Minder and Pfluger Respond to “Electromagnetic Fields and Cancer in Railway Workers” by Savitz

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We are grateful for the opportunity to reply to Dr. David Savitz’s comments (1) on the findings of our study (2). Of necessity, we will concentrate on certain points, omitting others equally worthy of discussion.

Dr. Savitz outlines, with admirable clarity, an ideal epidemiologic study of the health effects of electromagnetic field (EMF) exposures. By such exacting standards, any real-life study is bound to fail, and the interesting question is how and to what extent. We believe that studies of railway personnel permit epidemiologists to get close to Dr. Savitz’s ideal: Railway engine drivers have stable and predictable exposures to extremely low frequency EMFs (≤100 Hz), other carcinogens are largely absent, and sufficient person-years are available for study. Perhaps comparability of occupations is least guaranteed.

Dr. Savitz questions the contribution of studies of 16⅔-Hz exposure to the larger question of population exposure. We think that studies of 16⅔-Hz exposure are relevant, because there is solid evidence that exposure to extremely low frequency EMFs causes resonance phenomena in cells, affecting calcium ion concentrations (3–6). Resonance may initiate a biologic pathway even with low energies. In addition, large populations are exposed to 50-Hz (namely, triple 16⅔-Hz) extremely low frequency EMFs from household currents. Resonance effects observed at 16⅔ Hz mean that resonance will occur with exposures at or near low multiples (60 Hz) of this frequency as well. This conclusion is strengthened by the elevated leukemia risks found at the 16⅔ Hz level as well as at 50 Hz and 60 Hz.

Dr. Savitz argues that the weak point of EMF research is the lack of a biologically plausible pathway. A review of the literature has convinced us otherwise. There is extensive evidence linking extremely low frequency EMF exposure to calcium ion concentrations (3–6), apoptosis (7), melatonin levels (8, 9), chromosome damage, and cancer (see discussion below). There is also evidence linking calcium ion balance to apoptosis (10), melatonin levels (11), chromosome damage, and cancer. For brevity, we shall restrict ourselves here to the evidence on chromosome damage, a precursor of cancer. Nordenson et al. (12), Valjus et al. (13), and Skyberg et al. (14) have all found elevated levels of chromosome damage among workers exposed to extremely low frequency EMFs. Nordenson et al. (15, 16), Rosenthal and Obe (17), Khalil and Qassem (18), and Garcia-Sagredo and Monteagudo (19) have found elevated levels of chromosome damage in human cell lines after exposure to extremely low frequency EMFs. El Nahas and Oraby (20) found an extremely low frequency EMF dose-dependent increase in micronuclei of mouse somatic cells.

This biologic evidence, combined with the ample epidemiologic evidence of dose-response relations between leukemia and extremely low frequency EMF exposure, seems to us to satisfy Hill’s criteria for causality (21). Thus, Dr. Savitz’s suggestion of “a possible modest association” (1, p. 838) could be misinterpreted. The risk ratios found for leukemia and extremely low frequency EMF exposure are similar in magnitude to those for lung cancer and passive smoking. Both of these associations may be modest in size; in our opinion, both are well established. The research cited above suggests that the association between extremely low frequency EMFs and leukemia may be causal. If this is the case, its public health impact will not be modest.

We agree with Dr. Savitz that “black box” epidemiology has made its contribution to solving this problem. We feel that now is the time to impartially assemble all of the available evidence on the biology and health effects of extremely low frequency EMF exposure. It would be fitting for the Society for Epidemiologic Research to initiate such an effort.

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