(range, 1.38–1.63). During the same period, the annual age-adjusted mortality rates were, on average, 1.99-fold higher among Black men than among White men (range, 1.82–2.15). The twofold increase in the death rate among Black men compared with White men is only partially explained by the higher incidence among Black men. On the basis of available evidence, it seems that the additional increase in the mortality rate among Black men is the result of their poorer survival because of a combination of later-stage diagnosis for Blacks and differences in treatment between Blacks and Whites (5, 6). Racial differences in stage at diagnosis and treatments are likely to stem from cultural and socioeconomic differences between the two ethnic groups (6, 7).

Similarly, men in typical high-magnetic-field-exposure occupations (e.g., linemen) could also systematically differ in many social, economic, and cultural ways from other, unexposed workers. If any of those characteristics is associated with survival, the observed difference in prostate cancer mortality between exposed and unexposed workers may not reflect real differences in prostate cancer incidence rates.

Although the study (1) was well designed and executed, and the results were clearly presented, the value and interpretation of the results are questionable. Because of the use of mortality data, these results provide no reliable support for a possible etiologic relation between magnetic field exposure and prostate cancer development. Interpretation of the results becomes even more problematic when we consider the lack of convincing laboratory evidence in support of a relation between magnetic field exposure and carcinogenesis in general, and prostate cancer development in particular (8).

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THE FIRST TWO AUTHORS REPLY

In his letter to the editor, Dr. Mezei (1) raises an important issue regarding a limitation of our study (2), which investigated risk factors for prostate cancer mortality rather than incidence. Although the distinction between cancer incidence and mortality is likely to be familiar to most readers of the *Journal*, we acknowledge that prostate cancer mortality is affected by various factors, including the ones cited by Dr. Mezei, and agree that a short description of the limitations of mortality data in our study would have been helpful to some readers.

However, we disagree with his assertion that "the value and interpretation of the results are questionable" (1, p. 929) because the study was based on mortality data. For differential survival to confound associations of prostate cancer with exposure to magnetic fields, substantial differences in prostate cancer survival would have to be associated with cumulative exposure. However, our analyses included only those workers employed by five large companies within a single industry. Large differences in culture and socioeconomic position are less likely to confound associations in such internal comparisons than in community-based studies that sample from the population at large. Moreover, access to medical care is likely to be relatively uniform within a cohort of workers from the same industry.

Undoubtedly, additional studies designed to control for biases from several sources would have to be conducted before it can be concluded that electromagnetic field exposure is an etiologic agent for prostate cancer development. Our study (2) makes an important contribution to the literature on prostate cancer mortality, and it adds another layer to the foundation of studies from which epidemiologists and laboratory scientists can build to further investigate whether a causal relation exists between exposure to electromagnetic fields and development of prostate cancer.

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