MICRO WAVE NEWS

A Report on Non-Ionizing Radiation

Faulty DNA Repair May Explain EMF Role in Childhood Leukemia

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This could be a breakthrough, a major breakthrough. It could explain how power lines promote childhood leukemia. It could identify which children are at greatest risk. And it could shed new light on the pivotal role played by EMF-induced DNA breaks.

Chinese researchers have found that children who carry a defective version of a gene that would otherwise help repair damaged DNA are much more likely to develop leukemia if they also live near power lines or transformers. Xiaoming Shen and coworkers at the Jiao Tong University School of Medicine in Shanghai have reported that children with this genetic variant —known as a polymorphism or snp (pronounced "snip") —and who lived within 100 meters of these sources of EMFs had over four times more leukemia than neighboring children with a fully functional version of the same gene.

After so many have spent the last 30 years trying to understand how EMFs could promote childhood leukemia, Shen has come up with a relatively simple explanation: Children exposed to power-line EMFs suffer more DNA breaks, but those who carry this modified gene cannot repair the damaged DNA, making them more susceptible to cancer. Childhood leukemia is now generally believed to be initiated by a chromosomal rearrangement that occurs in the womb, followed by some environmental insult after birth. The power-line-induced DNA breaks could be one of many such possible secondary events.

Writing in the December issue of *Leukemia & Lymphoma,* Shen warned that children who carry this snp "could be particularly sensitive to the carcinogenic effects of EMF." He went on to suggest that it may be possible to prevent some leukemias by eliminating these children's exposures to EMFs. You Yang is the first of six authors of the paper.

As early as 1997, Henry Lai and N.P. Singh showed that power-line EMFs lead to higher levels of DNA breaks in rats. Even though their work has been repeated in a number of labs over the last decade —for instance, by Britt-Marie Svedenstål in Sweden, Hugo Rüdiger in Austria and Primo Schär in Switzerland— it remains controversial, partly because some others have failed to see the same effect and partly because some physicists continue to insist that the only way to accumulate DNA breaks is by breaking chemical bonds and EMFs don't have the necessary photon energy to do so (even this latter argument has been challenged).

In that first 1997 paper, Lai and Singh suggested that what they might be seeing is a modulation of DNA repair rather than broken DNA bonds. "A possible explanation of the present observations," they wrote, "is that 60 Hz magnetic fields affect enzymatic processes involved in DNA repair, leading to an accumulation of DNA strand breaks." Their hypothesis was later supported by a group led by Kim O'Neill in Utah. Shen's new finding introduces a genetic component and raises the possibility that there are two independent processes that impair DNA repair.

This gene in question is called *XRCC1* and is one of many known to help repair DNA damage. The variant studied by Shen has the same sequence as its more common, wild-type version except for the substitution of one single base, an adenine (A) for a guanine (G). This snp is known by a variety of designations: "rs25489," "Ex9+16G>A" and "Arg280His."

Others have previously shown that the same Ex9+16G>A polymorphism makes its carriers more likely to develop breast and prostate cancer. For instance, researchers in Cyprus, France and the U.S. have all reported that women with this snp have higher rates of breast cancer. Women who smoked cigarettes were typically at the greatest risk —here, the chemical carcinogens in cigarette smoke would cause the DNA breaks. Similarly, male smokers in China who carry this same snp have been found to have higher rates of prostate cancer. (For more on polymorphisms and cancer, see reviews from IARC and the Fred Hutchinson Cancer Center; both are free downloads.)

Shen's finding may also explain why children in Mexico City have one of the highest incidences of leukemia in the world. According to statistics compiled by the Centers for Disease Control (CDC), Mexican-Americans are much more likely to carry the Ex9+16G>A snp than non-Hispanic (white and black) Americans: over 20% compared to 6-8%. Juan Manuel Mejía-Aranguré has also reported that children in Mexico City also have higher magnetic field exposures than those in other countries, often more than 0.6 μT (6 mG).

Shen told *Microwave News* that 4-6% of his study population, who live within 500 km of Shanghai carry the Ex9+16G> polymorphism.

One last, but far-reaching, implication of Shen's study is that, if proved correct in further studies, it would implicate extremely weak magnetic fields, certainly less that 0.1 μT (1 mG). Shen found that the leukemia risk among carriers of the Ex9+16G>A snp extended

out to at least 100 meters. Even at 500 meters more of these children had leukemia, but here the increase was no longer significant. Though this finding is surprising, it is consistent with a large 2005 study by Gerald Draper in the U.K., who found higher risks among children living 200 meters from high-voltage power lines. Draper also saw a risk out to 600 meters, which only just attained significance. (The British and Chinese used different epidemiological techniques and their risk estimates are not comparable.)

In his response to a barrage of attacks, Draper wrote: "We have been criticized for publishing alarming results that we cannot explain. We should have preferred to delay publication until we could analyze magnetic field exposure data and, if possible, explain our results. It would have been unethical, however, not to publish results of potential health significance." The results of Draper's magnetic field analysis are expected next year.

We await for the reaction to Shen's breakthrough. He has tied together so many loose ends, it simply cannot be ignored.

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