Evaluation of Chronic Bronchitis, Chronic Obstructive Pulmonary Disease, and Ventilatory Function Among Workers Exposed to 2,3,7,8-Tetrachlorodibenzo-p-dioxin[1-3]

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SUMMARY 2,3,7,8-Tetrachlorodibenzo-p-dioxin (TCDD) is produced as an undesirable contaminant in the manufacture of 2,4,5-trichlorophenol (TCP) and its derivatives. There is considerable concern about the health effects that may be associated with exposure to TCDD-contaminated substances. A cross-sectional medical study that included a comprehensive medical history, medical examination, and measurement of pulmonary function was conducted on workers employed more than 15 yr earlier in the manufacture of NaTCP and its derivatives at two chemical plants. The workers had substantial exposure to substances contaminated with TCDD, as evidenced by a mean serum TCDD level, lipid adusted, of 200 ppt compared with a mean of 7 ppt in the unexposed reference group. The comparison group consisted of individuals with no occupational exposure to phenoxy herbicides who lived in the same communities as the workers. A total of 281 workers and 260 unexposed referents participated in the medical examination. Logistic and linear regression analyses, which contained categorical and continuous measures of TCDD exposure, were performed to control for important confounders, including cigarette and alcohol consumption. No difference was found between workers and referents in the risk for chronic bronchitis or COPD. Analysis of the ventilatory function data revealed no association between history of exposure to substances contaminated with TCDD and the forced expiratory volume at one second (FEV_1), forced vital capacity (FVC), or the ratio of FEV_1 to FVC (FEV_1/FVC%).

AM REV RESPIR DIS 1991; 144:1302-1306

Introduction

The last few years have witnessed major scientific and epidemiologic efforts to assess the possible chronic effects arising from exposure to chemicals contaminated with 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD). Pulmonary system effects due to TCDD exposure is one area of concern.

Studies of long-term exposure to TCDD in Sprague-Dawley rats (1, 2), B6C3F1 mice (3), Swiss-Webster mice (4), and rhesus monkeys (5) reported changes in bronchiolar or alveolar tissue ranging from epithelial hyperplasia and metaplasia to squamous cell carcinomas. The hyperplastic and metaplastic changes observed in exposed animals are similar to the pathologic picture of chronic bronchitis in humans (6).

There is conflicting evidence from epidemiologic studies regarding an association between chronic respiratory system effects and human exposure to substances contaminated with TCDD. One study of workers involved in the production of trichlorophenol (TCP) and 2,4,5-trichlorophenoxyacetic acid (2,4,5-T) suggested that TCDD exposure reduces ventilatory function (7). A study of Air Force personnel who sprayed Agent Orange, a herbicide mixture contaminated with TCDD, failed to demonstrate such an association (8, 9). The study of Air Force personnel did find a slight, but statistically nonsignificant, elevation in abnormalities of the thorax and lung on physical examination among those veterans who sprayed Agent Orange (9).

To evaluate the long-term health effects of occupational exposure to chemicals and materials contaminated with TCDD, the National Institute for Occupational Safety and Health (NIOSH) conducted a cross-sectional medical study. This cross-sectional study compared living individuals (workers) employed more than 15 yr earlier in the production of sodium trichlorophenol (NaTCP), 2,4,5-trichlorophenoxyacetic ester (2,4,5-T ester), or hexachlorophene, all of which were substances contaminated with TCDD, with an unexposed comparison group. The workers were employed at one of two chemical plants located in Newark, New Jersey and Verona, Missouri. This report describes the evaluation of these individuals for chronic bronchitis, chronic obstructive pulmonary disease (COPD), ventilatory function, and thorax and lung abnormalities detected on physical examination.

Methods

A total of 490 workers were employed at the New Jersey facility from 1951 through 1969 in the production of NaTCP, 2,4,5-T ester, and other chemicals. At the facility in Verona, Missouri, 96 individuals were involved in the production of NaTCP, 2,4,5-T ester, or hexachlorophene. Production of NaTCP and 2,4,5-T ester occurred for approximately 4 months in 1968, and production of TCP and hexachlorophene occurred from April 1970 to January 1972. Both plants produced a variety of other industrial chemicals.

To constitute the comparison group, one individual with no self-reported occupational exposure to TCDD-contaminated substances was sought from within the neighborhood of each worker, who matched the worker by age (within 5 yr), ethnicity, and sex. A detailed description of the referent selection process is reported elsewhere (10).

Information on worker and referent health status was collected through a comprehensive set of standardized interviews and standardized medical examinations. Interviewers and examiners were blinded to the exposure status (worker or referent) of the study participants. Interviewer-administered questionnaires elicited from each participant a lifetime medical and occupational history (including information on dust exposure) and demographic and life-style information, including smoking habits, alcohol consumption, education, religion, and income.

Each participant received a standardized medical examination in addition to spirometry and blood tests. Serum samples collected from examined participants were analyzed for TCDD. All TCDD levels were adjusted for lipid as described by Patterson and colleagues (11).

The spirometric tests were administered by trained technicians using a Med Science 570 (Med Science, St. Louis, MO) wedge spirometer. The spirometer was calibrated weekly. Lung volumes were corrected to body tem-

(Received in original form April 1, 1991 and in revised form July 17, 1991)

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perature pressure saturated (BTPS). Each subject was required to perform at least two acceptable trials. Data collected included the forced expiratory volume at one second (FEV_1), the forced vital capacity (FVC), and the ratio of FEV_1 to FVC (FEV_1/FVC%). The trial with the largest sum of FEV_1 and FVC was considered the best trial and was used in the analysis. Each participant's best trial was evaluated by a board-certified pulmonologist. Participants found to have unacceptable flow-volume loop tracings were required to repeat the pulmonary function test until an acceptable tracing was obtained. For quality control purposes, at least one participant per day was randomly selected to undergo independent, repeat spirometry under the direction of a different technician. Overall, there was good reproducibility since after performing a paired t test no statistically significant differences were found between the original and repeat observations for FEV_1 ($p = 0.56$) and FVC ($p = 0.18$).

The case definition for chronic bronchitis was adapted from the American Thoracic Society (ATS) (6). A case of chronic bronchitis was defined as the presence of cough productive of sputum for a minimum of 3 months per year that occurred in at least 2 different years. An individual was excluded from the chronic bronchitis analysis if any self-reported medical condition that can mimic the symptoms of chronic bronchitis was diagnosed by a physician before the commencement of cough productive of sputum. Furthermore, the commencement of cough productive of sputum must have occurred after the worker began TCDD exposure at the New Jersey or Missouri plant. Referents were excluded if onset of their cough productive of sputum preceded the matched worker's first date of TCDD exposure.

The case definition for COPD was adapted from the ATS (12). An individual was considered to have COPD if the individual had an abnormal FEV_1 and an abnormal FEV_1/FVC%. A measured spirometric parameter was considered abnormal if it fell below the one-tailed lower 95% confidence interval for the predicted value (13). The predicted FEV_1 and FEV_1/FVC were calculated using Hankinson's prediction formulas, which correct for height, age, ethnic group, and sex (13). A participant was excluded from the COPD analysis if the individual reported ever having been diagnosed by a physician to have another medical condition that can also lead to an abnormal FEV_1 and FEV_1/FVC.

Analysis of Data

To evaluate the overall risk of chronic bronchitis, COPD, and thorax and lung abnormalities among workers compared with referents and to evaluate for the presence of selection bias, unadjusted odds ratios (OR) were calculated and significance testing was performed using a chi-squared test for association (14). To evaluate the measured means for each spirometric parameter between workers and referents, Student's t tests were performed (14).

Regression analysis was performed to assess the relationship between the outcome of interest and exposure to TCDD-contaminated substances while controlling for several potential confounders and/or effect modifiers (14). For the categorical outcomes (chronic bronchitis and COPD), separate regression analyses were conducted using methods appropriate for both matched and unmatched data. With the matched analysis, exclusion of 25% (70 workers) of the examined workers and 19% (49 referents) of the examined referents was required because their matched partners did not participate in the medical examination. Although the study was originally designed as a matched study, an unmatched analysis was also performed to increase the power of the analysis and to avoid bias associated with the large number of unmatched participants excluded from the matched analysis. For the continuous outcomes (ventilatory measures) only unmatched regression analyses were performed. The potential confounders evaluated in the regression analyses for each of the outcomes of interest are listed in table 1. All potential confounders found to be statistically significant for a particular outcome were retained in the final model of that outcome. Interactions between the exposure variable and statistically significant potential confounders were also examined.

Exposure to TCDD-contaminated materials was analyzed as a dichotomous exposure variable (defined as being a worker or referent). Additionally, to assess a possible dose-response relationship, regression analyses were conducted in which the dichotomous exposure variable was replaced by the lipid-adjusted serum TCDD level, a continuous measure of exposure. Additional dose-response analyses were conducted using the TCDD levels at the time of termination from occupational TCDD exposure (half-life-extrapolated levels). The half-life levels were calculated elsewhere (15). Eight workers and one referent were excluded from the dose-response analyses because their serum TCDD levels were not obtained.

Results

Of the 586 workers at the two plants who were eligible for the study, 400 (68.3%) workers were determined to be alive and could be located. A total of 142 (24.2%) workers were deceased, and 44 (7.5%) could not be located. All 400 workers from the two plants who were living and located at the start of the study were invited to participate in the study; 281 (70%) were examined. A total of 938 referents were invited to participate in the study, of whom 260 (28%) were examined.

Descriptive information on the study participants examined is provided in table 2. Workers were found to have a statistically significantly higher mean lipid-adjusted and half-life-extrapolated serum TCDD level ($p < 0.001$). Referents were found to have a significantly higher lifetime alcohol consumption as measured by alcohol-years ($p = 0.011$). No other statistically significant differences were found in the demographic characteristics between workers and referents.

TABLE 1

POTENTIAL CONFOUNDERS EVALUATED IN THE REGRESSION ANALYSES BY OUTCOME OF INTEREST

	Chronic Bronchitis	COPD	Ventilatory Function
Age	×	×	×
Gender	×	×	×
Ethnic group (white, nonwhite)	×	×	×
Current smoker*	×	×	×
Former smoker†	×	×	×
Pack-years‡	×	×	×
Current drinker§	×	×	×
Former drinker‖	×	×	×
Alcohol-years¶	×	×	×
Height, cm	×	×	×
Other lung disease**	×	×	×
Occupational dust exposure (self-reported)	×	×	×
Location of TCDD exposure (New Jersey or Missouri plant)	×	×	×
Other medical conditions††			×

* Reported smoking within the past year.

† Denied smoking within the past year.

‡ Calculated by multiplying the average number of cigarette packs smoked per day by the number of years cigarettes were smoked.

§ Reported drinking within the past year.

‖ Denied drinking within the past year.

¶ Calculated by multiplying the average number of alcoholic drinks consumed per day by the number of years alcohol was consumed.

** A self-reported, physician-diagnosed history of sarcoidosis, pulmonary emboli, irritant gas inhalation, lung mass, pneumothorax, pleuritis, or pulmonary edema.

†† A self-reported, physician-diagnosed history of asthma, tuberculosis, pneumoconiosis, congestive heart failure, or chest surgery.

TABLE 2

CHARACTERISTICS OF THE STUDY PARTICIPANTS EXAMINED

	Worker (*n* = 281)	Referent (*n* = 260)
Age, yr	55.4	56.0
Range	(33–82)	(31–78)
Ethnic group (%)		
White	250 (89.0)	231 (89.9)
Nonwhite	31 (11.0)	29 (11.1)
Sex, %		
Male	95	93
Female	5	7
Height, cm	173.4	173.2
Range	(150–199)	(146–193)
Mean pack-years	26	29
Range	(0–140)	(0–175)
Smoking status (%)		
Current	94 (33)	85 (33)
Former	123 (44)	114 (45)
Nonsmoker	64 (23)	57 (22)
Mean alcohol-years*	41.4	62.1
Range	(0–405)	(0–719)
Alcohol status (%)		
Current	181 (64)	164 (63)
Former	76 (27)	62 (24)
Nondrinker	24 (9)	34 (13)
Mean lipid-adjusted serum 2,3,7,8-TCDD Level†	220	7
(range)	(ND–3,400)‡	(ND–20)
Half-life–extrapolated serum 2,3,7,8-TCDD level†	1,896	6
(range)	(2–32,400)	(2–20)

* Significant difference between workers and referents using a Student's *t* test ($p = 0.011$).
† Significant difference between workers and referents using a Student's *t* test ($p < 0.001$).
‡ ND, not detected.

TABLE 3

NUMBER OF WORKERS AND REFERENTS EXCLUDED FROM THE CHRONIC BRONCHITIS ANALYSIS BY TYPE OF EXCLUSION

Exclusion	Workers (%) (*n* = 281)	Referents (%) (*n* = 260)
Dates of symptoms not reported	3 (1.1)	4 (1.5)
History of asthma before commencement of cough productive of sputum	4 (1.4)	1 (0.4)
History of congestive heart failure before commencement of cough productive of sputum	1 (0.4)	0
Commencement of cough productive of sputum preceded TCDD exposure of the worker	2 (0.7)	2 (0.7)
Total	10 (3.6)	7 (2.7)

TABLE 4

LOGISTIC REGRESSION MODEL FOR CHRONIC BRONCHITIS USING THE LIPID-ADJUSTED TCDD LEVEL AS THE CONTINUOUS EXPOSURE MEASURE*

Variable	Coefficient	Standard Error of the Estimate	Odds Ratio	95% CI†
Serum TCDD level, per ppt‡	-1.6×10^{-3}	1.8×10^{-3}	0.45§	0.08, 2.57
Per pack-year	0.02	5.7×10^{-3}	1.63‖	1.16, 2.27
Per alcohol-year	3.3×10^{-3}	1.4×10^{-3}	1.18¶	1.03, 1.35

* There were 513 observations.
† The 95% confidence interval.
‡ Parts per trillion.
§ The odds ratio for a participant with a TCDD level of 500 ppt compared with a participant with a TCDD level of 7 ppt.
‖ The odds ratio for a participant with a 30 pack-year history of smoking compared with a participant who never smoked.
¶ The odds ratio for a participant with a 50 alcohol-year history of drinking compared with a participant who never drank alcohol.

Chronic Bronchitis

A total of 10 workers and seven referents were excluded from the chronic bronchitis analysis because they did not meet critieria for inclusion (table 3). Of the remaining 271 workers and 253 referents, 11 (4.1%) and 13 (5.1%), respectively, met the case definition for chronic bronchitis (OR = 0.78, 95% CI = 0.34, 1.78). In the logistic regression models, the risk of chronic bronchitis was not significantly associated with any measure of TCDD exposure, including serum TCDD level ($p = 0.37$) (table 4), half-life–extrapolated TCDD level ($p = 0.45$), or status as a worker versus a referent (OR = 1.04, 95% CI = 0.43, 2.48). Finally, the matched logistic regression analysis did not reveal an association between exposure to substances contaminated with TCDD and chronic bronchitis (odds ratio = 0.70, 95% CI = 0.27, 1.84).

To evaluate whether bias may have been introduced into the chronic bronchitis analysis by the participants who were excluded, we reanalyzed the chronic bronchitis data by including all the participants except those who could not recall the year their symptoms began or the year of their most recent episode. No association between chronic bronchitis and TCDD exposure was found.

COPD

A total of 82 individuals did not meet the criteria for inclusion in the COPD analysis (48 workers and 34 referents) (table 5). Among the remaining 233 workers and 226 referents after exclusions, 11 (4.7%) and seven (3.1%), respectively, had COPD (OR = 1.55, 95% CI = 0.59, 4.05). In the logistic regression analyses, the risk of COPD was not significantly associated with any measure of TCDD exposure, including serum TCDD level ($p = 0.66$) (table 6), half-life–extrapolated TCDD level ($p = 0.67$), or status as a worker versus a referent (OR = 1.58, 95% CI = 0.59, 4.25). The matched logistic regression analysis did not reveal a statistically significant association between exposure to substances contaminated with TCDD and COPD (odds ratio = 2.33, 95% CI = 0.60, 9.02). Among those with a self-reported history of occupational dust exposure, the risk for COPD was borderline significant ($p = 0.053$) (table 6). Information collected from the participants was not adequate to determine the specific dust exposure(s) responsible for this finding.

To evaluate whether bias may have been introduced into the COPD analysis by the large number of participants who were excluded, we reanalyzed the COPD data without excluding any of the

TABLE 5

NUMBER OF WORKERS AND REFERENTS EXCLUDED FROM THE COPD ANALYSIS BY TYPE OF EXCLUSION

Exclusion (History)	Workers (%) (*n* = 281)	Referents (%) (*n* = 260)
Asthma	17 (6.0)	18 (6.9)
Chest surgery	8 (2.8)	6 (2.3)
Tuberculosis	4 (1.4)	7 (2.7)
Occupational lung disease	5 (1.8)	2 (0.8)
Congestive heart failure (CHF)	6 (2.1)	0 –
Lung cancer and chest surgery	1 (0.4)	1 (0.4)
Both asthma and occupational lung disease	4 (1.4)	0 –
Both CHF and chest surgery	2 (0.7)	0 –
Both CHF and tuberculosis	1 (0.4)	0 –
Total	48 (17.1)	34 (13.1)

TABLE 6

LOGISTIC REGRESSION MODEL FOR CHRONIC OBSTRUCTIVE LUNG DISEASE USING THE LIPID-ADJUSTED TCDD LEVEL AS THE CONTINUOUS EXPOSURE MEASURE*

Variable	Coefficient	Standard Error of the Estimate	Odds Ratio	95% CI†
Serum TCDD level, per ppt‡	3.7×10^{-4}	8.5×10^{-4}	1.21§	0.52, 2.78
Current cigarette smoker (yes = 1, no = 0)	1.04	0.49	2.83	1.08, 7.46
Current alcohol drinker (yes = 1, no = 0)	2.37	1.04	10.71	1.39, 82.3
Occupational dust exposure (yes = 1, no = 0)	0.97	0.50	2.63	0.99, 7.00

* There were 454 observations.
† The 95% confidence interval.
‡ Parts per trillion.
§ The odds ratio for a participant with a TCDD level of 500 ppt compared with a participant with a TCDD level of 7 ppt.

TABLE 7

MEAN ACTUAL SPIROMETRIC PARAMETERS FOR WORKERS AND REFERENTS

	Workers (SD)* (*n* = 281)	Referents (SD) (*n* = 260)
Measured FEV_1, L	2.84 (0.71)	2.85 (0.71)
Measured FVC, L	3.48 (0.81)	3.49 (0.79)
Measured FEV_1/FVC, %	81.3 (0.82)	81.6 (0.89)

* Standard deviation.

TABLE 8

DISTRIBUTION OF THORAX AND LUNG ABNORMALITIES ON PHYSICAL EXAMINATION

	Workers (%) (*n* = 281)	Referents (%) (*n* = 260)	OR* (95% CI)
Asymmetric chest excursion	2 (0.7)	1 (0.4)	1.86 (0.17, 19.89)
Abnormal chest shape	17 (6.1)	16 (6.2)	0.98 (0.49, 1.99)
Abnormal chest expansion	8 (2.9)	5 (1.9)	1.50 (0.49, 4.60)
Hyperresonant lungs	10 (3.6)	20 (7.7)	0.44 (0.21, 0.95)
Dullness to percussion	2 (0.7)	2 (0.8)	0.93 (0.13, 6.62)
Diminished breath sounds	11 (3.9)	9 (3.5)	1.14 (0.46, 2.79)
Crackles on auscultation	14 (5.0)	8 (3.1)	1.65 (0.69, 3.98)
Wheezes on auscultation	8 (2.9)	2 (0.8)	3.78 (0.88, 16.20)
Pleural friction rub	0 (0.0)	0 (0.0)	–
Asymmetric chest excursion	2 (0.7)	1 (0.4)	1.86 (0.17, 19.89)
Any abnormality (includes all these abnormalities)†	43 (15.3)	41 (15.7)	0.97 (0.61, 1.54)

* Odds ratio.
† Because some participants had more than one thorax and lung abnormality on physical examination, the sum of the individual lung and thorax abnormalities exceeds the totals in this group.

participants. No association between COPD and TCDD exposure was found.

Ventilatory Function and Abnormalities on Physical Examination

No participants were excluded from these analyses. There were no significant differences between workers and referents for any of the spirometric parameters (table 7). Linear regression revealed that even after controlling for all important covariables, exposure to substances contaminated with TCDD (using the dichotomous or either of the continuous exposure variables) did not significantly affect FEV_1, FVC, or FEV_1/FVC%.

The results of the analysis of the thorax and lung abnormalities that were detected on physical examination are presented in table 8. Workers were not found to have an elevated risk for any of the thorax and lung abnormalities.

Discussion

This is the first epidemiologic study of TCDD-exposed individuals to examine the presence of chronic bronchitis or COPD. Our interest in chronic bronchitis and COPD arose from studies that demonstrated that TCDD-exposed animals developed changes in their respiratory tract that resembled the pathologic changes observed in humans with chronic bronchitis. Our study did not find an association between previous occupational exposure to TCDD-contamination and elevation in the incidence of chronic bronchitis or elevation in the prevalence of COPD.

Our finding that ventilatory function was similar between exposed workers and the unexposed referents was consistent with a study of TCDD-exposed Air Force veterans who sprayed Agent Orange (8, 9). However, our findings are not consistent with those of Suskind and Hertzberg (7). Suskind and Hertzberg found that TCDD exposure was associated with a lower mean percentage of predicted FVC and FEV_1/FVC% even after controlling for smoking. The disparity between their results and ours may be related to two differences. The first difference involves the age of the unexposed populations. In the Suskind and Hertzberg study, the exposed workers were, on average, 10 yr older than the unexposed workers. Although the authors indirectly adjusted for age by analyzing age-adjusted ventilatory measures, it is not clear if these adjustments can completely control for a 10-yr difference in age. In our study, the difference in mean age between the exposed and unexposed groups was 0.6 yr. The second difference

TABLE 9

COMPARISON OF SELF-REPORTED CHRONIC BRONCHITIS AND EMPHYSEMA BETWEEN PARTICIPANTS AND REFUSANTS BY EXPOSURE STATUS

	Chronic Bronchitis (%)	Emphysema (%)
Examined workers (n = 281)	11 (4)	11 (4)
Refusant workers (n = 68)	4 (6)	4 (6)
Odds ratio (95% CI)	0.65 (0.18, 2.52)	0.65 (0.18, 2.52)
Examined referents (n = 260)	10 (4)	7 (3)
Refusant referents (n = 99)	5 (5)	4 (4)
Odds ratio (95% CI)	0.75 (0.23, 2.60)	0.66 (0.20, 2.23)

involves the potential for 2,4,5-T acid dust exposure at the plants studied. The 2,4,5-T acid that was produced at the plant studied by Suskind and Hertzberg was finished as a powder. At the plants we studied, the 2,4,5-T acid was finished as a liquid. Therefore, the potential for exposure to 2,4,5-T acid dust was greater at the plant studied by Suskind and Hertzberg compared with the plants we studied. Although we are not aware of any published reports supporting an association between ventilatory function and 2,4,5-T acid exposure, a respiratory burden of particles, in the absence of a specific toxic agent, can be a probable cause of ventilatory function declines (16).

The physical examination of the thorax and lungs can provide valuable clues to the presence of pulmonary disease. The only evidence that TCDD exposure is associated with abnormalities at these sites comes from a study of Air Force veterans who sprayed Agent Orange; however, the elevation was of borderline statistical significance (9). Our study of workers whose TCDD exposures were higher than those of the Air Force veterans did not find evidence for an association between TCDD exposure and thorax and lung abnormalities.

To assess the effect of selection bias in our study, the workers who refused to be examined and a 10% random sample of the referents who refused to be examined were invited to be interviewed by phone. Of the 115 refusant workers and 129 refusant referents who were contacted, 68 (57%) and 99 (77%), respectively, agreed to be interviewed. These individuals were asked several questions, which were similar to questions asked in our medical study, including, "Has a doctor ever told you that you had any type of serious lung disease such as chronic bronchitis or emphysema?" The proportions who reported these pulmonary conditions were not statistically significantly different between the refusant workers and the examined workers (table 9). Similar results were found for the referents (table 9). These findings suggest that selection bias played little or no role in the outcome of our study.

Results from a recent mortality study of U.S. workers involved in the production of TCDD-contaminated substances, including workers from the two plants we studied (17), suggest that the potential for survivor bias in our study was minimal. Fingerhut and coworkers (17) found that exposed workers were not at elevated risk for death from nonmalignant chronic diseases of the respiratory system (standardized mortality ratio (SMR) = 94, 95% CI = 72, 122).

It is unlikely that the statistical power of this study was a limitation. This study had 87% power to detect a 5% difference in mean spirometric parameters between workers and referents. The power to detect a twofold rise in risk for chronic bronchitis or COPD among workers compared with referents was 65% and to detect a threefold rise in risk the power was greater than 95%.

In summary, in this population with high exposure to substances contaminated with TCDD, an association was not found between prior occupational exposure to chemicals contaminated with TCDD and elevation in the incidence of chronic bronchitis, elevation in the prevalence of COPD, declines in ventilatory function, or elevations in the prevalence of lung and thorax abnormalities.

Acknowledgment

The writers express their thanks for the assistance provided by Lance Cameron, Barbara Connally, Jolene Schoettelkotte, Kyle Steenland, Brent Thompkins, Julie Tolbert, Laurie Piacitelli, and David Marlow and the many other NIOSH personnel too numerous to list individually. The authors also acknowledge their colleagues at the Center for Environmental Health and Injury Control for performing the TCDD analyses on the serum samples.

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