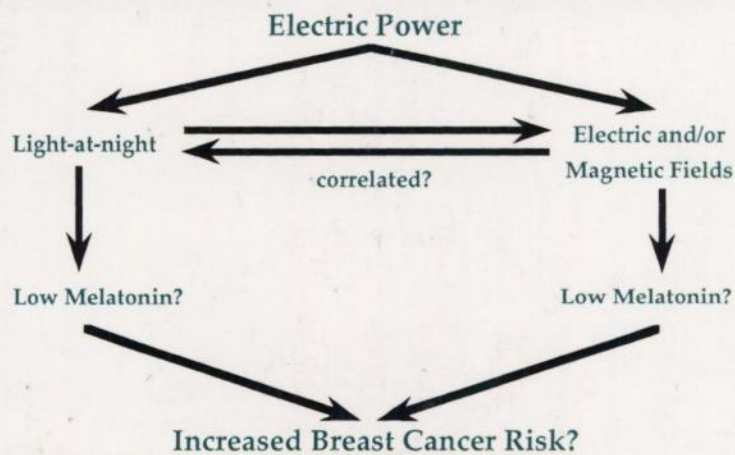
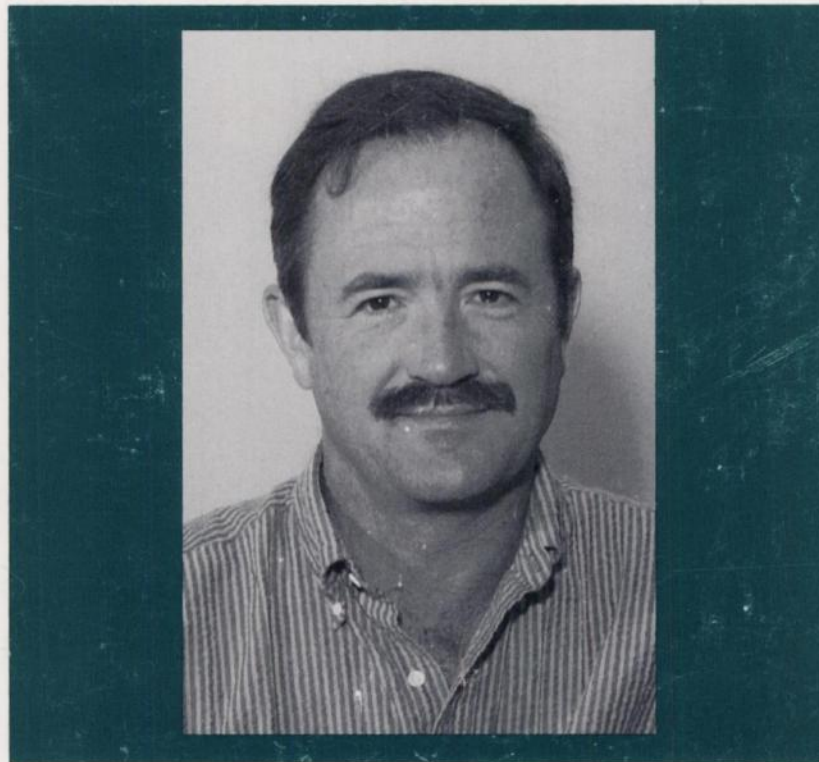




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# CARCINOGENESIS FROM ENVIRONMENTAL POLLUTION: ASSESSMENT OF HUMAN RISK AND STRATEGIES FOR PREVENTION

Joint Meeting Organized by the  
American Association for Cancer Research (AACR)  
and the International Agency for Research on Cancer (IARC)

With the Collaboration of the Hungarian Cancer Society



October 6-9, 1996  
Hotel Gellért  
Budapest, Hungary



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## SCIENTIFIC PROGRAM

### Keynote Address

Curtis C. Harris / Bethesda, USA

### Cancer Incidence and Etiology

Witold A. Zatoński / Warsaw, Poland

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### Air, Water, Food, and Soil Contamination

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### Ambient, Environmental, and Occupation Exposure and Cancer Risk

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Ivan Plesko / Bratislava, Slovakia

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Paolo L. Vineis / Turin, Italy

Stephen S. Hecht / Valhalla, USA

Krystyna Frenkel / New York, USA

Bernadette Schoket / Budapest, Hungary

### Strategies for Prevention

Waun Ki Hong / Houston, USA

I. Bernard Weinstein / New York, USA

Anna Tompa / Budapest, Hungary

### Roundtable Discussion

Paul Kleihues / Lyon, France

Hans-Olov Adami / Uppsala, Sweden

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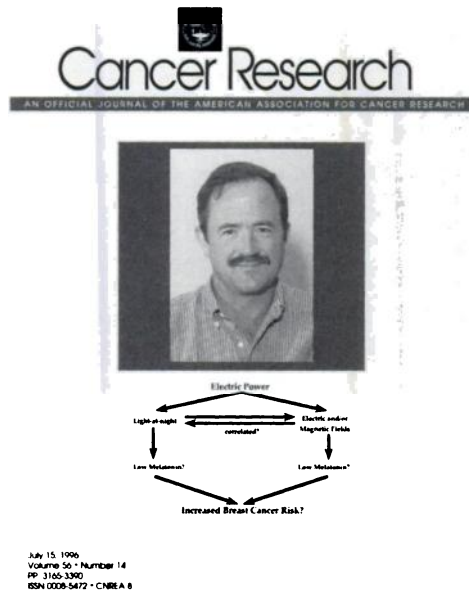
*Applicants are encouraged to submit abstracts  
for poster presentation.*

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### Information and Application Forms

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# COVER LEGEND



Breast cancer is a disease of modern life. As societies industrialize, the risk of breast cancer seems to increase; for example, the historically low risk in Japan has been rising fast in recent decades. Yet, it is unclear which of the myriad changes coming with industrialization might account for the increase in breast cancer in modern societies. There is little consensus among researchers on the primary causes of breast cancer; for example, whereas reproductive factors are acknowledged to be important, the debate on dietary fat continues to rage (*Epidemiol. Rev.*, 15: 110–132, 1993).

A hallmark of modern life is the pervasive use of electric power. Over a decade ago, in 1985, Richard “Bugs” Stevens (*cover*) made a presentation at the Department of Energy’s (DOE) Annual Review of Electromagnetic Fields Bioeffects in Alexandria, VA, suggesting that two products of electric power might play a part in explaining the mystery of breast cancer. In a series of publications, Dr. Stevens and colleagues have provided details on the hypothesis and its implications (*Am. J. Epidemiol.*, 125: 556–561, 1987; *FASEB J.*, 6: 853–860, 1992; *Environ. Health Perspect.*, 104(1) (Suppl.): 135–140, 1996). Electric power produces light-at-night (LAN) and anthropogenic electromagnetic fields (EMF), both relatively new exposures in the human environment that might suppress pineal function and its primary hormone melatonin, and thereby perhaps increase the risk of breast cancer (*see cover illustration*).<sup>1</sup> The circumstantial case for this hypothesis has three aspects to it: (a) light effects on melatonin; (b) EMF effects on melatonin; and (c) melatonin effects on breast cancer. The strongest of these aspects is light effects on melatonin. It is clear that the normal nocturnal melatonin rise in humans can be suppressed by light of sufficient intensity. (Whether ambient night time illumination typical in women’s homes can

<sup>1</sup> Reprinted with permission from Stevens, R. G. Electric power and risk of hormone-related cancers. In: D. O. Carpenter (ed.), *Biologic Effects of Electric and Magnetic Fields*, Vol. 2, pp. 263–268. Orlando, FL: Academic Press, Inc., 1994.

affect melatonin is unknown.) The evidence for an effect of melatonin on breast cancer in experimental animals is strong, but the evidence in humans is scant and difficult to gather. The most limited aspect of the circumstantial case is EMF effects on melatonin. Whereas half a dozen independent laboratories have published findings of suppression in animals, there are inconsistencies among those findings, and there are no published data in humans.

The direct evidence bearing on this hypothesis is sparse but provocative. Two laboratories have published data reporting substantial increases in chemically induced breast cancer in rats by a weak AC magnetic field (*Cancer Lett.*, 61: 75–79, 1991; *Cancer Lett.*, 71: 75–81, 1993; *Carcinogenesis*, 16: 119–125, 1995), and one has reported an EMF-induced reversal of melatonin’s oncostatic action (*J. Pineal Res.*, 14: 89–97, 1993). The epidemiological evidence is very limited but has offered some support (*J. Natl. Cancer Inst.*, 86: 921–925, 1994; *Am. J. Epidemiol.*, 142: 446–448, 1995) as well as negative findings (*J. Natl. Cancer Inst.*, 86: 885–886, 1994). Results from the first two large epidemiological studies to be focused specifically on this hypothesis will be reported early in 1997 from the Fred Hutchinson Cancer Research Center in Seattle, WA, and the Karolinska Institute in Stockholm, Sweden. Several more large studies are just getting under way.

Dr. Stevens received his B.S. in Genetics in 1973 from the University of California, Berkeley, and his Ph.D. in Epidemiology from the University of Washington in 1985. He came to the Pacific Northwest National Laboratory (PNNL), operated for DOE by Battelle Memorial Institute, in 1984. One of his responsibilities was to manage an epidemiological study of EMF and leukemia in adults sponsored by the New York Power Lines Project (*Am. J. Epidemiol.*, 128: 10–20, 1988). He became curious whether any bioeffects from low intensity EMF exposure had been shown in the laboratory. There was then, and continues today, to be serious doubt that it is even possible (*Phys. Rev. A*, 43: 1039–1048, 1991).

In the 1970s, DOE began a research program on EMF bioeffects and potential hazards which is now managed by DOE’s Office of Utility Technologies. Supported by this program, Bary Wilson and colleagues at PNNL published work reporting a suppression of the normal nocturnal rise in melatonin in rats by exposure to a 60-Hz electric field (*Bioelectromagnetics*, 2: 371–380, 1981), which is analogous to work by Peter Semm *et al.* from Germany showing an effect of an artificial static magnetic field on pineal activity in guinea pigs (*Nature*, 288: 607–608, 1980). Melatonin, in turn, may be important in breast cancer etiology (*Cancer Res.*, 41: 4432–4436, 1981; *Cancer Res.*, 48: 6121–6126, 1988; *J. Cancer Res. Clin. Oncol.*, 117: 526–532, 1991). Putting the two together yielded the electric power/breast cancer hypothesis. In addition, other factors that might disrupt pineal function, such as alcohol, shift work, and the lighted environment in buildings, may also be surmised to affect long-term risk of breast cancer.

To date, the body of evidence is sufficient to bind electric power over for trial, but not nearly adequate to render a verdict. Although the hypothesis is in its early stages of evaluation, it may be resolved within 5 years because, sadly, breast cancer is so common in industrialized societies that many large studies can be conducted simultaneously.

Sidney Weinhouse