Paternal Occupational Exposure to 2,3,7,8-Tetrachlorodibenzo-p-dioxin and Birth Outcomes of Offspring: Birth Weight, Preterm Delivery, and Birth Defects

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Agent Orange is a phenoxy herbicide that was contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). We studied pregnancy outcomes among wives of male chemical workers who were highly exposed to chemicals contaminated with TCDD and among wives of nonexposed neighborhood referents. For exposed pregnancies, we estimated serum TCDD concentration at the time of conception using a pharmacokinetic model. The mean TCDD concentration for workers’ births was 254 pg/g lipid (range, 3–16,340 pg/g). The mean TCDD concentration for exposed pregnancies was assigned to pregnancies fathered by workers before exposure. A total of 1,117 live singleton births of 217 referent wives and 176 worker wives were included. Only full-term births were included in the birth weight analysis (≥37 weeks of gestation). Mean birth weight among full-term babies was similar among referents (n = 604), preexposure workers’ babies (n = 221), and exposed workers’ babies (n = 292) (3,420, 3,347, and 3,442 g, respectively). Neither continuous nor categorical TCDD concentration had an effect on birth weight for term infants after adjustment for infant sex, mother’s education, parity, prenatal cigarette smoking, and gestation length. An analysis to estimate potential direct exposure of the wives during periods of workers’ exposure yielded a nonstatistically significant increase in infant birth weight of 130 g in the highest exposure group (TCDD concentration > 254 pg/g) compared with referents (p = 0.09). Mothers’ reports of preterm delivery showed some suggestive evidence of an association with paternal TCDD (log) concentration (odds ratio = 0.8, 95% confidence interval, 0.6–1.1). We also include descriptive information about reported birth defects. Because the estimated TCDD concentrations in this population were much higher than in other studies, the results indicate that TCDD is unlikely to increase the risk of low birth weight or preterm delivery through a paternal mechanism.

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A questionnaire was used to collect information on health status and risk factors. Subjects were also asked to participate in a medical examination, which included drawing blood for determination of serum TCDD. The methods of serum collection, analytical methods, and quality control standards have been published previously (Fingerhut et al. 1989, 1991; Patterson et al. 1986, 1989; Turner et al. 1989). The study was voluntary, and informed consent was obtained from all study subjects.

Current and former wives/partners (hereafter referred to as wives) of male participants were contacted and administered a telephone interview, which collected detailed information on reproductive history, medical history, lifestyle factors, and occupational factors. Data for 14 women who worked at the plants and their referents were not included in the analysis because of the small number of births.

**Outcome definitions.** We analyzed three outcome variables for this study: birth weight, preterm delivery, and birth defects. Only live singleton births were included in the birth weight and preterm delivery analyses. Furthermore, the birth weight analysis was conducted only among full-term births. Although previous studies of birth weight and environmental or occupational exposure have included preterm births, the most current literature recommends analyzing birth weight only among full-term births for two reasons: An exposure that affects fetal growth does not necessarily affect preterm delivery, and among term births, the influence of gestational age is minor (Wilcox 2001). For the birth defects analysis, only singleton births and stillbirths were included. Pregnancies not fathered by the study males were excluded in all analyses.

We requested birth certificates for all births, as well as neonatal death certificates and medical records where applicable. Birth certificates were obtained for 82% of the births (86% for worker births and 77% for referent births). Birth weight as recorded on the birth certificates was used when available. If no certificate was available, then the mother’s report of her child’s birth weight was used. Among women who had both birth certificates and mother’s report, the correlation coefficient was 0.91. We analyzed birth weight in grams to be consistent with previous literature.

We defined full-term birth as a live birth of ≥ 37 completed weeks from last menstrual period (LMP) or no more than 3 weeks before due date. For gestational age, we used the mother’s response to the question “Was the baby born on time, early, or late,” and if early or late, “by how many weeks?” We compared the mother’s report of gestational age to what was reported on the birth certificate, when available, and found that 49% were in agreement within the exact week and an additional 25% agreed within 1 week, leaving 26% who disagreed by > 1 week. We found little difference among exposure groups when comparing the percentages of those who disagreed by > 1 week (referents, 25%; preexposure workers, 27%; postexposure worker pregnancies, 28%).

We used the criteria described by Erickson et al. (1984) to define infants with major birth defects—that is, those defects that would affect survival, require substantial medical care, or result in marked physical or psychological challenges.

**Statistical analysis.** For the birth weight analysis, we modeled the primary independent variable, TCDD concentration, both as a continuous variable using the log and as a categorical variable (referents, < 20 pg/g, 20 to < 255 pg/g, ≥ 255 pg/g) using dummy variables. Unlogged TCDD and log-transformed TCDD gave similar results, but the model fit better with logged data, indicating a transformation was necessary. Therefore, the log-transformed TCDD data are presented to linearize the regression model. We selected the < 20 pg/g category as the lowest TCDD category because all of the referent serum samples were < 20 pg/g. The other categories were created based on the distribution of the TCDD estimates in our previous report (Schnorr et al. 2001). The pregnancies fathered by the workers fell into the four exposure categories as follows: 20% of pregnancies had serum values < 20 pg/g; 20% of pregnancies had serum values ≥ 1,120 pg/g; and the remaining 60% of the pregnancies were split equally into two categories, 20 to < 255 and 255 to < 1,120. For the analyses in this report, the upper two categories are combined into > 255 pg/g.

Repeated-measures analyses of variance were performed for the birth weight analysis using the SAS PROC MIXED procedure (SAS Institute, Inc., Cary, NC, USA) to account for the lack of independence among multiple pregnancies per mother. Restricted maximum likelihood was used to estimate the parameters, and a compound symmetric variance-covariance structure was assumed.

To analyze preterm birth, we dichotomized gestational age into two categories: < 37 weeks or ≥ 37 weeks. The primary independent variable, TCDD, was modeled as a continuous (log) variable (too few subjects in the preterm category prevented analysis by TCDD categories). Repeated-measures analyses of variance were performed using the SAS PROC GENMOD procedure to account for the lack of independence among multiple pregnancies per mother.

For the birth weight and preterm analyses, we used univariate analyses to search for medical, lifestyle, and exposure factors that could
potentially confound multivariate analyses. Potential confounders included mother’s medical conditions, prenatal medication use, alcohol consumption, cigarette smoking, maternal age at conception, parity, year of the birth, and prenatal weight gain; length of gestation; infant sex; accident, injury, or falls during pregnancy; maternal workplace factors such as occupational exposure to chemicals, prolonged standing, and heavy lifting; and mother’s and father’s education, race, and ethnicity. A variable was considered a potential confounder and retained for further modeling if it was significantly related to both log TCDD concentration and the outcome \((p < 0.20)\) or changed the TCDD estimate by more than 15%. The variables that were retained for multivariate modeling for the birth weight analysis were sex of the infant, education of the mother, parity, cigarette smoking during pregnancy, and length of gestation. Inclusion in the model of the squared term for gestational length did not improve the model. The variables retained for the preterm birth analysis included having an accident, injury, or fall during pregnancy; mother’s age; and cigarette smoking and medication use during pregnancy.

For the birth defects analysis, we reviewed 1,153 live births and 13 stillbirths for a total of 1,166 births. We attempted to confirm all birth defects with medical records and/or vital statistics of the referent births and preexposure and exposed births. Mothers of preexposure births were younger than mothers of referent or exposed births \((p < 0.01)\). Births conceived during or after fathers’ exposure to TCDD had a higher average exposure to cigarette smoke compared with referents \((p = 0.04)\), although the percentages of mothers who smoked were similar. Alcohol use was higher in exposed pregnancies compared with referent and preexposure births, although the differences were not statistically significant.

The median estimated TCDD concentration for exposed births was 254 pg/g \((\text{range}, 3–16,340 \text{ pg/g}; \text{Table 1})\). The median TCDD serum concentration for referent fathers who participated in the medical exam was 6 pg/g \((\text{range}, 2–19)\), which was the value assigned to all referent and preexposure births. As reported previously \((\text{Schnorr et al. 2001})\), there was a lower percentage of male births in the preexposure group \((50.7\%)\) than in the referent \((54.5\%)\) and exposed \((56.2\%)\) groups, although these differences were not statistically significant.

The same percentage of referent and worker wives worked during their pregnancies \((28\%)\), and few reported exposure to chemicals or radiation during the pregnancies \((0.3–3.0\%; \text{data not shown})\). A portion of the wives worked in jobs that involved physical stressors such as heavy lifting, continuous standing, or use of vibrating tools \((12\% \text{ referent births, 10\% preexposure births, and 8\% exposed births})\).

Mean birth weights were similar in the three exposure groups \((\text{mean ± SD: referent births, 3,420 ± 490 g; preexposure births, 3,347 ± 485 g; exposed births, 3,442 ± 507 g})\). Figure 1 shows average birth weight by length of gestation for referent births and preexposure and exposed worker births. The graph is limited to gestational length of 37–43 weeks because of small numbers in the other weeks, as shown in Table 2. The three exposure groupings are shown in Figure 1.
groups presented in Figure 1 show similar mean birth weight patterns.

The results of a crude analysis show a small but statistically significant birth weight increase of 25 g with each increase in the log TCDD concentration (p = 0.03). In the categorical analysis (Table 4), the highest category of paternal TCDD (≥ 255 pg/g) showed an increase of 130 g (SE = 76) compared with the referents (p = 0.09). When we further restricted the analysis to pregnancies in which the entire pregnancy occurred during the father’s occupational exposure, or dates of plant operation for the referents, the results were similar to those shown in Table 4.

Because there were some differences among referent mothers, mothers of preexposed births, and mothers of exposed births in variables such as maternal age and parity (Table 1), we conducted an analysis of birth weight using the final adjusted model but limited only to exposed births (i.e., pregnancies conceived by workers during or after exposure; n = 287). This analysis showed a statistically significant increase in birth weight of 38 g with each increase in the log of TCDD concentration (p = 0.04).

There were 59 worker wives who had births in both exposure periods (preexposure and during/after exposure), with a total of 219 births (110 conceived preexposure and 109 conceived during or after exposure). An analysis of this subset showed that the mean birth weight for preexposure births was lower (mean ± SD, 3,388 ± 444 g) compared with exposed births (3,524 ± 563 g), similar to the main analysis. As would be expected, mean parity was also different between preexposure and exposed pregnancies (0.9 and 2.6, respectively). The difference in birth weight between the two exposure periods was not statistically significant after adjusting for parity. There was no difference in length of gestation or sex ratio between the two exposure periods in this subset.

To examine preterm births, we included all live births in the cohort (n = 1,153), consisting of 618 referent births, 238 preexposure births, and 297 exposed births. Of the 51 preterm births in the cohort (4.4%), 22 were among the 618 referent births (3.6%), 21 were among the 238 preexposure births (8.8%), and 8 were among the 297 exposed births (2.7%). A crude analysis of the preterm births showed a somewhat protective association with TCDD (log) concentration at LMP [odds ratio (OR) = 0.8; 95% confidence interval (CI), 0.6–1.0]. Results changed little when adjusted for accident during pregnancy, age of the mother, cigarette smoking during pregnancy, and medication use during pregnancy (OR = 0.8; 95% CI, 0.6–1.1; results not shown).

Table 5 shows selected birth defects that were reported on the maternal interviews, stratified by whether or not the father was exposed. The birth defect categories include central nervous system (CNS) defects, cardiovascular defects, genitourinary defects, clubfoot, hip and lower limb defects, orofacial clefts, and Down syndrome.
Six major CNS defects were reported: one case of spina bifida, two anencephalus cases, two hydrocephalus cases, and one case of multiple congenital anomalies. Two CNS cases were referents, two were in the background category (< 20 pg/g), one was in the lowest exposure category (20 to < 255 pg/g), and one case (spina bifida) was in the highest exposure category (≥ 1,120 pg/g). Four of these cases were confirmed. Two cases were not confirmed, specifically the spina bifida case and one of the hydrocephalus cases, because the records were not available. The mother of the spina bifida case reported that the child was diagnosed at age 5, with “neurologic impairment, spina bifida, and cerebral palsy.”

There were three cardiovascular defects, including tetralogy of Fallot, single ventricle, and Wolf-Parkinson-White syndrome. All three cases were in the referent or background (< 20 pg/g) categories.

**Discussion**

The results of our analyses do not support a causal relationship between low birth weight and high paternal TCDD exposure. In fact, subanalyses showed a positive association between paternal dioxin exposure and the birth weight of offspring. There are few previous studies of paternal exposure to TCDD and birth weight, Michalek et al. (1998) studied Vietnam veterans who were exposed to Agent Orange and TCDD during Operation Ranch Hand. The median estimated TCDD concentration at the time of conception among Ranch Hand veterans was 79 ppt, with a range from 0 to 1,425 ppt (Michalek et al. 1998). The risk of intrauterine growth retardation was not increased in any Ranch Hand exposure category. In a study of sawmill industry workers, no increase was found in risk for lower birth weight in wives of men occupationally exposed to dioxin-contaminated chlorophenols, as measured by expert raters’ estimations of hours of exposure (Dimich-Ward et al. 1996). Although the chlorophenols in that study were not contaminated with TCDD, the workers were exposed to other polychlorinated dioxins. A study of Australian veterans who served in Vietnam showed a higher prevalence of low birth weight (< 2,500 g) among veterans than among controls (5.8% and 3.7%, respectively), although the difference was not statistically significant (Field and Kerr 1988). Multivariate analyses were not presented in that study, and no data on exposure were collected. Studies of maternal TCDD exposure and birth weight have had contradictory results (Eskenazi et al. 2003; Kimbrough and Krouskas 2001).

Overall, the proportion of preterm births in the present population was low (4.4%), and it was lower among exposed births (2.7%) than among preexposure births (8.8%) or referent births (3.6%). Multivariate analysis showed a somewhat protective effect of paternal TCDD concentration with respect to preterm birth. In a previous study of the Ranch Hand veterans, there was a moderate increase in risk of preterm birth among children in the high and background (unexposed) categories compared with referents, although there was no increased risk in the low exposure category, suggesting that the risk was not likely due to TCDD exposure (Michalek et al. 1998).

Our data were too limited to present statistical analysis of reported birth defects and paternal TCDD exposure, although descriptive information on birth defects did not suggest any relationship to TCDD. The NAS concluded in 1996 that there was limited suggestive evidence of an association between paternal exposure to herbicides and spina bifida, although the evidence was inadequate regarding other birth defects (NAS 1997). The NAS conclusion was based on three high-quality studies of Vietnam veterans: the Centers for Disease Control and Prevention (CDC) Birth Defects Study (Erickson et al. 1984), the CDC Vietnam Experience Study (CDC 1989), and the Ranch Hand study (Wolfe et al. 1995). All three studies suggested an association between herbicide exposure and an increased risk of spina bifida in offspring. In our study, there was one reported case of spina bifida. The medical records were not available to confirm the case, and the mother reported that it was not diagnosed until age 5; however, it is interesting to note that the father's exposure was in the highest category of TCDD.

Many previous occupational studies of paternal exposure to dioxin and birth defects suffer from small numbers and inexact exposure methods. Two studies did not find any association between birth defects and occupational exposure to TCDD or 2,4,5-T, but both acknowledged limited statistical power (Smith et al. 1982; Townsend et al. 1982). In a study of sawmill workers, paternal exposure to dioxin-contaminated chlorophenol was associated with increased risks for congenital anomalies of the eye, anencephaly, spina bifida, and genital defects (Dimich-Ward et al. 1996).

Our study’s limitations include a lengthy recall period and the reliance on maternal report of gestational age, birth defects, and birth weight when birth certificates or medical records were not available. However, previous literature shows that mothers’ recall of birth weight is generally found to be accurate (Sanderson et al. 1998; Tomeo et al. 1999; Troy et al. 1996). Correlation coefficients between mother’s report of birth weight and birth weight recorded on the birth certificate ranged from 0.84 to 0.89, even when recall was two to four decades after birth (Sanderson et al. 1998; Troy et al. 1996) with a mean difference in birth weight of ~25 g (Tomeo et al. 1999). When we conducted a subanalysis that excluded those individuals (18%) for whom birth weight was not confirmed by certificate, the results were similar. We conducted another subanalysis of birth weight in which the definition of full term was estimated from birth certificates rather than mothers’ questionnaires (excluding 250 additional births) that also showed similar results. Another limitation of our study is that serum TCDD was measured several years after the pregnancies, and therefore, the concentrations at LMP are estimates. However, we used a pharmacokinetic model to estimate concentrations at LMP, allowing for changes in individual body burden over time.

Because of the random sequencing procedure used in this study, we could not calculate a true participation rate for male referents. To address participation bias, a telephone interview was attempted with all of the male workers who refused to be examined and a 10% random sample of the referents who refused to participate. Of the 115 refusant workers and 129 refusant referents who were contacted, 68 (57%) and 99 (77%), respectively, agreed to be interviewed. The differences in reporting of selected chronic diseases between participants and nonparticipants were not statistically significant (Calvert et al. 1991, 1992, 1999). No significant difference in age was found between participants and refusants (Calvert et al. 1999). In addition, because male subjects agreed to participate in a cross-sectional medical health study and then later were asked to enroll in the reproductive health study, we do not anticipate that male refusants would differ from participants with respect to reproductive outcomes.

The men in our occupational study group were exposed to TCDD at substantially higher concentrations than other cohorts, with estimated concentrations at the time of conception ranging up to 16,340 pg/g. The strengths of our study include biologic measurements of internal dose and a pharmacokinetic modeling technique. Another strength is that we were able to adjust for confounding variables that were collected with a telephone interview. In addition, most of our outcome data were verified by birth certificate and/or medical records.

In conclusion, these results do not support a relationship between paternal TCDD exposure and lowered birth weight or preterm delivery. Because the estimated TCDD concentrations in this population were much higher than in other studies, the results indicate that TCDD is unlikely to increase the risk of low birth weight or preterm delivery through a paternal mechanism.
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