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Reproductive Outcome in Offspring of Parents Occupationally Exposed to Lead in Norway

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In Norway, great efforts have been made to protect both male and female employees against teratogenic exposures. Associations between occupational lead exposure and reproductive outcome in the offspring were studied. All births in Norway 1970–1993 with possible maternal or paternal occupational lead exposure were compared with a reference population of offspring of parents without occupational lead exposure. Offspring of lead exposed mothers had an increased risk of low birth weight (RR = 1.34; CI = 1.12–1.60) and neural tube defects (RR = 2.87; CI = 1.05–6.38). Effects on birth weight and gestational age showed significant dose-response associations. Offspring of lead exposed fathers had no increased risks of any of the analyzed reproductive outcomes. However, decreased risks were observed of low birth weight (RR = 0.91; CI = 0.86–0.96) and preterm birth (RR = 0.89; CI = 0.86–0.93). Further efforts seem to be needed to protect the offspring of lead-exposed mothers. Am. J. Ind. Med. 34:431–437, 1998. © *1998 Wiley-Liss, Inc.*

KEY WORDS: human; lead; offspring; occupation; birth defect; birth weight; reproductive outcome

INTRODUCTION

Lead is absorbed by inhalation and ingestion, accumulates in the bone and soft tissues, and is known to have harmful acute and long-term health effects in humans. Lead has also been shown to pass the placenta barrier, affecting the fetus' growth and development. As well as teratogenic effects, reduced fertility and sperm quality have been observed. Most studies have focused on paternal lead exposure related to malformations and to semen [Henderson et al., 1986; Hass et al., 1991; Sallmén et al., 1992; Kristensen et al., 1993; Olshan and Faustman, 1993; Andrews et al., 1994]. Few studies have focused on maternal occupational lead exposure and reproductive outcome.

In Norway, regulations have been enforced to protect both male and female employees from teratogenic exposure [Directorate of Labour Inspection, 1996]. In particular, pregnant women who work in a harmful environment have the right to be transferred to a safe workplace or, if this is not possible, to take sick leave. Exposure to lead varies greatly from occupation to occupation. [Gundersen et al., 1977; Johnsen, 1995; Osvoll and Woldbæk, 1995]. During the last decades, the Norwegian Occupational Health and Safety Authorities have lowered the accepted levels of lead exposure (administrative norm, threshold limit value (TLV); the administrative norm is based on industrial hygiene recommendation, political and economic evaluations). Up to 1979 Norway had no occupational lead level. The level set in 1979 (0.10 mg/m^3) was further reduced in 1981 (0.05 mg/m^3), and there are indications, based on blood as well as air measurements, that occupational lead exposure has decreased slightly. This decrease may be accounted for by more widespread use of protective equipment and better ventilation at the workplace. The aim of the present study was to assess whether offspring of parents occupationally exposed to lead in Norway had an increased risk of adverse reproductive outcome.

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TABLE I. Exposure Matrix for Lead Exposure Based on Job Title With Code Number and Description of Occupation[a]

Code	Occupation
Occupations with low/moderate lead exposure	
753	Engine and motor repair, branch 951. Repair of motor vehicle, domestic equipment and goods for personal use
755	Plumber
801	Compositor
811	Glassblower and additional workers
812	Formers (ceramic production)
813	Oventenders (glass and ceramic production)
814	Decorators, glaziers (glass and ceramic production)
819	Additional occupations in occupational group 81. Other groups probably exposed to small amount of lead
090	Painter, artist
Occupations with high lead exposure	
731	Smelter workers
732	Warmers, hardeners, glowers
737	Foundry workers
739	Additional occupations in occupational group 73
769	Additional occupations in electro work 76
783	Industry sprayer in branch 384, production of means of transport
811	Glass cottage workers in the municipalities Jevnaker and Eidsskog
813	Oventenders in the municipalities Jevnaker and Eidsskog
814	Decorators, glaziers in the municipalities Jevnaker and Eidsskog
819	Additional occupations in occupational group 81in the municipalities of Jevnaker and Eidsskog

[a]Nordic Occupational Classification [1965].

TABLE II. Birth Defects Referred to As ''Serious Birth Defects'' in the Analysis

Diagnosis	Modified ICD8 codes
Anencephalus	7409
Spina bifida	7410 7419
Hydrocephalus	7429
Encephalocele	7430
Microcephalus	7431
Other brain defect	7432
Anoftalmi	7440
Microftalmi	7441
Heart failure	7460-9
Circulation system defects	7470-9
Lung defects	7480 7483-6
Esophagus atresia	7502
Abdominal cyst	2399
Peritoneal cyst	2289
Ileum-, analatresia	7511-2
Kidney defects	7530-9
Limb reduction defects	7552-4 7558-9
Skeleton defects	7560 7564-6
Omfalocele	5514
Diaphragmatic hernia	5513
Gastroschisis	7567
Malformations of endocrine organs	7583
Situs inversus, conjoined twins, chromosomal anamolies, tuberous sclerosis	7590-6
Other specified syndromes	7598

MATERIAL AND METHODS

Based on compulsory notification, the Medical Birth Registry of Norway (MBRN) comprises, since 1967, all births of 16 weeks of gestation or more in the country. [Medical Birth Registry of Norway, 1993]. Data on parents' job titles have so far not been registered. Thus, to establish the parents' occupations, all records of the MBRN 1970–1993 were linked to population census records of 1970, 1980, or 1990. The information included job title, branch, and education. Information from the census prior to the birth was used. Linkage was obtained for approximately 1.2 million records. All infants of fathers or mothers not classified as lead exposed were used as the reference population.

Classification of lead exposure was based on a Nordic occupational list [Nordman, 1979]. On the basis of the occupational code, the branch, and the municipality code of selected factories with lead exposure, a Norwegian exposure matrix was established (Table I). Workers were classified to be of high, moderate/low, and not exposed to lead.

Altogether, 1,886 infants were born to mothers classified as lead-exposed, including 83 of high exposure and 1,803 of low/moderate exposure. The number of infants to fathers classified as lead-exposed was 35,930, including 2,128 of high exposure and 33,802 of low/moderate exposure.

Among offspring of lead exposed parents, 180 children had both parents exposed. Since the exposed groups were relatively small, the power of detecting rare reproductive outcomes was low. Thus, the study was focused on risk of low birth weight, preterm birth, perinatal death, male proportion among offspring, serious malformations, in addition to some of the more common birth defects, such as

TABLE III. Prevalence at Birth of Selected Reproductive Outcomes in Offspring of Mothers Exposed to Lead and in Offspring of Nonexposed Reference Mothers, Norway, 1970–1993[a]

Reproductive outcome, category	Nonexposed in category	Exposed per 10,000	Nonexposed per 10,000	Odds ratio	95% confidence interval
Low birth weight	128	679.0	522.0	1.34	1.12–1.60
Preterm birth	234	1,241.0	1,137.0	1.13	0.98–1.29
Neural tube defect	5	26.51	9.36	2.87	1.05–6.38
Down syndrome	2	10.60	10.39	1.09	0.18–3.60
Serious birth defects	21	111.35	72.70	1.25	0.80–1.90
Perinatal mortality	13	68.93	65.94	1.05	0.59–1.76
Male infant (RR)	987	5,236.0	5,142.0	1.02	0.97–1.06

[a]All outcomes, except male infant, are adjusted for maternal age and education; low birth weight is also adjusted for gestational age.

Down syndrome, neural tube defects, isolated cleft palate, and cleft lip.

All malformations diagnosed during the first week of life are to be recorded in the MBRN. Perinatal mortality as used in the present study included all stillbirths of more than 15 weeks of gestation as well as deaths during the first week of life. Preterm birth was defined as less than 37 weeks and low birth weight as less than 2,500 g. Serious birth defects are defined in table II.

Rare events (malformations, deaths, neural tube defect, clefts, and Down syndrome) were analyzed by an exact method from several 2×2 tables for calculating Mantel-Haenzel odds ratio with exact confidence intervals [Vollset, 1991]. For birth weight and gestational age, odds ratios were calculated by logistic regression (BMDP Release: 7.1 SUN/UNIX) [Dickson, 1990]. All reproductive outcomes except sex ratio were adjusted for maternal age and maternal or paternal educational level. In addition, low birth weight was adjusted for gestational age. Crude relative risks for male offspring were calculated directly from a 2×2 table using BMDP (4F).

RESULTS

Offspring of lead-exposed mothers (1,886) had an increased adjusted risk of low birth weight, an increased adjusted risk of neural tube defects, and a tendency toward increased risk of malformations (adjusted), but here the numbers were small (Table III). Dose-dependent associations were observed for low birth weight and preterm birth (Table IV). The risk of neural tube defects was increased in the low/moderate exposure group, but no cases were observed in the high exposure group. No cases were seen since 1980. One case had both parents lead-exposed. The risk of serious malformations was increased in the low/moderate exposure group, but no cases were observed in the high exposure group. Only one case of cleft lip and one of palate were seen among exposed mothers. Adjustment for place of birth to control for possible differences in birth defects ascertainment resulted in a minor change only in serious malformations (1.57; CI = 0.99–2.36).

Offspring of fathers classified as lead-exposed (35,930) had no increased risk of any of the analyzed reproductive outcomes. On the contrary, decreased risks of low birth weight and preterm birth and a tendency toward decreased perinatal mortality were observed (Table V). Offspring of men with moderate/low (33,802) exposure had a decreased risk of low birth weight, preterm birth, and perinatal death (Table VI). For serious malformations, a decreasing risk was seen with increasing exposure, but the exposed groups were small. For the other reproductive outcomes, no effects were observed. Adjustment for place of birth to control for differences in birth defects ascertainment showed no changes.

No secular trends were observed for low birth weight and gestational age in offspring of lead-exposed mothers and fathers. Due to small numbers this could not be analyzed for birth defects and perinatal death.

DISCUSSION

Even in a country like Norway, with strict regulations regarding occupational lead exposure, adverse reproductive outcomes in terms of low birth weight, preterm birth, and birth defects were observed in the offspring of exposed mothers. Since lead passes through the placenta, offspring can be exposed in fetal life. Associations between high maternal blood level or environment with high lead level and low birth weight as well as preterm birth have been observed [Factor-Litvak et al., 1991; Recnor et al., 1997]. One study [West et al., 1996] did not find a negative association between third semester lead level and birth weight among people with low lead exposures. Increased risk of neural tube defects was observed in an English study of mothers exposed to lead in the environment [Bound et al.,

TABLE IV. Prevalence at Birth of Selected Reproductive Outcomes in Offspring of Mothers Exposed to Lead by Level of Exposure and in Offspring of Nonexposed Reference Mothers, Norway, 1970–1993[a]

Reproductive outcome	High exposure[b]	Low/moderate exposure[b]	No exposure[b]
Low birth weight[c]	13	115	62,900
	1566.0	638.0	522.0
	3.47 (1.84–6.12)	1.25 (1.03–1.51)	1
Preterm birth[d]	16	218	136,868
	1,928.0	1,209.0	1,137.0
	1.93 (1.09–3.28)	1.10 (0.95–1.26)	1
Neural tube defects	0	5	1,127
		27.73	9.36
		3.00 (1.10–6.68)	1
Down syndrome	0	2	1,251
		11.09	10.39
		1.14 (0.19–3.77)	1
Serious birth defects	0	21	8,754
		116.47	72.70
		1.63 (1.03–2.46)	1
Perinatal mortality	2	11	7,940
	241.0	61.01	65.94
	3.74 (0.62–12.72)	1.14 (0.19–3.78)	1
Male infant (RR)	39	948	618,873
	4,699.0	5,261.0	5,142.0
	0.91 (0.75–1.13)	1.02 (0.98–1.07)	1

[a]All outcomes, except male infant, are adjusted for maternal age and education; low birth weight is also adjusted for gestational age.
[b]Number, per 10,000, odds ratio (CI).
[c]Dose-response relationship: $p < 0.005$.
[d]Dose-response relationship: $p < 0.008$.

TABLE V. Prevalence at Birth of Selected Reproductive Outcome in Offspring of Fathers Exposed to Lead and in Offspring of Nonexposed Reference Fathers, Norway, 1970–1993[a]

Reproductive outcome	Number of exposed	Exposed per 10,000	Nonexposed per 10,000	Odds ratio	95% confidence interval
Low birth weight	1,702	474.0	519.0	0.91	0.86–0.96
Preterm birth	3,615	1,006.0	1,123.0	0.89	0.86–0.93
Neural tube defects	33	9.18	9.27	0.97	0.68–1.36
Down syndrome	37	10.30	10.33	1.13	0.80–1.54
Isolated cleft palate	24	6.68	5.05	1.32	0.86–1.96
Cleft lip	48	13.36	13.89	0.96	0.71–1.27
Serious birth defects	224	62.34	71.64	0.94	0.82–1.08
Perinatal mortality	191	53.16	63.88	0.87	0.75–1.01
Male infant (RR)	18,407	5,123.0	5,142.0	1.00	0.99–1.01

[a]All outcomes, except male infant, are adjusted for maternal age and fathers education; low birth weight is also adjusted for gestational age.

1997]. A U.S. study showed a nonsignificant increased risk of stillbirth and an increased risk of preterm birth in offspring of occupational lead exposed mothers [Savitz et al., 1989]. To our knowledge, no study has looked at associations between maternal occupational lead exposure and birth defects.

Admittedly, classification of occupational exposure based on job title and branch implies risks of misclassification. In

TABLE VI. Prevalence at Birth of Selected Reproductive Outcomes in Offspring of Fathers Exposed to Lead by Level of Exposure and in Offspring of Nonexposed Reference Fathers, Norway, 1970–1993[a]

Reproductive outcome	High exposure[b]	Low/moderate exposure[b]	No exposure[b]
Low birth weight	101	1,601	59,369
	475.0	474.0	519.0
	0.88 (0.72–1.07)	0.92 (0.87–0.97)	1
Preterm birth	223	3,392	128,556
	1,048.0	1,003.0	1,123.0
	0.90 (0.78–1.03)	0.89 (0.86–0.93)	1
Neural tube defects	2	31	1,061
	9.40	9.17	9.27
	0.99 (0.17–3.29)	0.97 (0.67–1.37)	1
Down syndrome	0	37	1,183
		10.95	10.33
		1.20 (0.85–1.65)	1
Isolated cleft palate	0	24	578
		6.68	5.05
		1.41 (0.92–2.09)	1
Cleft lip	3	45	1,594
	14.10	13.31	13.89
	1.01 (0.26–2.75)	0.96 (0.71–1.28)	1
Serious birth defects[c]	11	213	8,202
	51.69	63.01	71.64
	0.74 (0.39–1.29)	0.95 (0.82–1.09)	1
Perinatal death	17	174	7,313
	79.89	51.48	63.88
	1.20 (0.72–1.88)	0.85 (0.73–0.99)	1
Male infant (RR)	1,069	17,338	588,518
	5,023.0	5,131.0	5,142.0
	0.98 (0.94–1.02)	1.00 (0.99–1.01)	1

[a]All outcomes, except male infant, are adjusted for maternal age and fathers education; low birth weight is also adjusted for gestational age.
[b]Number, per 10.000, odds ratio.
[c]Dose response relationship: p value <0.047.

the censuses, a three digit coding system has been used, thus offering rather broad groups.

A job matrix, as employed in the present study, will provide even cruder groups with additional possibility of misclassification. In addition, grouping exposure on the basis of a job matrix will provide a cohort exposed to lead and several other exposures. The three digit coding system that has been used made adjustment for other exposures difficult. Studies have shown that occupational exposure in men and women with the same job title will vary [Messing et al., 1994]. Moreover, the use of the job title provided in the census up to 10 years prior to the offspring's conception may further increase the possibility of misclassification. Thus, there are indications that the actual number of lead-exposed mothers in Norway is lower than those included in this study (data not shown). However, the main consequence of this misclassification would be a dilution of the exposed group in the analyses and thereby an attenuation of true effects. Analyzing a subset born only a few years after the census would have reduced occupational misclassification, but also reduced the numbers considerably.

In spite of possible misclassification, the study showed significant findings in lead-exposed mothers, and thus the effects are possibly greater than those observed. Still, in the interpretation of the results, the possibility that the lead-exposed group might also be exposed to other agents should be considered.

Misclassification of the outcome variables might also occur. Information on birth weight is considered to be valid. For gestational age, misclassification may be more frequent, but by using a dichotomized variable, this problem is considerably reduced. While ascertainment, especially of minor congenital malformations, represents a problem, the data on perinatal mortality are considered to be reliable.

TABLE VII. Prevalence at Birth of Isolated Cleft Palate and Cleft Lip in Offspring of Fathers Exposed to Lead and in Offspring of Nonexposed Reference Fathers, Norway, 1970–1993[a]

Reproductive outcome	Number of exposed	Exposed per 10,000	Nonexposed per 10,000	Odds ratio	95% confidence interval
Isolated cleft palate					
Male	14	7.61	4.25	1.73	0.97–2.89
Female	10	5.71	5.76	1.00	0.50–1.80
Cleft lip					
Male	34	18.49	18.13	1.02	0.71–1.41
Female	14	8.00	9.41	0.86	0.49–1.43

[a]Both outcomes are adjusted for maternal age and father's education.

Misclassification of gender is usually not a problem for full-term births, but in preterm deaths, the rate of misclassification might be higher. However, since such a misclassification is nondifferential, it will probably dilute true effects.

In this study, maternal age and maternal or paternal educational level were used to adjust for possible confounding factors, using educational level as a proxy for social class. Confounding factors relevant to sex ratio of offspring have not yet been established.

A confounding factor relevant to low birth weight is maternal smoking that might be more prevalent among lead-exposed workers. Data on smoking were not available. During the last decades, smoking seems to be increasingly concentrated in lower social classes [Cnattingius et al., 1992] in Scandinavia. Admittedly, adjustment for education as a proxy for social class might not fully adjust for maternal smoking. Still, the dose-dependent increase of low birth weight in offspring of lead-exposed mothers adjusted for maternal age and maternal or paternal education might, to some extent, be attributable to lead exposure. This increase was not reduced during the observation periods.

In spite of problems with misclassification that might attenuate true effects, offspring of women classified as lead-exposed had increased risks of low birth weight, short gestational age, neural tube defects, and serious birth defects for low birth weight and preterm birth with significant dose-response associations. Due to small numbers, only the most frequent birth defects were analyzed and dose-response associations for malformations were not assessed.

Sons of lead-exposed fathers had a nonsignificant increased risk of isolated cleft palate, but an increased risk of cleft lip was not observed (Table VII). An earlier Norwegian study reported an increased risk for cleft lip (with and without cleft palate) among sons of lead-exposed men, but due to small numbers in that study, evaluation of risk for cleft palate was not possible [Kristensen et al., 1993].

Offspring of lead-exposed fathers had a reduced risk for some of the reproductive outcomes that were analyzed, i.e., low birth weight and preterm birth. An increased risk of low birth weight has been reported in a case control study from the U.S. among men exposed to high lead levels; no increased odds were observed at low level of exposure [Min et al., 1996].

Apart from an elevated risk for isolated cleft palate in our study, no increased risks of any of the malformations analyzed were seen. However, nonsignificantly elevated risks for a number of malformations were seen in a Finnish study [Sallmén et al., 1992].

The increased perinatal mortality observed in an earlier Norwegian study among offspring of exposed fathers [Kristensen et al., 1993] was not found in the present study, rather the contrary.

One might speculate that lead exposure could reduce the number of nonfit spermatocytes or increase early abortion of adverse outcomes, mechanisms that might reduce the occurrence of adverse outcomes. The mechanisms have previously been proposed by others [Henderson et al., 1986; Irgens et al., 1991; Lie et al., 1992; Anttila and Sallmén, 1995]. A reduced sex ratio, i.e., a reduced male proportion, among offspring of lead-exposed fathers might relate to a similar mechanism. A reduced sex ratio has been observed in some studies [Dickinson et al., 1994] but not in others [Min et al., 1996, 1997]. In the present study, such a tendency seemed to exist in the high exposed group.

CONCLUSION

The findings of the present study suggest that men and especially women occupationally exposed to lead should be informed of the harmful reproductive effects of lead. They should be encouraged to take precautions in advance of planning a pregnancy. In Norway, approved regulations for the workforce's reproductive health offer ways of dealing with this problem. It is, however, important that future parents make use of the regulations. Most developmental malformations occur already during the first eight weeks of gestation. Thus, a leave of absence after a pregnancy is confirmed might be too late because the half-life of accumulated lead has a range from days to several years. Since an increased blood lead level will persist for some time due to

lead's large half-life range, the fetus might be exposed even weeks after exposure has ceased. Of great importance is the intention of the authorities to further reduce the set levels of lead in the future.

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