Electromagnetic field exposures and childhood leukaemia in New Zealand

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A nationwide case-control study of childhood leukaemia in New Zealand included measurements of electric and magnetic fields in children's homes. There was no significant association between leukaemia and the time-weighted average of the 50 Hz magnetic or electric fields in the bedroom and living (or daytime) room combined.

The controversy about whether childhood cancers are linked to electromagnetic field exposures has lasted for 20 years. Recent childhood leukaemia case-control studies have involved measurements of electromagnetic fields.[1–5] Two of these were large studies.[1,4] A nationwide New Zealand study was reported last year.[3] This communication arises from a suggestion for further analysis of the New Zealand data (personal communication from R Doll, Sept 9, 1998), to facilitate comparisons between studies.

The New Zealand study was population-based with comprehensive case ascertainment.[3] Controls were selected from birth records and matched individually to cases by age and sex. Information was collected by maternal questionnaires and measurements in the home occupied at the time of interview. Maternal interviews were available for 121 of the 131 eligible children with leukaemia (92%). The mothers of 89 of the 121 matched first-choice controls gave interviews (74%); replacement controls were found for the other 32. Electric and magnetic fields (50 Hz) were measured in the child's bedroom and living room, with positron dosimeters recording every 60 s for 24 h in each room. Measurements were completed for 115 of 121 interviewed leukaemia cases and 117 of their 121 matched controls.[3] Results of case-control analyses for the arithmetic mean magnetic and electric fields were presented for the bedroom and living room separately.[3]

Further analysis involved combining measurements from the two rooms into a time-weighted average, to estimate overall residential exposure. The measurement data were weighted using the questionnaire data on the lengths of time the child spent in the bedroom and living room. When a child had missing data for "length of time spent in the room" (about 77% of the total for each room), we used the relevant average value from the combined case and control sample. The main magnetic field results are given for categories defined by the a priori cut-points of 0·1 and 0·2 μT. Few children were in the category ⩾0·2 μT, so additional analyses were done with the data divided into thirds based on the distribution among the controls (lowest, middle, and highest). In the absence of prior information about appropriate cut-points for the electric field data, we categorised those data into thirds based on the distribution among the controls. Two sets of analyses were conducted, for all available case-control pairs with measurement data (113 pairs), and for those where both members of the pair had been resident in the house 2 years before the diagnosis or reference date (40 pairs). The measurements for the latter group are more likely to represent past exposures. While this approach leads to the exclusion of some children because they were under 2 years of age on the reference date, only the results for these 40 pairs are tabulated. We adjusted for the same confounders as before,[3] but there could be residual confounding because we had small numbers.

The analyses of all 113 matched pairs (children in any house with measurements at the time of interview) showed no association with magnetic field exposure of ⩾0·2 μT versus <0·1 μT (adjusted OR 1·4, 95% CI 0·3–6·3). The adjusted results for the 40 matched pairs who were resident in the same house 2 years before the reference date (table) showed a raised, but non-significant, OR of 3·3 for the highest category of the measured arithmetic mean magnetic field. This was lower than the ORs we reported earlier for the bedroom and daytime room separately.[3] The CI was wide, because few children were in the highest exposure category.

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Details of exposure	Categories	No of cases	No of controls	Unadjusted OR (CI)†	Adjusted OR‡ (CI)
Magnetic field, arithmetic mean (μT)	<0·1	31	33	1·0	1·0
	0·1–<0·2	4	5	0·9 (0·2–3·3)	1·5 (0·3–7·2)
	⩾0·2	5	2	2·5 (0·5–12·8)	3·3 (0·5–23·7)
Lowest third*	<0·0214	13	13	1·0	1·0
Middle third	0·0214 to <0·0590	12	18	0·7 (0·2–1·9)	0·6 (0·2–2·1)
Highest third	⩾0·0590	15	9	1·6 (0·6–4·9)	1·5 (0·4–5·7)
Electric field, arithmetic mean (V/m)					
Lowest third*	<5·05	10	13	1·0	1·0
Middle third	5·05 to <14·00	12	11	1·4 (0·4–4·3)	2·0 (0·4–10·1)
Highest third	⩾14·00	18	16	1·4 (0·5–3·6)	1·3 (0·2–6·7)

*The splitting into thirds was done on the basis of the combined bedroom and daytime room data for all the available controls (the total number of controls was 117, including those whose matched cases had no dosimetry). †CI=95% confidence interval. ‡The following confounders were included in the conditional logistic regression models,and these were determined using the full sample of 113 matched pairs. For magnetic field arithmetic means: mother's education, mother's smoking in pregnancy, and residence of the child on a farm. For electric field arithmetic means: mother's education and home ownership status, mother's smoking in pregnancy, household crowding, and residence of the child on a farm.

Childhood leukaemias and the time-weighted average of residential 24-h electric and magnetic field measurements in the bedroom and living room (overall measurement values combining both rooms). Restricted to case-control pairs where each child lived in the same house as that monitored 2 years before the reference date

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4 McBride ML, Gallagher RP, Thériault G, et al. Power-frequency electric and magnetic fields and risk of childhood leukemia in Canada. *Am J Epidemiol* 1999; **149:** 831–42.
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Diabetes as a risk factor for sudden death

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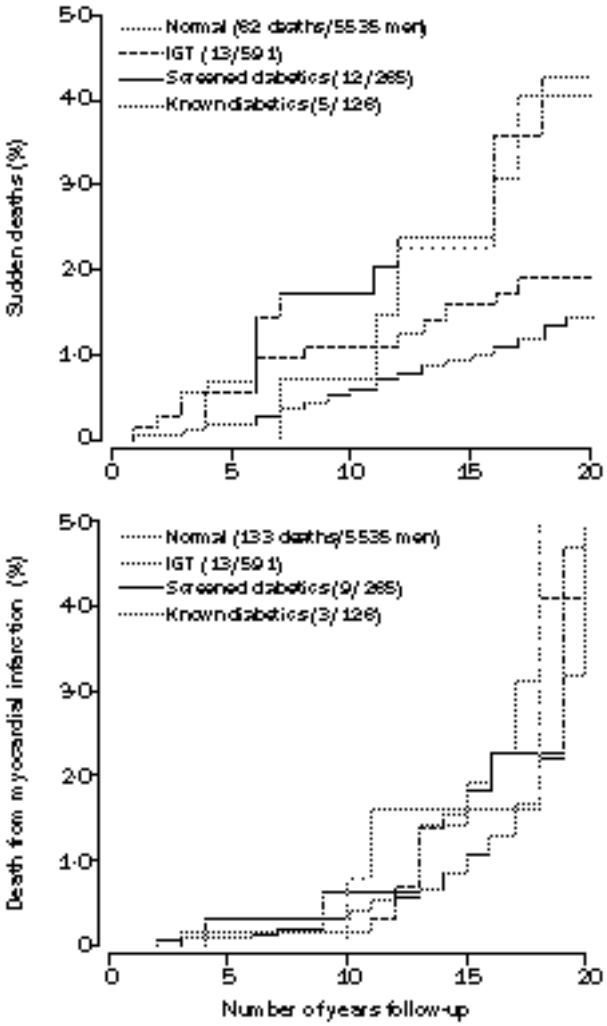
Although a family history of sudden death was a risk factor for sudden death in the Paris Prospective Study I, diabetes was also a strong risk factor, with a similar risk after accounting for other cardiovascular risk factors. Diabetes, however, was not a risk factor for death by myocardial infarction.

The Paris Prospective Study I reported that parental sudden death was a predisposing factor for sudden death in the middle-aged men.[1] In this analysis, a number of other cardiovascular risk factors were reported, including diabetes. In fact diabetes carried a hazard ratio of 3·23 (95% CI: 1·64–6·37) for sudden death, which was reduced to 2·21 (1·10–4·44) after adjustment for other cardiovascular risk factors; in contrast there was no significant association between diabetes and death from myocardial infarction (hazard ratios of 1·47 [0·69–3·12]) and 1·18 [0·55–2·52]) before and after adjustment for other risk factors.

We analysed the relationship between diabetes and risk of sudden death, and death from myocardial infarction. In the study a diagnosis of diabetes was based on a self-completed questionnaire. At the first follow-up examination, participants who were not diabetic were screened for diabetes with a 75 g oral glucose-tolerance test and diabetic status of those who said they were diabetic was verified from their anti-diabetic treatment. The definition of diabetes we use includes both known and screened diabetic participants. In this analysis we use only those deaths occurring before the end of 1988, when the causes of death were obtained from enquiries to physicians, hospitals, and family. Sudden death



Mortality curves for sudden death and for death by myocardial infarction, according to glucose tolerance status. The Paris Prospective Study I

was defined as death within 1 h of onset of symptoms. Causes of death were coded according to the 8th revision of the International Classification of Disease by a medical panel: myocardial infarction codes 410 to 414; sudden death 795. The 6539 men with complete data had a follow-up for causes of death for an average of 17·5 years.

Of these 6539 men, 128 (2·0%) were confirmed to have diabetes by their treatment (men treated with insulin were excluded), 285 (4·4% were found to be diabetic by screening (fasting glucose ⩾7·0 mmol/L and/or 2-h glucose ⩾11·1 mmol/L) and 591 (9·0%) had impaired glucose tolerance (non-diabetic but 2 h glucose ⩾7·8 mmol/L), using the 1998 World Health Organization proposed recommendations for the definition of diabetes and impaired glucose tolerance[2] (table).

Characteristics	n (%)	Hazard ratios (95% CI)			
		Sudden death		Myocardial infarction	
		Not adjusted	Adjusted*	Not adjusted	Adjusted*
Diabetes					
Non diabetic	6126 (83·7%)	1	1	1	1
Screened or known	413 (6·3%)	2·91 (1·74–4·88)	1·82 (1·04–3·17)	1·45 (0·82–2·55)	0·96 (0·53–1·73)
Family history					
Myocardial infarction	446 (6·8%)	1·54 (0·83–2·88)	1·28 (0·68–2·43)	2·06 (1·29–3·29)	2·13 (1·33–3·43)
Sudden death	453 (6·9%)	1·97 (1·12–3·45)	1·80 (1·02–3·20)	0·73 (0·36–1·49)	0·63 (0·31–1·29)

*Adjusted for age, BMI, smoking, heart rate, systolic blood pressure, cholesterol, triglycerides, diabetes, family history of myocardial infarction and for sudden death.

Characteristics, n (%) of the 6539 44–55-year old men from the Paris Prospective Study I and hazard ratios (95% CI)