Can we close the book on the issue of magnetic fields and breast cancer? No. This is the rational answer to a question possibly implied by the very large study by Forssén et al., which found “no evidence for an increased risk of breast cancer among women working in occupations with high magnetic field exposure” (1, p. 255). Indeed, that this—and prior studies—did not detect an increased risk does not readily imply that there is none.

Here is why: In studies of possible links between nonionizing radiation and breast cancer, exposure misclassification is of paramount concern. In their introduction, Forssén et al. (1) start out from the so-called melatonin hypothesis that suggests a biologically plausible and credible (2) mechanism for possible causal associations between exposures to extremely-low-frequency electric and/or magnetic fields and breast cancer in women via impaired pineal secretion of melatonin (3). A “lack of melatonin can reasonably be anticipated to be a human carcinogen” (4, p. 313), and “this hypothesis has thus far withstood 15 years of critical scrutiny and empirical research, inside the laboratory and out in the epidemiologic field” (5, p. 73). While it is to the credit of Forssén et al. that they improved assessment of women’s exposure to magnetic fields, it must be emphasized that the study’s underlying hypothesis implicates two distinct frequencies: extremely-low-frequency electric and/or magnetic fields and visible light. Of the two, light inhibits melatonin much more strongly and more reproducibly than magnetic fields may. It follows that light could be a much more likely culprit—at work and at home—than magnetic fields (6).

Intriguingly, findings from first studies of blind people and of shift workers are compatible with the notion that visible light may affect breast cancer risks; that is, risks may be reduced in the blind (7–11) and increased in female shift workers (12–15). Importantly, light could be a significant cause of breast cancer because the ubiquitous nature of visible radiation implies the possibility that even small risk elevations could lead to a substantial population burden. Moreover, since the possibly strong risk factor light was not controlled for, it remains conceivable that magnetic fields do increase breast cancer risks but that this effect has been assessed erroneously in many (or all) epidemiologic studies to date. To illustrate this crucial point with a provocative analogy: we would be prone to miss, underestimate, or overestimate possible effects of most known lung carcinogens if we did not control for differential smoking habits in study subjects (16).

A look at experimental work may be important here. In recent years, Thun-Battersby et al. (17) were able to substantiate earlier observations that magnetic field exposures enhance the development and growth of mammary tumors in the 7,12-dimethylbenz[a]anthracene (DMBA) model of breast cancer in female Sprague-Dawley rats. Experimental
work could also explain why a similar study conducted in the United States yielded different results, in particular because the substrains of rats used in the two laboratories differed in their susceptibility to DMBA-induced mammary cancers (18). Possibly, the control of light (indeed, all animals were exposed to the same light regime) may have allowed the experimental researchers to detect magnetic field effects that could be masked in the existing epidemiologic studies by uncontrolled light exposures.

It remains to be emphasized that if we decide to find out whether the melatonin hypothesis and associated predictions are valid, epidemiologic studies must collect information about magnetic fields and light (6). If we continue to rely on exposure assessments of magnetic fields alone, investigations shall not be sensibly interpretable and the important question for public health concerning whether magnetic fields and/or visible light may increase breast cancer risks will not be answered convincingly.

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REFERENCES