I read the obituary on Morton Levin by Drs. Haroutune K. Armenian and Moyses SzklO (1) with great interest and appreciation. Let me add a little more to our knowledge of the web of causation that led to the publication in 1950 of the two landmark papers on cigarette smoking and lung cancer in the Journal of the American Medical Association (JAMA) (2, 3). At the time the two papers were submitted to JAMA, Dr. Levin was working at the American Medical Association (AMA) offices in Chicago as Director of the Commission on Chronic Illness. This commission had been established by the AMA, the American Hospital Association, the American Public Health Association, and the American Public Welfare Association after the publication of their joint statement on "Planning for the Chronically Ill" (4, 5). Dr. Levin told me that the editor of JAMA explained to him that he could not publish the papers because they were "just statistical." In the course of the discussion, Dr. Levin was able to convince him to reverse his position and go ahead and publish the papers.

The web of causation started with Assistant Surgeon-General Joseph Mountin, chairman of the Subcommittee on Medical Care of the American Public Health Association, who took the leadership in initiating the joint statement; then the Commission on Chronic Illness was formed, and Dr. Levin moved to Chicago to work at AMA headquarters. This gave him the opportunity to convince the editor of JAMA to reverse his rejection of the papers.

REFERENCES

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Editor’s note: Drs. SzklO and Armenian thank Dr. Terris for supplying the above clarification of the "web of causation" that led to publication in 1950 of the two landmark papers on cigarette smoking and lung cancer in JAMA.

Recently, Davis et al. (1) examined the sex differences in high density lipoprotein (HDL) cholesterol in six countries. They do not discuss the possible importance of the dietary intake of saturated fat as an explanation of the observed sex differences.

In 1985, we noted (2) that the sex differences in serum HDL cholesterol levels commonly found in Western populations were absent in Oriental populations, such as in China and Korea. This was confirmed by an ecologic survey performed in China in 1983 in all 65 Chinese counties (3), which found no difference in HDL cholesterol levels between men and women (3). In a study of cardiovascular disease mortality in women (4), we offered as an explanation that women who consume a diet high in saturated fat increase their sex hormone levels, resulting in a higher HDL cholesterol level. This could explain the difference in HDL cholesterol levels, because Oriental women have a lower intake of saturated fat.

The level of serum HDL cholesterol is influenced independently by body mass index, smoking, alcohol intake, physical exercise, and dietary lipids (5). There are indications that alcohol increases and smoking decreases the HDL cholesterol levels to a greater extent in women (5), which confirms the data of Davis et al. (1). Reducing the level of saturated fat intake in women can reduce their HDL cholesterol levels to the levels observed in men (6). We further studied this problem and found that the female-to-male ratio of serum HDL cholesterol obtained from 21 countries increased significantly by the level of saturated and monounsaturated fat intake and decreased by the ratio of dietary saturated to polyunsaturated fat intake (P/S ratio) (7). In Singapore, which was not included in the analysis, a higher level of serum HDL cholesterol was found in women of Chinese origin compared with men. This difference was markedly higher than in other Oriental populations and was accompanied by higher total cholesterol levels (8), presumably due to a high intake of saturated fat (palm oil).

The data of Davis et al. (1) were age-adjusted, but age plays only a minor role as a determinant of the serum HDL cholesterol level (5). In our opinion, differences in the level of saturated fat intake offer at least a partial explanation of the sex difference in HDL cholesterol level between populations, and we therefore believe this possibility warrants further investigation.

REFERENCES
In a letter to the editor, Erren (1) pointed to five studies, three occupational and two residential, where exposure to electromagnetic fields of various types showed an association with lung cancer. Although there has been no hypothesis regarding a specific carcinogenic effect of electromagnetic field exposure on the lung, Erren pointed out that because lung cancer in the United States is epidemic, a link between electromagnetic field exposure and lung cancer would have considerable public health relevance.

Research at Bristol University has recently demonstrated (2) the ability of the electric field component of power frequency electromagnetic fields to attract and concentrate airborne radon progeny in their vicinity. Radioactive radon progeny atoms when formed rapidly attract water molecules in air growing into a so-called ultrafine aerosol around 10 nm in size. Depending on availability, such aerosols may then attach to larger aerosol particles up to 1 μm in size. The Bristol observations are therefore indicative of the behavior of aerosols in general, and they demonstrate that airborne chemical pollutant aerosols, bacteria, and viruses would be expected to be similarly concentrated by electric fields.

Two main physical processes govern these observations: the oscillation of electrically charged aerosols, which mainly affect the ultrafine aerosols and the movement up field gradients by the polarization force affecting mainly larger aerosols. These processes lead naturally to possible mechanisms of increased skin deposition, inhalation, and lung retention of aerosols as well as transport to all body organs. In the case of radon progeny aerosols, the dose to red bone marrow following inhalation has been specifically modeled (3). The possibility of increased lung cancer in relation to electromagnetic field exposure constitutes one prediction from the Bristol observations. Therefore, the finding of lung cancer in several studies suggests the possibility of a causal relation.

Most epidemiologic studies of electromagnetic field exposure have concentrated on magnetic field exposure because, unlike electric fields, they readily penetrate the human body. The Bristol findings, however, suggest the importance of electric fields. Miller et al. (4) reported a nonsignificant odds ratios for leukemia incidence of 1.6 in Ontario electric utility workers exposed to magnetic fields, but this increased to 11.2 (95 percent confidence interval 1.3–97.2) when combined exposure to electric and magnetic fields was considered. Furthermore, there was a suggestion of a dose-response curve in relation to electric field exposure as well as a suggestion of increased risk of melanoma. As has been pointed out elsewhere (5), the leukemia risks are some of the highest ever reported in a major epidemiologic study. Miller et al. (4) conclude that their analysis shows associations for all leukemia and its subtypes with increasing electric field exposure, with a dominant effect of electric field exposure on leukemia when both electric and magnetic field exposures are considered together.

The findings of Miller et al. therefore represent a further epidemiologic test of an electric field/aerosol interaction. This strengthens the possibility that the findings are causal. The above considerations suggest that an urgent assessment of electric field exposure in both previous and future epidemiologic studies of the possible health effects of electromagnetic field exposure is warranted.

REFERENCES


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Editor’s note: In accordance with Journal policy, Dr. Erren was given the opportunity to reply to the above letter, but he chose not to do so.