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Comparison of industrial emissions and carpet dust concentrations of polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans in a multi-center U.S. study

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Abstract

Proximity to facilities emitting polychlorinated dibenzo-*p*-dioxins and polychlorinated dibenzofurans (PCDD/F) has been associated with increased risk of non-Hodgkin lymphoma (NHL). There is limited information about whether proximity to industrial sources leads to indoor PCDD/F contamination of homes. We measured carpet dust concentrations (pg/g) of 17 toxic PCDD/F congeners and calculated their toxic equivalence (TEQ) in 100 homes in a population-based case-control study of NHL in Detroit, Los Angeles, Seattle, and Iowa (1998–2000). We took global positioning system readings at residences and obtained coordinates and PCDD/F emissions (ng TEQ/yr) from an Environmental Protection Agency database for 6 facility types: coal-fired electricity generating plants, cement kilns burning non-hazardous waste, hazardous waste incinerators, medical waste incinerators, municipal solid waste incinerators, and sewage sludge incinerators. For each residence, we computed an inverse distance-squared weighted average emission index (AEI [pg TEQ/km^2/yr]) for all facilities within 5 km from 1983–2000. We also computed AEIs for each of the 6 facility types. We evaluated relationships between PCDD/F dust concentrations and the all-facility AEI or categories of facility-type AEIs using multivariable linear regression, adjusting for study center, demographics, and home characteristics. A doubling

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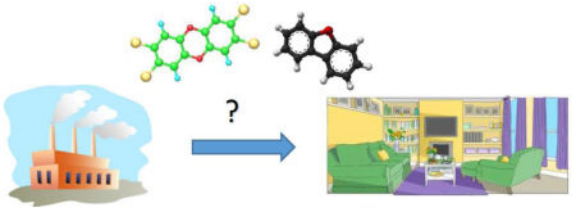
of the all-facility AEI was associated with a 4–8% increase in PCDD/F dust concentrations of 7 of 17 PCDD/F congeners and the TEQ (p-value<0.1). We also observed positive associations between PCDD/F dust concentrations and facility-type AEIs (highest vs. lowest exposure category) for municipal solid waste incinerators (9 PCDD/F, TEQ), and medical waste incinerators (7 PCDD/F, TEQ) (p<0.1). Our results from diverse geographical areas suggest that industrial PCDD/F emission sources contribute to residential PCDD/F dust concentrations. Our emissions index could be improved by incorporating local meteorological data and terrain characteristics. Future research is needed to better understand the links between nearby emission sources, human exposure pathways, and health risks.

Graphical abstract



Keywords

dioxins; furans; geographic information systems; dust; air pollution; environmental exposure; non-Hodgkin lymphoma (NHL)

1. Introduction

Polychlorinated dibenzo-*p*-dioxins and dibenzofurans (PCDD/Fs) are persistent organic pollutants and are classified as human carcinogens by the International Agency for Research on Cancer (IARC) based on epidemiologic studies of all cancers combined, lung cancer, and non-Hodgkin lymphoma (NHL) (IARC 2012). PCDD/Fs are also associated with other health effects including immunotoxicity (Gascon et al. 2013), endocrine disruption (Fernandez-Gonzalez et al. 2013; Schug et al. 2011), and adverse pregnancy outcomes (Vinceti et al. 2008; WHO 2014). Most evidence of the carcinogenic effects of PCDD/F comes from populations with relatively high exposures in occupational settings or via accidental release (Baan et al. 2009; IARC 1997, 2012; NTP 2014; Pesatori et al. 2009; Warner et al. 2011). However, results from a few epidemiologic studies in France (Floret et al. 2003; Viel et al. 2008b; Viel et al. 2011) and the United States (De Roos et al. 2005; Pronk et al. 2013) suggest that lower-level environmental PCDD/F exposures may increase risk of NHL.

PCDD/F are primarily emitted from industrial combustion facilities such as waste incinerators, metal smelters, cement kilns, and coal-fired power plants (U.S.EPA 2006). They are emitted as mixtures; the carcinogenic potency of the mixture is expressed as a toxic equivalence (TEQ), a summed metric that weights the concentrations of the individual congeners relative to the potency of the most potent congener, 2,3,7,8-tetrachlorodibenzodioxin (TCDD), using toxic equivalency factors (TEFs). The predominant

PCDD/F exposure pathway in the general population is considered dietary ingestion of animal products, which accumulate the lipophilic PCDD/F compounds (Dougherty 2000, EPA 1994, USDA 2015, Charnley and Doull 2005). Non-dietary exposure pathways, such as non-dietary ingestion, inhalation, and dermal contact of indoor dust, have received relatively little attention, as they are estimated to contribute less than 10% of total exposure in the U.S. general population (Lorber et al. 2009). However, exposures from dust could be important contributors to total PCDD/F exposures in certain populations, such as those living in close proximity to PCDD/F-emitting sources, those who do not consume a lot of animal products, those who spend a lot of time at home, or children who spend a large portion of time on the floor and exhibit hand-to-mouth contact (Tue et al. 2013). Several studies have documented concentrations of PCDD/F in samples collected in the vicinity of incinerators and other combustion sources including measurements in soils (Domingo et al. 2000; Floret et al. 2006; Oh et al. 2006), and house and attic dust (Dahlgren et al. 2007; Deziel et al. 2012; Gonzalez et al. 2011; Hensley et al. 2007; Feng et al. 2011; Tue et al. 2013). However, these prior studies have generally been limited by small sample sizes or a focus on a single facility.

In France, significantly elevated NHL incidence rates were observed in communities near a high PCDD/F-emitting municipal solid waste incinerator (Viel et al. 2000). Two subsequent case-control analyses of NHL were conducted among community residents living in the vicinity of the same incinerator. Floret et al. (2003) observed that residents who were estimated to have the highest environmental PCDD/F exposure based on a Gaussian dispersion model of emissions from the same municipal solid waste incinerator had a 2-fold increased risk of NHL. Viel et al. (2011) reported an increased risk of NHL with each 10 pg/g lipid increase in serum TEQ within same study population. Another French study of 13 municipal solid waste incinerators used similar Gaussian dispersion modeling to estimate air concentrations of PCDD/F and observed significant increased risk of NHL in areas estimated to have the highest environmental PCDD/F exposures (Viel et al. 2008). Furthermore, in a subset of the National Cancer Institute Surveillance, Epidemiology and End Results Non-Hodgkin Lymphoma (NCI-SEER NHL) Study, a population-based case-control study in four areas in the United States, NHL risk was significantly increased with each 10 pg/g lipid increase in plasma TEQ (De Roos et al. 2005). In addition, NHL risk was significantly increased among those with the highest versus lowest quartile of summed plasma furans (De Roos et al. 2005). In that same study, risk of marginal zone lymphoma (a subtype of NHL) was higher among those in the highest versus lowest quartile of an emission index that captured both proximity to and emissions from a range of nearby combustion facilities (Pronk et al. 2013). NHL risk was also higher among those living within 3 km of cement kilns compared to those living farther away (Pronk et al. 2013).

Carpet dust is a reservoir for many environmental pollutants including PCDD/F, which may persist for years due to their stability and the limited sunlight, moisture, and microbial activity. PCDD/F concentrations may be a useful indicator of environmental exposure around the home (Butte and Heinzow 2002; Franzblau et al. 2009; O'Connor and Sabrsula 2005). Previously, we conducted a pilot study in the NCI-SEER NHL Study and measured 17 toxic PCDD/F congeners in carpet dust from 40 homes, half of which were within 5 km of at least one type of PCDD/F-emitting industrial facility (Deziel et al. 2012). In the pilot

study, we observed that homes within 3 km or 5 km of a cement kiln had 2 to 9 times the dust concentrations of specific PCDD/F ($p<0.1$). Those findings were based on a small number of residences and only a few types of combustion facilities. Here, we conducted a larger study within the NCI-SEER NHL study to determine whether proximity to a broader range of facility types was associated with PCDD/F in carpet dust.

2. Methods

2.1 U.S. Environmental Protection Agency Database of PCDD/F Emissions

We obtained a U.S. Environmental Protection Agency (EPA) database (Personal communication, D. Cleverly, U.S.EPA 2001) that contained addresses, coordinates, and emissions for 10 types of industrial combustion facilities, which accounted for >85% of industrial emissions in the United States from 1987 to 2000. The facility types were: coal-fired electricity generating plants, cement kilns burning non-hazardous waste, hazardous waste incinerators, medical waste incinerators, municipal solid waste incinerators, sewage sludge incinerators, secondary copper smelters, cement kilns burning hazardous waste, iron ore sintering plants, and industrial boilers. For the purpose of this analysis, we defined “facility” as a unique emission source (i.e., stack). Therefore, there were some instances in the database where an address had more than one “facility” on site.

2.2 Study population

The current exposure study included 100 participants of the NCI–SEER NHL case-control study, selected based on the availability of carpet dust samples and the presence of an industrial PCDD/F source within 5 km of the home. The 5-km distance was chosen to conservatively account for dispersion based on fate and transport models and soil measurements taken at multiple distances from facilities (Domingo et al. 2000; Floret et al. 2003; Lorber et al. 1998; Viel et al. 2008). The NCI-SEER NHL Study has been described previously in detail (Colt et al. 2005; Pronk et al. 2013). In brief, 2,378 individuals (1,321 cases and 1,057 controls) from Detroit, Iowa, Los Angeles County, and Seattle, U.S.A, were interviewed in their homes from 1998 to 2000 about demographics, residential and occupational histories, pesticide use around the home and garden, and other factors. Participants were asked to provide a dust sample at the time of interview if they had used their home vacuum cleaner in the past year and had owned at least half of their rugs or carpets for 5 years or more (n=1,106). Of these, 43 individuals were ineligible for the current study because they had already been included in our pilot study or had insufficient dust available for analysis.

Among those eligible (1,063), 285 (26.8%) individuals lived within 5 km of a medical waste incinerator, 31 (2.9%) near a coal-fired plant, 28 (2.6%) near a sewage sludge incinerator, 16 (1.5%) near a municipal solid waste incinerator, 15 (1.4%) individuals lived within 5 km of a cement kiln, and 11 (1.0%) near a hazardous waste incinerator. Some people lived near more than one facility type. We selected all of these participants who lived within 5 km of cement kilns, coal-fired plants, hazardous waste incinerators, municipal solid waste incinerators, and sewage sludge incinerators, for a total of 85 participants with potential exposure. Because medical waste incinerators were quite common, 45 of the 85 selected individuals also lived

within 5 km of medical waste incinerators. No homes in the NCI-SEER NHL study were within 5 km of the other four facility types (secondary copper smelters, cement kilns burning hazardous waste, iron ore sintering plants, and industrial boilers). We also selected 15 participants who lived >5 km of all facility types yielding a total of 100 participants.

2.3 All-Facility and Facility-Type Annual Emissions Index (AEI)

Global positioning system (GPS) readings were collected outside the interview home for over 95% of the participants in the NCI-SEER NHL case-control study. Because most readings were collected before the end of selective availability (the intentional alteration of public GPS signals for national security purposes), GPS coordinates were checked against geocoded addresses and those that differed by more than 200 m were corrected using a combination of digital orthophotography, Census Bureau street files, road maps, and driving to the residence to collect new GPS coordinates (Seattle, Los Angeles, Iowa) (De Roos et al. 2010; Pronk et al. 2013). As described in Pronk et al. (2013), addresses and coordinates of the industrial facilities were quality-checked and corrected based on comparisons with web-based aerial photographs and ancillary information (Google Inc. Mountain View, CA; Environmental Systems Research Institute, Redlands, CA, USA).

The emissions data for each facility from the EPA database were expressed as a TEQ based on the sum of the PCDD/Fs weighted by their WHO 1998 TEFs (U.S.EPA 2010; Van den Berg et al. 2006). As previously described (Pronk et al. 2009, Pronk et al. 2013), we calculated an annual average emission index (AEI) in units of pg TEQ/km^2/yr for each residence that was equal to the annual emissions from each facility within 5 km (Q_F) divided by the square of the distance in km between the facility and the residence (d_F), summed over all facilities (F) for each year (t) the person lived in the residence up to a maximum of 15 years before the interview (Eq. 1).

Annual emissions data were available for all facilities for the year 1995 and for municipal solid waste incinerators, facility-specific emissions were also available for the year 1987. In addition, we obtained national average emission levels for each facility type for the years 1987, 1995, and 2000 from an EPA survey (U.S.EPA 2006). As described in detail by Pronk et al. (2013), we estimated the linear rate of change in emissions for each facility type between 1987 and 1995, and between 1995 and 2000. We applied the appropriate rate of change to each facility's 1995 emission level to estimate emissions between 1987 and 1994 and 1996 and 2000. We assumed constant emission levels from 1983 to 1987 because air pollution controls were not required prior to 1987. We averaged the yearly AEIs for all facility types over the duration of residence in the interview home (t), up to a maximum of 15 years, to derive the average AEI for each residence ("all-facility AEI") (Eq. 1). In addition to the AEI computed for all facilities, we computed AEIs for each of the six facility types of interest separately ("facility-type AEIs"). Finally, we created dichotomous facility-type proximity metrics, which captured whether a home was within 5 km of at least one of a specific facility type (facility-type AEI=0 or >0) to compare with the metrics that were used in the epidemiologic analyses of NHL risk (Pronk et al. 2013). The 15 homes located >5 km from any facility had an AEI=0.

$$AEI=1/t\sum_{1}^{t}\sum_{F=1}^{n}Q_F/(d_F)^2 \quad (1)$$

2.4 Traffic Proximity Metrics

Vehicle exhaust is another source of PCDD/F emissions (U.S.EPA 2006). Therefore, we evaluated proximity to traffic sources as potential determinants of PCDD/F concentrations in house dust. Replicating the method we reported in Deziel et al (2012), we computed Euclidean distance between residences and major roadways or freight routes using the Streetmap software (TeleAtlas Dynamap Transportation version 5.2, 2003) and used these as continuous variables in our models. Participants living more than 1000 m from a major road or freight route were assigned a distance of 1000 m. Major roadways were classified as inter-state, inter-metropolitan area, or intra-state/intra-metropolitan area/inter-metropolitan area. Freight routes were identified using Freight Analysis Framework 2.2 Network Machine Readable Data Files (Federal Highway Administration Office of Freight Management and Operations, Washington DC, 2009).

2.5 Demographic, Residential, and Occupational Characteristics

We examined whether the following demographic and residential characteristics were determinants of PCDD/F concentrations in house dust: study center, case-control status, year the sampled residence was built, population density of residence census block (U.S. Census Bureau; 2000), and smoking status (current, former, never). Smoking is a potential indoor source of PCDD/F (Lofroth and Zebuhr 1992; Wilson et al. 2008). Because smoking is not a major NHL risk factor, status was ascertained in only half of the NCI-SEER parent study participants and 45% of the current study participants. Among those with smoking information available, current or former smoking was not associated with higher concentrations of PCDD/F in the dust compared to never smokers in either this study or the pilot study (Deziel et al. 2012). Detailed information on whether participants ever worked in industries with high potential for exposure to PCDD/F (mills, refineries, or refuse industry) for at least 12 months was available from occupational histories. Para-occupational sources likely had limited impact on concentrations of PCDD/F in carpet dust in our population. Only two subjects were employed in relevant industries; the jobs were held prior to 1955 and 1975, respectively, limiting the relevance to dust samples collected in 1998–2000.

2.6 Dust Sample Collection and Analysis

During the home visit (occurring between 1998 and 2000), interviewers removed the used bag from the household vacuum cleaner and placed it in a sealable polyethylene bag; for bagless vacuum cleaners, the loose dust was put directly in the polyethylene bag. Dust collected using the home vacuum cleaner was previously demonstrated to have concentrations of organochlorine and other chemicals that were comparable to those in dust sampled using a standardized high-volume small surface sampler (Colt et al. 2008). The dust was sieved (<150 μm), stored at −20° C and initially analyzed for other chemicals of interest (pesticides, other organochlorines, polycyclic aromatic hydrocarbons) at Southwest Research Institute (San Antonio, TX) from 1999 to 2001. Remaining dust was stored at −20°

C at the National Cancer Institute Repository. In 2012, 100 dust samples were shipped on ice to Southwest Research Institute.

A total of 3 g of sieved dust was analyzed for the 17 EPA-designated toxic congeners of PCDD/F (7 PCDD and 10 PCDF) with an isotope-dilution method using a high resolution gas chromatograph/high resolution mass spectrometer (HRGC/HRMS) operated in positive electron ionization mode at ≥ 10,000 mass resolution, in accordance with EPA Method 8290 (U.S.EPA, 2007). A J&W 60 m × 0.32 mm I.D. DB-5 column with a 0.25 μm film thickness (Agilent, Santa Clara, CA, USA) was used as the primary analytical column. The secondary confirmation column (for separation of 2,3,7,8-TCDF from its nearest eluting isomers) was a J &W 30 m × 0.32 mm I.D. DB-225 column with a 0.25 μm film thickness (Agilent, Santa Clara, CA, USA). For concentrations of analytes with co-eluting interferences, we assumed the interfering compounds (i.e., those inflating the ratio between the two ions monitored for calculation of congener concentrations) influenced only one of the monitored ions. The target analyte concentration was then estimated based on the assumed non-interfered ion and the expected ratio between the two.

Quality control (QC) samples included matrix spikes (spiked with a known quantity of target PCDD/F congeners), matrix spike duplicates, and laboratory control sample (a clean matrix). Each of the study samples and QC samples were spiked with 5.00 μL of ^{13}C-labeled internal standard mixture prior to extraction. Samples were cleaned with a sulfuric acid wash, silica gel column chromatography, alumina column chromatography, and activated carbon on silica column chromatography. Samples were reconstituted in 5 uL of recovery standard (in nonane) plus 15 uL of nonane to reduce matrix interferences and analyzed via HRGC/HRMS. The concentration of each compound was determined using an isotope dilution method. All samples had internal standard recoveries within the QC limits of 40–135%, demonstrating acceptable extraction efficiency. The recoveries of the PCDD/F congeners from the matrix spike and matrix spike duplicates generally met the QC limits of 60–140%, though recoveries for some of the higher concentration congeners (1,2,3,4,6,7,8-HpCDD, OCDD, OCDF) were outside the QC limits. This occurred in samples for which the native amounts of PCDD/F congeners were 1 to 2 orders of magnitude greater than the spiked amounts, and therefore the spiked portion was not easily quantifiable. The relative percent differences in measured concentrations between matrix spikes and duplicates were <25% for all measurements, demonstrating good precision in the method. We observed a mean relative percent difference of 7.4% between three pairs of laboratory blind duplicates across the congeners, with a range of 0% (both samples <detection limit) to 38%. We calculated a method quantitation limit for each congener as the lowest calibrated concentration for a compound, adjusted for effective extract volume and sample weight. This serves as a conservative estimate of the limit of detection.

The dust samples were stored frozen at −20° C for approximately 13 years, which far exceeds the 1-yr recommended hold time in EPA Method 8290 (U.S.EPA 2007). However, PCDD/F are extremely stable compounds with half-lives on soil surfaces ranging from 1 to 15 years and half-lives in subsurface soil ranging from 12 to 100 years (U.S.EPA 2003). Estimated half-lives of the structurally similar polychlorinated biphenyls measured in repeated carpet dust samples (i.e., at room temperature) over a period of 3 to 8 years range

from 5 to 18 years (Whitehead et al. 2014). Suppliers of PCDD/F analytical standards routinely monitor their standard lots out to 10 years with no signs of degradation when stored at room temperature in the dark (Cambridge Isotope Laboratories 1997, personal communication).

We calculated the TEQ in house dust using both the 1998 and 2005 World Health Organization TEFs. We present only the 2005 TEQs for the house dust, because they were highly correlated with the 1998 TEQs (Spearman r=0.996, p<0.0001), reflect the most up-to-date toxicity information (U.S.EPA, 2010; Van den Berg et al., 2006), and are comparable to published results from the pilot study (Deziel et al. 2012).

2.7 Statistical Analysis

All toxic congeners were above the limit of quantitation in ≥ 70% of samples except 1,2,3,7,8,9-HxCDF, which we excluded from further analyses due to its low quantitation rate (26%) (Table 1). We used a single imputation approach to assign values to samples with concentrations below the quantitation limit with a randomly drawn value assuming a lognormal distribution defined by the distribution of the measurements above the detection limit using SAS (Cary, NC, USA). This is an unbiased approach when ≤ 30% of samples are below the detection limit (Lubin et al. 2004).

We evaluated the associations between all-facility and facility-specific AEIs (independent variables) and dust PCDD/F concentrations (dependent variables) using multivariable regression models for each of the AEIs and the 17 toxic congeners and the TEQ. We used step-wise backward elimination to construct models with covariates of p-values <0.1. An exception was study center, which we retained in all models regardless of statistical significance due to its potential role as a proxy for other sources or characteristics specific to the location not accounted for by the other determinants. We examined the interaction between study center and the all-facility AEI for each PCDD/F; results from interaction tests were not statistically significant at the p<0.1 level.

For the all-facility AEI models, we evaluated the relationships between the natural log-transformed PCDD/F concentrations and the natural log-transformed all-facility AEI (substituting 0.0001 for AEI=0) because both variables were highly positively skewed and the transformations yielded an approximate linear relationship, based on visual inspection. The facility-type AEIs were modeled as categorical variables due to the high prevalence of residences with 0 pg TEQ/km^2/yr for specific facility types. Categories were facility-type AEI=0 (reference), and ≤ or > the median of the facility-type AEI. We tested for linear trends in the facility-type AEIs using a continuous variable derived from the median value within each category. Finally, we constructed models for facility-type proximity metrics (yes/no home within 5 km of one of a specific facility type) used in the epidemiologic analyses of NHL risk (Pronk et al. 2013). All regression analyses were conducted using STATA 13 (StataCorp LP, College Station, TX), and all p-values are two-sided with no correction for multiple testing; results are considered significant at alpha<0.1, a benchmark commonly used in exposure determinants analyses.

3. Results

The median concentration of the TEQ in the dust was 24 pg/g (IQR: 12, 40). The most toxic and lower chlorinated congeners, TCDD and PeCDD (TEFs=1), had median dust concentrations of 0.63 pg/g (interquartile range [IQR]: 0.20–1.1) and 2.0 pg/g (IQR: 1.2–3.9), respectively (Table 1). The highly chlorinated congeners were most abundant, such as OCDD (median concentration=7540 pg/g), 1,2,3,4,6,7,8-HpCDD (median= 768 pg/g), and OCDF (median=291 pg/g). The Spearman correlation coefficients among the congeners ranged from 0.25 to 0.95, with median of 0.67 (Supplemental Table 1).

The demographic and residential characteristics of the 100 participants and their interview homes are presented in Table 2. The homes were approximately evenly distributed across the four study centers. A total of 74% of homes were single family homes. There were 43 homes ≤ 5 km from a medical incinerator, 31 homes ≤ 5 km of coal-fired power plants, and 28 homes ≤ 5 km of sewage sludge incinerators. The other facility types had 11 to 16 homes within 5 km. Forty-eight percent of homes had more than one facility type within 5 km. The median year that study homes were built was 1956, with 75% of homes built in 1970 or earlier.

Table 3 describes the number of facilities and the distributions of the AEIs by study center. Medical waste incinerators and coal-fired plants had moderate PCDD/F emissions and were the most prevalent (n=36 and n=23 facilities, respectively). Municipal solid waste incinerators had the highest emissions but were less common (n=7). Sewage sludge incinerators, cement kilns, and hazardous waste incinerators had lower emissions than the other facility types and were also relatively uncommon (n=7, n=5, and n=2, respectively). The specific facility types were not evenly distributed among the study centers. For example, most of the medical waste incinerators were located in Detroit, the majority of the coal-fired plants were in Iowa, and the two hazardous waste incinerators were both located in Los Angeles. In general, the highest AEIs were observed in Detroit, followed by Iowa, Seattle, and Los Angeles County, which reflected differences in the numbers and types of facilities and proximities to study homes.

Results from multivariable models evaluating associations between PCDD/F dust concentrations (pg/g) and the all-facility AEI (pg TEQ/km2/yr) are presented in Table 4. The all-facility AEI was associated with higher concentrations of 7 of the 17 PCDD/F congeners and the TEQ ($p<0.1$). For those 7 congeners and the TEQ, a doubling of the AEI (on the arithmetic scale) was associated with a 4.4 to 7.9% increase in the PCDD/F dust concentrations. The variability in the log-transformed PCDD/F dust concentrations explained across all models (model r^2) was on average 19%, with the greatest variability explained for 2,3,4,7,8-PeCDF 1,2,3,4,6,7,8-HpCDF, 2,3,7,8-TCDF, and 1,2,3,4,6,7,8-HpCDD (23 to 27%). Study center was a predictor ($p<0.1$) of 6 of the 17 PCDD/F and the TEQ, but the direction of associations with study centers differed across congeners. Older age of the home (those with an earlier year built) was significantly associated with higher concentrations of 13 PCDD/F congeners and the TEQ ($p<0.05$). Single family homes had higher concentrations of 10 PCDD/F in the dust ($p<0.05$). Proximities to major roadways

and freight routes were not associated with higher concentrations of any PCDD/F in the dust.

Results from final multivariable models evaluating associations between the facility-type AEIs and PCDD/F dust concentrations are presented in Table 5. These models explained an average of 22% of the variability in PCDD/F dust concentrations. The highest category of the AEI for medical waste incinerators (>median AEI vs. 0) was associated with higher dust concentrations of 6 PCDD, 1 PCDF, and the TEQ. Tests for trend were statistically significant (p-value<0.05) in two models. For example, homes in the highest category compared to the reference (medical waste AEI=0) had 2.2-times higher dust TCDD concentrations (95% confidence interval [CI]: 1.3, 3.8) and 1.6 times higher dust TEQ (95%CI: 1.0, 2.6). The AEI for municipal solid waste incinerators was associated (p<0.1) with elevated dust concentrations of 1 PCDD, 8 PCDF, and the TEQ; tests for trend had p-values <0.05 for six of these associations. For example, homes in the highest category of the municipal solid waste incinerator AEI had 1.9 times (95% CI: 0.96, 3.7) higher dust TEQs compared to homes with no facilities of this type within 5 km. The coal-fired power plant AEI and the sewage incinerator AEI (middle category vs. reference) were associated with elevated concentrations of 2 and 1 PCDF congeners, respectively; the overall trends were not monotonic. The cement kiln and hazardous waste AEIs were not associated with any PCDD/F. In these models, study center was a predictor (p<0.1) of 9 of the 17 PCDD/F and the TEQ; older homes and single family homes were associated with higher dust concentrations of PCDD/F congeners and the TEQ. Results from models using dichotomous facility-specific proximity metrics were generally consistent with the facility-type AEIs (Table 5, Supplemental Tables S2), except that relationships between emissions from municipal solid waste incinerators, comparatively heavy-emitting facilities, and dust concentrations of PCDD/F were not evident in a few models when using proximity alone.

4. Discussion

In this analysis in four areas of the United States, an all-facilities emission index that incorporated residential proximity to and emissions from industrial combustion sources was associated with higher carpet dust concentrations of 7 of 17 PCDD/F congeners and the TEQ. These relationships were driven mainly by medical waste incinerators and municipal solid waste incinerators, based on analyses of facility-specific indices. Municipal solid waste incinerators had the greatest emissions and medical waste incinerators were the most prevalent in our study population. Certain home characteristics, such as the age and type of home, were also consistently associated with dust PCDD/F levels.

The median house dust concentration of PCDD/F in our study homes (24 pg TEQ/g) was similar to floor dust concentrations in homes located downwind of a former incinerator and pesticide producer (27 pg TEQ/g) and slightly higher than levels in homes sampled from the general population in the United States, Australia, and Korea (Table 6). The concentrations in the current study were 4 to 20 times lower than studies measuring PCDD/F in attic dust, perhaps because attic dust may integrate over a longer time period than vacuum bag dust, capturing higher, historical exposures (Ilacqua et al. 2003).

Few studies have investigated the relationship between proximity to industrial facilities and PCDD/F levels in house dust. A cross-sectional analysis of 30 homes in Australia observed a non-significant, positive relationship between self-reported proximity to industries and dust concentrations of PCDD/F in homes (Hinwood et al. 2014). However, it was not possible to compare their results with our findings, because the specific types of industry queried and emission levels were not provided in their publication. Several descriptive studies have measured PCDD/F in air, soil, and vegetation in the vicinity of combustion facilities; many of these were conducted for risk assessment purposes or for the purpose of examining temporal, not spatial trends, and did not sample at a wide range of distances (e.g., Rovira et al. 2010, Rovira et al. 2014). However, studies that compared dispersion model-based estimates of ambient concentrations and measurements of PCDD/F in air or soil have reported positive associations or good agreement (Floret et al. 2006, Huang et al. 2007).

Studies comparing serum levels of PCDD/Fs in populations near (e.g., within 2 to 5 km) and far (e.g., within 20 km) from specific PCDD/F–emitting facilities generally reported no associations (e.g., Chen et al. 2004; De Felip et al. 2008; Fierens et al. 2007; Huang et al. 2007; Leem et al. 2006; Reis et al. 2007; Zubero et al. 2009). These studies used proximity only and did not incorporate dispersion models; therefore, differences in facility type, facility age, air pollution control technologies, residential distances, and/or meteorological conditions could be contributing to inconsistencies between environmental and biomonitoring studies. In addition, the lack of associations between proximity to facilities and serum concentrations could be due to the contribution of dietary intakes.

Study center was a predictor ($p<0.1$) of many PCDD/F in our models with the all-facility AEI and the facility-type AEIs, suggesting that site-specific demographic, geographic, geophysical, and meteorological characteristics could be important predictors of fate and transport of PCDD/F emissions. More site-specific studies with improved characterization of these variables are needed to better understand their influence on PCDD/F concentrations in homes.

Older homes consistently had higher PCDD/F concentrations in the dust. This variable may be capturing higher emissions from combustion facilities prior to the widespread adoption of air pollution control technologies after 1987 (U.S. EPA 2006). These higher emissions would not be captured by our AEI metrics, in which we used a rate of change in emissions 1987–1995 to estimate annual AEI only back to 1983 (Pronk et al, 2013). Homes built in 1983 and earlier (n=94) had statistically significantly higher dust TEQ compared to those built after 1983 (n=6) ($p=0.006$). The year the home was built could also be capturing other sources that were more common in the past, such as outdoor trash burning. Further, older homes may have uncharacterized indoor sources of PCDD/F emissions (e.g., fireplaces) contributing to house dust contamination over time (Franzblau et al. 2009). Studies have observed home age to be associated with increased dust concentrations of other persistent pollutants such as polychlorinated biphenyls in U.S. homes (Colt et al. 2005; Whitehead et al. 2014) and PCDD/F in Australian homes (Hinwood et al. 2014). Single family homes consistently had higher PCDD/F concentrations compared to other home types, perhaps because they are impacted more by secondary transport of ground-level dust compared to other home types. The lowest concentrations observed were in apartments in buildings,

possibly because they are less susceptible to secondary transport of ground-level dust due to their elevation.

We explored whether the relationship between dust concentrations of specific congeners and the facility-specific AEIs were consistent with the typical congener profiles of emission sources (EPA 2006; Cleverly et al. 1997). Source allocation was challenging, because all facility types that we studied emit all 17 toxic congeners, but at varying proportions, and the relative concentrations depend on several factors, such as design and features of processing operations (e.g., furnace type, combustion temperature) and air pollution controls. The EPA database provided emissions data in the form of the TEQ, not emissions of individual congeners, and did not contain facility-specific information on existing air pollution control technology. The associations we observed were somewhat consistent with expected emissions profiles. For example, the emissions profile of municipal solid waste incinerators is generally dominated by octachlorodibenzodioxin (OCDD), octachlorodibenzofuran (OCDF), 1,2,3,4,6,7,8 heptachlorodibenzodioxin (1,2,3,4,6,7,8-HpCDD), and 1,2,3,4,6,7,8-heptachlorodibenzofuran (1,2,3,4,6,7,8-HpCDF). Dust concentrations of OCDD, 1,2,3,4,6,7,8-HpCDF, and OCDF (but not HpCDD) were associated with the municipal solid waste AEI. Medical waste incinerators tend to be dominated by OCDD, 1,2,3,4,6,7,8-HpCDF, and OCDF; however, we did not observe associations with dust concentrations of these congeners and the medical waste incinerator AEI. Because the dominant congeners are similar across the combustion sources in our analysis, it is not possible to identify a specific congener that would be uniquely attributable to a given facility type. Further, house dust integrates a broad array of sources indoor, industrial, traffic, and other combustion sources, and it may not be possible to discern a signature source congener profile from the dust samples.

We observed important differences in results between the current study and the previously-conducted pilot likely due to differences in facility prevalence, emission levels, and statistical power. In the current study, the cement kiln AEI was not associated with dust concentrations of PCDD/F, based on 15 homes that were located near 5 kilns. In the pilot study, proximity to cement kilns burning non-hazardous waste was associated with concentrations of several PCDD/Fs, but this was based on 2 homes located near a single kiln. Emissions were similar for kilns in the pilot and the current study, suggesting that difference in our findings were likely attributable to a difference in meteorological, geophysical, and other fate-transport factors, other emission sources in the area, or chance. Additionally, in the current study, the medical incinerator AEI was associated with higher levels of multiple PCDD/F based on 43 homes near 36 facilities, whereas in the pilot, proximity to medical incinerators was not associated with PCDD/F dust concentrations based on 18 homes near 27 facilities. The medical waste incinerators near homes in the current study included several with higher emissions than those in the pilot, which may explain these differences.

We did not observe any associations between proximity to freight routes and major roadways and higher concentrations of furans, in contrast to our pilot study, despite similar distributions of distances to roads. For example, in the current study, the median (inter-quartile range [IQR]) of the distance to nearest freight routes (meters) was 393 (203, 693),

and in the pilot it was 387 (129, 675) (Deziel et al. 2012). Our metric did not specifically account for traffic density or meteorology. Two previous studies observed elevated concentrations of PCDD/F in samples of soil immediately adjacent to highways, but found that levels decreased substantially at distances > 20 m (Benfenati et al. 1992; Sidlova et al. 2009). However, these studies were not designed to capture diesel-fueled truck traffic, which was the intent of the restricted classification of roadways (major roadways or freight routes) used in our proximity metric. Furthermore, these null findings for our traffic-related variables may not be generalizable to other regions, such as Europe and developing countries, where diesel-fueled personal and trucking vehicles are more common and release greater dioxin emissions compared to gasoline (UNEP 1999, Chang et al 2004, Zhou and Levy 2007). Studies incorporating more robust characterization of source, fate, and transport of dioxins emitted by diesel-fueled vehicles and traffic are needed to assess this potential contribution to environmental PCDD/F levels and related adverse health impacts.

The use of the facility-type AEI metric revealed associations with municipal solid waste incinerators that were not apparent with a metric based only on Euclidian distance between residences and these facilities (Table 5, Supplemental Table S2). This suggests that incorporation of emissions data in health studies may improve the exposure assessment. For example, the exposure methodology used in studies in France employed Gaussian-type dispersion models and demonstrated associations between estimated ambient PCDD/F concentrations and PCDD/F levels in soil and human serum samples in the same area (Floret et al. 2003, Floret et al. 2006). In our study, we used a relatively simple inverse-distance-squared weighted metric for the AEI because required input data for a standard air pollution model such as stack height, fuel input, and meteorological data were not available. Further evaluations are needed to determine if this simpler model might be of value in exposure assessment for risk assessment, but the correlation with dust levels is promising. Our metric could be improved by incorporating local meteorological data and terrain characteristics.

Our study findings may have relevance for exposure patterns in other regions with similar combustion sources. However, our study likely underestimates the contribution of these sources to residential levels in homes in areas with less stringent emission controls than those in the United States. Other countries may have different emission sources than those discussed here. For example, the emission of PCDD/F is of increasing concern for developing countries in which open burning of electronic waste is a fast-growing industry (Chan et al. 2013). One important aspect to consider is that our AEI metrics and analyses are reflecting pre-2000 emissions. In the United States, emissions decreased after implementation of improved air pollution control technologies resulting from regulations in the late 1980s and early 1990s (U.S.EPA 2006). An area for future research is the inclusion of measurements of polybrominated dibenzodioxins and furans, which have been shown to contribute significantly to the total TEQ value of indoor dust and may have different emission sources than the PCDD/F (Tue 2013).

The strengths of this study include the analysis of house dust as an exposure indicator for PCDD/F across geographically heterogeneous study areas for a relatively large sample of homes. Multiple PCDD/Fs were universally detected in homes. We observed fairly consistent relationships between proximity to facilities and concentrations of PCDD/F in

dust by study center. Because this analysis was an addition to the original retrospective exposure assessment within a population-based case-control study of NHL, we were constrained with our exposure study design. In spite of this fact, our analysis provides evidence to support and interpret results from a growing body of literature on PCDD/F exposure and its association with risk of adverse health impacts, including diseases such as NHL.

One limitation of the use of PCDD/F levels in house dust alone as an exposure indicator is that it does not include dietary sources, and diet has been cited as the dominant source of PCDD/F in non-occupationally exposed individuals (IARC 1997; Liem et al. 2000). However, consumption of locally grown food may also be impacted by local industrial emission sources. Non-dietary ingestion of house dust may also be an important contributor of exposure, particularly among children who have a propensity to engage in hand-to-mouth activity (Harrad et al. 2003). House dust is an important contributor to exposure to dioxin-like persistent pollutants with indoor sources, such as polybrominated diphenyl ethers, for which inhalation, ingestion, and dermal contact with house dust contributes more to aggregate exposure than diet in several populations (Bramwell et al. 2016). More exposure studies are needed to address the relative contribution of dust versus diet for the PCDD/Fs.

Other limitations of this analysis include the use of a single dust sample to characterize exposure over a longer period of time, and we do not know the variability in levels over time. However, high repeatability has been observed for carpet dust concentrations of the structurally similar polychlorinated biphenyls, with intra-class correlation coefficients>0.7 reported for samples taken 3 to 15 months apart (Whitehead et al. 2012) and Spearman correlation coefficients of 0.61–0.72 for samples collected from the same homes 6 to 8 years apart (Whitehead et al. 2014). Also, we lacked data on other factors that could contribute to indoor dust PCDD/F concentrations, such as outdoor trash burning (Jiang et al., 2011; Knutson et al., 2007a; Knutson et al., 2007b) or indoor sources, such as presence of fireplaces. In addition, we were limited in power to evaluate certain facility types, such as hazard waste incinerators and cement kilns which were less prevent and had lower emissions compared to medical and municipal waste incinerators, and we had limited power to examine study center-specific differences. Finally, we constructed models for each of the PCDD/F congeners, and therefore some associations would be expected due to chance. However, the relationships observed were consistent across congeners that were not strongly correlated with each other, providing support for the results.

Conclusions

Our results suggest that outdoor emission sources of dioxins and furans including several types of incinerators and coal-fired plants contribute to indoor levels of PCDD/Fs, providing support for our exposure metrics and results from epidemiologic studies that observed increased risk of NHL among those residing near these types of industrial facilities. These findings may be important for other diseases known or suspected to be associated with exposure to dioxin-like compounds. Our emissions index could be applied to populations in other geographic areas with similar available information. The index could be improved by incorporating local meteorological data and terrain characteristics. More research is needed

to understand how proximity to industrial and other major PCDD/F sources impacts human exposure and risk of adverse health outcomes.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

AEI	average emission index
EPA	Environmental Protection Agency
GIS	geographic information system
GPS	global positioning system
IARC	International Agency for Research on Cancer
IQR	inter-quartile range
LOD	limit of detection
NCI	National Cancer Institute
NHL	non-Hodgkin lymphoma
PCDD	polychlorinated dibenzo-*p*-dioxins
PCDF	polychlorinated dibenzofurans
SEER	Surveillance, Epidemiology and End Results
TCDD	2,3,7,8-tetrachlorodibenzo-*p*-dioxin
TEF	Toxic Equivalence Factor
TEQ	Toxic Equivalence

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Highlights

- Knowledge of impacts of industrial PCDD/F emissions on home dust levels is limited.
- We created emissions indices for industrial facilities using data from the USEPA.
- We compared industrial PCDD/F emission indices and PCDD/F measured in 100 homes.
- Municipal & medical waste incinerator emissions increased PCDD/F levels in dust.
- More research on industrial emissions and potential exposure pathways is needed.

Table 1

Distributions of PCDD/F concentrations in 100 house dust samples from the NCI-SEER NHL study.

Congener	TEF	Median MQL	% >MQL	Median (pg/g)	Inter-Quartile Range (pg/g)
PCDD					
2,3,7,8-TCDD	1	0.4	66	0.63	(0.20, 1.1)
1,2,3,7,8-PeCDD	1	1	83	2.0	(1.2, 3.9)
1,2,3,4,7,8-HxCDD	0.1	1	94	3.5	(2.2, 6.7)
1,2,3,6,7,8-HxCDD	0.1	1	100	24.7	(12, 46)
1,2,3,7,8,9-HxCDD	0.1	1	100	15.5	(7.9, 26)
1,2,3,4,6,7,8-HpCDD	0.01	1	100	768	(410, 1470)
OCDD	0.0003	2	100	7540	(4500, 14000)
PCDF					
2,3,7,8-TCDF	0.1	0.4	97	2.4	(1.4, 3.9)
1,2,3,7,8-PeCDF	0.03	1	87	1.9	(1.1, 3.2)
2,3,4,7,8-PeCDF	0.3	1	92	3	(1.7, 5.4)
1,2,3,4,7,8-HxCDF	0.1	1	100	7.6	(4.8, 14)
1,2,3,6,7,8-HxCDF	0.1	1	100	5.3	(2.9, 9.8)
1,2,3,7,8,9-HxCDF[a]	0.1	1	26	0.51	(0.50, 0.51)
2,3,4,6,7,8-HxCDF	0.1	1	96	5	(2.5, 9.8)
1,2,3,4,6,7,8-HpCDF	0.01	1	100	110	(62, 230)
1,2,3,4,7,8,9-HpCDF	0.01	1	76	5.3	(1.0, 10)
OCDF	0.003	2	100	291	(160, 480)
TEQ[b]				24	(12, 40)

MQL, method quantitation limit; TEF, Toxic Equivalency Factor; TEQ, toxic equivalence; TCDD, tetrachlorodibenzodioxin; PCDD, pentachlorodibenzodioxin; HxCDD, hexachlorodibenzodioxin, HpCDD, heptachlorodibenzodioxin; OCDD, octachlorodibenzodioxin; TCDF, tetrachlorodibenzofuran; PeCDF, pentachlorodibenzofuran; HxCDF, hexachlorodibenzofuran, HpCDF, heptachlorodibenzofuran; OCDF, octachlorodibenzofuran

[a]Excluded from further analyses due to infrequent detection.

[b]TEQ calculated using the 2005 World Health Organization toxic equivalency factors.

Table 2

Demographic and residential characteristics of the study population (n=100).

Characteristic	Number/percent[a]
Study center	
Detroit	27
Iowa	28
LA	20
Seattle	25
Case	60
Male	55
White	92
Smoking status	
Never	19
Former	17
Current	9
Not ascertained	55
Single family home	74
Number of homes ≤ 5 km of PCDD/F facilities:	
Medical waste incinerators	43
Coal-fired power plants	31
Sewage sludge incinerators	28
Municipal solid waste incinerators	16
Non-hazardous waste cement kilns	15
Hazardous waste incinerators	11
>1 Facility Type	48
	Median (IQR)
Census block population density (ppsm)[b]	5154 (2393, 7296)
Year residence built	1956 (1945 (1970)
Distance to freight route (meters)[c]	393 (203, 693)
Distance to major road (meters)[c]	1000 (418, 1000)

[a] The numbers add up to 100; therefore number=percent.

[b] 2000 U.S. Census of Population and Housing; ppsm=persons per square mile

[c] Residences > 1000 m from a freight route or major road were assigned a distance of 1000 m.

Table 3

Number of facilities, emissions, and average emission indices (AEI) by facility type and study center, for all facilities within 5 km of the 100 study homes.[a]

	Total		Detroit		Iowa		Los Angeles County		Seattle	
Facility Type	**# Facilities**	**Avg 1995 Emissions (pg TEQ/yr)**	**# Facilities**	**AEI Median (IQR) (pg TEQ/km2/yr)**	**# Facilities**	**AEI Median (IQR) (pg TEQ/km2/yr)**	**# Facilities**	**AEI Median (IQR) (pg TEQ/km2/yr)**	**# Facilities**	**AEI Median (IQR) (pg TEQ/km2/yr)**
Cement Kiln	5	170	1	18 (5.9, 30)	2	91.5 (39, 140)	0	0	2	8.8 (5.3, 16)
Coal-Fired Plant	23	200	9	67 (29, 170)	13	34 (26, 42)	1	0.33 (0.25, 1.1)	0	0
Hazardous Waste Incinerator	2	100	0	0	0	0	2	7.5 (0.40, 14)	0	0
Medical Waste Incinerator	36	410	29	220 (92, 560)	4	23.2 (16, 68)	0	0	3	14 (3.4, 21)
Municipal Solid Waste Incinerator	7	14200	3	3350 (2700, 5200)	1	280 (280, 280)	2	6.1 (3.8, 7.7)	1	4.0 (3.9, 4.4)
Sewage Sludge Incinerator	7	110	5	7.2 (3.5, 13)	1	3.1 (2.8, 3.4)	0	0	1	6.2 (3.5, 6.7)
All Facilities	80	NA	47	330 (8.8, 1520)	21	57 (34, 140)	5	0.88 (0.37, 8.1)	7	8.8 (4.9, 26)

IQR, inter-quartile range; TEQ, toxic equivalence

[a]Facility is defined as an emission source. Therefore, a given address or industrial site could have more than one unique facilities.

Table 4

Increase in PCDD/F dust concentrations (pg/g) and the TEQ (pg/g) associated with average exposure indices (pg TEQ/km^2/yr), based on multivariable regression models.

PCDD/F in Dust	Regression Coefficient (β)[a]	95% CI	% Increase in PCDD/F Dust Concentration with 2-fold increase in the AEI[b]	R^2 of Multivariable Model
PCDD[c]				
2,3,7,8-TCDD[d]	0.110	(0.039, 0.181)**	7.9	0.21
1,2,3,7,8-PeCDD[d,e]	0.042	(−0.032, 0.117)	3.0	0.13
1,2,3,4,7,8-HxCDD[f]	0.037	(−0.040, 0.114)	2.6	0.10
1,2,3,6,7,8-HxCDD[d,f,g]	0.066	(−0.011, 0.143)*	4.7	0.22
1,2,3,7,8,9-HxCDD[f,h]	0.067	(−0.007, 0.141)*	4.8	0.17
1,2,3,4,6,7,8-HpCDD[f,h,g]	0.062	(−.009, 0.132)*	4.4	0.23
OCDD[f,h,g]	0.046	(−0.026, 0.117)	3.2	0.19
PCDF[c,i]				
2,3,7,8-TCDF[f,g]	0.073	(0.019, 0.127)**	5.2	0.26
1,2,3,7,8-PeCDF[d,f,g]	0.066	(−0.004, 0.137)*	4.7	0.22
2,3,4,7,8-PeCDF[d,e,f]	0.041	(−0.030, 0.112)	2.9	0.27
1,2,3,4,7,8-HxCDF[d,f]	0.065	(−0.005, 0.136)*	4.6	0.19
1,2,3,6,7,8-HxCDF[d,e,f]	0.021	(−0.062, 0.104)	1.5	0.16
2,3,4,6,7,8-HxCDF[e,f]	0.000	(−0.081, 0.081)	0.0	0.15
1,2,3,4,6,7,8-HpCDF[d,f,g]	0.029	(−0.048, 0.106)	2.0	0.27
1,2,3,4,7,8,9-HpCDF[d]	0.036	(−0.073, 0.145)	2.5	0.13
OCDF[d,f]	0.028	(−0.049, 0.105)	1.9	0.16
TEQ[f,h,g]	0.063	(−0.003, 0.130)*	4.5	0.20

CI, confidence interval; TEQ, toxic equivalence; TCDD, tetrachlorodibenzodioxin; PeCDD, pentachlorodibenzodioxin; HxCDD, hexachlorodibenzodioxin, HpCDD, heptachlorodibenzodioxin; OCDD, octachlorodibenzodioxin; TCDF, tetrachlorodibenzofuran; PeCDF, pentachlorodibenzofuran; HxCDF, hexachlorodibenzofuran, HpCDF, heptachlorodibenzofuran; OCDF, octachlorodibenzofuran

[a] Regression models compare ln(PCDD/F) concentration in dust (dependent variable) versus ln(AEI) and other covariates with $p<0.1$ (independent variables)

[b] % increase in dust concentration on per each 2-fold change in AEI calculated as 2.0^{β}

[c] All models adjusted for Study Center

[d] Adjusted for home type (single family home vs. other)

[e] Adjusted for race

[f] Adjusted for year the home was built

[g] Study center variable had $p<0.1$

[h] Adjusted for population density

[I] 1,2,3,7,8,9-HxCDF excluded due to low detection rate

[*] p<0.1

[**] p<0.05

Table 5

Relative increase in PCDD/F dust concentrations with respect to categories of facility-type annual average inverse-distance-weighted index (AEI), based on multivariable linear regression models.[a]

Congener	Facility-Type AEI	Category (Reference: AEI=0)	Relative Increase (95% CI)	P-Value for Trend	R^{2b}
PCDD[b]					
2,3,7,8-TCDD[c,d]	Medical Waste Incinerator	≤median	1.82 (1.09, 3.04)	0.01	0.25
		>median	2.21 (1.27, 3.84)		
1,2,3,7,8-PeCDD[c,d,e]	Medical Waste Incinerator	≤median	1.58 (0.93, 2.66)	0.17	0.18
		>median	1.59 (0.9, 2.81)		
1,2,3,4,7,8-HxCDD[d,f,g]	Medical Waste Incinerator	≤median	1.61 (0.95, 2.71)	0.10	0.17
		>median	1.74 (0.98, 3.09)		
1,2,3,6,7,8-HxCDD[c,d,g]	Medical Waste Incinerator	≤median	1.47 (0.87, 2.5)	0.05	0.24
		>median	1.89 (1.07, 3.35)		
1,2,3,7,8,9-HxCDD[d,f]	Medical Waste Incinerator	≤median	1.53 (0.92, 2.54)	0.05	0.20
		>median	1.84 (1.06, 3.2)		
1,2,3,4,6,7,8-HpCDD[d,f,g]	Medical Waste Incinerator	≤median	1.37 (0.84, 2.23)	0.13	0.23
		>median	1.58 (0.93, 2.7)		
OCDD[c,d,g]	Solid Waste Incinerator	≤median	1.06 (0.52, 2.13)	0.10	0.17
		>median	1.81 (0.89, 3.68)		
PCDF[b]					
2,3,7,8-TCDF[d,g]	Medical Waste Incinerator	≤median	1.35 (0.91, 2)	0.10	0.29
		>median	1.46 (0.97, 2.19)		
	Solid Waste Incinerator	≤median	1.26 (0.74, 2.14)	0.01	
		>median	1.74 (1, 3.02)		
1,2,3,7,8-PeCDF[c,g]	Solid Waste Incinerator	≤median	1.53 (0.77, 3.03)	0.00	0.14
		>median	3.11 (1.58, 6.1)		

Congener	Facility-Type AEI	Category (Reference: AEI=0)	Relative Increase (95% CI)	P-Value for Trend	R^{2b}
2,3,4,7,8-PeCDF[d,e]	Solid Waste Incinerator	≤median	1.49 (0.74, 2.98)	0.08	0.26
		>median	1.94 (0.93, 4.05)		
1,2,3,4,7,8-HxCDF[c,d]	Solid Waste Incinerator	≤median	1.35 (0.68, 2.66)	0.01	0.22
		>median	2.54 (1.28, 5.06)		
1,2,3,6,7,8-HxCDF[c,d]	Solid Waste Incinerator	≤median	1.16 (0.52, 2.59)	0.05	0.16
		>median	2.23 (0.99, 5.01)		
1,2,3,4,6,7,8-HpCDF[c,d,g]	Solid Waste Incinerator	≤median	0.63 (0.3, 1.3)	0.01	0.35
		>median	2.85 (1.35, 6)		
	Coal-Fired Plant	≤median	1.63 (0.93, 2.87)	0.25	
		>median	1.38 (0.77, 2.5)		
1,2,3,4,7,8,9-HpCDF[c]	Solid Waste Incinerator	≤median	0.48 (0.16, 1.4)	0.02	0.19
		>median	3.41 (1.17, 9.93)		
OCDF[d,e,g]	Solid Waste Incinerator	≤median	1.04 (0.49, 2.22)	0.06	0.28
		>median	3.42 (1.42, 8.24)		
	Sewage Waste Incinerator	≤median	2.05 (1.04, 4.02)	0.83	
		>median	1.11 (0.51, 2.41)		
	Coal-Fired Plant	≤median	1.83 (1.02, 3.27)	0.52	
		>median	1.18 (0.63, 2.2)		
TEQ[c,g]	Medical Waste Incinerator	≤median	1.19 (0.74, 1.93)	0.07	0.25
		>median	1.59 (0.98, 2.6)		
	Solid Waste Incinerator	2	1.08 (0.56, 2.05)	0.03	
		3	1.89 (0.96, 3.72)		

CI, confidence interval; TEQ, toxic equivalence; TCDD, tetrachlorodibenzodioxin; PeCDD, pentachlorodibenzodioxin; HxCDD, hexachlorodibenzodioxin, HpCDD, heptachlorodibenzodioxin; OCDD, octachlorodibenzodioxin; TCDF, tetrachlorodibenzofuran; PeCDF, pentachlorodibenzofuran; HxCDF, hexachlorodibenzofuran, HpCDF, heptachlorodibenzofuran; OCDF, octachlorodibenzofuran

[a]Regression models compare ln(PCDD/F) concentration in dust (dependent variable) versus categorical facility-type AEIs and other covariates with p<0.1 (independent variables). AEI not associated with dust concentrations of 2,3,4,6,7,8-HxCDF; 1,2,3,7,8,9-HxCDF excluded due to low detection rate.

[b]All models adjusted for Study Center

[c]Adjusted for home type (single family home vs. other)

[d] Adjusted for year the home was built

[e] Adjusted for race

[f] Adjusted for population density

[g] Study center variable had p<0.1

Table 6

Comparison of Toxic Equivalences (TEQs) of house dust from the current study and prior studies.

Study Location (Reference)	N	Median (pg TEQ/g)[a]	Range (pg TEQ/g)[a]	Study Period	Dust Sample Source	Study Population
LA, Detroit, Seattle, Iowa (current study)	100	24	3.0, 169	1998–1999	home vacuum cleaner	general population
Western Australia (Hinwood 2014)	30	<0.10	<0.10, 4.8	2009–2011	home vacuum cleaner	general population
Busan, Korea (Kim 2013)	46	4.4	0.086, 170	2007	home vacuum cleaner	general population
New York State (Tue 2013)	21	4.4	0.086, 9.4	2005–2006	home vacuum cleaner	general population
LA, Detroit, Seattle, Iowa (Deziel 2012)	40	20	5.4, 260	1998–1999	home vacuum cleaner	general population
Sauget, Illinois (Gonzalez 2011)	14	504	29, 14000	2008	attic	<2 miles of former PCB/pesticide manufacturer & secondary copper facilities
Louisiana, Mississippi, Alabama (Feng 2011)	60	139	8.2, 13936	not provided	attic	<2 miles of former wood treatment facilities
Michigan (UMDES 2008)	198	11	1.6, 1060	2004–2005	high-volume small-surface samplers	downwind from former incinerator/pesticide manufacturer[b]
Michigan (UMDES 2008)	37	27	7.6, 96	2004–2005	high-volume small-surface samplers	downwind from former incinerator/pesticide manufacturer[b]
Southern Alabama (Hensley 2007)	11	84	8.4, 502	2006	attic	<1 mile of former wood treatment facility
Mississippi (Dahlgren 2007)	38	75	0.91, 22000	2007	attic	<2 miles of former wood treatment facility

WHO, World Health Organization; TEQ, Toxic Equivalency; UMDES, University of Michigan Dioxin Exposure Study; PCB, polychlorinated biphenyls

[a] TEQs weighted by WHO 2005 TEFs, except for Hinwood study for which we present only the values for TCDD.

[b] Based on aerosol dispersion model; distances from source not provided.