ORIGINAL ARTICLE

Risk of birth defects by parental occupational exposure to 50 Hz electromagnetic fields: a population based study

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Objectives: To study the risk of birth defects by parental occupational exposure to 50 Hz electromagnetic fields.
Methods: The Medical Birth Registry of Norway was linked with census data on parental occupation. An expert panel constructed a job exposure matrix of parental occupational exposure to 50 Hz magnetic fields. Exposure to magnetic fields was estimated by combining branch and occupation into one of three exposure levels: <4 hours, 4–24 hours, and >24 hours/week above approximately 0.1 μT. Risks of 24 categories of birth defects were compared across exposure levels. Out of all 1.6 million births in Norway in the period 1967–95, 836 475 and 1 290 298 births had information on maternal and paternal exposure, respectively. Analyses were based on tests for trend and were adjusted for parents' educational level, place of birth, maternal age, and year of birth.
Results: The total risk of birth defects was not associated with parental exposure. Maternal exposure was associated with increased risks of spina bifida (p=0.04) and clubfoot (p=0.04). A negative association was found for isolated cleft palate (p=0.01). Paternal exposure was associated with increased risks of anencephaly (p=0.01) and a category of "other defects" (p=0.02).
Conclusion: The present study gives an indication of an association between selected disorders of the central nervous system and parental exposure to 50 Hz magnetic fields. Given the crude exposure assessment, lack of comparable studies, and the high number of outcomes considered, the results should be interpreted with caution.

Several studies of possible effects of low frequency electromagnetic fields on human health have been carried out.[1] Most attention has been given to the occurrence of cancer, in particular leukaemia and cancers of the nervous system among children. Some smaller studies provide data on pregnancy outcomes.

Sources of exposure to magnetic fields are numerous. The hypothetical relevance of any source that provides fields of a particular wavelength makes a complete exposure assessment extremely difficult. Therefore, most studies focus on particular sources of exposure.

Studies concerning specific sources of exposure relative to fetal development and pregnancy outcome have included the use of electric blankets, heated waterbeds, power lines, video display terminals, and other occupational sources. Several of these studies were reviewed by Robert,[2] but present no convincing evidence that exposure to electromagnetic fields in pregnant women or their partners is associated with reproductive outcomes. However, the previous studies are small and would not have enough statistical power to detect effects on risks of specific birth defects. Animal models do not indicate that exposure to low frequency electromagnetic fields has serious reproductive effects.[3]

The present study is based on information on all births in Norway since 1967. By record linkage with data from recent population censuses, we obtained job titles of parents. An expert panel established a job exposure matrix by categorising job types into three levels of exposure to 50 Hz magnetic fields. The data provided sufficient power to study a range of specific types of birth defects.

Main messages

- The Medical Birth Registry of Norway provides a good oportunity to evaluate birth outcomes in workers' offspring.
- An increased risk of spina bifida and anencephalus were shown after mothers and fathers potential exposure to electromagnetic fields.
- Chemical exposure may be an alternative explanation for the increased risk found for spina bifida.

Policy implications

- Our findings have no support in the scientific literature, a possible biological mechanism still needs to be determined, and further research is needed.
- The reported incidence of spina bifida and anencephalus in Norway in 1998 was 5/10 000 and 2/10 000, respectively.

METHOD

The Medical Birth Registry of Norway comprises all Norwegian births with at least 16 weeks of gestation since 1967. Notification is compulsory, and is performed by midwives within the first week after birth. Any diagnosis available at that time should be reported. Ascertainment of serious visible birth defects such as spina bifida and cleft lip is probably as high as 80%. Some other conditions may have less complete reporting.[4] The 24 birth defects included in the analyses were anencephaly, spina bifida, hydrocephaly, other central nervous system defects, eye defects, facial defects, cardiac defects, circulation system defects, respiratory system defects, isolated cleft palate, total cleft lip, oesophageal defects, other gastrointestinal defects, genital system defects, urinary system defects, clubfoot, limb defects, hip dysplasia, gastroschisis, musculoskeletal

Abbreviations: ICD-8, eighth revision of the international classification of diseases

Table 1 Number of births among mothers and fathers at different levels of exposure in selected categories of demographic data in Norway 1967–95

	Mother's exposure			Father's exposure		
	<4 h	4–24 h	>24 h	<4 h	4–24 h	>24 h
Total births (n)	772790	58265	5420	1128986	114000	47312
Education (y, %):						
<10	13.2	22.1	38.0	19.0	24.3	29.6
10–12	52.0	54.7	52.8	50.8	61.8	59.6
>12	34.3	22.8	8.4	29.7	13.3	10.3
No education	0.005	0	0	0.01	0.02	0.04
Unknown	0.4	0.5	0.8	0.4	0.6	0.6
Place of birth (%):						
Oslo or Akershus	21.8	24.9	6.6	20.3	14.5	7.7
Hordaland	9.9	11.6	17.2	10.5	11.9	12.6
Sør-Trøndelag	6.4	4.7	3.8	6.1	5.1	5.0
Others	61.9	58.9	72.4	63.1	68.5	74.7
Age of mother (%):						
⩽20	5.2	8.4	12.2	6.5	8.8	9.9
21–25	28.8	37.2	41.1	31.1	36.6	36.1
26–30	36.4	35.0	29.0	35.4	33.1	31.7
31–35	21.5	15.0	12.8	19.2	15.4	15.7
>35	8.0	4.4	5.0	7.9	6.0	6.6
Year of birth (%):						
1967–75	28.5	40.8	42.0	39.0	40.8	43.8
1976–85	35.5	36.4	31.4	33.2	31.1	31.3
1986–95	36.0	22.8	26.6	27.8	28.1	24.9

system defects, skin/hair/nail defects, "other defects", Down's syndrome, and "other syndromes". Children could be registered with up to three different types of malformations. We did not consider multiple disorders as a separate category. However, when considering spina bifida and hydrocephalus, those with a recorded anencephalus were not included. Similarly, children with hydrocephalus combined with spina bifida were not counted as cases of spina bifida.

The Norwegian personal identification number is recorded for child and mother of all births in the registry. The identity of 92% of fathers is also recorded.

Norway's population censuses are based on self administered questionnaires with information on job title, branch of industry, education, and income. The most recent censuses were undertaken in 1970, 1980, and 1990. Information on occupation and industrial activities are coded according to the Nordic version of the international standard classification of occupations[5] and the international standard industrial classification of all economic activities.[6]

Census information on parents from the three most recent censuses were attached to all 1 688 263 birth records of the Medical Birth Registry from the period 1967–95 by record linkage with the national identification numbers. Information on occupation, branch of industry, education, and income for parents in a particular calendar year was obtained from information in the census that was closest in time. The 1990 census did, however, not include the whole Norwegian population, and 1980 information was used when 1990 information was missing (occupational information on 179 965 mothers and 231 929 fathers). In the analyses, we excluded births that lacked occupational information on both parents. To evaluate the effect of using data from the 1980 census for some births all the way up to 1995, we attempted separate analyses before and after 1986. Such analyses were only performed for outcomes where an indication of an exposure effect was found. These separate analyses would also account for possible bias introduced by the general availability of ultrasound screening after 1987.

We organised an expert panel to assess exposure to magnetic fields in relevant occupations with a practical modification of a method described by Flynn *et al*.[7] adapted to Norwegian conditions. The expert panel consisted of one occupational physician, one physicist, and two industrial hygienists, all with broad experience in measurements and exposure assessment related to occupational exposure to electromagnetic fields. Each of the members of the expert panel first made their own classification of combinations of branch of industry and occupation into one of three exposure levels measured by hours/week in a potential magnetic field above a background level. They then met and discussed their results. When the panel did not reach agreement on exposure category for a particular occupation, branch organisations were contacted to obtain more information. The background field was not measured, but was expected to be similar to office environment or homes, in Norway, approximately 0.1 μT.[8] The following exposure categories above background level were used: less than 4 hours/week, 4–24 hours/week, and above 24 hours/week. Occupations in the melting industry, welders, machinists, pilots, some occupations in textile industries, woodworking factories, working with electricity, glass, and ceramics were the main contributors to the highest exposure category. The resulting job exposure matrix with a specification of exposure categories for all job categories may be obtained from the authors. The number of children with exposure information available on either mother or father is presented by categories of demographic variables in table 1. When the mother or father were not registered with an occupation in the census closest to the birth, the child was excluded.

A series of potential confounders were considered. Those included are parents' highest educational level, parents' social status, parents' total income, maternal age, place of birth, and year of birth. Exposure to certain potentially harmful chemicals (metals, solvents, and polycyclic aromatic hydrocarbons) is higher in some occupations than in the general population. Such exposure may also be correlated with exposure to electromagnetic fields and produce confounding. Based on the work from a previous Norwegian study,[9] we classified all occupations into one of two categories: likely and unlikely exposure to harmful chemicals. This classification was used in an attempt to adjust for confounding from these occupational exposures. The classification matrix can also be obtained from the authors.

Odds ratios (ORs) for the middle and highest exposure categories compared with the lowest with 95% confidence intervals (95% CIs) were obtained from logistic models adjusting for confounders. The estimated effects of maternal and paternal exposure were derived separately for each particular

Table 2 Odds ratios of total and selected birth defects by maternal occupational exposure to magnetic fields above 0.1 μT in Norway 1967–95

Category of birth defects	Hours exposed/week	Cases (n)	Crude			Adjusted†		
			OR	95% CI	p Value*	OR	95% CI	p Value*
Any birth defect	<4	22066	1.00		0.47	1.00		0.13
	4–24	1703	1.02	0.97 to 1.08		1.06	1.01 to 1.12	
	>24	119	0.76	0.64 to 0.92		0.92	0.77 to 1.11	
All CNS defects	<4	1158	1.00		0.12	1.00		0.36
	4–24	95	1.09	0.88 to 1.34		1.03	0.84 to 1.28	
	>24	13	1.60	0.93 to 2.77		1.41	0.81 to 2.44	
Anencephaly	<4	306	1.00		0.96	1.00		0.55
	4–24	21	0.91	0.58 to 1.42		0.82	0.53 to 1.29	
	>24	3	1.40	0.45 to 4.36		1.11	0.35 to 3.48	
Spina bifida	<4	402	1.00		0.02	1.00		0.04
	4–24	38	1.25	0.90 to 1.75		1.21	0.86 to 1.69	
	>24	7	2.48	1.18 to 5.25		2.33	1.10 to 4.94	
Hydrocephaly	<4	310	1.00		0.18	1.00		0.13
	4–24	20	0.86	0.54 to 1.35		0.82	0.52 to 1.30	
	>24	0	0.03		1	0.03		
Other CNS defects	<4	147	1.00		0.03	1.00		0.05
	4–24	16	1.44	0.86 to 2.42		1.44	0.86 to 2.43	
	>24	3	2.91	0.93 to 9.13		2.55	0.81 to 8.06	
Cardiac defects	<4	1744	1.00		0.03	1.00		0.33
	4–24	118	0.90	0.74 to 1.08		1.00	0.83 to 1.21	
	>24	4	0.33	0.12 to 0.87		0.40	0.15 to 1.05	
Respiratory system defects	<4	490	1.00		0.02	1.00		0.05
	4–24	24	0.65	0.43 to 0.98		0.90	0.59 to 1.36	
	>24	1	0.30	0.04 to 2.07		0.50	0.07 to 3.59	
Isolated cleft palate	<4	397	1.00		0.01	1.00		0.01
	4–24	17	0.57	0.35 to 0.93		0.57	0.35 to 0.92	
	>24	1	0.37	0.05 to 2.56		0.34	0.05 to 2.34	
Total cleft lip	<4	1042	1.00		0.10	1.00		0.12
	4–24	85	1.08	0.87 to 1.35		1.07	0.86 to 1.34	
	>24	13	1.78	1.03 to 3.08		1.73	1.00 to 2.99	
Genital system defects	<4	3208	1.00		0.56	1.00		0.19
	4–24	273	1.13	1.00 to 1.28		1.15	1.02 to 1.31	
	>24	13	0.58	0.34 to 1.00		0.71	0.41 to 1.22	
Clubfoot	<4	5051	1.00		0.02	1.00		0.04
	4–24	460	1.21	1.10 to 1.33		1.13	1.03 to 1.25	
	>24	24	0.68	0.45 to 1.01		0.92	0.62 to 1.38	
Other defects‡	<4	58	1.00		0.63	1.00		0.45
	4–24	4	0.91	0.33 to 2.52		0.76	0.27 to 2.11	
	>24	0	0.07			0.03		
Down's syndrome	<4	871	1.00		0.02	1.00		0.29
	4–24	51	0.78	0.59 to 1.03		0.93	0.70 to 1.24	
	>24	2	0.33	0.08 to 1.31		0.43	0.11 to 1.70	

*Test for trend across three exposure categories; †adjusted for highest family educational level, place of birth, mothers age, and year of birth; ‡refers to the ICD-8 category 758.

outcome. Tests for trend with maternal and paternal exposure were obtained from logistic models treating exposure categories as a continuous variable. The analyses were carried out for a pooled category of all birth defects, and for the 24 categories of specific birth defects separately. Significance was set at 5% for each hypothesis test, and no attempts were made to adjust for multiple comparisons. The statistical software used was SPSS for Windows.[10]

RESULTS

Altogether 836 475 and 1 290 298 children had information on maternal and paternal exposure, respectively. Among the 24 categories of defects that were studied for association with either maternal or paternal exposure, the categories gastroschisis, eye defects, facial defects, non-cardiac circulatory system defects, oesophageal defects, other gastrointestinal defects, urinary system defects, limb defects, hip dysplasia, musculoskeletal system defects, skin/hair/nail defects, or syndromes other than Down's syndrome, showed no associations with parental exposure, neither in crude nor in adjusted analyses. Results on these 12 categories are not shown, but may be obtained from the authors.

The pooled category of "any birth defect" showed a slightly increased risk in the intermediate category for maternal exposure after adjustment for parents' education, maternal age, place of birth, and year of birth (table 2). However, no trend across the exposure categories was found. The pooled category of all central nervous system defects did not show any association with maternal exposure. However, both spina bifida and other central nervous system defects (international

Table 3 Odds ratios of total and selected birth defects by paternal occupational exposure to magnetic fields above 0.1 μT in Norway 1967–95

Category of birth defects	Hours exposed/week	Cases (n)	Crude			Adjusted†		
			OR	95% CI	p Value*	OR	95% CI	p Value*
Any birth defect	<4	28937	1.00		0.0002	1.00		0.11
	4–24	2831	0.97	0.93 to 1.01		1.03	0.99 to 1.07	
	>24	1088	0.90	0.84 to 0.95		1.03	0.97 to 1.09	
All CNS defects	<4	1430	1.00		0.06	1.00		0.22
	4–24	168	1.16	0.99 to 1.37		1.13	0.96 to 1.32	
	>24	68	1.13	0.89 to 1.45		1.07	0.84 to 1.37	
Anencephaly	<4	347	1.00		0.0004	1.00		0.01
	4–24	57	1.63	1.23 to 2.15		1.52	1.15 to 2.02	
	>24	23	1.58	1.04 to 2.41		1.39	0.91 to 2.13	
Spina bifida	<4	535	1.00		0.29	1.00		0.24
	4–24	54	1.00	0.76 to 1.32		0.98	0.74 to 1.30	
	>24	16	0.71	0.44 to 1.17		0.70	0.42 to 1.15	
Hydrocephaly	<4	383	1.00		0.54	1.00		0.71
	4–24	37	0.96	0.68 to 1.34		0.94	0.67 to 1.32	
	>24	20	1.25	0.79 to 1.95		1.19	0.75 to 1.86	
Other CNS defects	<4	172	1.00		0.21	1.00		0.27
	4–24	21	1.21	0.77 to 1.90		1.17	0.74 to 1.84	
	>24	10	1.39	0.73 to 2.62		1.35	0.71 to 2.57	
Cardiac defects	<4	2184	1.00		0.74	1.00		0.42
	4–24	228	1.03	0.90 to 1.19		1.09	0.95 to 1.25	
	>24	84	0.92	0.74 to 1.14		1.01	0.81 to 1.26	
Respiratory system defects	<4	565	1.00		0.01	1.00		0.50
	4–24	49	0.86	0.64 to 1.15		1.02	0.76 to 1.36	
	>24	12	0.51	0.29 to 0.90		0.75	0.42 to 1.33	
Isolated cleft palate	<4	543	1.00		0.43	1.00		0.66
	4–24	63	1.15	0.89 to 1.49		1.11	0.85 to 1.44	
	>24	24	1.05	0.70 to 1.59		1.00	0.66 to 1.51	
Total cleft lip	<4	1513	1.00		0.17	1.00		0.22
	4–24	163	1.07	0.91 to 1.25		1.06	0.90 to 1.25	
	>24	73	1.15	0.91 to 1.46		1.14	0.90 to 1.44	
Genital system defects	<4	4265	1.00		0.06	1.00		0.65
	4–24	384	0.89	0.80 to 0.99		0.96	0.87 to 1.07	
	>24	167	0.93	0.80 to 1.09		1.09	0.93 to 1.28	
Clubfoot	<4	6770	1.00		0.00005	1.00		0.92
	4–24	638	0.93	0.86 to 1.01		1.05	0.96 to 1.13	
	>24	206	0.73	0.63 to 0.83		0.95	0.83 to 1.09	
Other defects‡	<4	83	1.00		0.03	1.00		0.02
	4–24	15	1.79	1.03 to 3.10		1.92	1.10 to 3.36	
	>24	6	1.72	0.75 to 3.95		1.85	0.80 to 4.27	
Down's syndrome	<4	1205	1.00		0.44	1.00		0.52
	4–24	103	0.85	0.69 to 1.04		0.97	0.79 to 1.18	
	>24	52	1.03	0.78 to 1.36		1.16	0.88 to 1.54	

*Test for trend across three exposure categories; †adjusted for highest family educational level, place of birth, mothers' age, and year of birth; ‡refers to the ICD-8 category 758.

classification of diseases, 8th revision (ICD-8) category 743) showed a more than twofold increased risk in the highest exposure category, and the tests for trend were of borderline significance for both (p=0.04 and p=0.05 respectively).

Clubfoot (table 2) showed a weak positive association with maternal exposure (p=0.04). Still, the point estimate of the OR of the highest exposure category was not consistent with an increase. Maternal exposure seemed to have a lower prevalence for several types of defects. For isolated cleft palate a consistent trend was found (p=0.01) with a threefold reduction in the highest exposure category (OR 0.34, 95% CI 0.05 to 2.34).

An increased risk of anencephaly (table 3) was found among offspring of exposed fathers. The association was weaker but still significant after adjustments (p=0.01). For the category other defects (ICD category 758) a significantly increased risk was found in the second exposure category (OR 1.92, 95% CI 1.1 to 3.4), and the test for trend was significant even after adjustment (p=0.02). An unadjusted analysis of paternal exposure showed a lower prevalence of birth defects in general (p<0.001). However, adjustment removed the association. An apparently lower prevalence was also found in unadjusted analyses of the categories respiratory defects and clubfoot, but in each category adjustment removed the association (table 3).

Among the variables adjusted for, educational level had the greatest impact on the estimates. After further adjustment for potential confounding from chemical exposures, the association between maternal exposure and risk of spina bifida was

no longer significant (p=0.55, intermediate category: OR 0.82, 95% CI 0.53 to 1.29, high category: OR 1.12 (95% CI 0.35 to 3.53). The adjustment did not alter any of the other results significantly. Analysis of risk of spina bifida after chemical exposure of the mother alone did not show any effect (p=0.70, OR 1.06, 95% CI 0.77 to 1.46). Adding an interaction term between potential maternal exposure to electromagnetic fields and chemicals did not change the results. However, for anencephaly and paternal exposure this procedure increases the association with magnetic fields (p=0.01, intermediate category OR 1.75, 95% CI 1.52 to 2.64, high category OR 2.25, 95% CI 1.11 to 4.56). The interaction term showed a significant downward trend for increasing combined exposure (p=0.04). Analyses within specific subperiods of calendar time and with 1980 census data for some births all the way up to 1995, did not have enough power to show any risk pattern. Excluding all births after 1985, avoiding use of the 1990 census, did not alter the associations for spina bifida after potential maternal exposure or the association for anencephaly after potential paternal exposure to 50 Hz magnetic fields.

DISCUSSION

The most intriguing findings in this study were the increased risks of anencephaly and spina bifida by paternal and maternal exposure, respectively. The association found for spina bifida is weakened by the fact that adjusment for exposure to harmful chemicals removes the association. This is not the case for the anencephalus result. Increased risks of central nervous system defects in general, however, were not found.

An association between anencephaly, spina bifida, and other central nervous system defects and exposure to electromagnetic fields has not previously been reported. However, studies have indicated an increased risk of neural tube defects[11] and spina bifida[12] in offspring of fathers in electrical occupations. Brant and Nielsen[13] reported an excess risk of hydrocephalus among children of women working with video display terminals, but other investigators have not reported a similar excess. Studies of chick embryos have shown open neural tube and microcephaly after exposure to magnetic fields.[14] A previous study linking the Norwegian Birth Registry to census data from 1970, 1980, and 1990 did not show any increased risks of any birth defect in offspring of men and women in industries with assumed exposure to strong electromagnetic fields.[9] However, this was a smaller study based on a job exposure matrix adopted from a Swedish study.[15]

Spina bifida occurs during the fourth week of prenatal development. Folic acid deficiency is known to increase the risk.[16] Retinoic acid has been shown to alter axial and brain formation.[17] Mechanisms involving an interference of magnetic fields on vitamin effects could therefore be hypothesised.[18] Another more plausible explanation is direct confounding from vitamin intake. If parental education is associated with vitamin intake, confounding from vitamin intake could also explain why adjustment for parental education further reduced the effects.

The decreasing risk for isolated cleft palate by maternal exposure has no strong support in previous studies. Dlugosz *et al*,[19] however, showed a non-significant reduction for this outcome and mothers' use of electric bed heating.

The indication of an increased risk of clubfoot in the intermediate maternal exposure category, is interesting in the light of a reported cluster of clubfoot among children of fathers who were exposed to microwave and radio frequency fields on a Norwegian Navy vessel.[20] However, paternal exposure was not associated with increased risk in our data. Experimental studies have reported a possible effect of maternal exposure on development of fetal limbs. A study has evaluated the effects of pulsing electromagnetic fields with a tension of 0.6 V/m on the in vitro development in preimplanted mouse embryos and early somite rat embryos as well as in vivo development of rat embryos. Absence of telencephalic, optic, and otic vesicles and forelimb buds were found.[21]

In general, it is difficult to interpret associations between reproductive outcomes and paternal exposure. A genetic effect transmitted by the sperm is one possible explanation of an association of paternal exposure to electromagnetic fields with the health of their children. A study of men with abnormal semen quality found no association between occupationally related categories of magnetic fields and sperm morphology, motility, and concentration.[22] A Swedish study investigating effects at delivery of offspring of men exposed to electric and magnetic fields around the time of spermatogenesis, found no clear cut effects.[23]

Even if some animal studies have reported window effects of low frequency magnetic fields[24 25] or have shown that window effects can be accounted for in a biological system,[26] results lacking a dose-response relation should be interpreted with caution. Most occupational exposures are also far below the exposure windows used in animal studies. A study with data from previously published epidemiological investigations on early pregnancy loss to evaluate possible dose-response patterns, did not support intensity windows, and a threshold type dependence on field strength seemed to be more plausible than a linear relation.[27]

The strength of this study is the large number of births included. However, some of the outcomes had zero or very few cases in the highest exposure groups, particularly for maternal exposure. The Norwegian birth registry only includes birth defects identified at the maternety ward of the hospital during the first week after birth. Most birth defects are probably detected shortly after birth. However, defects diagnosed later, such as cardiac defects, are less likely to be detected and might therefore be seriously underreported in the registry.

Our analyses covered 24 categories of birth defects for maternal and paternal exposure separately. Also we did some supplementary analyses. Some false positive associations would therefore be expected.

The Medical Birth Registry of Norway uses the ICD-8 codes when birth defects are categorised. For some disorders like heart and limb defects this categorisation is broad. An effect that is present only for a small subcategory of any of our broad categories is therefore likely to be missed.

When comparing results from the crude and the adjusted analyses, stronger associations were found in the unadjusted analyses, indicating potential confounding from the variables adjusted for in our analyses, particularly educational level.

The main limitations of this study are the crude exposure classification, the crude information of other occupational exposures, and the lack of information on residential exposures. The exposure classification was only based on job titles and branch of industry. This approach may lead to misclassification, which in this cohort study may lead to a reduction in the estimated ORs. On the other hand, job title could be a better marker for exposure to magnetic fields than measurement at the workplace,[28] as personal exposure may vary considerably during working hours. Kromhout *et al*[29] found that the time weighted average varied more on a day to day basis for an individual worker than between workers in a larger measurement study including five electricity companies. The exposure classification method we used has previously been evaluated by Flynn *et al*.[7] They compared expert judgment with personal monitoring of exposure to magnetic fields and concluded that an expert panel was able to differentiate current job titles with regard to exposure to 60 Hz magnetic fields. Those job titles were more detailed than the job titles used in this study. Exposure in this study was defined by duration. A job with a higher cumulative exposure might be placed in a lower exposure category. However, it is not known whether the cumulative exposure, the intensity, or the duration of exposure is the most important for reproductive disorders. As the information on employment history was limited to three points in time represented by the three censuses, we also expect some misclassification of job titles.

This also makes it impossible to take into account possibly critical exposure windows during pregnancy. Furthermore, we had no information about part time jobs. A pregnant woman would be more likely to work part time than her partner. This will result in a higher misclassification among mothers.

Another limitation is that our analyses did not account for a slight dependence between siblings in our data. Such dependence would have the effect that our calculated p values were slightly too low and that the calculated 95% CI was slightly too narrow. Given the low number of sibships affected more than once in our data,[30] the effect of such dependence is probably low or not estimable. A tendency of a higher risk in the intermediate exposure category was found for several birth defects (table 2). This pattern was also found for total defects and mothers' exposure. Corresponding results have been found for leukaemia among children living close to power lines.[31] However, in a situation where no possible mechanisms of an effect of magnetic fields are known, it is unclear whether these findings are of biological importance. The findings should therefore be interpreted with caution. The small numbers in the highest exposure category for several outcomes also make estimates in this category imprecise.

We found indications of lower prevalences of several defects. An effect of electromagnetic fields on the tendency towards spontaneous abortion of a fetus affected by a defect could hypothetically produce such reverse effects. Other selection effects could also contribute. Children of women with birth defects have been shown to have a higher risk of getting the same birth defect.[32] Several occupations classified in the higher exposure categories could be more physically demanding than the occupations in the lowest exposure category. The possibility therefore exists that mothers who had a birth defect that implied a physical handicap were more likely to work in occupations in the lowest exposure category. This potential selection of affected mothers and the increased risk of recurrence of that defect in the offspring might hypothetically produce a small increase in the risk among offspring of unexposed mothers. A similar effect could be hypothesised for fathers.

In conclusion, the present study gives an indication of an association between selected disorders of the central nervous system and parental exposure to 50 Hz magnetic fields. However, the association shown for potential maternal exposure to electromagnetic fields disappeared when potential exposure to chemical agents was included in the model, indicating that chemical exposure may be an alternative explanation for the increased risk for spina bifida. The results should be interpreted with caution due to the crude exposure assessment and possibilities of residual confounding.

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