Childhood Cancer: Overview of Incidence Trends and Environmental Carcinogens

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An estimated 8000 children 0 to 14 years of age were diagnosed with cancer in 1993 in the United States (1). According to population-based data from the National Cancer Institute’s Surveillance, Epidemiology and End Results program, which covers about 10% of the U.S. population (1), leukemia and brain tumors (gliomas and meningiomas) are the most common childhood malignancies, accounting for 30 and 20% of newly diagnosed cases, respectively. From 1975 to 1978 to 1987 to 1990, cancer among white children increased slightly from 12.8 to 14.1/100,000. Increases are suggested for leukemia, gliomas, and, to a much lesser extent, Wilms’ tumor. There are a few well-established environmental causes of childhood cancer such as radiation, chemotherapeutic agents, and diethylstilbestrol. Many other agents such as electromagnetic fields, pesticides, and some parental occupational exposures are suspected of playing roles, but the evidence is not conclusive at this time. Some childhood exposures such as secondhand cigarette smoke may contribute to cancers that develop many years after childhood. For some exposures such as radiation and pesticides data suggest that children may be more susceptible to the carcinogenic effects than similarly exposed adults. — Environ Health Perspect 103(Suppl 6):177–184 (1995)

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Introduction

An estimated 8000 children 0 to 14 years of age were diagnosed with cancer in 1993 in the United States (1). According to population-based data from the National Cancer Institute’s Surveillance, Epidemiology and End Results program, which covers about 10% of the U.S. population (1), leukemia and brain tumors (gliomas and meningiomas) are the most common childhood malignancies, accounting for 30 and 20% of newly diagnosed cases, respectively (Table 1). Almost 80% of childhood leukemia cases are acute lymphocytic leukemia (ALL). For most childhood malignancies, incidence is highest between 0 and 4 years of age. Incidence rates for non-Hodgkin’s lymphoma (NHL), Hodgkin’s disease, osteosarcoma, and Ewing’s sarcoma, however, increase with age. Boys are more likely than girls to be diagnosed with cancer, primarily due to the excess among males of ALL, NHL, soft tissue sarcoma, and Hodgkin’s disease (Table 2). Wilms’ tumor, on the other hand, is slightly more common among girls than boys. Racial differences in cancer incidence are also evident among children. For all cancers combined, whites have a 23% higher incidence (13.5/100,000) than blacks (11.0/100,000). Leukemia and, to a lesser extent, gliomas and meningiomas, neuroblastoma, NHL, and Hodgkin’s disease are more common among whites than blacks, whereas Wilms’ tumor is slightly more common among black children. Ewing’s sarcoma, rare among whites, is virtually nonexistent among blacks. From 1975 to 1978 to 1987 to 1990, cancer among white children increased slightly, from 12.8 to 14.1/100,000.

Table 1. Estimated number of incident cases annually in the United States for selected cancers among children (0–14 years of age) based on data from the SEER program (1975–1990) and the American Cancer Society.†

| Cancer                  | Estimated annual U.S. cases |
|-------------------------|----------------------------|
| Leukemia                | 2370                       |
| Acute lymphocytic       | 1850                       |
| Acute myeloid           | 230                        |
| Other leukemia          | 290                        |
| Glioma and meningioma   | 1590                       |
| Neuroblastoma           | 660                        |
| Wilms’ tumor            | 520                        |
| Soft tissue sarcoma     | 460                        |
| Non-Hodgkin’s lymphoma  | 500                        |
| Hodgkin’s disease       | 400                        |
| Retinoblastoma          | 230                        |
| Osteosarcoma            | 190                        |
| Ewing’s sarcoma         | 150                        |
| Other                   | 930                        |
| Total                   | 8000                       |

†American Cancer Society, Cancer Facts and Figures—1993.

Increases are suggested for leukemia, particularly ALL, gliomas, and, to a much lesser extent, Wilms’ tumor (Figure 1). At least part of the increase in ALL likely is due to increasing cell-type specificity; total leukemia incidence rose less rapidly. Incidence rates for brain and nervous system cancers among adults have also risen, particularly among the elderly; improved diagnostic technology and application has played a role in this trend. Data are inadequate to evaluate incidence trends among black children. Cancer mortality among children has decreased over time due largely to dramatic improvements in treatment, particularly for acute leukemia and Hodgkin’s disease. Cancer remains, however, the most common cause of death, after accidents, among children, accounting for approximately 1500 deaths annually.

Environmental Carcinogens

Environmental exposures that contribute to cancer etiology among children include most of the same exposures known to cause cancer in adults, such as radiation, certain medications, and some industrial and agricultural chemicals (2) (Table 3). Some childhood exposures such as secondhand cigarette smoke may contribute to cancers that develop many years after childhood. There are also factors suspected of playing a role in childhood cancer but for which the evidence to date is inconsistent or speculative; for example, electromagnetic fields (EMF). For some exposures, such as radiation and pesticides, data suggest that children may be more susceptible to the...
carcinogenic effects than similarly exposed adults. There are also suggestions of possible interactions between environmental carcinogens and genetic susceptibility.

Radiation

The most well-established cause of childhood cancer is radiation. High-dose radiation exposure, such as that experienced by atomic bomb survivors and children receiving radiation therapy for cancer, enlarged thymus, tinea capitis (ringworm of the scalp), and other conditions, has caused increases in acute leukemia, chronic myelogenous leukemia, osteosarcoma, thyroid cancer, breast cancer, and soft tissue sarcoma (2–4). The effects of lower dose radiation exposure are more controversial, however. Many studies have shown a small increase of leukemia after low-dose prenatal irradiation (5–8). The apparent association may not be causal, however, but may reflect selection factors related to the medical reasons for the prenatal X-rays (9).

One study that strongly supports a causal relation investigated childhood cancer among twins, who are often X-rayed to verify twinship or to determine fetal position rather than for medical conditions that might independently be associated with cancer (10). Twins who were X-rayed prenatally were found to have twice the risk of leukemia compared to twins who were not X-rayed. In contrast, atomic bomb survivors exposed in utero have not shown excess cancer (11,12). Fallout from nuclear weapons tests has unequivocally been linked to thyroid cancer in children exposed to high doses in the Marshall Islands (2). Lower exposure to fallout in Utah has shown no association with thyroid cancer (13). An apparent association between fallout and childhood leukemia found in some studies (14) but not in others (15) has been challenged based on a lower than expected cancer rate in the low-exposure population, small numbers of deaths, and for an unexplained deficit of other childhood cancer deaths (3,16). Some studies have suggested that residence near a nuclear facility was linked to clusters of childhood leukemia and lymphoma in other countries (17–19), but more rigorous studies have not demonstrated increased risks (20–25). Paternal employment at nuclear facilities, particularly prior to the child’s conception, was suggested as a risk factor for childhood cancer in one study (19) but not in others (26,27). One study found paternal exposure to radionuclides but not external radiation to be associated with leukemia and NHL (27). Other possible explanations for the increases in the vicinities of nuclear facilities include chance, boundaries for the areas under study being determined by the existence of cases, outbreaks of an infectious disease, and exposure to some other unidentified environmental agent (20,25,28–32). To date, the evidence is not convincing that extremely low doses of radiation from fallout or from residing near nuclear facilities are associated with childhood cancers. The studies have been limited by the lack of detailed exposure information for the individuals under study, which can lead to underestimation of risks in epidemiologic studies. Radon, a radioactive decay product of radium that leaches out of the soil into air and groundwater, has been linked to lung cancer in

Table 2. Number of cases and incidence rates for selected cancers by race and gender among children (0–14 years of age), based on data from the National Cancer Institute’s Surveillance, Epidemiology, and End Results (SEER) program for 1975 to 1990.

| Cancer                  | White males |       | White females |       | Black males |       | Black females |       |
|-------------------------|-------------|-------|---------------|-------|-------------|-------|---------------|-------|
|                         | Count       | Rate  | Count         | Rate  | Count       | Rate  | Count         | Rate  |
| Leukemia                | 1472        | 4.41  | 1181          | 3.70  | 130         | 2.55  | 127           | 2.51  |
| Acute lymphocytic       | 1159        | 3.46  | 936           | 2.92  | 91          | 1.78  | 94            | 1.63  |
| Acute myeloid           | 125         | 0.38  | 122           | 0.39  | 16          | 0.32  | 24            | 0.49  |
| Other leukemia          | 188         | 0.56  | 123           | 0.39  | 23          | 0.45  | 19            | 0.39  |
| Gliomas and meningiomas| 943         | 2.89  | 778           | 2.50  | 117         | 2.31  | 112           | 2.25  |
| Neuroblastoma           | 376         | 1.06  | 346           | 1.03  | 43          | 0.77  | 46            | 0.89  |
| Wilms’ tumor            | 254         | 0.74  | 291           | 0.86  | 42          | 0.78  | 54            | 1.02  |
| Soft tissue sarcoma     | 262         | 0.80  | 211           | 0.68  | 46          | 0.91  | 39            | 0.90  |
| Non-Hodgkin’s lymphoma  | 415         | 1.30  | 142           | 0.46  | 35          | 0.72  | 18            | 0.36  |
| Hodgkin’s disease       | 253         | 0.81  | 188           | 0.64  | 34          | 0.72  | 14            | 0.30  |
| Retinoblastoma          | 116         | 0.32  | 124           | 0.36  | 23          | 0.41  | 23            | 0.41  |
| Osteosarcoma            | 99          | 0.32  | 93            | 0.31  | 18          | 0.38  | 22            | 0.47  |
| Ewing’s sarcoma         | 97          | 0.31  | 89            | 0.30  | 1           | 0.02  | 2             | 0.04  |
| Other                   | 451         | 1.37  | 534           | 1.74  | 57          | 1.34  | 93            | 1.94  |
| Total                   | 4738        | 14.33 | 3967          | 12.58 | 556         | 10.90 | 552           | 10.99 |

*Per 100,000 person-years, age-adjusted using the 1970 U.S. standard population.

Table 3. Selected environmental exposures and associated cancers among children.

| Exposure                  | Cancer                                           |
|---------------------------|--------------------------------------------------|
| Radiation                 | Leukemia, thyroid, brain, breast, skin, melanoma, soft tissue sarcoma, osteosarcoma |
| Electromagnetic fields    | Leukemia, brain, lymphoma, soft tissue sarcoma   |
| Diethyldithostrobol       | Vagina                                           |
| Phenootin                 | Neuroblastoma, soft tissue sarcoma               |
| Alkylating agents         | Leukemia, osteosarcoma                           |
| Chloramphenicol           | Leukemia                                          |
| Immunosuppressive therapy | Non-Hodgkin’s lymphoma, Hodgkin’s disease, skin, soft tissue sarcoma, lung cancer |
| Tobacco                   | Oral cancer, leukemia, rhabdomyosarcoma, lymphoma, lung cancer |
| Pesticides                | Leukemia, brain cancer, neuroblastoma, Ewing’s sarcoma, Wilms’ tumor, lymphoma |
| Epstein-Barr virus        | Burkitt’s lymphoma                                |

*Usually develops in adulthood. †Evidence to date is inconsistent or preliminary. ‡Cigarette smoking is unequivocally linked to lung cancer. The evidence to date for childhood passive smoking as a causal agent of subsequent lung cancer in adulthood is inconclusive.

Figure 1. Trends in childhood cancer incidence among white children 0 to 14 years of age, SEER Program, 1975 to 1978 to 1987 to 1990.
uranium miners and is thought to play a role in lung cancer in adults in the general population (33). Two reports found a correlation between indoor radon and acute myeloid leukemia (34,35), although this malignancy has not been found to be elevated in uranium miners. A study that evaluated childhood cancer county mortality rates and radon concentrations in drinking water in North Carolina found a dose-related association with leukemia (36). More research is needed to determine the role of radon in cancer etiology among both adults and children. Ultraviolet radiation causes skin carcinomas and melanomas (37). These tumors rarely appear in childhood because of the long latent period involved, but evidence is increasing that the exposures sustained during childhood are important determinants of risk, particularly for melanoma. The number of blistering sunburns experienced before 20 years of age, especially by fair-skinned, blue-eyed persons, is a strong determinant of risk for melanoma later in life (38).

**Electromagnetic Fields**

The role of EMF generated by power lines, electrical appliances, and large electrical machinery in the development of cancer is controversial. Adults exposed occupationally to EMF have consistently been found to have increased risk for all leukemia, acute myeloid leukemia, and brain cancer, but the workers usually were also exposed to other potential carcinogens, such as solvents (39,40), leaving the role of EMF unclear. Studies of residential EMF exposure have shown associations with leukemia and brain cancer among children (41-43), but generally not among adults (39,44-46). The most puzzling aspect is that the association between EMF and childhood leukemia appeared stronger when EMF was indirectly estimated by evaluating wiring code configurations and appeared weaker when EMF was directly measured (43,47), contrary to what would be expected if the association were causal. On the other hand, as suggested by Theriault (40), perhaps the wiring code configurations provide a better indication of long-term exposure than short-term direct measures of EMF. Parental employment in occupations involving EMF was linked to neuroblastoma among children in two studies (48,49) but not in another (50). Ongoing studies, some of which involve monitoring exposures throughout the subject children's day (i.e., residence, school, daycare, etc.) (51,52) plus wiring configuration codes, may help clarify the role of EMF and childhood cancer.

**Medications**

Transplacental carcinogenesis was established by the discovery in 1971 of vaginal adenocarcinoma in the daughters of women who took the hormone diethylstilbestrol (DES) during pregnancy to avoid miscarriages (53). This very rare cancer has been detected in girls as young as 7 years old, with most affected between 15 and 22 years of age (54). There are concerns that at older ages the exposed daughters may also have increased risk of squamous carcinomas of the vagina and cervix and cancers of the breast (55-59) and that exposed sons may have excess testicular and prostate cancer (55,60,61). Continued followup of the DES-exposed daughters and sons is ongoing at the National Cancer Institute and may provide further information on the late effects of DES and on transplacental carcinogenesis in general (62). Suspected, but less well-established, of being a transplacental carcinogen is phencytoin, an antiepileptic drug. There are reports of neuroblastoma (63-65) and soft tissue sarcoma (66) in children exposed in utero to phencytoin. There have also been reports of excess brain tumors, neuroblastomas, leukemia, and retinoblastomas in children of women who used antinausea medications (e.g., Bendectin) during pregnancy (67-71). This issue had received considerable publicity, however, which may have affected recall of use by study subjects. One study used medical records, not subject recall, to assess exposure and did not show any associations (72). There is one report of excess Wilms' tumor among Swedish children whose mothers were exposed to penthrane (methoxyfluorane) anesthesia during delivery (73). The excess risk was higher in females and increased with age at diagnosis. Some medical treatments received during childhood also play a role in the development of childhood cancer. Chemotherapy and radiation therapy received for an initial childhood cancer can dramatically increase the risk for second cancers (74,75). For example, in one study children treated with alkylating agents for cancer have a 5-fold risk of subsequently developing leukemia (76). At high doses, the risk was increased as much as 25 times the expected rate of leukemia (76). Bone sarcomas were also elevated in children treated with radiation and chemotherapy (77). The potent antibiotic chloramphenicol, given to treat life-threatening infectious conditions, has been linked to excess acute lymphocytic leukemia and acute nonlymphocytic leukemia in children in Shanghai (78). This association with leukemia is consistent with a report of bone marrow depression following use of chloramphenicol (79). Parental use of illegal drugs has been linked to childhood cancer in a few reports. Marijuana use was associated with rhabdomyosarcoma (80), leukemia (71), and brain tumors (67). Cocaine use was also associated with rhabdomyosarcoma (80). These exposures are difficult to study accurately and need further research, but prevention efforts clearly must continue for noncancer-related reasons even in the absence of convincing data on childhood cancer.

**Tobacco**

Tobacco, the single exposure responsible for the largest proportion of cancers among adults, is also important to consider in a discussion of cancer among children. At ages 16 to 19, 16% of boys and 15% of girls were current smokers during 1985 (81). Smoking prevention programs must be vigorous and start at young ages. It is also important to recognize that a large proportion of children are exposed to tobacco by-products from parental smoking during pregnancy and during childhood. Several studies of leukemia and lymphoma have reported increased risks associated with parental cigarette smoking (82-85). In one study, risk increased if more than one parent smoked (82). The evidence is less convincing that tobacco plays a role in the etiology of other childhood cancers. Rhabdomyosarcoma was associated with paternal but not maternal smoking in one study (86). No associations between smoking and soft tissue sarcoma in general or rhabdomyosarcoma specifically were seen in other studies (87,88). Neuroblastoma (69), brain tumors (85,89-91), and Wilms' tumor (92) also have not shown any association with parental smoking. Use of smokeless tobacco by children has been increasing at an alarming rate (93). In 1985, 30% of white males 12 to 17 years of age reported having used smokeless tobacco, approximately twice the prevalence of use in men over age 35 (94). Use is primarily among males except for Native Americans, among whom 45% of teenage girls also use smokeless tobacco (95). One study of junior and senior high school students reported that 55% of smokeless tobacco users began use...
before age 13 (96). Smokeless tobacco use is strongly associated with oral and pharyngeal cancer (97), and cases of oral mucosal changes and cancer have been diagnosed in teenage boys (98).

**Alcohol**

Excessive alcohol use has been linked to adult cancers of the oral cavity, pharynx, esophagus, larynx, and liver, with suggestive evidence for increased risk of colorectal and breast cancer (99). Alcohol is known to cross the placenta, with heavy maternal drinking resulting in fetal alcohol syndrome, a constellation of deformities and impairments (100). It is not known if transplacental exposure to alcohol also increases risk of childhood cancer or subsequent cancers in adulthood.

**Pesticides**

Many pesticides are carcinogenic in laboratory animals, and several have been associated with cancer in adults (101). Phenoxyacetic acid herbicides have been linked to lymphoma and soft tissue sarcoma. Organochlorine insecticides have been associated with lymphoma, leukemia, soft tissue sarcoma, neuroblastoma, and cancers of the pancreas, breast, and lung. Organophosphate insecticides have been reported to increase the risk of lymphoma and leukemia. Arsenicals appear to cause lung and skin cancers, while triazine herbicides have been associated with ovarian cancer. Most of the human data come from studies of farmers, licensed pesticide applicators, other agricultural workers, and manufacturing populations. Children are potentially exposed to pesticides from use in homes, gardens, and yards, through the diet, and through contaminated drinking water. Children of farmworkers are also often heavily exposed while accompanying their parents to the fields, while in housing contaminated by direct pesticide spray or drift from nearby fields, and through their own farmwork (102). Beginning in the late 1970s, there have been several case reports of cancer among children exposed to pesticides. Pre- and postnatal exposure to the termiticide chlordane was associated with neuroblastoma and childhood leukemia (103–105). Cases of organophosphate insecticide exposure linked to aplastic anemia and acute leukemia were reported in children (106,107). Nine of 13 extremely rare cases of colorectal cancer were found among children exposed to insecticides used in the production of cotton and soybeans (108). A cluster of cancers including leukemia, lymphoma, neuroblastoma, and Wilms' tumor was noted among farmworker children in California (109). Case-control studies have appeared to confirm the leads generated by these case reports. Maternal employment in agricultural occupations (OR = 1.8) or reported exposure to pesticides during pregnancy (OR = 3.5) was associated with acute lymphocytic leukemia in a case-control study in China (78). Occupational exposure to pesticides by either parent and use of pesticides in the home or garden during childhood was linked to acute myeloid leukemia in U.S. children (110). Parental use of pesticides in the home or garden during pregnancy (father or mother) or nursing (mother only) was associated with 3- to 9-fold increases in childhood leukemia in a case-control study in Los Angeles County, CA (111). Brain cancer has also been linked to pesticide exposure of children (112,113) or the mother (112,114) or father (115) during pregnancy. In one study, paternal employment in agriculture (OR = 9) or in any occupation with exposure to pesticides (OR = 6) was strongly associated with Ewing's sarcoma (116). Another study had similar findings but reported lower levels of increased risk (117). Wilms' tumor (92) and childhood NHL (J Buckley, unpublished data) have also been associated with household or garden insecticide use in one case-control study each. Many of the case-control studies of pesticides and childhood cancer are limited by the small number of exposed subjects and in some instances by the possibility of recall bias, but it is striking that many of the reported increased risks are of greater magnitude than those observed in studies of pesticide-exposed adults. These reports suggest that children may be a particularly sensitive subgroup of the population with respect to possible carcinogenic effects of pesticides. This is of concern, given the children employed in farmwork and the high prevalence of pesticide use in the home in the general population. The U.S. EPA has estimated that 82% of U.S. households use pesticides annually (75% insecticides inside the home, 22% insecticides or herbicides in the yard or garden) (118). Another survey reported that approximately one-third of single-family households treat their lawns with herbicides (119), a practice that is estimated to be increasing 5 to 8% annually (120). The possible carcinogenic effects, particularly to children, of this liberal use of pesticides within and around the home must be evaluated further.

**Other Industrial Chemicals and Physical Agents**

Children who have been exposed to the carcinogen asbestos carried home on their fathers' workclothes (121) or by playing near open pits at an asbestos mine (122) have developed mesothelioma decades later. These findings have raised concern about the potential exposure of children to deteriorating asbestos ceilings in schools built in the United States between 1950 and 1973 (2). The roles of other environmental or parental occupational exposures in the development of childhood cancer are unclear. There have been several studies evaluating parental occupation, for example, with conflicting results. Fabia and Thuy (123) found a greater proportion of the fathers of children with Wilms' tumor to have been employed in jobs involving lead and hydrocarbon exposures, but these findings were not confirmed in a later study by Wilkins and Sinks (124). Fabia and Thuy (123) also reported an association between hydrocarbon exposures and brain cancer. Some later studies found a similar increase (115,125), but other studies did not (68,126–130). In most if not all the studies to date of childhood cancer and parental occupation, potential exposures were determined either by examination of parental job title alone or by use of relatively crude job-exposure matrices that contained data on known and suspected carcinogens only. More comprehensive and sensitive methods of assessing exposures by expert industrial hygienists (131) are needed to improve the quality of this line of research and possibly resolve the conflicting results concerning childhood cancer and parental occupational exposures.

**Air and Drinking Water**

Some general environmental exposures via drinking water and air have been investigated with respect to childhood cancers. Possible carcinogenic effects related to fluoridation of municipal drinking water supplies have been evaluated thoroughly many times, most recently using 36 years of cancer mortality data and 15 years of cancer incidence data (132). Osteosarcoma was a cancer of particular interest because a 2-year bioassay had reported a small number of osteosarcomas in male rats but not in female rats or mice of either gender (133). The human cancer incidence data revealed increases over time of osteosarcoma in
young males under age 20 that were more prominent in fluoridated areas than in nonfluoridated areas. The increases were not related to the timing of fluoridation, however, so the authors concluded there was no link of cancer to fluoridation (134). This conclusion was consistent with several earlier expert evaluations of fluoride and cancer (135–137). A cluster of leukemia cases detected in 1979 in Woburn, Massachusetts, was thought by some researchers (138) but not by others (139) to be related to contamination of the town drinking water supply by trichloroethylene from a nearby chemical plant. Other general environmental exposures have been studied less extensively. One case-control study (140) and two correlational studies (141,142) have suggested that motor vehicle exhaust may increase risk of childhood leukemia. 

Infectious Agents

Infectious agents, generally viruses, are included in the category of environmental exposures and have been linked to a few types of cancer in adults. There is little evidence, however, of viral- or bacterial-induced cancer in children. An exception would be Burkitt’s lymphoma, which in Africa is related to infection with Epstein-Barr virus (143). Considerable attention is being given currently to the hypothesis that the excesses of leukemia seen in populations near nuclear facilities in England and other areas where large-scale population mixing occurred may, in fact, be due to some infectious agent as yet unidentified (29–32). Not all areas with similarly large influxes of children or their parents have demonstrated increases in childhood leukemia, however (28).

Future Research Recommendations

There are a few well-established environmental causes of childhood cancer, such as radiation, chemotherapeutic agents, and diethylstilbestrol. Many other agents such as EMF, pesticides, and some parental occupational exposures are suspected of playing a role, but the evidence is not conclusive at this time. There is a need to research and better quantify these exposures. Studies must entail sophisticated exposure assessment, such as that used in epidemiologic studies of occupational exposures and adult cancers, and consideration of possible genetic and environmental interactions.

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