1) This Preliminary Opinion omits important information on ELF-EMF and melatonin/tamoxifen studies as they relate to breast cancer. The Opinion should be revised by the Scientific Committee on Toxicity, Ecotoxicity and the Environment (CSTEE) before the report is submitted to the European Commission for the purpose of updating its 2001 opinion on “Possible Effects of Electromagnetic Fields (EMF), Radiofrequency Fields (RF) and Microwave Radiation on Human Health”.

The CSTEE is charged with identifying and discussing major new research initiatives that confirm findings of previous investigators related to genotoxicity of ELF-EMF. These studies should be included and appropriate conclusions drawn about the consistency and strength of this evidence for an association between ELF-EMF exposures at environmental levels and breast cancer.

The constellation of relevant scientific papers providing mutually-reinforcing evidence for an association between power-frequency electromagnetic fields (ELF-EMF) and breast cancer should be thoroughly and completely reported in the CSTEE report. ELF-EMF at environmental levels negatively affects the oncostatic effects of both melatonin and tamoxifen on human breast cancer cells. Numerous epidemiological studies over the last two decades have reported increased risk of male and female breast cancer with exposures to residential and occupational levels of ELF-EMF. Animal studies have reported increased mammary tumor size and incidence in association with ELF-EMF exposure.
Tamoxifen and ELF-EMF

Girgert et al (2005) reported that “the anti-estrogenic activity of tamoxifen is reduced in two subclones of MCF-7 cells under the influence of ELF/EMF to different extent. Dose-response curves of the growth-inhibitory effect of tamoxifen are shifted towards higher concentrations leading to a reduced growth inhibition at a given concentration. Our observations confirm results from a previous report describing a reduced inhibitory effect of tamoxifen at $1^{-7}$ M from 40% to only 17% by exposure to an EMF of 1.2 $\mu$T” (Harland et al, 1997). Further, Girgert et al conclude that “From a medical point of view, it is disturbing that maximal induction of cell proliferation by tamoxifen at a field strength of 1.2 $\mu$T is observed at concentration of $10^6$ M. This is exactly the serum concentration achieved in BC patients under standard oral therapy.” (De Cupis et al, 1997).

The Girgert et al paper confirms prior findings that environmental level ELF-EMF inhibits the antiproliferative action of tamoxifen in MCF-7 human breast cancer cells. Four other papers reporting this effect include Liburdy et al, 1997; Harland et al, 1997; Harland et al, 1999; and Blackman et al, 2001).

References


Melatonin and ELF-EMF

Evidence which supports a possible mechanism for ELF-EMF and breast cancer is the consistent finding (in five separate labs) that environmental levels of ELF-EMF can act at the cellular level to enhance breast cancer proliferation by blocking melatonin’s natural oncostatic action in MCF-7 cells (Liburdy, 1993; Luben et al, 1996; Morris et al, 1998; Blackman et al, 2001; Ishido, et al, 2001). ELF-EMF levels between 0.6 and 1.2 µT have been shown to consistently block the protective effects of melatonin.

The series of papers reporting increased breast cancer cell proliferation when ELF-EMF at environmental levels negatively affects the oncostatic actions of melatonin in MCF-7 cells should be reported; and the consequence of this important series of papers should be highlighted in the CSTEE,

References


Ishido et al, 2001. Magnetic fields (MF) of 50 Hz at 1.2 µT as well as 100 µT cause uncoupling of inhibitory pathways of adenylyl cyclase mediated by melatonin 1a receptor in MF-sensitive MCF-7 cells.


Animal Studies and ELF-EMF


Thun-Battersby, S., M. Mevissen, et al. (1999). Exposure of Sprague-Dawley rats to a 50 Hz, 100 uTesla magnetic field for 27 weeks facillitates mammary tumorigenesis in the

**Epidemiological Studies on Breast Cancer and ELF-EMF**

**Female Breast Cancer Studies**

**References**


**Male Breast Cancer Studies**

References


