

dying under age ten of leukemia in the years 1948–1960 by county. Using 1950 and 1960 census population for denominators, I found that in 1950, New York state had a childhood leukemia death rate of 4.3 per 100,000 people, but Oneida County alone had an elevated rate of 19.9 per 100,000 people. Between 1950 and 1951, Rome, Oneida County, New York, had a greatly elevated childhood leukemia rate of 36.1 per 100,000 people. I verified the Rome, New York, diagnoses through both pathological information and interviews with the next of kin. One father told me that his wife had also died of acute leukemia at age twenty-eight, one year after their son had died. Unfortunately, at the time, I could find no common denominator to explain the cases (Milham 1963). In 1996, when researcher Stanislaw Smiegelski showed that Polish military personnel working with radar had an increased incidence of leukemia, I remembered that many of the homes I visited in Rome, New York, were in line-of-sight with the large rotating radar arrays of Griffth Air Force Base. This offered further support of my suspicion that leukemia might be caused by environmental electrical exposure.

In reviewing the epidemiology literature about childhood leukemia, I came across a remarkable 1961 paper by Michael Court-Brown and Sir Richard Doll showing that a peak in childhood leukemia at ages two through four had emerged anew in the United Kingdom in the 1920s; and in the United States white population in the 1930s (Court-Brown & Doll 1961). Childhood leukemia was then a rapidly and completely fatal disease, which made death records an efficient way to count cases, since records would be recent and would count all cases. I reasoned that if we could find something that had changed at about the time the peaks appeared, we could solve the childhood leukemia mystery. It took about forty years, but I finally was able to show that the childhood leukemia peak was caused by some facet of residential electrification (Milham & Ossiander 2001). More about that later.

CONGENITAL DEFECT SURVEILLANCE

Shortly after starting work at the New York State Department of Health, I thought it would be interesting to do a real-time analysis of congenital defects. At the time, all of the vital records from the upstate population passed over just a few desks in Albany each month to be manually coded before being keypunched and stored. Very detailed annual reports were generated from this information, with a two or three year delay. I wanted real-time analysis so I set up a simple birth record review system. The problem was that rare events like congenital defects and childhood leukemia are so dispersed in the population, that no single doctor would be likely to notice an excess incidence in his practice. For birth defects surveillance, I asked the coders to make me a copy of all the birth and stillbirth records that listed a congenital defect, and did a monthly analysis of malformation incidence. I used a previous year of malformation experience for a baseline.

A few months after starting the system, there were a higher number of infants born with missing arms or legs than expected. I called the mothers, and found that almost all of them had been out of the country and had taken thalidomide. Before I had a chance to complete the investigation, the phocomelia-thalidomide connection appeared in the medical literature. It was thrilling to know that my simple system picked up the association in its early stages.

My system had another rigorous test when a pandemic of German measles swept the state. The virus can cause eye defects in fetuses if the mother is infected. The system picked up the eye defects with no problem, and the stillbirth rate increased and the live birth rate decreased, showing that a lot of the impact of the pandemic on births happened early in pregnancy before vital record ascertainment was possible.

When I left the health department four years later, the surveillance system left with me, since no one at the department was interested in maintaining it. When I joined the Washington State Department of

Health in 1968, after a short tour in Hawaii, one of my first projects was to set up a similar birth record surveillance system.

In 1970–71, eleven infants were born in Washington State with scalp defects reported on their birth records (Milham & Elledge 1972). The lesions were single, circular, punched-out ulcer-like midline defects at the top or over the back of the head. A query of the mothers revealed that two of them had taken methimazole (Tapazole, manufactured by Eli Lilly) during pregnancy for hyperthyroidism. One of the mothers delivered fraternal twins, both of whom had the defect.

I published a note in the journal *Teratology* asking if others had noticed this association. A number of physicians around the world had seen the same scalp defects in the infants of women taking methimazole, and one physician reported that a mother taking carbimazole had had an infant with a scalp defect. Carbimazole is metabolized by the body into methimazole, strengthening the association. Additionally, some of these infants were reported to have an umbilical cord defect called patent urachus.

I reported my findings to the drug company and to the Food and Drug Administration. A warning was added to the drug insert, and gradually other overactive-thyroid drugs replaced methimazole. I think this was the first, and possibly only, time that routine birth record malformation surveillance identified a human teratogen. This was probably the only way the association could have been made.

TWIN STUDIES

Girtlesohn and I also conducted a large study of twins (Girtlesohn & Milham 1965) in New York State, linking the birth records of single births into a twins pair file. We wound up with 21,128 twin pairs among all births between 1950 and 1960. There are two types of twins: identical or monozygotic (one egg), and fraternal or dizygotic (two egg). In our study, we examined fetal survivorship, sex, birth-weight, zygosity

(one egg or two eggs), maternal age, and birth rank. To me, our most interesting finding was that for any maternal age, the fraternal twinning rate increased systematically with birth rank. This held true for any maternal age with the number of previous pregnancies determining the probability that a woman would have fraternal twins. In other words, the more previous pregnancies a woman had, the higher her fraternal twinning rate became.

I knew that two-egg twinning was the result of polyovulation (more than one egg). A drug called clomiphene, given to infertile mothers, caused polyovulation and consequently many multiple births. Ovulation is under the control of the pituitary gonadotrophic hormones, follicle stimulating hormone (FSH), and leutinizing hormone (LH). I suspected that the pituitary gland had to be somehow responsible for the birth rank/fraternal twinning relationship.

My aha! moment came when I found a reference in my medical school pathology textbook showing that the weight of the pituitary gland of women increases with successive pregnancies. In 1964, I published a medical hypothesis in *The Lancet*, stating that fraternal twinning was caused by multiple ovulation, which in turn was caused by excessive production of the pituitary gonadotrophic hormones FSH and LH (Milham 1964). Years later, when FSH and LH levels could be accurately determined, my hypothesis was confirmed. This turns out to be true for others species, too. The litter size in dogs and cats increases with the number of litters.

LEUKEMIA IN HUSBANDS AND WIVES

In the early 1960s, while visiting at the Albany Medical College, a hematologist complained he was having trouble treating his leukemia patients. There was some scientific speculation at the time that human leukemia might be caused by a virus and could be spread by personal contact. This speculation created problems among families and clinicians

alike. He asked me if there was any way to examine the question. Three years later, I had his answer.

I studied adult leukemia in husbands and wives, selecting all leukemia deaths in the years 1951–1961 where the marital status of the deceased was stated as “widow” or “widower.” Of the 1,241 cases, a spouse’s death certificate was located for 876 of them. Out-of-state death was the major reason for failure to find a spouse’s death record. A matched death record control for each spouse was selected. There were seven leukemia cases in the spouses of the cases and five in the controls. The cause of death distribution of the spouses and their controls were similar. I was happy to conclude that adult leukemia is not contagious in the usual sense (Milham 1965).

HODGKIN’S DISEASE IN WOODWORKERS

My last study in New York before I left for Hawaii set the course for the rest of my career, by showing me the power of occupational studies. One of my medical school professors was dying of stomach cancer, and his college-age daughter was back in Albany to be closer to him. She volunteered to help in a study, which she selected from my “to do” list. We reviewed the death records of 1,549 white males who had died of Hodgkin’s disease and their matched controls, and found a two-to-one excess of woodworking or wood-exposure occupations in the Hodgkin’s cases (Milham & Hesser 1967). In Hodgkin’s disease, the exposure was to wood. How many other cancers might actually be attributable to other environmental or occupational factors?

UNIVERSITY OF HAWAII AND THE WASHINGTON STATE DEPARTMENT OF HEALTH

SHORTLY AFTER COMPLETING the Hodgkin’s disease study, I headed to Honolulu to take a job teaching epidemiology in the school of public health and the new medical school at the University of Hawaii.

The Hawaii job seemed ideal, but my oldest son, Richie, became chronically ill there with a low-grade fever and allergies. On a trip back to the West Coast during our first summer, he was well, only to become ill again on our return to Hawaii. His health problems would be a major factor in our relatively short stay.

I received a small grant from the Hawaii Cancer Society to replicate the Hodgkin’s disease study in the states of Oregon and Washington, where they have lots of woodworkers. West Coast woodworkers also had a Hodgkin’s disease excess, so the New York Hodgkin’s woodworker findings were published in *The Lancet* (Milham & Hesser 1967). While visiting the Washington State Department of Health in Olympia, Washington, during the course of the study, I found an advertisement

for the job that would take us back to the West Coast in May 1968. It would finally be our last job move.

My brief time in Hawaii was an eye-opener for me. The people I worked with were smart, and there always was something to be learned in other disciplines in the university environment. I loved teaching, and had a wonderful friend and boss in Bob Worth, head of the epidemiology department. We had moved from upstate New York in the dead of winter to a sunny and warm tropical paradise. The week before we left, I bundled the kids up on a sparkling cold winter's day to go sledding in the Helderberg Mountains near Albany, with a thermos of hot chocolate and cookies. Soon after that, we were in the tropics.

We spent a couple of nights at the Kaimana Beach Hotel while buying two old cars and renting a nice house in Hawaii Kai near Haunauma Bay. My children were the only Caucasians in the Koko Head elementary school and have been racially colorblind ever since. Lorna took a job teaching at a local college. This was during the Vietnam War, and my time at Tripler Army Hospital still haunts me. I saw many eighteen-year-old boys returning from that wretched war with permanent injuries that included paraplegia, double amputation, blindness, and brain damage.

WASHINGTON STATE HEALTH DEPARTMENT

We arrived in Olympia, Washington, in May 1968 to begin my job at the State Department of Health. I would eventually retire from there in 1992.

All the moving around had taken its toll. In 1971, Lorna and I would divorce. The children, by then ages eight, ten, and twelve, stayed with me in Washington State. Lorna went to California. In time, after the kids were off to college, I had other close relationships with smart, good women, one of whom died of a malignant melanoma at age forty-eight in 1990 after we had been together for ten years. She had

unfortunately used tanning beds. Tanning bed use has subsequently been linked to malignant melanoma. Some jurisdictions are attempting to restrict or outlaw tanning bed use.

OCCUPATIONAL MORTALITY STUDIES

The New York Hodgkin's study and its association with the woodworking occupations lead me to think that a lot of unknown occupational disease associations might turn up through a systematic analysis of the occupational and cause-of-death information contained in the Washington State death records. The intuitive appeal of occupational diseases is that they are, by definition, preventable. Disease prevention is every bit as important as treatment after disease appears. However, prevention is rarely noticed, funded, or rewarded.

I did some small case-control studies of single cancers and single occupations. In the course of these studies, I developed some computer software to automate the studies. One program is still in use at the Hutchinson Cancer Center in Seattle, some thirty-five years later. Soon I concluded that the piecemeal approach made no sense, and decided to examine all occupations and all causes of death. This approach was used in the United Kingdom by the Registrar General and in the United States by the National Center for Health Statistics. Both studies examined many causes of death in many occupations. The Registrar General's study is done every ten years, while the U.S. study was done only once. The United States study used a proportionate mortality analysis, but used an age sixty-four cut off, so it missed most of the deaths. Before jumping into a massive, long-term commitment to occupational coding of deaths, I thought it was smart to see just how comprehensive and accurate the occupational and industry information was on the death record.

I was the thesis advisor for a doctoral student named Gerald Petersen at the University of Washington School of Public Health.

Petersen interviewed the next-of-kin of men who died from Hodgkin's disease and found that the death certificate statement for occupation was identical to that obtained in the interview in 75 percent of cases, and gave a related occupation in 10 percent of cases. We found the same results for men who died from bladder cancer.

I had also conducted a large population-based mortality study of members of the AFL-CIO United Brotherhood of Carpenters and Joiners of America (Milham 1974) and found that their death certificate occupational statements nearly always listed a trade covered by the union. With the assurance that the occupational information was quite good, my secretary and I spent a year abstracting and coding adult male Washington State deaths. We used a modification of the United States Census Bureau's Occupational code, adding codes for occupations specific to Washington State.

The National Institute for Occupational Safety and Health (NIOSH) published our first report in 1976, covering male deaths from 1950 to 1971 (Milham 1976). A first update was published in 1983 covering the years from 1950 to 1979; and before I retired, we reported an online analysis of 588,090 white male deaths from 1950 to 1989, as well as 88,071 white female deaths from 1974 to 1989.

With a study spanning forty years of deaths, three different International Classifications of Disease (ICD) codes for cause of death were in use. I remember living with the three different colored ICD books for about three months to make sure that we had an accurate correspondence between the ninth, eighth, and seventh ICD codes. (You can go to <https://fortress.wa.gov/doh/occmort/> to look up your occupation and see its occupational mortality pattern). We used an age, sex, and year-of-death proportionate mortality analysis program, which calculates expected deaths based on the percent distribution of a given cause of death of all deaths in all occupations. A detailed cause of death analysis is available for 161 causes of death in 219 occupational categories for men, and sixty-eight occupational categories for women.

We knew we were on the right track when all the expected

occupational mortality associations were present in our data analysis: pilots die in plane crashes, loggers are struck by falling objects (trees), roofers fall, and power linemen are electrocuted. Most of the occupational mortality associations in the scientific literature were also present. Hard rock miners die of silicosis, asbestos and insulation workers die of lung cancer and pleural mesothelioma, and funeral directors and embalmers have increased leukemia mortality. The point of the study was to look for new occupational mortality associations, and a number of them emerged.

Below are some of the new occupational mortality associations we discovered.

MULTIPLE MYELOMA IN HANFORD PROJECT WORKERS

Workers at the Hanford Project, where weapons-grade plutonium for our nuclear weapons was made, had a small increased number of multiple myeloma deaths. I contacted Dr. Thomas Mancuso of the University of Pittsburgh, who headed a government-supported study of the health of these workers, and gave him my Hanford workers file. I later sat on an advisory group that oversaw the health projects at Hanford. The myeloma excess held up, and it still shows up in multi-site analyses and is related to ionizing radiation exposure in workers measured with film badges.

To make sure that the populations living near the Hanford facility were not being adversely impacted by ionizing radiation from the site, I studied the local populations through their vital records, school records, and parental employment records. When the government admitted intentionally releasing radio iodine (RI) from the site, I spent a lot of time visiting the "downwinder" families, and searching for any evidence of illness or death. Happily, I found nothing, but that wasn't what some people wanted to hear. Since radio iodine is a thyroid seeker, the downwinders expected that they would have increased thyroid

cancer and thyroid disease. Due to public pressure, the government then funded a large project to search for thyroid morbidity. It also found nothing. Large populations have been given RI therapeutically and diagnostically over many years, with no evidence that it causes thyroid disease or cancer.

LEUKEMIA AND LYMPHOMA IN ALUMINUM REDUCTION PLANT WORKERS

There were six aluminum reduction plants in Washington State where aluminum is made from aluminum ore (alumina or bauxite). The plants were located in the state during World War II because of its cheap hydroelectric power at that time.

The process involves the electrolytic reduction of alumina in an anode, cathode process and uses enormous amounts of electrical power. These workers had increased mortality rates from leukemia and non-Hodgkin's lymphoma in the Washington State occupational mortality study. I telephoned the medical directors of the companies, and was dismissed by all of them except for the medical director of Kaiser Aluminum, Dr. James Hughes. Kaiser had a reduction plant in Tacoma, and another in Mead near Spokane. They also had an aluminum rolling mill in Spokane. I explained my findings, and when I suggested a cohort study in the Mead worker population, Dr. Hughes agreed (Milham 1979).

The plant was built in 1946, and 25 percent of the workers were hired that year. There was a rumor that some of the worker records had been purged, but I found a proverbial "little old lady" in the personnel department who had saved a three by five card on every worker who had ever worked at the plant. These cards had all the information needed to do a good study. Using Richard Monson's modified life table computer program for analysis, I found increased mortality due to lymphatic and hematopoietic (blood) cancers, including leukemia, non-Hodgkin's

lymphoma, benign brain tumors, pancreatic cancer, and pulmonary emphysema.

The Mead plant was a pre-bake-type plant, in that the anodes used in the electrolytic reduction process were baked before being deployed. This process minimizes air pollution in the potrooms, the place where the electrolytic reduction takes place. The Tacoma plant was a horizontal stud Soderberg-type plant, where the anodes were baked in place in the potrooms. The anodes and cathodes were made of coke with a coal tar pitch binder. Coal tar pitch gives off volatile substances when burned, which include known carcinogens like benzo-a-pyrene. The industry position was that the coal tar pitch volatiles (CTPVs) were responsible for the cancer excess seen in these workers. I doubted it, because men working in coke oven environments had much higher exposures to CTPVs and no excess of lymphoma or leukemia. They did, however, have a lung cancer excess.

I was suspicious that the strong magnetic fields in the potrooms might be a contributing factor. Dr. Hughes had his engineers characterize the EMF fields that the workers were exposed to.

I wanted the workers to know what we had found. There were three work shifts at the plant, so instead of making three personal presentations, Dr. Hughes and I made a video of our findings and recommendations. It was shown at the Mead plant, at the Tacoma plant, and at another Kaiser plant in Chalmette, Louisiana. A couple of days after the video was shown, I received a phone call from Don Kropp, the health and safety officer of the steelworkers union, which covered the Tacoma plant workers. "Hey doc, we have a lot of lymphoma here," he said. He was right. They had five cases of B-cell lymphoma diagnosed in a seven-year period at the plant. Dr. Hughes flew up from Oakland, and we took histories and examined the men. We also verified the pathology with referee pathologists at the University of Wisconsin medical school.

Dr. Hughes asked me to represent Kaiser in the Tripartite multi-plant study of mortality in the aluminum industry. It was called that

because the aluminum companies, the steelworkers union, and the government were represented. They paid my way to meetings, but as a state employee, I couldn't take any money for my services. I was able to meet all the aluminum company medical directors and their epidemiologists. I also attended a party at the Hughes' home in Oakland, California. I was surprised that Sir Richard Doll, the English researcher, and his wife, Lady Joan, were there.

I later learned that the management of Kaiser had retained Doll to neutralize my cancer findings. I had previously spent time with Doll at Oxford University while on a lecture tour of UK medical schools. (I had an International Agency for Cancer Research fellowship and a United States technology transfer award). I was disturbed that he didn't seem to appreciate the epidemiologic strengths of my Washington State occupational mortality system. I was also puzzled by work that he and co-authors presented at a Cold Spring Harbor conference in 1981, stating that occupational exposures caused only 4 percent of all cancers and environmental exposures caused only 2 percent. I presented hard data at the same conference showing that the occupational component caused more than 10 percent of cancers.

Though confusing at the time, some of Sir Richard Doll's seemingly pro-industry stance made sense years later. After he died, it was revealed that Monsanto had paid him \$1,500 per day for many years.

LEUKEMIA IN ELECTRICAL WORKERS

In 1979, Nancy Wertheimer and Ed Leeper published a study suggesting that residential magnetic fields could cause childhood cancer. They didn't actually measure magnetic fields, but noticed that the electrical distribution wires at residences with childhood cancer cases were thicker than at control houses. Thick wires carry more current, and magnetic fields are proportional to current flow.

At that time, the Kaiser engineers had measured 100 gauss (G) of

static (steady) DC magnetic fields, and more than 100 milligauss (mG) of alternating AC magnetic fields in the potrooms (1,000 milligauss = 1 gauss). Most homes measure below 4 mG. Kaiser Tacoma operated on 60,000 amperes (amps) of alternating current (AC) power that was changed into direct current (DC) to supply the anodes and cathodes. I thought that if magnetic fields could cause cancer in children, what about adults working in strong fields?

When my first state occupational mortality tables became available, I looked at cancers in men who worked in jobs with a connection to electricity. These included electricians, power and telephone linemen, aluminum workers, radio and TV repairmen, welders, power station workers, and so on. These workers turned out to have increased mortality due to leukemia, especially acute leukemia, lymphoma, and brain tumors. When the letter I wrote on these findings was published in the *New England Journal of Medicine* (Milham 1982), I received a number of calls from colleagues around the country saying that there was no way these weak fields could cause cancer. My answer was simply, "Prove me wrong." But unfortunately, over the next few months, they and others proved me right. The last time I looked, there were over fifty residential and one hundred occupational studies that now associate power frequency magnetic field exposures with cancer.

Leukemia in Amateur Radio Operators

After my letter was published, I received a note from an amateur radio operator named Andrew Sobel, W2EVE (each amateur radio operator is assigned a unique call sign). He suggested that I study mortality in members of the American Radio Relay League (ARRL). ARRL is a group of amateur "ham" radio operators who are exposed to electromagnetic fields, including radio frequency radiation (RFR).

Recent ARRL deaths are reported in the "Silent Keys" section of *QST*, ARRL's monthly magazine. A quick study of the deaths reported in the Silent Keys column who were residents of Washington State and California, where I could get quick access to the death records, showed twice as many leukemia deaths as expected.

Though I had been invited by an AARL member to investigate, I did not receive the support of the organization. I negotiated unsuccessfully with AARL for nearly a year to get access to their membership files so I could do a national population-based study. In the course of learning about the ham radio hobby, I learned that all amateur radio operators in the United States are federally licensed. I purchased the 1984 Federal Communications Commission (FCC) license file and did a cohort study of 67,829 Washington State and California amateur radio licensees (Milham 1988). Again, I chose those two states because I had ready access to death records. AARL members did have increased mortality due to lymphoma and acute myeloid leukemia.

After the study had been published, I received a call from the National Cancer Institute (NCI) asking why I hadn't done a license class analysis too, since the exposures would be different depending on the frequency and intensity of a licensee's use. The reason I hadn't included that was because the data sheet I had received from the FCC had an undecipherable entry, "... ass," which turned out to stand for "license class." Interestingly, when I did a subsequent license class analysis, it showed very little increased mortality in the novice entry level license class, the youngest of the five license classes, who were limited in transmitter power and restricted to certain transmission frequencies. But with increasing International Morse Code speed and theoretical knowledge, they could graduate to higher license classes and higher exposures. The higher class licensees had most of the cancer excess in the cohort (Milham 1988).

KAISER TACOMA IMMUNE STATUS STUDY

Over a ten-year period, the Centers for Disease Control assigned a series of physicians to me for a two-year training period. One of them, Dr. Robert Davis, helped me study the immune status of workers at the

Kaiser Tacoma plant in work related to the initial studies we had done there.

The steelworkers union reported additional lymphoma cases had been diagnosed since Dr. Hughes and I had examined the original group of cases. B-cell lymphoma is more common in both congenital and acquired immunodeficiency. Children born with combined immune deficiency syndrome, people with AIDS, and people treated with immunosuppressive drugs for organ transplants all have an increased incidence of B-cell lymphoma.

Dr. Hughes at Kaiser Tacoma had by then retired, and unfortunately the "risk managers" who succeeded him at Kaiser refused to cooperate with additional studies. We wanted to see if healthy aluminum reduction plant workers had any evidence of immune system problems that could account for the excess of B-cell lymphoma in the worker population. We had the support of the workers, so the steelworkers union rented a senior citizen's center near the plant. We were able to draw blood samples to study the blood lymphocytes of twenty-three volunteers from the Kaiser Tacoma workforce. The University of Washington's transplant lab did a battery of blood studies on fresh blood drawn two samples at a time, which was delivered to them once a week over a twelve-week period. This meant getting up very early in the morning to meet the workers either before or after their work shift, and driving a total of one hundred twenty miles per day.

At the end of the study (Davis & Milham 1990), I received a call from the lab stating that the Tacoma workers we were studying had very abnormal T4 and T8 counts. The laboratory recommended redrawing the blood samples. With no more funds and no desire to repeat my early morning, one hundred twenty mile per day round trips, I initially refused. Later, I agreed to repeat the study if all the blood work could be drawn at one sitting with no further expense.

Dr. Davis and I took blood samples from the group again and got identical results. The T8 and T4 counts in these workers were very high, and they had abnormal T4/T8 ratios because of the high T8 counts.

My interpretation of these results was that the body was mounting an immune response to the EMF fields in the potrooms. I postulated that after time from such chronic immune system stimulation, the immune system fatigues and fails, allowing the lymphoma to occur. A later study of rats exposed to pulsed radio frequency radiation (microwaves) showed a very similar immune system pattern, and the rats also had a large cancer excess. That study is covered in detail in chapter 7.

MULTIPLE SCLEROSIS AND VITAMIN D

Early in 1980, I worked on another interesting study while at the health department. My occupational mortality data clearly showed that multiple sclerosis (MS), a remittent demyelinating central nervous system disease, was more common in indoor workers than in outdoor workers. To make sure that it wasn't a matter of self-selection into sedentary jobs, I looked at indoor and outdoor jobs in men dying of chronic rheumatic heart disease. They showed no indoor vs. outdoor mortality difference. The epidemiology of MS was fascinating. The disease was uncommon at the equator, but increased in incidence with increasing north and south latitude. Average annual hours of sunshine had a nearly perfect negative 90 percent correlation with MS incidence and mortality. This means that the more sunshine where you live, the less MS there is. The geographic exceptions were also interesting. Populations in dark regions with high fish consumption had low MS rates.

Vitamin D is really a powerful hormone, and with the exception of fatty fish and fish oils, is almost absent from the diet. Sunlight or ultraviolet light falling on the skin generates vitamin D in our bodies. Ultraviolet light is a part of the EMF spectrum that our species evolved with and obviously needs. One hour in tropical sun generates about 10,000 international units (IU) of vitamin D. In spite of this, the recommended daily dose of vitamin D is about 400 IUs. To compound the problem, sunscreens have been recommended to prevent skin

cancers, cutting down on vitamin D production when outdoors. In dark climates, 3,800 IUs is a reasonable daily dose of vitamin D.

I wrote a two-page hypothesis suggesting that MS, like rickets, was a vitamin D deficiency disease. *The Lancet* rejected the paper at the time, but in the last five years, Scandinavian studies have verified all my theories about the relationship of MS to vitamin D.

At about that time my first cousin, then in his early twenties, developed MS. I explained my theory to him and suggested that he take an outdoor job. He did, and he's had minimal disease since then.

RADIAL TIRE FIELDS

My interest in electromagnetic fields grew after the Kaiser Tacoma work, and I'd followed EMF epidemiology after Wertheimer and Leeper published their 1979 study on residential magnetic field exposures and childhood cancer. I bought my first magnetic field meter in the 1990s and started attending EMF meetings to keep up on what was going on in the field. Noting that secretaries and typists had a breast cancer increase in my occupational data, and remembering the old electric typewriters used in the pre-computer era, I found that many of them had very high magnetic fields at the chest level of the typist (Milham & Osslander 2007). The one exception was the popular IBM Selectric, which had low fields.

One day, I made the mistake of turning my magnetic field meter on in the car while driving. To my surprise, the fields were especially high (more than 20 mG) on the seat between my legs and highest near my left foot. The fields were only present when the car was in motion. Turning the engine off at sixty mph did not change the fields. It gradually became apparent that the fields were coming not from the engine, but from the spinning tires, which have a mile of fine magnetic steel wire woven into belts. I later ran a compass over the tread of a tire and watched the compass needle spin because of the magnetic domains in

the belts. Electromagnetic fields can be generated by spinning magnets, which is what steel-belted radial rotating tires are.

To study the magnetic structure of the steel belts, I cut up a tire, clamped a segment down flat, covered the inside of it with white paper, and “visualized” the fields by sprinkling iron filings on the paper, which would demonstrate the magnetic pattern created by the metal belts.

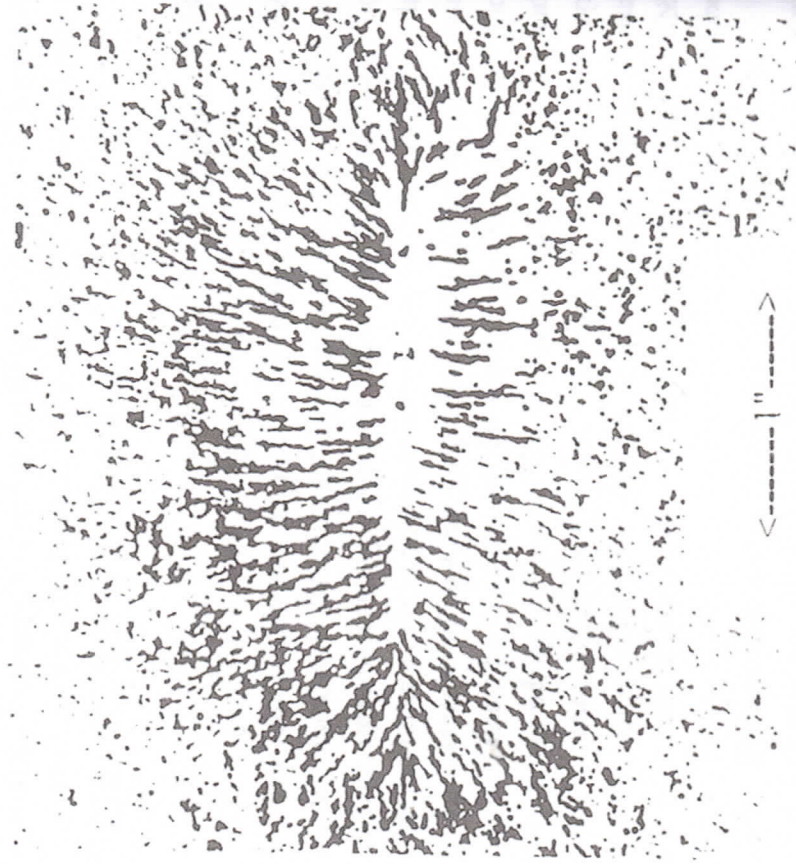


FIGURE 1
Radial tire iron filings

An X-ray of a tire segment was beautiful, showing the weave pattern of the steel belts. The radiologist smiled when I asked him to take an X-ray of a piece of tire.

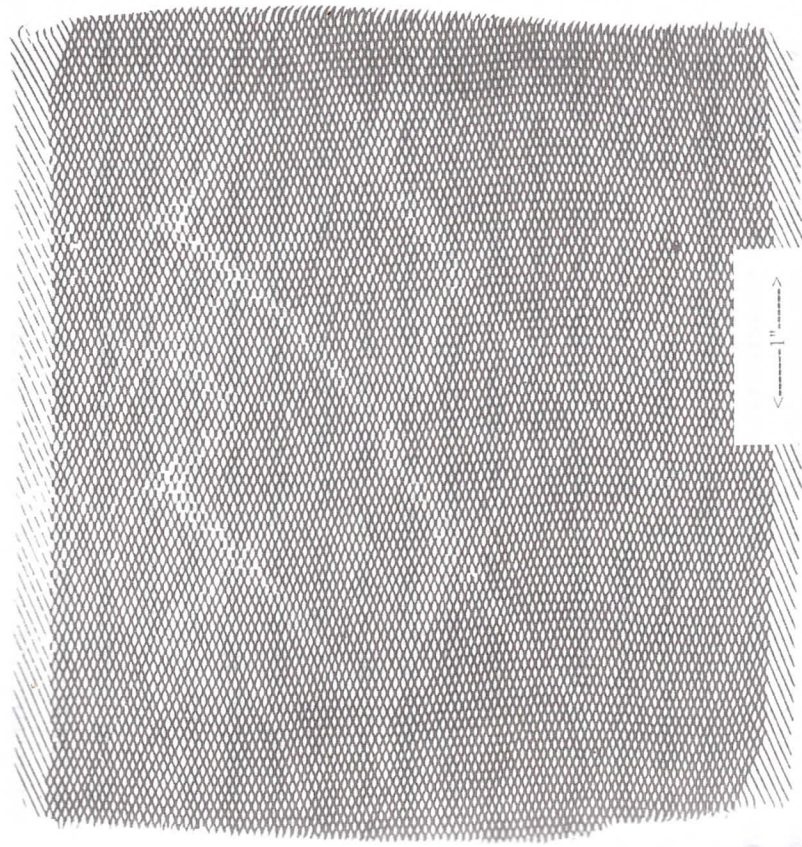


FIGURE 2
Radial tire x-ray

A trip to a tire store with a compass and my inexpensive magnetic field gauss meter, along with a more expensive Emdex meter borrowed from the University of Washington, answered a lot of questions. High-end gauss meters like the Emdex have on-board computers, which allow customized long-term magnetic field sampling.

Most tires by then were steel-belted radials. My compass picked out the one fiberglass belted tire still in the shop. A spinning steel-belted radial tire on a tire balancing machine gave high magnetic field readings on my meter about a foot from the tire, but amazingly, nothing was shown on the Emdex meter. The tire frequencies are determined by the frequency of rotation, and are well below the narrow band of frequencies

that the expensive Emdex meter was designed to detect. I recall that at sixty miles an hour, my Honda's tires were rotating at ten cycles a second and generating 10 Hz magnetic fields. My cheaper meter was clearly capable of picking up a wider range of frequencies.

This is of special interest, since similar high-end meters were and continue to be used in most of the human health EMF epidemiologic studies. Since these meters routinely reject all exposures below and above the power frequencies band, these studies could be missing critical exposure data. A child sitting in a baby seat over a rear tire could get a higher magnetic field exposure in an hour riding in a car than in twenty-four hours at home. The expensive meters, designed to examine a narrow band of frequencies around 60 Hz, will routinely miss fields below 30 Hz and above 300 Hz.

Eventually, I enlisted the help of electrical engineers Richard Tell and James Hatfield, who had the electrical knowledge and equipment to characterize the radial tire fields in detail. We published a paper about it in *Bioelectromagnetics* (Milham et al 1999).

I wrote to the tire manufacturers, suggesting that they could solve the problem by using non-magnetic steel wire in the belts, but got no response. I contacted the "Car Talk" radio program on National Public Radio and wrote to *Car and Driver* magazine, also with no response.

LEAVING THE HEALTH DEPARTMENT

Sometime in the mid-1980s I was informed that the health department intended to eliminate my occupational coder position. Eric Ossiander, an accomplished epidemiologist and statistician in my office, helped me automate the system. We ended up with a system that precisely matched the keyed death record occupation with those on a list. The system improved over time by adding occupational titles. It is still being used to code the decedent's occupation on the death certificate, and parental

occupations on the birth certificate. It codes correctly more than 90 percent of the time. Eric Ossiander does the back up coding.

Near the end of my time at the Washington State Health Department, there was a downsizing of employees and I was demoted. I made a futile appeal to the state personnel board. In the last two years there, my office had been moved four times. I decided that since the handwriting was on the wall, it was time to leave. I resigned on the day I turned sixty, in 1992.

from being built temporarily, but knew that the utilities would ultimately prevail, as they typically did.

I saw the same handful of utility “experts” in all of these cases. I often wondered how they could look themselves in the mirror after making misleading comments under oath, saying that power line EMFs were safe. It was also galling that they were so well paid with rate-payer money to testify against the rate-payers’ best interest.

At breakfast one morning during one of the trials, a utility expert I’d known for twenty years admitted that our side was right. But it did not stop him from pocketing a reported quarter of a million dollars a year as an expert witness for the utility companies, in addition to his full university professor’s salary.

MALE BREAST CANCER AND EMF EXPOSURE

I testified in a trial for two of three men who had developed breast cancer from sitting in the same office in a county office building in Albuquerque New Mexico, where they were exposed to strong magnetic fields from a nearby electrical switchgear room (Milham 2004). A dozen previous studies had linked male breast cancers (MBC) to EMF exposures, so these cases should have been a slam dunk. The plaintiff’s attorney had already won a nice settlement for one of the three men, but didn’t seem to care about the other two cases. He didn’t cross-examine the defendant’s EMF expert, and cut my testimony short to celebrate a religious holiday. Without a good attorney, an expert witness is both helpless and useless.

There are reports of an epidemic of male breast cancer at the Marine Corps Base Camp Lejeune in North Carolina in the environmental news right now. The Marines had identified fifty-five MBC cases and thought the epidemic was caused by solvent contamination of drinking water at the base. While there are studies that link solvents with a few cancers, the more likely culprit is EMFs. There have been fifteen studies

7 ON MY OWN

AFTER I RETIRED, I was often approached to be an expert witness, mostly testifying for people trying to prevent a power line from being sited near their homes. Like so many others who had experienced it, I did not like the legal adversarial system, or the fact that the deck was always stacked against the “little guys” who were trying to defend their personal worlds.

The utility companies had built-in rate-payer money at their disposal and hired the best attorneys, as well as very well coached expert witnesses to present their side of the case. Many times I worked for no money, only airfare and a place to stay. Unfortunately, the plaintiff’s lawyers, often just local attorneys, were no match for the high-paid legal specialists from big outside firms who did nothing but EMF cases.

In one case, I was hired by the Pennsylvania Public Utility Commission to evaluate citizens’ complaints about a number of proposed high voltage transmission lines. This was the only case that I was involved in where taxpayer money was actually used to support and evaluate taxpayers’ concerns. I helped stop a few power line corridors