data on childhood cancer cases were obtained from New York State, Wisconsin, and Denver, all of which operated established tumor registries by the late 1970s. Adding these three regions to SEER nearly doubled the population included for study, to about 19 percent of the nation. The analysis is restricted to cancers diagnosed in children aged 0–9, adding precision to the report while still retaining about three-fourths of cancers diagnosed in children under age 15. The study begins in 1980 (when all registries had complete reporting) and concludes in 1993, the last year for which complete data are available. Invasive malignancies of all sites (standard ICD-9 codes 140–208) were included, except for those diagnosed as in situ. To obtain population-based rates, the 1980 and 1990 U.S. Censuses were used to calculate estimated populations for 1981 to 1989. Annual population estimates made by the Census Bureau are used for 1991, 1992, and 1993. The 1980–82 and 1991–93 average populations are used as denominators for computing cancer rates in this report. Data on live births, also used in the report to obtain rates for children under 1 year, are collected by state and local health departments, and published in the National Center for Health Statistics annual compilation *Vital Statistics of the United States.*

Each cancer registry has maintained long-standing policies for collecting data and ensuring its accuracy and completeness. While each registry may differ somewhat in its methods, all registries report that their rules have been consistent over time, making trend analysis feasible.

For comparative purposes, childhood cancer rates in England and Wales were obtained from the United Kingdom's Office of Population Census and Surveys in London, along with population estimates based on the 1981 and 1991 census population counts and estimates for all other years. British cancer data (also ICD-9 140–208) have been collected nationally since 1962, and reporting is generally considered complete; analyzing trends in Britain may help in appraising completeness of cancer data collected by the 11 U.S. registries. Nearly 7 million American children under 10 years of age live in the areas studied, roughly equivalent to all children residing in England and Wales.

RESULTS

Table 1 indicates that U.S. cancer incidence rates for age groups 0–4 and 5–9 rose considerably from the early 1980s to the early 1990s. From 1980–82 to 1991–93, the earliest and latest three-year periods studied, incidence rose from 16.11 to 22.68 per 100,000 persons (a 40.8 percent increase) for the 0–4 age group; the rate for age group 5–9 advanced from 9.38 to 12.22 per 100,000 (up 30.3 percent). Very little change in rates occurred after 1991. The overall rate for age group 0–9 increased 36.6 percent. All changes are highly significant ($P < .00001$).[1]

The trend in childhood cancer incidence for the 11 U.S. cities and states is contrasted with that in England and Wales in Figure 1. Unlike the steady rise in the United States, Britain's childhood cancer rate rose sporadically and more gradually, advancing only 16.2 percent from 1980–82 to 1990–92, less than half of the U.S. increase of 36.6 percent. In some years, the British rate drops sharply, a phenomenon that never occurred in the United States. The British incidence rate was below the U.S. rate in the early 1980s, and the gap has widened since then. Such a contrast may validate the completeness of U.S. data, since the British national registry is recognized as complete and consistent over time (7).

An especially large upturn in cancer rates from the early 1980s to the early 1990s occurred in infants aged 0–1. From 1980–82 to 1991–93, the rate of cancer cases diagnosed in children before their first birthday soared 53.6 percent, from 17.86 to 27.43 per 100,000 population. The annual number of cases in the studied areas for this age group is now about 200, up from 120 a decade before. The rate continues to climb steadily in the 1990s, to a high of 31.64 per 100,000 in 1993.

Another way of examining these same data is by birth cohort. Figure 2 (on p. 398) shows sharp rises for two cohorts, namely children born in 1982–83 and in 1986–87. These increases occurred for cancers diagnosed before ages 1, 3, and 5 years. There was a downturn in rates in 1984–85, suggesting that the jump for 1982–83 births was a one-time phenomenon. However, rates for children born after 1987 remain similar to the 1986–87 averages, suggesting that the reason(s) for the change continues to affect children born subsequently.

Using the above data, a rough estimate can be made of how many U.S. children under 10 years old are diagnosed with cancer each year. The 1990 Census shows that children in the 11 areas studied made up 18.6 percent of that age group nationwide, compared with 19.7 percent in 1980. Thus, the annual number of cases in the early 1980s for the entire country would be 4,286, compared to 6,487

[1] Calculation of the significance of cancer incidence changes in Table 1 employs the standard formula $(O - E)/\sqrt{(O^2 + E^2)/N}$, found in the annual publication *Vital Statistics of the United States.* O = observed incidence rate 1991–93; E = expected 1991–93 rate (the 1980–82 rate); and N = number of reported cancer cases in 1991–93. The computation yields the number of standard deviations by which the observed rate differs from the expected, enabling the area under the normal curve and statistical probability to be reached.

Table 1

Cancer incidence in age group 0–9, number of cases and rate per 100,000 population in 11 U.S. states and cities[a]

	Age 0–4		Age 5–9		Total age 0–9	
Year	Cases	Rate	Cases	Rate	Cases	Rate
1980	544	15.92	300	9.71	844	12.97
1981	553	16.11	300	9.66	853	13.04
1982	562	16.29	274	8.77	836	12.72
1983	617	17.81	292	9.30	909	13.76
1984	612	17.58	303	9.59	915	13.78
1985	654	18.70	363	11.43	1,017	15.24
1986	687	19.56	342	10.71	1,029	15.35
1987	645	18.28	354	11.03	999	14.82
1988	668	18.85	359	11.12	1,027	15.17
1989	740	20.78	393	12.11	1,133	16.65
1990	698	19.52	418	12.81	1,116	16.32
1991	792	22.59	406	12.39	1,198	17.66
1992	807	22.73	399	12.09	1,206	17.61
1993	811	22.72	405	12.17	1,216	17.63
980–2	1,659	16.11	874	9.38	2,533	12.91
991–3	2,410	22.68	1,210	12.22	3,620	17.63
ercent change	40.8%		30.3%		36.6%	

[a]Connecticut, Denver, Detroit, Hawaii, Iowa, New Mexico, New York, San Francisco, Seattle, Utah, and Wisconsin.

the early 1990s. An unchanged cancer rate, coupled with a slightly growing opulation, would put the expected number of such cases in the early 1990s 4,750 per year. This computes to an 11-year excess of about 9,500 of the pproximately 63,000 cancer cases for the 0–9 age group diagnosed in the years 983 to 1993.

One issue raised by the data is the consistency of the trend in childhood cancer itionwide. Table 2 shows the change from 1980–82 to 1991–93 in each state or etropolitan area in the analysis. While each of the 11 recorded an increase, the agnitude of changes varied. Rates in Hawaii, Iowa, and Utah all rose less than percent, far below Detroit (116.3 percent), New York (55.4 percent), and enver (37.9 percent). The 1991–93 rate in each of the 11 regions is well above e 1980–82 U.S. average. Childhood cancer rates in the eight SEER areas creased 24.5 percent, only about half of that in the three non-SEER regions 7.5 percent).

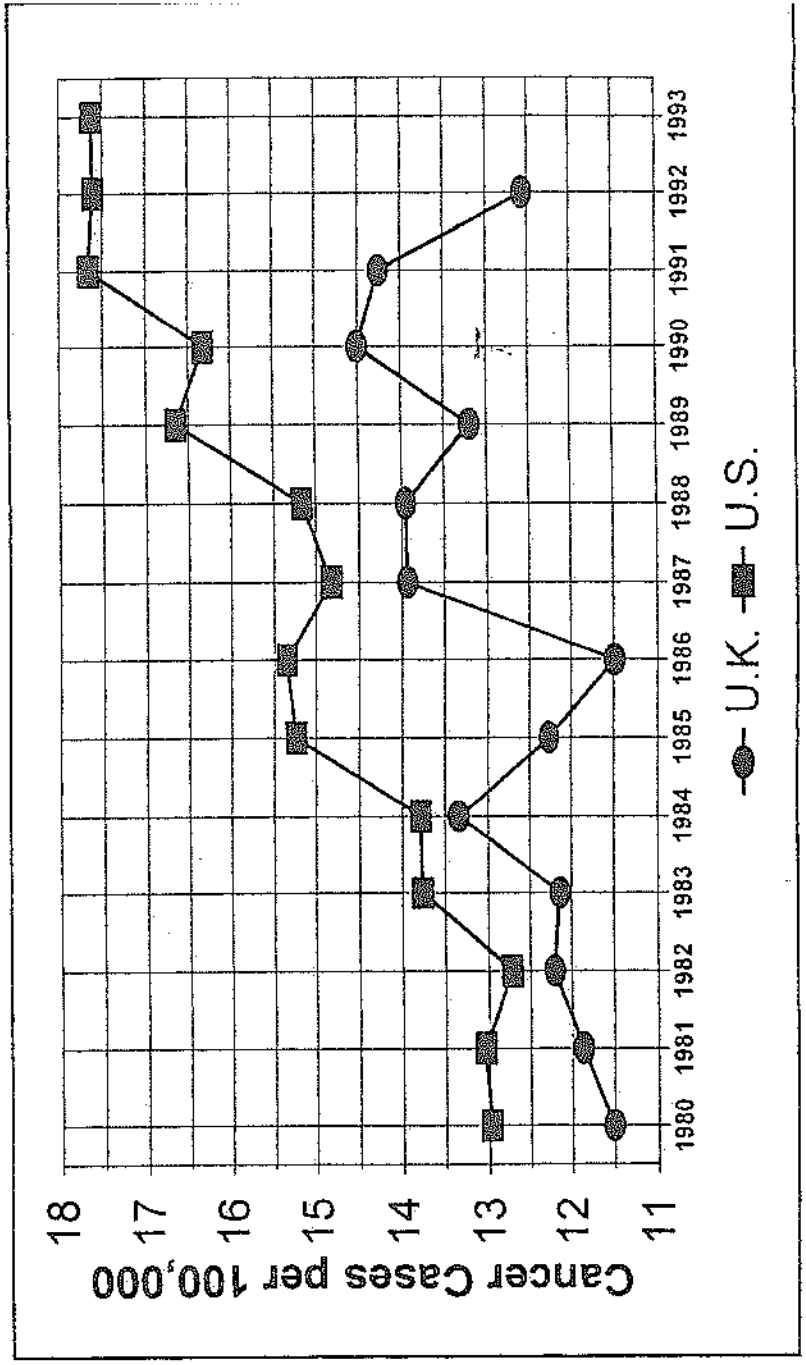

Figure 1. Cancer incidence in the 0–9 age group, 11 U.S. states and cities and the United Kingdom, 1980–1993.

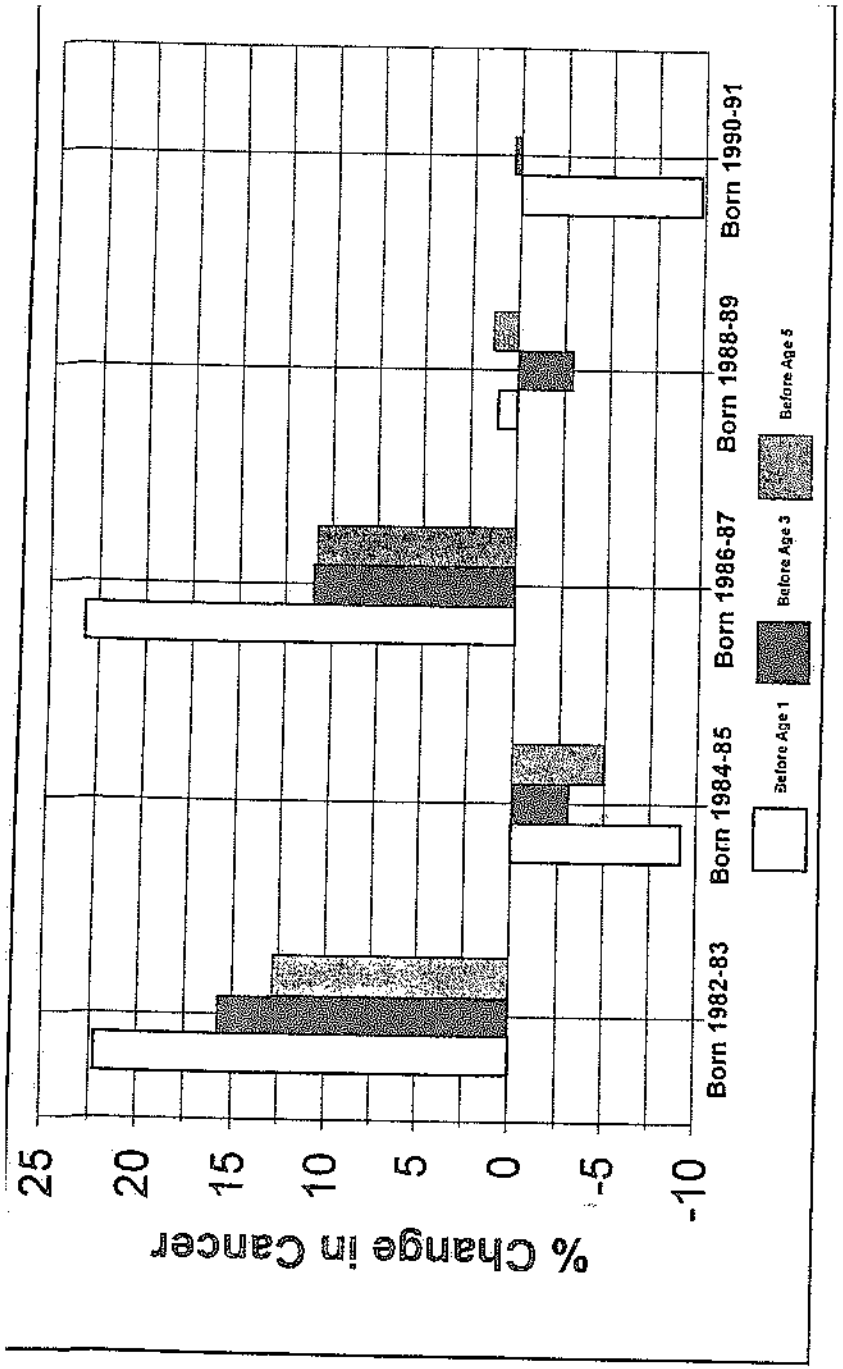

Figure 2. Cancer incidence by birth year in children under 5 years old, 11 U.S. states and cities, 1982–1993.

Table 2

Cancer incidence in age group 0–9, in 11 U.S. states and cities, 1980–82 to 1991–93

State/city	No. of cases		Rate, per 100,000		Percent change
	1980–82	1991–93	1980–82	1991–93	
Connecticut	169	225	14.24	16.57	16.3%
Denver[a]	83	142	11.56	15.95	37.9
Detroit	139	290	7.73	16.73	116.3
Hawaii	73	84	15.98	16.32	2.1
Iowa	189	185	14.64	15.74	7.5
New Mexico	73	112	10.69	14.00	31.0
New York[a]	994	1,539	12.89	20.03	55.4
San Francisco	178	254	15.06	16.45	9.3
Seattle	169	263	14.28	16.72	17.1
Utah	151	163	14.90	15.46	3.7
Wisconsin[a]	315	363	13.14	16.46	25.2
Total	2,533	3,620	12.91	17.63	36.6%
8 SEER	1,141	1,576	12.98	16.16	24.5%
3 Non-SEER	1,392	2,044	12.86	18.96	47.5

[a]Non-SEER area.

DISCUSSION

The data presented contain a number of new findings on recent trends in U.S. childhood cancer incidence between the early 1980s and early 1990s.

1. *Beginning the analysis in 1980 better shows the actual trend.* From 1980–82 to 1991–93, childhood cancer incidence in the U.S. rose 36.6 percent, or about 3 percent per year, according to data from the sample of 11 states and cities. Several reports mentioned earlier have described a 1 percent annual rise from 1973 to 1991; however, virtually all of the recent increase began after 1982, so starting the analysis in 1980 more accurately describes the current trend. A look at SEER-generated cancer rates for age group 0–14 shows little change from 1973–75 to 1980–82 (12.4 to 12.6 per 100,000), but an 11 percent jump from 1980–82 to 1990–92, when the rate reached 14.0 per 100,000 (6). Thus, using 1973 as a base year may understate the magnitude of the present trend.

2. *U.S. data are likely to be accurate.* The U.S. childhood cancer increase of 36.6 percent is more than double the 16.2 percent rise in England and Wales for the same time period. The U.S. rate has consistently been higher than the U.K. rate, even though the United Kingdom, with a centralized, well-established cancer registry, probably has little undercount of cases, if at all. Thus, despite the

need to combine information from multiple tumor registries, there is no evidence suggesting U.S. cancer cases are under-reported.

3. *SEER alone is not representative.* Adding New York, Wisconsin, and Denver to the SEER areas changed the analysis dramatically. Cancer in the 0–9 age group in these three regions rose 47.5 percent, versus only 24.5 percent for SEER. While this report only included 19 percent of all American children under age 10, it is clear that SEER, with about 10 percent, cannot be assumed to be a reliable proxy for national patterns. Federal officials should perhaps consider maintaining records for a second, expanded SEER, in addition to continuing the nine-area SEER begun in 1973.

4. *Increases are greatest in the very young.* The analysis found an inverse correlation between rising cancer incidence and age at diagnosis. The rate for children under 1 year at diagnosis rose 53.6 percent, compared to 40.8 percent for those 0–4 and 30.3 percent for those 5–9. Thus, analyzing only the 0–14 age group as a whole may conceal specific patterns in childhood cancer incidence.

5. *Higher rates are evident in all parts of the nation.* In all 11 states and municipalities in the report, higher levels of cancer incidence for age group 0–9 were reported in 1991–93 than in 1980–82, making the trend a national one. The Detroit area had the largest increase (116.3 percent), although its rate for the early 1980s was much lower than any other state or city studied, perhaps suggesting a previous under-reporting of cancer cases.

All trends are, of course, dependent on how completely tumor registries report newly diagnosed cancer cases. It is possible that better detection plays a role in the upward trend; for example, more CAT and other diagnostic medical scans may uncover brain and central nervous system cancers more easily. However, several factors indicate that better detection does not fully explain the rise in childhood cancer. Children are not subject to any routine cancer screening, as adult women are for breast and cervical cancer, for example. As always, physicians only begin to search for childhood cancer in response to symptoms and/or lab test abnormalities. In addition, Great Britain has improved its ability to detect tumors but experienced a more modest rate of increase in cancer incidence. While the degree to which increase is due to better detection remains unresolved, the search for other etiologies must proceed.

The data in this report, while revealing, need to be reviewed more extensively, such as by gender, race, and cancer site. Furthermore, data from the 18 states that developed new registries in the 1980s should also be considered for inclusion in future studies. Among these states are the highly populated California, Florida, Illinois, Massachusetts, North Carolina, and Pennsylvania, which together accounted for 79 million Americans (31 percent) in 1990 (8). Not all of these states have cancer information that is useful in trend analysis of rates. Some maintain voluntary rather than mandatory (complete) reporting, others are still struggling to refine methods of verifying data accuracy and completeness, and still others are not yet capable of producing computerized reports of cancer cases. Nonetheless, even several years of reliable data from more states in the 1990s will bring us closer to understanding the actual rates and trends in U.S. childhood cancer.

RADIATION EXPOSURE: EXAMINING ONE POSSIBLE CAUSE

The logical question posed by these new findings is, what factor(s) have caused them? Any etiological search, especially in a broad area such as all childhood cancers combined, consists of laborious and detailed examinations of each potential reason. The disturbing trends presented here demand that more epidemiological studies, however resource intensive, should be pursued, followed by formulation of hypotheses and clinical research testing.

Of the many potential causes of excess childhood cancer, one possibility that has been raised is that of polluting chemicals in the environment. Several high-ranking federal health officials and other scientists are now concerned about the potential linkage between increasing childhood cancer rates and toxic chemicals (9). Their concern is partly theoretical, but is also grounded in previous scientific discovery. For example, children diagnosed with cancer and leukemia in Denver from 1976 to 1983 were found to have greater exposure to pesticides and herbicides than did healthy children (10).

One type of environmental pollutant that is an established risk factor for childhood cancer is exposure to low levels of ionizing radiation. In the 1950s, British researcher Alice Stewart and colleagues (11, 12) first established the connection between fetal exposure due to pelvic X-rays and excess cancers developed before age 10. During the same period, a growing number of scientists, including Linus Pauling and Andrei Sakharov, became concerned that fallout from atmospheric atomic-bomb testing was entering the food chain, and subsequently causing increases in childhood cancer and other diseases. In Connecticut, the only U.S. state with an established tumor registry during that time, the cancer rate for children aged 0–14 rose from 8.8 to 13.2 per 100,000, a 50 percent increase, between the early 1940s and early 1960s. Children aged 0–4 were particularly affected, as their cancer rate soared 61.5 percent during this time (13). After atmospheric nuclear testing was banned in 1963, interest turned to potential cancer increases in children living near nuclear power plants. In the three Pennsylvania counties closest to Three Mile Island, site of a near-meltdown of a reactor in 1979, cancer deaths in the 0–9 age group soared from a rate 33 percent below to 33 percent above the U.S. average between 1970–74 (before the plant began operations) and 1980–84 (after the accident) (14). A 1990 study by the National Cancer Institute found an excess of cancer cases for children aged 0–9 near nuclear installations in Connecticut and Iowa after the plants began operations (14). A recent 10-month span produced reports showing an excess of leukemia diagnosed in children under 1 year born in 1986 and 1987 in Greece,

ıe United States, and the former West Germany (15–17). These findings were ıe first to link Chernobyl fallout to unexpectedly high levels of disease outide the area closest to the accident. All of the aforementioned forms of radiaon exposure (in utero pelvic X-rays, bomb test fallout, local releases from the hree Mile Island accident, routine emissions from nuclear power plants, and hernobyl fallout far from the accident) represent low levels of radioactivity.

If low-level radiation exposure is to be considered as a potential factor behind sing childhood cancer rates in the 1980s and 1990s, one must pinpoint a link etween specific *new* exposures (not just radioactivity left over from nuclear perations of years before) and subsequent cancer rates. Americans were newly posed from several sources in the past two decades:

Chernobyl. The Chernobyl disaster produced fallout that remained in the merican diet for several months (for short-lived products such as iodine-131) or long as several years (for long-lived products such as cesium-137) (18).

Routine emissions from power plants. The number of operating nuclear power actors in the United States increased in the 1980s and early 1990s; the Nuclear egulatory Commission reports emissions from 90 reactor sites in 1993, up from ; in 1980 (19). Radioactive emissions from these reactors continue to routinely nit small amounts of radioactivity into the environment (20). Some low-level leases are larger than others. For example, the Indian Point plant, 35 miles north New York City, emitted 14.03 curies of airborne effluents in 1985–86, virtually entical to the 14.20 curies released during the Three Mile Island mishap in 79. (The 14.03 figure from Indian Point was later changed to 1.90 due to a nscribing error, according to the U.S. Nuclear Regulatory Commission.) From 80 to 1983, Brunswick reactors 1 and 2 (near Wilmington, North Carolina) and esden reactors 2 and 3 (near Chicago, Illinois) each released over 11 curies of lioactivity; most other U.S. plants emitted under 0.5 curies during this time. e Oyster Creek 1 plant near Tom's River, New Jersey, released nearly 8 curies m 1980 to 1985 (20). While it is difficult to thoroughly review any connection tween such releases and subsequent childhood cancer rates, given the absence established cancer registries in many states, such a link may well exist. It is ssible that the larger emissions at many plants could have had a belated effect childhood cancer since isotopes such as strontium-90 normally take two to ır years after release to peak in the diet.

Accidental releases. Accidents at nuclear plants also release radioactivity into environment. While no major accident has taken place since the 1979 partial ltdown at Three Mile Island, several smaller-scale events have occurred. For mple, the Pilgrim reactor near Plymouth, Massachusetts, accidentally released stantial amounts of radioactive waste in June 1982.

Bomb testing. Underground atomic bomb testing in Nevada continued through the 1980s before ceasing in September 1992. The average annual number of onations and yield from the explosions were very similar to those in the late 70s and early 1980s (21). However, several of the shots experienced leaks, from which radioactivity escaped into the atmosphere. Two of these malfunctions occurred during the Misty Rain (April 6, 1985) and Mighty Oak (April 10, 1986) tests.

Have American children been exposed to increasing levels of radioactivity in the 1980s and early 1990s? The calculation of precise dose-absorption data is a lengthy, nearly impossible task, especially on a national level. However, a relatively simple method of estimating relative changes in radioactivity is possible by using measurements in the food chain. This information has been collected and publicly issued since 1959 when concerns over levels of atmospheric bomb test fallout in food and water led President Dwight Eisenhower to issue an executive order mandating its collection and reporting; this procedure is the responsibility of the U.S. Environmental Protection Agency (EPA).

The EPA periodically publishes levels of various forms of radioactivity in the air, water, and food. Perhaps the most meaningful of these measurements are monthly readings of radioactivity in pasteurized milk for three radioisotopes—barium-137, cesium-137, and iodine-131—taken from each of approximately 50 to 60 U.S. cities. Unfortunately, when the EPA assumed authority for these measurements from the U.S. Public Health Service in the mid-1970s, the majority of the monthly readings were expressed as negative numbers. Although the EPA insists that such levels essentially represent nondetectable amounts (22), annual national averages well below zero, based on hundreds of readings, cast doubt on this belief. In a large database, the variation from zero should have about the same number of positive as negative numbers. Barium-140 data still continue to produce mostly negative numbers, making trend analysis useless, but by 1983, Cs-137 and I-131 readings were mostly positive. However, the EPA ceased publication of any readings of these three isotopes at the end of 1990, stating that any large-scale effort to post minimal levels was not worthwhile (22).

Despite the variety of obstacles, trends in average Cs-137 and I-131 levels in milk in the (continental) United States from 1983 to 1990 provide an opportunity to examine trends across the United States. The average readings from 50 to 60 cities in most states making up the sample create a fairly good representation of the United States. Half-year averages include over 300 readings, providing a sound basis for the reliability of such an average. Looking at Cs-137 (biological half-life of 30 years) and I-131 (8.05 days) also permits an analysis of trends in a long-lived and a short-lived radioisotope.

Figure 3 shows national trends in radioactivity levels in pasteurized milk for both isotopes from 1983 to 1990, in half-year increments. From 1983 to 1985, levels for both increased; for Cs-137, the average moved from about 1.5 to 2.5 picocuries per liter (pCi/L) of milk; while the upsurge for I-131 was from –0.5 to 2.5 pCi/L. (A picocurie is one-trillionth (10^{-12}) of a curie, a measure of disintegrations in the high-energy radioactive particles.) Fallout from the plume that encircled the globe after the Chernobyl accident was deposited throughout

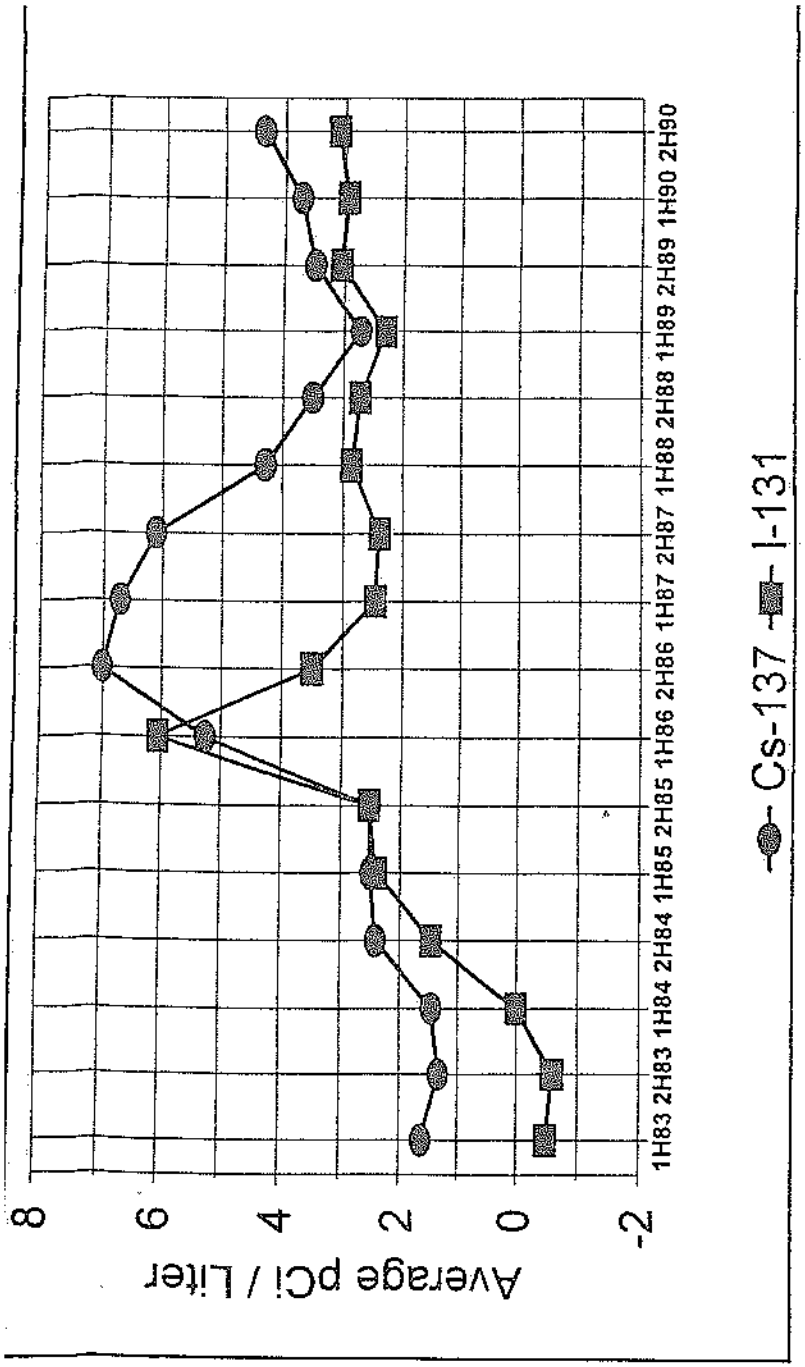

Figure 3. Radioactivity levels of pasteurized milk (cesium-137 and iodine-131), United States, 1983–1990. (Data shown for first half (1H) and second half (2H) of each year.)

the United States via precipitation in May 1986, pushing first-half 1986 average radioactivity readings to 6–7 pCi/L, more than double the 1985 average. The Chernobyl fallout decayed, quickly for I-131 (levels returned to normal by early 1987) and for Cs-137 (normal by early 1989). In 1989 and 1990, however, readings were increasing before the EPA ceased publication of the data in December 1990.

These figures may be relevant to the trends observed in childhood cancer incidence. Rising radioactivity levels in the food chain, the primary means by which low-level radioactivity is ingested by humans, mean greater levels of in vivo accumulation. Iodine-131 kills and impairs cells in the thyroid gland, which may lead to thyroid diseases including cancer, and may retard physical and mental development in fetuses, infants, and children. Cesium-137 accumulates in all soft tissue as well as in bone, which is crucial to the developing immune system. Moreover, the increased presence of Cs-137 and I-131 alone should not be viewed as a comprehensive summary of additional radioactivity absorbed internally by Americans. Radioactivity from fissioning is a cocktail often consisting of dozens of isotopes, and the 1983 to 1990 trend probably points to a similar pattern for other chemicals in the radioactive mixture.

Any additional radioactivity will always affect fetal or infant cells most significantly. The fetal cell, which is developing more rapidly than at any other stage of life, needs just one-eighteenth of the I-131 dose to cause damage equivalent to that in an adult cell, while infants need just one-fifth of the dose (23). A fetus conceived on April 1, 1989, would be exposed to 333 additional pCi of I-131 in utero compared with a fetus conceived on April 1, 1983 (0.5 L/day × 180 days × [3.10 – (–0.60 pCi/L)]). This assumes that the fetal thyroid is not present in the first trimester of pregnancy, and that the mother consumes 0.5 liters of milk each day. The excess picocuries of I-131 in the first year of life (infancy) is 420 (0.5 L/day × 365 days × [3.03 – 0.73 pCi/L)]). These figures can be translated into a total dose of I-131 absorbed by the thyroid gland during the fetal and infant stages. The calculation employs a formula developed during the late 1950s, when scientists became concerned about bomb test fallout's effects on humans (24). The formula for uptake of I-131 by the thyroid is

$$R = (.039 \times L \times C \times U) / M$$

where R = thyroid dose rate in rads per year; L = average daily iodine levels; C = daily consumption of milk, in liters; U = fraction of ingested radioiodine taken up by the thyroid, estimated to be about 30 percent; and M = mass of the thyroid gland, about 0.15 g for fetuses and 1.5 g for infants. Employing the *excess* average of 3.70 pCi/L (that is, 3.10 – (–0.60)) for persons conceived on April 1, 1989, versus April 1, 1983, the formula provides an estimated additional number of rads absorbed by the thyroid gland:

Fetus: (.039 × 3.7 pCi/L × 0.5 L/day × 0.3 × 0.5 yr) / 0.15 g = .07215 rads

Infant: (.039 × 3.7 pCi/L × 0.5 L/day × 0.3 × 1.0 yr) / 1.5 g = .01443 rads

Total additional dose, fetus and infant = .08658 rads

The total additional dose to the fetus of 0.07215 rads is not much less than the approximate annual dose to the body from background (natural) radiation of 0.1 rads. Background radiation is emitted by a variety of radioisotopes, such as carbon-14, which often have long half-lives and decay at a very slow rate, limiting their damage to the human body. Studies show no conclusive proof that areas with elevated natural levels of radiation have a higher rate of cancer (25). However, background radiation is generally distributed throughout the body, unlike a synthetic chemical such as I-131 which concentrates in one particular organ, the thyroid gland. This difference between natural and man-made radiation raises the possibility that isotopes such as I-131, absorbed in doses similar to natural radiation, could have a harmful impact on humans. The cocktail of radioisotopes in radioactive emissions suggests that rates of various cancers may be affected.

Are such levels of radioactivity large enough to have contributed to excess childhood cancers during the 1980s and 1990s? In theory, experts generally agree that there is no safe threshold for exposure to ionizing radiation (26). The concept that low-dose effects are minimal has recently been challenged, especially in radiation-related health effects on newborns and in children. One recent meta-analysis shows that low-level Chernobyl fallout in the United States was linked with increases in infant mortality, perinatal mortality, congenital hypothyroidism, thyroid cancer, and infant leukemia (26). In addition, an increase of 5 percent in low-weight births for both blacks and whites from 1980 to 1993 (27) suggests increasing health problems in fetuses, which may show up in higher cancer rates prior to the age of 10.

It is interesting to note that childhood cancer incidence trends by birth cohort (Figure 2) parallel the trend of cesium and iodine in U.S. milk after 1985. The 1986–87 levels of radioactivity were sharply higher, mostly due to Chernobyl, and cancer rates for children born in these years increased well beyond the standard for those born in 1984–85. Thereafter, radioactivity levels returned to just above their pre-Chernobyl levels; but childhood cancer rates for babies born from 1988 to 1991 remained similar to those for "Chernobyl babies" (born in 1986–87). While no firm conclusions may be drawn from these patterns, a more thorough evaluation of any potential effect may be in order. Clinical studies, such as those comparing radioactivity levels in children with cancer and in those without, appear to be the best means of testing any hypothesis involving a low-dose radiation causal link with increasing rates of childhood cancer.

This report is only a first step in identifying the magnitude and characteristics of the childhood cancer issue. Possibly the next logical step would be to examine trends and potential causes by type of malignancy. About one-third of childhood cancers are leukemias, one-third are brain and central nervous system tumors, and one-third are all other malignancies combined. Since radiation exposure is known to increase the risk of leukemia, it is logical to focus on this condition first. For the 11 states and cities in this report, the leukemia incidence rate for age group 0–9 rose 14.9 percent (4.78 to 5.49 cases per 100,000 persons) from the early 1980s to the early 1990s. The increase is a significant one ($P < .001$), but is well below the 36.6 percent rise for all cancers.

Any environmental factors such as low-level radiation exposure that eventually are shown to contribute to rising childhood cancer rates must first pass a stiff test. The clinical association between cause and disease must demonstrate an exposure to *elevated* levels of a known carcinogen that would cause the childhood cancer rate to abruptly rise after the early 1980s, replacing the stable rates that had existed for at least a decade. This standard may limit the potentially offending substance. For example, pesticides can be carcinogenic, but there would have to be a demonstration that levels of environmental pesticides underwent an unusual rise in the late 1970s and early 1980s. The other potential scenario is that certain carcinogens work synergistically, or in combination with each other, to cause elevated cancer rates in young Americans. The continued search for causes presents a great challenge to medical and scientific professionals.

Acknowledgments — The assistance of Jay Gould, Ph.D., and Ernest Sternglass, Ph.D., in preparing this article is greatly appreciated.

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