Magnetic Field Exposure and Cardiovascular Disease Mortality among Electric Utility Workers

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Laboratory studies suggest that electric and magnetic field exposure may affect heart rate and heart rate variability. Epidemiologic evidence indicates that depressed heart rate variability is associated with reduced survival from coronary heart disease as well as increased risk of developing coronary heart disease. The authors examined mortality from cardiovascular disease in relation to occupational magnetic field exposure among a cohort of 138,903 male electric utility workers from five US companies over the period 1950–1988. Cardiovascular disease deaths were categorized as arrhythmia related (n = 212), acute myocardial infarction (n = 4,238), atherosclerosis (n = 142), or chronic coronary heart disease (n = 2,210). Exposure was classified by duration of work in jobs with elevated magnetic field exposure and indices of cumulative magnetic field exposure. Adjusting for age, year, race, social class, and active work status, longer duration in jobs with elevated magnetic field exposure was associated with increased risk of death from arrhythmia-related conditions and acute myocardial infarction. Indices of magnetic field exposure were consistently related to mortality from arrhythmia and acute myocardial infarction, with mortality rate ratios of 1.5–3.3 in the uppermost categories. No gradients in risk were found for atherosclerosis or for chronic coronary heart disease. These data suggest a possible association between occupational magnetic fields and arrhythmia-related heart disease. Am J Epidemiol 1999;149:135–42.

Arrhythmia; electromagnetic fields; heart rate

Research on potential adverse health effects of electric and magnetic field exposure has focused on cancer in both children and adults (1, 2) and secondarily on reproductive health (3) and neurobehavioral effects (4). However, despite a substantial amount of epidemiologic research concerning cancer, in particular, results are inconclusive and experimental evidence of biologic effects relevant to carcinogenesis is quite limited (2).

Concern with potential effects of electrical work on cardiovascular disease dates back to the mid-1960s, when reports from the Soviet Union attributed a range of symptoms to high electric field exposure among comparably exposed workers in Sweden (6) and Italy (7) found no indications of clinical cardiovascular abnormalities. Cohort studies of electric utility workers have found decreased mortality in the entire cohort (8–10) and among subsets of workers with elevated levels of electric and magnetic field exposure (9). There have been no analyses of subtypes of cardiovascular disease, presumably because of the absence of a rationale for suggesting that some forms would be likely to be affected by exposure to electric or magnetic fields.

Among the array of reported adverse health effects were changes in blood pressure and heart rhythm, though there is little documentation of research methods in these reports. More rigorous evaluations of comparably exposed workers in Sweden (6) and Italy (7) found no indications of clinical cardiovascular abnormalities. Cohort studies of electric utility workers have found decreased mortality in the entire cohort (8–10) and among subsets of workers with elevated levels of electric and magnetic field exposure (9). There have been no analyses of subtypes of cardiovascular disease, presumably because of the absence of a rationale for suggesting that some forms would be likely to be affected by exposure to electric or magnetic fields.

A hypothesis that exposure to power-frequency magnetic fields may influence specific subtypes of cardiovascular disease arises from an evaluation of acute cardiac effects of magnetic fields in human volunteers. In recent double-blind laboratory investigations (11), exposure to 20 μT of intermittent 60-Hz magnetic fields was found to reversibly reduce the normal periodicities of heart rate. Normally, healthy hearts do not beat with metronomic regularity but, as a result of interaction between sympathetic and parasympathetic activity, the beat-to-beat heart rate shows predictable periodicities over time.
In the past two decades, beat-to-beat variability (heart rate variability) has come to be recognized as a useful noninvasive method to quantitatively assess cardiac autonomic activity (12–14). Reductions in specific components of heart rate variability predict the development of heart disease over the subsequent several years in several large prospective cohort studies (15–18). Reduced heart rate variability is also associated with increased risk of all-cause mortality in myocardial infarction survivors (19–23) and with sudden cardiac death (24).

On the basis of these findings, we hypothesized that long-term exposure to magnetic fields might be associated with increased cardiovascular disease mortality, partially through the reduction of cardiac autonomic control. Under this hypothesis, one subgroup of particular interest is arrhythmia-related deaths. There is also clinical and physiologic evidence that cardiac autonomic imbalance may increase risk of acute myocardial infarction through increased heart rate (25), increased coronary vasoconstriction (26, 27), and increased shear stress leading to plaque fissure (28). In contrast, deaths from chronic coronary heart disease and atherosclerosis are the culmination of processes that develop over extended periods of time and are unlikely to be affected to the same extent by poor cardiac autonomic control.

To our knowledge, no epidemiologic studies have considered the potential cardiovascular effects of chronic exposure to elevated levels of electric and magnetic fields in detail. Data collected for a study of leukemia and brain cancer among electric utility workers provide information for addressing cardiovascular disease mortality in relation to indices of magnetic field exposure.

MATERIALS AND METHODS

We have reported detailed methods of the utility worker mortality study elsewhere (8, 29–31) and summarize them here. We conducted a cohort mortality study of men at five large US electric utility companies who worked for 6 months or more between 1950 and 1986, excluding women because they rarely received elevated exposures to electric and magnetic fields in the past. We excluded men with missing data and those who worked exclusively in the nuclear division, leaving 138,903 workers.

We organized job histories into occupational categories (29) for assignment of magnetic field exposure. A random sample of current workers, weighted by person-years and estimated exposure level, was selected for measurement of a work shift time-weighted average magnetic field exposure (30). Using the 2,842 complete measurements, we calculated arithmetic means for each of the occupational categories at each of the participating companies and then rank-ordered and grouped occupational categories to assign scores for the analysis (31). Expert panels at each company estimated exposure to workplace chemicals, particularly solvents and polychlorinated biphenyls, for the occupational categories.

Workers who left employment before the end of the study period were followed for mortality through 1988 using multiple tracking sources (8). We used the National Death Index for the period after 1979 and Social Security Administration records, Health Care Financing Administration data, credit bureau searches, drivers license records, state vital records searchers, and telephone tracing for earlier time periods. Of the 76,934 men who were no longer actively employed at the end of the study, we could determine vital status on all but 778. We obtained death certificates for 20,068 of the 20,733 deceased men.

Data analysis methods, also described in detail elsewhere (8), began with generation of standardized mortality ratios, comparing overall cardiovascular disease mortality of the cohort with that of the general population, using the National Institute for Occupational Safety and Health Life Table Analysis System (32). Then, we conducted analyses using internal referent groups and Poisson regression (33, 34) in which we modeled the mortality rate as a function of estimated exposure to magnetic fields, as well as covariates that included age, calendar decade, race, social class (based on job at hire, divided into upper white collar, lower white collar, skilled blue collar, and unskilled blue collar), and work status (active or inactive, with a 2-year lag). We controlled for work status in an effort to address the healthy worker survivor effect (35).

The primary analysis compared mortality among members of the cohort with differing histories of exposure to magnetic fields. We considered duration of employment in any of 18 occupational categories with greater than background exposure and duration of employment specifically as a lineman, electrician, or power plant operator, commonly held jobs with estimated mean magnetic field exposures of 0.65, 1.11, and 0.79 μT, respectively. For all analyses of exposure duration, the referent category consisted of person-years in occupational categories with no employment in jobs above background exposure. Intervals from >0 to <10, from 10 to <20, and from ≥20 years of employment were considered for the aggregation of exposed jobs and individual exposed jobs. We collapsed from the highest exposure categories downward to obtain at least four cases in each cell.

We also examined cumulative magnetic field exposure, accounting for job changes over time, and aggre-
gated across the total work history or selected time windows using units of “μT-years.” We considered several time windows of magnetic field exposure, including total career exposure with no lag, and exposure with lags of 5, 10, and 20 years. Categories for each of these indices were chosen to ensure adequately large numbers of events in the referent category and maximum separation in the upper end of the distribution. For total exposure with no lag and the 5- and 10-year lags, the referent category was below the 30th percentile, with intervals of 30–<50, 50–<70, 70–<90, and ≥90 percentiles. For the 20-year lag, the referent category was the absence of occupational exposure, with intervals of >0–<30, 30–<60, 60–<80, and ≥80 percentiles. In addition, we modeled exposure deciles as a continuous variable to estimate the rate ratio per μT-year, assuming a log-linear relation between exposure and mortality and fixing the lowest decile score as the intercept. For examining the effect of exposure in the previous 5 years (0- to 5-year window), we adopted a different approach since most men who died from cardiovascular disease were not employed in the 5 years prior to their death. We considered “not employed in the interval” as the referent category and compared mortality rates associated with work exclusively in low exposure jobs (those associated with measurements of <0.5 μT (31)) during the interval and rates associated with any work in high exposure jobs (≥0.5 μT) during the interval.

Spline regression was used to characterize the patterns of mortality in relation to magnetic field exposure, using the results with a 10-year lag. We conducted Poisson regression with cubic splines. Knots were placed at the 25th, 50th, and 75th percentiles of the exposure distribution for all deaths, corresponding to 0.27, 0.87, and 1.94 μT-years, and the plot was truncated at 2.5 μT-years, where the data become sparse. We classified the primary cause of death in the utility workers’ database according to the *International Classification of Diseases*, Ninth Revision (ICD-9) (36) and Eighth Revision (ICD-8) (37), separately, since one of the companies had previously conducted a cohort analysis using ICD-8. We then combined the cause of death from the two versions of the *International Classification of Diseases* in the analysis. The following four categories of cardiovascular death were developed to address the hypothesis of a potential effect of magnetic fields on heart rate variability: 1) arrhythmia related (ICD-8 code 427, ICD-9 code 426 or 427); 2) acute myocardial infarction (ICD-8 code 410, ICD-9 code 410); 3) atherosclerosis-related mortality (ICD-8 code 440, ICD-9 code 440); and 4) chronic/subchronic coronary heart disease (ICD-8 codes 411–413, ICD-9 codes 411–414). The first two groups were thought to be most likely reflective of changes in cardiac autonomic control, and the latter two groups were thought to be unlikely to be affected. We identified a total of 212 deaths that were arrhythmia related (184 from ICD-9 and 28 from ICD-8 codes), 4,238 deaths from acute myocardial infarction (3,381 from ICD-9 and 857 from ICD-8 codes), 142 deaths from atherosclerosis (127 from ICD-9 and 15 from ICD-8 codes), and 2,210 deaths from chronic coronary heart disease (1,986 from ICD-9 and 224 from ICD-8 codes).

### RESULTS

Overall, cardiovascular disease mortality was lower in the utility worker cohort compared with the US population for heart disease in total (standardized mortality ratio (SMR) = 0.74, 95 percent confidence interval (CI) 0.74–0.78) and for ischemic heart disease (SMR = 0.77, 95 percent CI 0.76–0.79) as is often found among employed cohorts. There was a small gradient of increasing risks going from lower to higher social class (SMRs ranging from 0.65 to 0.80 across levels). An anomalously high risk was found for nonwhite workers for ischemic heart disease (SMR = 1.74, 95 percent CI 1.64–1.84), also noted for total mortality and most other causes of death (8), which suggests an artifact of the assignment of race or the ethnicity of nonwhite workers in the cohort compared with those in the US population.

Duration of employment in exposed occupations in the aggregate was associated with increased rates of death from arrhythmia-related conditions and acute myocardial infarction (rate ratios of 1.4–1.5 in the longest employment intervals) (table 1). The gradient was stronger for electricians, and particularly power plant operators, and slightly weaker for linemen. In contrast, atherosclerotic conditions tended to show an inverse gradient by duration of employment, inconsistently across job categories. Chronic coronary heart disease was not associated with duration of employment in individual or aggregated exposed occupations.

The evaluation of cardiovascular disease mortality in relation to quantitative indices of magnetic fields generally corroborated this pattern (table 2). A dose-response gradient was observed for cumulative career magnetic field exposure for both arrhythmia-related deaths and deaths from acute myocardial infarction, with rate ratios in the highest category of 2.4 and 1.6, respectively. Risk generally rose across levels of exposure. A weak inverse gradient was again found for atherosclerotic conditions, with no association found for chronic coronary heart disease.

Restricting exposure to the 5 years prior to observation (a moving window) indicated reduced mortality.
rates for arrhythmia and atherosclerosis associated with employment and a slightly increased risk for acute myocardial infarction among employed men (table 2). Only for arrhythmia-related conditions was there evidence that employment in higher exposure jobs in the interval was associated with greater risk than employment in lower exposure jobs (rate ratio of 0.94 vs. 0.52), but the risk estimates for the high exposure group were imprecise.

With a lag of 20 years, which addresses a potential latency between accumulation of exposure and risk, the magnitude of association for arrhythmia was slightly greater than for the analysis with no lag period in the categorical analysis, but there was little effect of lagging exposure on results for acute myocardial infarction or chronic coronary heart disease (table 2). The inverse gradient in risk for atherosclerosis was eliminated with a 20-year lag. Risk based on continuous dose measures showed positive gradients but predicted lesser elevations in risk for the highest category than those observed in the categorical analysis. Spline regression for magnetic field exposure with a 10-year lag (figure 1) suggested that there was an increase in risk in the low end of the exposure range for arrhythmia and acute myocardial infarction, with little effect as exposure increased. The lack of more marked elevation in risk in the upper end of the exposure distribution explains why the rate ratio based on the continuous dose measure was only modestly elevated relative to the categorical analysis. Atherosclerosis was associated with a reduction in risk in the low exposure range, with a leveling off thereafter, and chronic coronary heart disease showed a rise and fall in risk in the low exposure range.

We examined the potential for confounding associated with specific company of employment (not shown), under the supposition that employees of different companies may have distinctive lifestyle or other factors related to risk of cardiovascular disease. The results for the association between magnetic field exposure with a 10-year lag adjusted for company showed some modest fluctuations. The results for arrhythmia-related conditions changed most notably, with reduced rate ratios in the two highest exposure groups; the rate ratio in the highest interval was reduced from 2.3 to 2.0. Results for acute myocardial infarction were essentially unchanged, those for atherosclerosis were more strongly inversely related to exposure, and a small elevation in the highest exposure group was found for chronic coronary heart disease (rate ratio of 1.2).

**DISCUSSION**

These results suggest an association between employment in jobs in the electric utility industry asso-
cated with elevated magnetic field exposure and mortality from arrhythmia-related causes and acute myocardial infarction. For both of these categories of cause of death, the rate ratios were elevated in relation to duration of employment in jobs with elevated exposure and in relation to indices of magnetic field exposure estimated for the entire work history. Lagging exposure had little effect, except for the 20-year lag that strengthened the association with arrhythmia-related deaths. Rate ratios rose, usually monotonically, to reach levels of 1.5–2.0 and higher in the uppermost categories, with good precision except for the evaluation of individual exposed occupations. The consistency of risks found for continuous dose measures and for categorical analyses and the large numbers of events strongly suggest that the pattern we have observed is not a product of random error.

The specificity of these associations is consistent with our hypothesis, with no clear pattern found for mortality from atherosclerotic conditions or chronic coronary heart disease. Given the diverse and subtle selection operating in occupational cohorts, particularly for cardiovascular disease (35, 38, 39), a gradient in risk related to changes in jobs resulting from subclinical cardiovascular disease is plausible. However, such health-related job changes might be expected to be strongest for chronic heart conditions, culminating in death from atherosclerosis or chronic coronary heart disease. Failure to observe gradients of increasing risk with increasing exposure for those categories of death that would not be expected to be associated with altered autonomic balance and cardiac conduction argues against health-related job changes as the basis for observed associations with arrhythmia and acute myocardial infarction.

The temporal pattern of the association, absent for the very recent exposures and consistent for career exposure and exposure lagged up to 20 years, could reflect an etiologic process with a delay between early exposure and eventual fatal outcomes. The temporal

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<th>10-year lag</th>
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* RR, rate ratio, adjusted for age, calendar year, race, social class, and work status (active/inactive); CI, confidence interval.

**TABLE 2.** Cardiovascular disease mortality in relation to magnetic field exposure, US utility worker mortality study, 1950–1988

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course of alterations in exposure, disease, and mortality from cardiovascular disease is not readily predicted even for established causal factors, such as hypertension or tobacco, and less so for an unproved potential contributor. The observed pattern may also be a result of subtle biases related to the healthy worker effect or random error. Complicating the interpretation of latency is the occurrence of 70 percent of the cardiovascular disease deaths at age 60 years or above, most of which presumably occurred among many men who were no longer employed.

Epidemiologic evidence pertinent to these results is severely limited. Previous cohort studies of utility workers have generally treated cardiovascular disease in the aggregate, noting that mortality is reduced compared with that in the general population (8–10). Some have tabulated results in relation to exposure to magnetic or electric fields (9) or in relation to occupational category (10). In the study of Southern California Edison workers (10), increased rates of major cardiovascular disease deaths were noted for all groups likely to have electric or magnetic field exposure (rate ratios of 1.3–1.8) relative to an aggregation of administrative, technical, and clerical occupations. Given the disparate patterns we observed across types of cardiovascular disease, comparable analyses of previously studied cohorts would be of interest.

Limitations in the study must be considered, including ascertainment of exposure, disease, and potential confounders. Though we based exposure assignment on expert insight into job activities and exposures in the electric utility industry, as well as extensive empirical surveys of worker exposures (30, 31), the assigned exposure values are likely to include substantial error. We focused on chronic magnetic field exposure, and neither the jobs nor the indices of exposure would necessarily reflect other, correlated exposures such as peak magnetic fields or any aspects of electric field exposure. For example, some electric utility workers are exposed occasionally to nuisance shocks and other currents. Nuisance shocks in the form of spark discharges can occur in high electric fields, and contact currents below perception may be associated with both electric and magnetic fields. However, no data are presently available to address such alternative etiologic hypotheses. Chemical hazards may also be encountered in the electric utility work environment, though none are presently known or strongly suspected to be associated with cardiovascular disease.

The ability to examine and control for confounding by factors other than age was quite limited. Lifestyle factors known to be associated with cardiovascular disease were not measured in this study and might act as confounders. However, cigarette smoking is unlikely to do so, given evidence that work in exposed jobs and indices of power-frequency magnetic field exposure are not associated with lung cancer mortality (40), for which the relative risks from smoking are much larger than those for cardiovascular disease. Diet, physical activity, blood pressure, and lipids all are unmeasured covariates, but the failure to find associations for chronic coronary heart disease and atherosclerosis calls into question whether these personal characteristics are likely to have introduced positive confounding exclusively for arrhythmia-related conditions and acute myocardial infarction.

Confounding associated with active employment (associated with reduced mortality and continued accumulation of exposure) was addressed by control-
ling for active work status (35). Active work status showed the expected relations with reduced mortality from cardiovascular disease, with rate ratios in the range of 0.4–0.7 for arrhythmia-related deaths, atherosclerosis, and chronic coronary heart disease, but a weaker association (rate ratio around 0.9) for acute myocardial infarction, presumably reflecting the lack of work-ending symptoms in the interval prior to the occurrence of fatal acute myocardial infarction. Adjustment for work status increased rate ratios slightly for the association between magnetic field exposure and arrhythmia-related deaths but had no discernible effect on the other categories of cardiovascular disease.

Our ability to accurately constitute subgroups of cases that are more and less likely to be affected by disorders associated with altered heart rate variability was limited by the information available on the death certificates. Error may be introduced in the assignment of cause of death and in the grouping of causes of death for analysis. The overall validity of assigning coronary heart disease on death certificates compared with assignment based on medical record review by physicians is high (41). The grouping of deaths is more probabilistic, with some of the deaths assigned as “arrhythmia related” not determined by cardiac conduction abnormalities and some assigned as “chronic coronary heart disease” or “atherosclerosis” affected in that manner. Coding in the Ninth Revision of the International Classification of Diseases (36), which was used for 83 percent of all cardiovascular disease deaths, is more suitable for this purpose than coding in the Eighth Revision (37). Unless there is an unusual pattern of error across the categories of cardiovascular disease deaths we examined, this problem would be predicted to dilute associations across categories, assuming that a subset of deaths are truly related to magnetic field exposure and others are not.

Although the motivation for this analysis came from observations in human laboratory studies of magnetic field exposure and heart rate variability (11), it is not possible to extrapolate from the acute, reversible, short-term effects on heart rate variability observed in laboratory settings to arrhythmia-related deaths or deaths from acute myocardial infarction that may result from prolonged exposure to elevated levels of electric or magnetic fields. The mechanisms that could link cumulative electric and magnetic field exposure with mortality 5–20 years later are highly speculative. Research to extend the experimental work to examine in more detail chronic exposure and endpoints of more direct clinical relevance is encouraged. Simultaneously, the study of acute cardiovascular responses (e.g., heart rate variability, blood pressure) to electric and magnetic fields in the laboratory should be continued and extended to the workplace through monitoring of cardiovascular variables and exposures in field settings. Opportunities to examine more specific cardiovascular health events, from changes in heart rate variability to nonfatal clinical outcomes, should be pursued to determine whether these findings are valid and, if they are, whether the hypothesized mechanisms involving heart rate variability are the relevant ones.

ACKNOWLEDGMENTS

This study was supported by contract RP-2964-05 from the Electric Power Research Institute (EPRI), Palo Alto, California.

The authors acknowledge the substantial contribution of the following people to the conduct and analysis of the study: University of North Carolina colleagues Dana Loomis, Michael Flynn, Lawrence Kupper, Stephen Rappaport, and Lori Todd; Hans Kromhout of Wageningen Agricultural University in the Netherlands; research assistants Stephen Browning, Kevin Chen, Gary Mihlan, Lucy Peipins, and Sandy West; computer programmers Richard Howard, Eileen Gregory, and Joy Wood; EPRI project officers Robert Black, Leeka Kheifets, and Kristie Ebi; EPRI scientific advisors A. A. Afifi, Patricia Buffler, James Quackenboss, T. Dan Bracken, Gary Marsh, and Thomas Smith; collaborating contractors J. Michael Silva and Richard Iriye of Enertech Consultants; William Kaune of EM Factors; Margaret Pennybacker of Battelle-Survey Research Associates; Judy Rayner of Westat, Inc., and William West; and Charles Graham and Mary R. Cook of Midwest Research Institute. In addition to those persons mentioned individually, a large number of electric utility employees from Carolina Power and Light, Pacific Gas and Electric, PECO Energy Company (formerly Philadelphia Electric Company), Tennessee Valley Authority, and Virginia Electric Power Company devoted a substantial amount of time assisting the authors with many aspects of the study, lending their expertise, time, and patience, for which they are most appreciative.

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