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The strontium 90 baby teeth study and childhood cancer

Rapporto fra stronzio 90 nei denti dei bambini e tumori infantili

J.M. Gould, E.J. Sternglass, J.J. Mangano, W. McDonnell, J.D. Sherman and J. Brown
Radiation and Public Health Project, New York, NY, USA

Summary

The Radiation and Public Health Project (RPHP) has collected and measured the amounts of radioactive strontium (^{90}Sr) in baby teeth in several areas in the United States. ^{90}Sr with its long half-life of 28 years is an accurate measure of radiation exposure, not influenced by race, income, occupation, smoking or lifestyle. ^{90}Sr follows the route of calcium and is taken up in the bones of adults, children, and the developing foetus. ^{90}Sr releases beta particles which damage cells in various ways. Documented increased levels of ^{90}Sr correlate with areas of increased childhood cancer including a small area where children have developed an excess of rhabdomyosarcoma. Current levels of ^{90}Sr, probably emitted by nuclear power facilities, compare with historic levels released from nuclear bomb testing. Eur. J. Oncol., 5, suppl. 2, 119-125, 2000

Key words: **Strontium-90, cancer, children, rhabdomyosarcoma**

Riassunto

Il Radiation and Public Health Project ha raccolto e misurato la quantità di stronzio radioattivo (^{90}Sr) nei denti dei bambini in diverse aree degli Stati Uniti. Lo ^{90}Sr, con la sua lunga emivita di 28 anni, è un indicatore attendibile dell'esposizione a radiazioni, non essendo influenzato da razza, reddito, professione, fumo o stile di vita. Lo ^{90}Sr libera particelle beta che danneggiano le cellule in vari modi. Un incremento di ^{90}Sr è stato osservato in aree con aumentata incidenza di tumori infantili, compresa una piccola area in cui i bambini hanno sviluppato un eccesso di radiomiosarcomi. I livelli attuali di ^{90}Sr, verosimilmente emerso da centrali nucleari, sono simili ai livelli storici provocati da test atomici. Eur. J. Oncol., 5, suppl. 2, 119-125, 2000

Parole chiave: **stronzio-90, cancro, bambini, rabdomiosarcoma, radiazioni nucleari**

Introduction

The global epidemic of cancer has not lessened despite improvements in diagnosis and treatment (Bailar III and Gornik, 1997). Cancer is costly to the person, the family, and society. Few resources are allocated for primary prevention. In this context, information gained from determining radioisotope levels in baby teeth may answer significant questions such as: Is there a link between strontium-90 (^{90}Sr) and cancer in adults and children? Which geographic areas have lesser or greater levels of ^{90}Sr? Does a child with elevated ^{90}Sr have medical problems such as cancer, learning disabilities, birth defects and other illnesses? Does the mother of such child have an increased risk for breast cancer or other illnesses? What interactions, chemical and radiologic, are fueling this epidemic?

Indirizzo/Address: Janette D. Sherman, PO Box 4605, Alexandria, VA 22303, USA

The chemical and biological characteristics of ^{90}Sr and its persistence makes it an ideal marker to measure radiation pollution. ^{90}Sr did not exist in nature until the advent of the atomic age. ^{90}Sr and a myriad of radioactive isotopes are produced by the fission of uranium during the detonation of nuclear bombs and emitted during operation of nuclear power plants. These isotopes are carried by the winds and fall to earth with rain and snow where they accumulate and are taken up in food.

Strontium belongs to the alkaline earth series of elements that includes beryllium, magnesium, calcium, barium and radium, and form ionic bonds with non-metallic elements, including oxygen (Faure and Powel, 1972). The stable isotopes of natural strontium (^{88}Sr, ^{87}Sr, ^{86}Sr and ^{84}Sr) make up less than 1% of the earth's rocks. The toxicity of ^{90}Sr was understood before the first atomic bomb was detonated, when Enrico Fermi proposed to use the bone-seeking isotope to poison the food supply of Germany (Rhodes, 1986). With a half-life of 28 years, ^{90}Sr is persistent in the environment and in the bodies of humans. The uptake of radioactive strontium (^{90}Sr) follows that of calcium and becomes deposited in bones and teeth.

The newborn's calcium and ^{90}Sr are derived from the mother's dietary intake and from her bone stores during pregnancy. Additional calcium and ^{90}Sr is contributed by the infant's diet during its year after birth (Reiss, 1961; Rosenthal, 1969). Deciduous teeth, usually shed at ages 5 to 12, represent a tissue similar to bone, with stable mineralization.

Measurements of ^{90}Sr deposited in bones and teeth began after the onset of above-ground nuclear bomb tests in Nevada and were carried out by various governments, including the US (Kulp, Eckelmann and Schubert, 1957; Weiss *et al.*, 1964; Barratta, Ferri and Wall, 1970; Klusek, 1984). An independent, comprehensive study by the Committee for Nuclear Information, measured ^{90}Sr levels in about 60,000 baby teeth collected from children in St. Louis, USA (Reiss, 1961; Commoner, 1972). Comparing 1954 births with those in 1964, ^{90}Sr levels increased in concentration from 0.77 pCi to 11.03 pCi per gram of calcium. The risk to health from this contamination and concern for the health of children worldwide led to a ban on above ground nuclear testing by the US and USSR, signed by Kennedy and Khrushchev.

More recent testing followed Chernobyl releases, when the Otto Hug Institute in Germany documented a ten-fold increase in ^{90}Sr levels in baby teeth for children born in 1987, compared with those born in 1983-85 (Scholz). These elevated levels are comparable to those documented in the St. Louis children at the height of above-ground nuclear bomb testing. In 1990, for unknown reasons, the US EPA programme of reporting levels of barium-140, cesium-137, and iodine-131 in pasteurized milk in sixty cities was discontinued after 33 years of monitoring (US EPA, 1991).

In the Chernobyl fallout area, compared to radiation-free territories with similar social and economic characteristics, ill health has been wide-spread. Children have a marked increase in thyroid dysfunction and thyroid cancer, an increased incidence of mental retardation and birth defects, and immunological impairment (Brulakova, 1996). Infant leukeamia following *in utero* exposure to Chernobyl fallout is reported at 2.6 times that of unexposed infants (Petridou *et al.*, 1996).

The cancer/radiation pattern occurs worldwide, involving exposure to both X-rays (Stewart *et al.*, 1970) and fission products (Sternglass and Gould, 1993). Thyroid cancer in children younger than 15 living near Chernobyl in the Ukraine rose seven-fold after the 1986 accident (Stajakhko, Tsyb and Tronko, 1995) and an increase in leukaemia in Greek, German and American infants rose by 160%, 48%, and 30% respectively for those born 1986-87 when low-level Chernobyl fallout was added to the environment (Petridou, *et al.*, 1997; Mangano, 1997; Michaelis and Kaletsch, 1997). Leukaemia in children younger than 10, living in four Connecticut/Iowa counties rose 20% after start-up of reactors compared to other counties without reactors (Jablon, Hrubec and Boice, 1991) and childhood leukaemia near the Sellafield nuclear processing plant in western England rose significantly after the plant opened (Gardner, *et al.*, 1990).

There was a near-doubling of breast cancer rates in Utah women who lived in the fall-out path from the Nevada bomb test site (Johnson, 1984) and increases in breast cancer in areas downwind from multiple US nuclear reactors (Gould, 1996). There was an increase in leukaemia, lung cancer and all malignancies in those living in the vicinity of the Three Mile Island nuclear reactor (Wing, *et al.*, 1997) and increased cancer incidence downwind from the US nuclear test site in Nevada (Johnson, 1984).

In Europe, there was a 40% increase in the age standardized inc dence of breast cancer in England and Wales for the period 197 1992 (Quinn and Allen, 1995), an increase in testicular canc in young men and leukaemia in children, and an 80 year-lon increase in mortality from breast cancer in Scotland (Swerdlow *al.*, 1998). Elsewhere in Europe, there was a 10% to 30% overa increase in cancer incidence for both sexes and various ag groups for the period 1974-1993 in the Swiss canton of Vau (Levi, Randimbison and La Vecchia 1996) and in France, a increase in large bowel and lung cancer in women, with breas cancer accounting for one third of all newly diagnosed cancer (Menegoz *et al.*, 1997). Curiously, the last citation, despite bein published in a journal with prevention in its name, states: "Th aims of the European Network of Cancer Registries (ENCR) ar to improve the quality, comparability and availability of cance registry data in Europe". Specific exposure data such as ^{90}Sr i the teeth of children are needed to buttress statistical data with *i vivo* exposures, and to overcome the lack of emphasis on prevention.

Methods

The Radiation and Public Health Project (RPHP) initiated the collection of baby teeth to assay for ^{90}Sr levels in 1996 (Brown *et al.*, 1999). The purpose of this study is to document present levels and historic trends of ^{90}Sr in children covering the time since the end of above-ground bomb tests. This will allow comparisons of historic ^{90}Sr levels from 35-50 years ago when most ^{90}Sr emanated from bomb fallout, with current ^{90}Sr levels, released primarily from nuclear power reactors.

Baby teeth are solicited and received from the entire US to provide various geographic areas for ^{90}Sr comparisons. Persons wishing information or to contribute teeth may contact RPHP via a toll-free telephone number (1-800-582-3716), and on the World Wide Web (www.radiation.org.).

The initial geographic study focused on Suffolk County, located at the eastern end of Long Island, New York, a 922 square mile area with a 1990 population of 1,321,864. This is an area with a documented high rate of breast cancer (Kulldorff *et al.*, 1997), which prompted the US government to commission the Long Island Breast Cancer Study Project (US Congress, 1993). For unknown reasons, assessment of radioactive materials is not a part of the study, despite the area being downwind from multiple nuclear power reactors and the home of Brookhaven National Laboratory, a major nuclear research facility. The latter is documented to have leaked radioactive materials into the air and water (New York State Environmental Radiation Monitoring Program, 1996; IT Corp 1998).

Families living in areas with reported increases in childhood cancer having potential for exposure from nuclear reactors were initially contacted. The areas included eastern Suffolk county on Long Island, mid-coastal New Jersey, and greater Miami, Florida. Notification was made via mass mailings to 15,000 randomly-selected households with children aged 6 to 12. A population with potentially less exposure included parts of Queens, New York, and northwestern New Jersey.

Each donor receives information describing the study. An envelope in which the donor can send teeth to RPHP is enclosed. Donors are assured of the confidentiality of all information

identifying mothers and children. The following information is requested:

- mother's name
- phone number
- address
- child's name
- child's birth date (month, day, year)
- birth weight (pounds, ounces)
- location where mother carried the baby (city, state, county, zip code)
- location where child was born (city, state, county, zip code)
- location during first, second and third years of life (city, state, county, zip code)
- water source (well, municipal, bottled water, other)
- mother's age at birth of child
- age of child when tooth was lost/or date of tooth loss.

Upon receipt of envelopes containing teeth, RPHP staff assigns a unique control number to each tooth and logs the information into a computerized data base. The teeth are coded and sent in batches to a radiochemistry laboratory in Waterloo, Ontario, Canada where personnel assay each tooth for ^{90}Sr activity in picocuries and calcium in weight (pCi/g Ca). Waterloo researchers are blinded as to any information concerning each tooth.

^{90}Sr readings for each tooth at the time it was shed by the child are converted to levels at birth, creating a standardized measure that can be compared regardless of the age of the individual tooth.

Results

As of 1 October 1999, approximately 1500 teeth have been received, and a total of 515 teeth have been analyzed for ^{90}Sr concentration. Of this number, 476 teeth were from children born during the years 1979-1994. Because of the 6+ year delay in children shedding teeth, only 15 teeth have been analyzed for the period beginning in 1993, an insufficient number to provide statistical reliability for these last years.

Table 1 shows the following: geographic distribution of the teeth, based on where the mother lived when pregnant with the child; the five areas that account for 422 of the teeth; and the average and maximum concentrations of ^{90}Sr.

The mean concentration for all 476 children's teeth is 1.50 pCi ^{90}Sr/g calcium, roughly equivalent to the amount in the children from St. Louis born in 1956, five years after atmospheric bomb testing commenced in Nevada.

More than 20% (106 teeth) had levels of 2.0 pCi or higher, which is 10 to 20 times greater than the projected values of 0.1 to 0.2 pCi/g Ca. Twenty six percent of the teeth measured the expected 0.2 pCi ^{90}Sr/g Ca, and given a standard error per tooth of ± 0.7, about one-third were below a value of 0.5. The largest single value was 17.87 pCi ^{90}Sr90/g Ca.

Table 1 - Baby teeth analyzed from children born 1979-1994 by geographic area (as of 1 October 1999)

Area	No. teeth	Average pCi ^{90}Sr/g Ca
Western Suffolk County, NY (ZIP 117)	185	1.56 ± 0.05
Eastern Suffolk County, NY (ZIP 119)	119	1.02 ± 0.06
New York City, NY	21	1.68 ± 0.15
Florida (mainly Dade County)	39	3.01 ± 0.10
New Jersey (mainly Ocean County)	55	1.64 ± 0.11
All other US areas	54	1.06 ± 0.10
All locations	476	1.50 ± 0.03

While the ^{90}Sr levels in teeth from Dade County are highest at 3.01 pCi/g Ca, the average for the entire period 1974-94 for all five areas closest to a local nuclear reactor is 1.55 ± 0.03, which greatly exceeds 0.1 to 0.2 pCi ^{90}Sr/g Ca the level that would be expected if post-Test Ban Treaty (1964-1970) declines observed in St. Louis teeth had continued (fig. 1).

Annual averages of ^{90}Sr levels in both baby teeth and adult diets reached peak levels in certain years for which there is an explanation: the peak reached in 1964 for both baby teeth and adult diet was the year following the termination of above-ground bomb tests. Fig. 2 shows that in 1964, the ^{90}Sr in baby teeth averaged 11 pCi/g Ca, or approximately one-third of the adult diet of level of 30 pCi/g Ca. In most years the level in baby teeth averaged between 20 and 50% of the adult level. From 1964-1970 both indicators declined each year at the rate on average of 15.7% in the absence of ^{90}Sr emissions from US/USSR above ground tests. Projecting this rate of decline after 1970, it is evident that by 1982 both indicators should be less than 1.0 pCi/g Ca.

Suffolk County children account for a sufficient number of teeth (304) to allow for analysis of their annual ^{90}Sr levels and the epidemic rise of childhood cancer in that county since 1984 (fig. 3). For the years 1983–1985 the Suffolk County cancer incidence rate for 41 children aged 0 to 4 years was 15.34 cases per 100,000

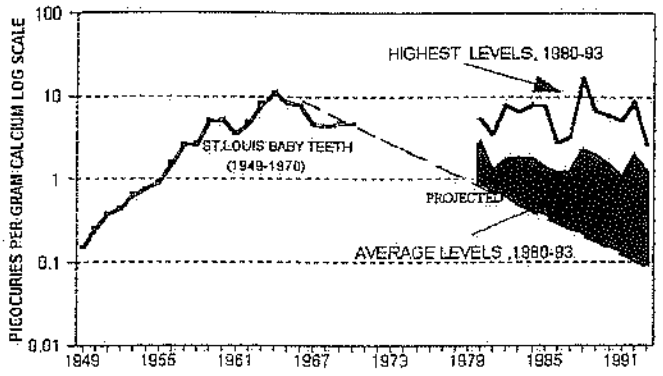

Fig. 1 - ^{90}Sr in St. Louis baby teeth, 1949-1970 and observed in 273 children, 1980-93).

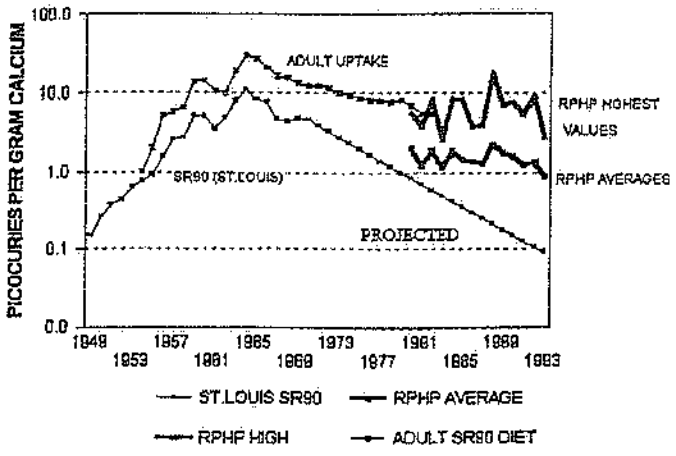

Fig. 2 - ^{90}Sr in baby teeth and adult diet trend. 367 RPHP baby teeth, 1980-93.

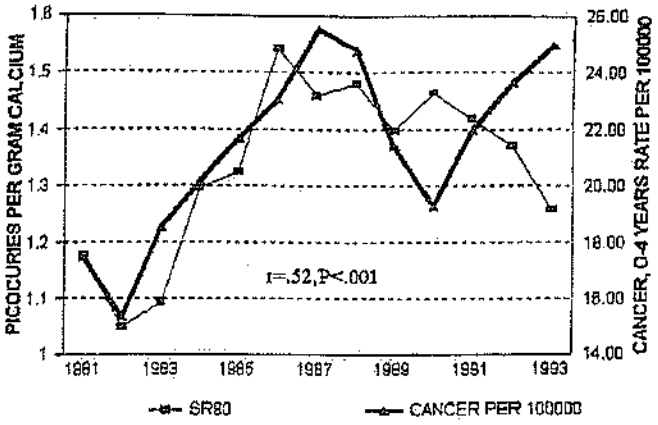

Fig. 3 - ^{90}Sr in Suffolk children, 1981-93 and Suffolk childhood cancer 0-4 years.

and rose 63% to 24.97 per 100,000 for 76 children diagnosed with cancer for the years 1994-96. With relatively small numbers, a three year moving average is used to diminish annual chance variation and preserve turning points. This is employed here because the average cancer rate in 1984 rose to an initial peak of 25.52 case per 100,000 in the years 1988-90 and then declined thereafter by 24% to 19.29 cases per 100,000 for children aged 0 to 4 diagnosed in 1991-93.

Assuming that it takes an average of three years for a child exposed *in utero* to carcinogens to develop a detectable cancer, we see that the Suffolk County ^{90}Sr baby teeth values exhibit a similar rise and fall but with a three year lag. Thus for 13 teeth from children born in the years 1981-83, centered in the year 1981 (three years before 1984), the average ^{90}Sr level was 1.01 pCi/g Ca, which rose by 50% to an average level of 1.50 for 70 children born in the years 1985-87 and then declined thereafter. This rise and fall closely matches the rise and fall in childhood cancer rates with a three year lag.

Fig. 3 displays a significant correlation (0.71, $p < 0.001$) between ^{90}Sr levels and childhood cancer rates since 1981 with the assumed three year lag between both indicators. The childhood cancer rate in Suffolk County rose again in the 1990s. ^{90}Sr levels are not yet available because children born in 1993 will not shed their teeth until the turn of the century.

Our data suggest that if RPHP had hundreds of teeth measured for each year, as was true of the St. Louis study, a three year moving average would not be needed to reduce statistical variation. A three year delay between ^{90}Sr measurements at birth and cancer incidence 0 to 4 years in Suffolk County gives the best statistical fit. This is in agreement with the observation that the major share of childhood malignancies (leukaemia and brain cancer) are most frequently diagnosed between the second and fifth years of life. Thus build up in the pregnant mother of ^{90}Sr and other short- and long-lived radioactive emissions for the period 1981-93 are matched with cancer incidence for 1984-96.

By contrast to nuclear bomb tests, which prolong the release of radioactive elements by dispersion into the stratosphere, emissions from nuclear power reactors are dispersed at low atmospheric levels and are brought down by rain and snow in a matter of days to weeks; thus, ^{90}Sr enters the water and food chain rapidly. ^{90}Sr is accompanied by isotopes such as argon, xenon, krypton, cesium, barium and iodine that have short-half lives and subsequent radioactive decay products that exposes the embryo and foetus to significant risk. ^{90}Sr's stability and long half-life is a reliable measure of contamination, and a proxy for other radioactive fission products from bomb fallout and power reactor emissions.

From 1964 to 1970, following the ban on above-ground bomb testing by the US and USSR, ^{90}Sr levels in baby teeth fell about 58%. Average pCi levels of ^{90}Sr per gram of calcium in Saint Louis baby teeth by year were: 1964 - 11.03; 1965 - 8.42; 1966 - 7.82; 1967 - 4.68; 1968 - 4.40; 1969 - 4.80; and 1970 - 4.60. However levels ceased declining from 1967 to 1970 when the baby tooth study was halted, suggesting other new sources were adding ^{90}Sr to the environment.

Radioisotopes accumulate in various parts of the body and produce damage as they decay by release of alpha, beta or gamma energy. An isotopes' path through the body and its site of accumulation is dependent upon the chemical family to which it belongs. Beta particles are quickly slowed by collisions with tissue elements and their kinetic energy converted to thermal energy (Faure and Powell, 1972). It is this energy, released during nuclear decay, that causes disruption of cellular membranes, alteration of DNA, changes in enzymes, and other adverse effects. The foetus is especially vulnerable to harm from exposure to toxic chemicals or radioactive isotopes. Throughout intrauterine life, the developing foetus undergoes rapid cell growth, self-programmed cell death (apoptosis), and cell rearrangement, all time- and space-dependent. The developing infant is particularly susceptible to cellular and metabolic damage. Unrepaired damage becomes magnified with time, increasing the risk of cancer, congenital malformations, impaired immunological and intellectual function, birth of underweight and premature babies, as well as foetal and infant deaths (Sherman, 1999)

At ten weeks of development when the foetus is a little over 4 cm (1,5 inches) in length, the enamel organs and dental papillae form. Bone formation begins two week earlier, when the foetus is less than an inch in length (Arey, 1954). Stem cells of haematopoietic system originate in the bone marrow beginning at about 12 weeks of prenatal development (Fitzgerald and Fitzgerald, 1994) giving rise to the B-lymphocytes whose progeny make humeral antibodies, and the T-lymphocytes involved in cellular immune responses (Carson, 1994).

All forms of cancer can be induced by radiation; the incidence increases with cumulative dose; and younger aged individuals, human and animal alike, are more sensitive to ionizing radiation than adults (Gofman and Tamplin, 1970; Gofman, 1981; Upton, 1985; Gofman, 1990).

Mesenchymal malignancies in children are of particular concern because they are aggressive and result in death of approximately 50% of those affected. Bone, teeth, blood cells, lymphoid tissue, fat cells, muscles, and fibrous tissue all originate from the mesenchymal layer during foetal development (Arey, 1954). Leukaemia and sarcomas are malignancies derived from mesenchymal cells (Gofman, 1981) the latter accounting for five percent of paediatric cancers (Rudolph, 1996). Rhabdomyosarcoma (RMS) is a rare form of sarcoma, derived from skeletal muscle cells (Helman, 1999) with an annual incidence of 4.5 per million children 15 years of age or younger (Dagher and Helman, 1999). An animal model with characteristics similar to nevoid basal cell carcinoma (Gorlin syndrome) and rhabdomyosarcoma found in humans can be induced in radiation-sensitive mice which subsequently develop both teratogenic and malignant changes (Hahn *et al.*, 1998). Rhabdomyosarcoma has been induced by exposure to

^{90}Sr beta radiation applied to the skin of test animals. Out of 50 mice, 27 developed skin and subcutaneous tumours, including 12 sarcomas. Nine of 12 tumours had rhabdoid histology and expressed biochemical characteristics establishing the diagnosis of rhabdomyosarcoma (Gupta *et al.*, 1999).
In a small area of Suffolk County, on New York's Long Island, at least 19 children have developed rhabdomyosarcoma (Table 2).

Table 2 - Rhabdomyosarcoma diagnosed in children in Suffolk County - Long Island

ID code of child	Year of diagnosis	Age at diagnosis	Postal zip code	Deceased
1	1994	16	11804	Yes
2	1996	3	11767 (a)	
3	1996	4	11780 (a)	
4	1994	4	11790 (a)	
5	1982	12	11741	Yes
6	1995	16	11968	Yes
7	Unknown	Unknown	11727	
8	1994	23	11752	Yes
9	1994	6	11767 (a)	
10	1994	10	11784	Yes
11	1996	7	11951	
12	1990	16	11784	
13	1997	1	11050	
14	1995	3	11949	
15	1996	6	11733 (a)	Yes
16	1997	13	11763	
17	Unknown	Unknown	11961/11727	
18	1998	15	11787	
19	1999	6	11780 (a)	

(a) Children from 8 postal zip codes near Brookhaven National Laboratory

The expected US rate of rhabdomyosarcoma in children ages 0 to 14 years is 4.5 per million, and for children 0 to 10 years the rate 3 per million. Seven children younger than 10 years diagnosed with rhabdomyosarcoma, from 1994 to 1999, lived in an area of 8 postal zip codes located 10 miles west-north-west of Brookhaven National Laboratory.
According to the 1990 census the total population of this area was 119,150. Children ages 0 to 10 years made up approximately 15% of that population, or 17,873 individuals, thus the expected rate of rhabdomyosarcoma was 3/1,000,000 X 17.873 X 6 years, or 0.32 cases. The actual annual rate is 65.3 cases per million, indicating a significant increase rhabdomyosarcoma in this area: 22 times greater than expected.

Discussion

In addition to ^{90}Sr and accompanying nuclear releases, other factors adversely impacting children, adults and the unborn are chemicals released from various industrial processes, pesticides, and emissions from incinerators and dumps. Harm from contaminants and radionuclides are each dependent upon the environmental half-life, as well as the path of intake, deposition within and excretion from the body. Repeated small exposures to radionuclides and to many industrial and agricultural chemicals produce additive and synergistic effects, resulting in greater toxicity than a single equivalent dose. Polychlorinated biphenyls (PCBs), dioxins and pesticides such as DDT, chlordane, heptachlor, lindane, chlorpyrifos, dibromochloropropane (DBCP), etc, are lipid-soluble. These chemicals have environmental and biological half-lives varying form years to decades, and accumulate within the fatty parts of the body for extended periods of time, producing changes in enzyme systems, immunological and neurological impairment, alterations of hormonal function, and irreversible damage to cells, resulting in cancer, and birth defects (Sherman, 1994).
Geographical deposition of radioisotopes is not uniform, whether it is derived from bomb testing, nuclear power plants, or catastrophes, such as Chernobyl and Three Mile Island. Various measurements of radioactivity in rain, soil, air, and in food have confirmed the uneven distribution of ^{90}Sr fallout on the ground (US Congress, 1959). Measurements of ^{90}Sr, ^{137}Cs, and plutonium fallout from the Chernobyl reactor demonstrate a gradient of deposition related to distance from the source, and depending upon wind, rain and geography (Ilyin, 1989).
The ^{90}Sr deposited in bones and teeth releases beta radiation as it decays to radio-yttrium (^{90}Y). The rate of radioactive decay is proportional to the number of parent atoms present (Faure and Powell, 1972) thus there is an increase in ^{90}Y as ^{90}Sr decays. ^{90}Y is also unstable and decays, releasing beta radiation as it becomes dispersed in the soft tissues of the body, including the breast and the pituitary gland, the latter governing hormone function throughout the body. The commonly held notion that reactions to chemicals and ionizing radiation follow a linear dose-response curve is not supported by fact. While a reaction may be proportional at high doses that impair or kill, a straight-line dose-response is not borne out at low-dose exposures (Staffa and Mehlman, 1979) nor when an insult occurs at a critical period of foetal development. Exposure to such chemicals and radionuclides below the level that kills a cell is critical: such sub-lethal exposures can alter cellular function or structure and if not repaired become expressed as cancer or functional alteration. The DES-daughters and sons are prime examples. Diethylstilbestrol (DES) was administered to pregnant women in the misguided idea that it would protect against foetal loss during pregnancy. Many children and grandchildren were born with anatomic and functional genital abnormalities and developed genital cancers when they reached adulthood (Sherman, 1994b).
Furthermore, the term "low-level" is misleading when the exposure is to radionuclides and chemicals with long half-lives. ^{90}Sr and other nuclides with long half-lives accumulate in the environment and in the body. While a 0.01 of a pCi is dismissed as an insignificant amount, a daily intake of this amount of isotope over a period of a year will accumulate 3.65 pCi, minus of course, that small amount that decays to Y90. When one considers intake from milk alone, and omits that in water, vegetables, meat and other food and drink, the EPA-measured intake is 1.5 pCi of ^{90}Sr-per day for a total of 544 pCi per year. This is based on an average of 1.49 pCi/litre of pasteurized milk measured in 45 cities in the continental US, with the highest reading at 3.5 pCi (National Air and Radiation Environmental Laboratory, 1993). Very low levels of radiation exposure have demonstrated an enhanced, supra-linear effect due to the release of free radicals, resulting in functional and physiological effects, not necessarily due to genetic or mutational alteration (Petkau, 1968; Sternglass, 1974).
Beta radiation is a high speed electron, a physical particle, released from the nucleus of an unstable atom undergoing decay. As the beta particle travels through tissue, it releases its energy

into the cells, causing damage. Beta particles can penetrate approximately the distance across 3 to 4 cells, altering or killing the cells as the particle passes through. Such energy has the potential to disrupt those portions of multicellular organisms that maintain tissue organization and cellular communication, the parenchyma or stroma of an organ. Radiation-induced injury is compatible with the thesis of Sonnenschein and Soto that "units of tissue maintenance and/or organization are present in all organs and are the ultimate targets of carcinogenic agents." They posit "that teratogenesis, developmental tumors, and 'spontaneous' and induced carcinogenesis, occur because of miscommunication among cells and tissues" (Sonnenschein and Soto, 1999). What more effective way to disrupt cellular communication than that of nuclear radiation!

The epidemic increase in childhood and adult cancer has occurred since World War II when both chemical and radiological pollution spread over the world. There is no longer any doubt that radioisotopes, in concert with industrial chemicals, have caused this epidemic (Sherman, 1999b). The RPHP has demonstrated increased ^{90}Sr levels in areas with increased childhood cancer. If the personal, social, moral, and economic costs of the world-wide epidemic of cancer are to be ended, we must invoke the precautionary principle and act on the considerable information we have already at hand. Prevention is imperative economically and ethically.

> "In this now universal contamination of the environment, chemicals are the sinister and little recognized partners of radiation in changing the very nature of the world - the very nature of life.
>
> Rachel Carson, *Silent Spring*, 1962.

References

Arey L.B.: Developmental anatomy, 6th Edition, 107, 344-345. W.B. Saunders, Philadelphia, 1954.

Ballar J.C. III, and Gornick H.L.: Cancer undefeated. N. Engl. J. Med., **336** (22), 1569-1574, 1997.

Barratta E.J., Ferri E.S., and Wall M.A.: Strontium-90 in human bones in the United States, 1962-1966. Radiol. Health Data and Reports, 183-186, April 1970.

Brown J. Gould J.M., Mangano J.J., *et al.*: Strontium-90 in baby teeth as a factor in early childhood leukemia and cancer. Int. J. Serv., 1999, in press.

Burlakova E.B.: Consequences of the Chernobyl catastrophe: human health. Centre for Russian Environmental Policy, Scientific Council on Radiology, Russian Academy of Sciences, Moscow, 1996.

Carson B.M.: Human embryology and developmental biology, 300. Mosby, St. Louis, 1994.

Commoner B.: The closing circle - nature, man and technology, 51-57, 188-200. Alfred A. Knopf, New York, 1972.

Dagher R., and Helman L.: Rhabdomyosarcoma: an overview. Oncologist, **4**, 34-44, 1999.

Faure G., and Powell J.L.: Strontium isotope geology, 1, 4, 5, 10. Springer-Verlag, New York, 1972.

Fitzgerald M.J.T., and Fitzgerald M.: Human embryology, 41. Bailliere Tindall, Philadelphia, 1994.

Gardner M.J., Snee M.J., Hall A.J., *et al.*: Results of a case control study of leukaemia and lymphoma among young people near Sellafield nuclear processing plant in West Cumbria. Br. Med. J., **300**, 423-429, 1990.

Gofman J.W.: Radiation and human health: a comprehensive investigation of the evidence relating low level radiation to cancer and other diseases. Sierra Club Books, San Francisco, 1981.

Gofman J.W.: Radiation-induced cancer from low-dose exposure: an independent analysis. CNR Books, San Francisco, 1990.

Gofman J.W., and Tamplin A.R.: Low doses radiation and cancer. IEEE Transactions on Nuclear Science, part 1, vol. NS-17, 1-9, 1970.

Gould J.M.: The enemy within: the high cost of living near nuclear reactors, 346. Four Walls Eight Windows, New York, and London, 1996.

Gupta A., Andrews K.L., McDaniel K.M., *et al.*: Experimental induction of rhabdomyosarcoma in mice with fractionated doses of B-radiation. J. Cancer Res. Clin. Oncol., **125**, 257-267, 1999.

Hahn H., Wojnowski L., Zimmer A.M., *et al.*: Rhabdomyosarcomas and radiation hypersensitivity in a mouse model with Gorlin syndrome. Nat. Med., **4** (5), 619-622, 1998.

Helman D.R.: Rhabdomyosarcoma: an overview. Oncologist, **4**, (1), 34-44, 1999.

Ilyin L.A.: Radio-contamination patterns and possible health consequences of the accident at the Chernobyl nuclear power station. J. Radiol. Protect., **10**, 3-29, 1989.

IT Corp: Fish Tissue Bioaccumulation Study Report: Brookhaven National Laboratory. December 1996 and May 1998.

Jablon S., Hrubec Z., and Boice J.D.: Cancer in populations living near nuclear facilities. JAMA, **265**, 1403-1408, 1991.

Johnson C.J.: Cancer incidence in an area of radioactive fallout downwind from the Nevada test site. JAMA, **251** (2), 230-236, 1984.

Klausek C.S.: Strontium-90 in human bones in the US-1982. EML-435, US Department of Energy, New York, 1984.

Kulldorff M., Feuer E.J., Miller B.A., *et al.*: Breast cancer clusters in the northeast United States: a geographical analysis. Am. J. Epidemiol., **146** (2), 161-170, 1997.

Kulp J.L., Eckelmann J., and Schulert A.R.: Strontium-90 in man. Science, **125**, 219-224, 1957.

Levi F., Randimbison L., and La Vecchia C.: Trends in cancer incidence and mortality in Vaud, Switzerland, 1974-1993. Ann. Oncol., **7** (5), 407-504, 1996.

Mangano J.J.: Childhood leukemia in US may have risen due to fallout from Chernobyl. Br. Med. J., **314**, 1200-, 1997.

Menegoz F., Black R.J., Arveux P., *et al.*: Cancer incidence and mortality in France, 1975-95. Eur. J. Cancer Prev., **6** (5), 442-466, 1997.

Michaelis J., and Kaletsch U.: Infant leukemia after the Chernobyl accident. Nature, **387**, 246-, 1997.

National Air and Radiation Environmental Laboratory: Environmental radiation data. Report 71. Montgomery, AL. US Environmental Protection Agency. 1993.

New York State Environmental Radiation Monitoring Program. Radioactive contamination in the Peconic River. NY State Department of Health, Septemebr 12, 1996.

Petkau A.: Effect of 22Na+ on phospholipid membrane injury to irradiated erythrocytes. Radiat. Res., **34**, 335-346, 1968.

Petridou E., Trichopoulos D., Dessyprist N., *et al.*: Infant leukaemia after *in utero* exposure to radiation from Chernobyl. Nature, **382**, 253, 1996.

Quinn M., and Allen E.: Changes in incidence of and mortality from breast cancer in England and Wales since introduction of screening. Br. Med. J., **311** (7017), 1391-1395, 1955.

Reiss L.Z.: Strontium-90 absorption by deciduous teeth. Science, **134**, 1669-1673, 1961.

Rhodes R.: The marking of the atomic bomb, 510-511, Simon and Schuster, New York, 1986.

Rosenthal H.L.: Accumulation of environmental 90-Sr in teeth of children. Proceedings of the Ninth Annual Hanford Biology Symposium, 163-171. Richland, WA, 1969.

Rudolph A.M.: Rudolph's Pediatrics, 1295. Appleton and Lange, Stamford, CT, 1996.

Scholz R.: Ten years after Chernobyl: the rise of strontium-90 in baby teeth. The Otto Hug Radiation Institute, Munich, Germany (available in English translation).

Sherman J.S.: Chemical exposure and disease. Pricenton Scientific Publishing Co., Priceton, NJ. 1994a.

Sherman J.D.: Structure-activity relationships of chemicals causing endocrine, reproductive, neurotoxic and oncogenic effects: a public health problem. Toxicol. Ind. Health, **10** (3), 163-179, 1994b.

Sherman J.D.: Birth defects and *in utero* exposure to Dursban-Report of 15 incidents, detailed evaluation of 8 cases and discussion of theory of action. Eur. J. Oncol., 4 (5), 1999a.

Sherman J.D.: Life's delicate balance. Causes and prevention of breast cancer. Taylor and Francis, New York, 1999b.

Sonnenschein C., and Soto A.: The society of cells, 103. Springer-Verlag, New York, 1999.

Staffa J.A., and Mehlman Mehlman M.A.: Innovations in cancer risk assessment (ED-01 Study). National Center for Toxicological Research, US Food and Drug Administration. Pathotox Publishers Inc., Park Forest South, IL, 1979.

Stajakhoko V.A., Tsyb A.F., and Tronko N.D.: Childhood thyroid cancer since accident at Chernobyl. Br. Med. J., **310**, 801, 1995.

Sternglass E.J.: Implication of dose-rate dependent cell-membrane damage for the biological effects of medical and environmental radiation. Proceedings Symposium on Population Exposures, Knoxville, TN, Oct. 21-23, 1974.

Sternglass E.J., and Gould J.M.: Breast cancer: evidence for relation to fission products in the diet. Int. J. Health Serv., **23**, 7883-7804, 1993.

Stewart A., *et al.*: Radiation dose effects in relation to obsetric x-ray and childhood cancers. Lancet, 7658, 1970.

Swerdlow A.J., dos Santos Silva I., Reid A., *et al.*: Trends in cancer incidence and mortality in Scotland: description and possible explanations. Br. J. Cancer, **77** (Suppl. 3), 1-54, 1998.

Upton A.C.: Biological basis for assessing carcinogenic risks of low-level radiation. Carcinogenesis, **10**, 381-401, 1985.

US Congress, Joint Committee on Atomic Energy. Summary Analysis of Hearings, US Government Printing Office, Washington, DC, page 12, May 5-8, 1959.

US Congress, Public Law 103-43, Section 1911, June 10, 1993.

US Environmental Protection Agency. Environmental Radiation Data, Montgomery, AL, 1991.

Weiss E.S., Land W.H., Falter K.H., *et al.*: Strontum-90 content of human bones, 1961-1963. Radiol. Health Data, 231-239, 1964.

Wing S., Richardson D., Armostrong D., *et al.*: A reevaluation of cancer incidence near the Three Mile Island nuclear plant: the collision of evidence and assumptions. Environ. Health Perspect., **105** (1), 52-57, 1997.