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Polycyclic aromatic hydrocarbons: determinants of residential carpet dust levels and risk of non-Hodgkin lymphoma

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Abstract

Purpose—To investigate the risk of non-Hodgkin lymphoma (NHL) associated with residential carpet dust measurements of polycyclic aromatic hydrocarbons (PAHs).

Methods—We evaluated the relationship between residential carpet dust PAH concentrations (benz(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, dibenz(a,h)anthracene, and indeno(1,2,3-c,d)pyrene, and their sum) and risk of NHL (676 cases, 511 controls) in the National Cancer Institute Surveillance Epidemiology and End Results multicenter case–control study. As a secondary aim, we investigated determinants of dust PAH concentrations. We computed odds ratios (OR) and 95 % confidence interval (CI) for associations between NHL and concentrations of individual and summed PAHs using unconditional logistic

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regression, adjusting for age, gender, and study center. Determinants of natural log-transformed PAHs were investigated using multivariate least-squares regression.

Results—We observed some elevated risks for NHL overall and B cell lymphoma subtypes in association with quartiles or tertiles of PAH concentrations, but without a monotonic trend, and there was no association comparing the highest quartile or tertile to the lowest. In contrast, risk of T cell lymphoma was significantly increased among participants with the highest tertile of summed PAHs (OR = 3.04; 95 % CI, 1.09–8.47) and benzo(k)fluoranthene (OR = 3.20; 95 % CI, 1.13–9.11) compared with the lowest tertile. Predictors of PAH dust concentrations in homes included ambient air PAH concentrations and the proportion of developed land within 2 km of a residence. Older age, more years of education, and white race were also predictive of higher levels in homes.

Conclusion—Our results suggest a potential link between PAH exposure and risk of T cell lymphoma and demonstrate the importance of analyzing risk by NHL histologic type.

Keywords

Polycyclic aromatic hydrocarbons; Non-Hodgkin lymphoma; T cell lymphoma; Dust; Case—control study

Introduction

Polycyclic aromatic hydrocarbons (PAHs) are a class of chemicals formed during incomplete combustion of organic materials. Major sources of PAH exposure for the general population include vehicle exhaust, electric power generation, waste incineration, wood smoke, tobacco smoke, and ingestion of grilled/charbroiled meats [1]. Occupational PAH exposure is high in the aluminum smelting, coal gasification, coal-tar distillation, and coke production industries. Several PAHs are known or probable human carcinogens [2, 3] based on associations between occupational exposures and cancers of the lung, skin, and bladder [2–4]. PAHs have also been shown to induce lymphomas in animals [5–8]; however, the relationship between PAH exposures and risk of non-Hodgkin lymphoma (NHL) in humans remains unclear.

NHL is the fifth most common cancer in men and women in the USA [9]. Immunodeficiency, such as HIV/ AIDS, is a known NHL risk factor; however, the etiologies of most lymphomas have yet to be identified [10, 11]. Epidemiologic studies of the association between PAH exposure and NHL risk have primarily focused on occupational exposures and mainly with work in the aluminum industry. A positive relationship between occupational benzo(a)pyrene exposure and NHL risk was observed in a Canadian cohort [12]; however, most studies of occupational PAH exposure have not observed significant associations with NHL risk [13–16]. Reports of associations between NHL and cigarette smoking, a major source of PAH in the general population [17], have been conflicting, with the majority of studies not showing an association with NHL overall [11, 18–20].

Although to date no clear association between PAH exposure and NHL risk has been identified, there can be substantial heterogeneity in the etiology of lymphoma subtypes [11, 21, 22]. In large pooled analyses in the International Lymphoma Epidemiology Consortium

(InterLymph), significant positive associations were found between smoking duration and follicular lymphoma [23] as well as lymphoplasmacytic lymphoma/Walderstrom macroglobulinemia, marginal zone, and peripheral T cell lymphomas [22]. In addition, an increased risk of T cell lymphoma was observed among men who had ever smoked in a Danish and Swedish population-based case–control study [19]. However, other studies of smoking and T cell lymphomas found no association [18, 20].

In this analysis, we evaluated the association between PAH concentrations in residential carpet dust and risk of NHL overall and by histologic subtypes. Because PAHs can accumulate in carpets and rugs over years or decades [24], dust PAH concentrations may represent long-term residential PAH exposure. To further understand sources of residential PAH exposure, we also explored whether home characteristics, demographic variables, geographic location, and environmental factors were determinants of PAH concentrations in the carpet dust samples.

Methods

Study population

The study population, which has been described in detail previously [25–27], included 1,321 first primary NHL cases aged 20–74 diagnosed between 1998 and 2000 from four Surveillance Epidemiology and End Results (SEER) registries: Detroit, Michigan metropolitan area (Macomb, Oakland, and Wayne counties); state of Iowa; Los Angeles County, California; and Seattle, Washington metropolitan area (King and Snohomish counties). HIV-positive cases were excluded. The participation rate among eligible cases was 76 %. Population controls were selected using random digit dialing (≥65 years of age) or from Center for Medicare and Medicaid Services files (≥65 years) and were frequency-matched to cases by age (within 5 years), sex, race, and study center. Of 2,046 eligible controls, 1,057 (52 %) participated. The study was approved by the Human Subjects Review Boards at the National Cancer Institute and each SEER center. Written informed consent was obtained from each participant.

Exposure assessment

A computer-assisted personal interview was conducted in participants' homes and included questions about demographics, lifestyle, housing characteristics, and other factors. As previously described, the study used a split-sample questionnaire design, in which participants were mailed one of two versions of a self-administered questionnaire [25]. Due to this design, some variables, such as smoking, were only asked for a subset of participants [25].

Dust samples were collected at the time of the interviews (February 1999–May 2001) from home vacuum cleaner bags for 695 cases and 521 controls that consented and met eligibility criteria [25, 26, 28]. Eligible cases and controls had used their vacuum cleaner within the prior year and had owned at least half of their carpets or rugs for 5 or more years. Dust samples were shipped overnight to Southwest Research Institute (San Antonio, TX) and stored in freezers at −20 °C. The dust was sieved (<150 um), and a 2-g sample of the fine

fraction was Soxhlet extracted for 16 h with 200 ml diethyl ether–*n*-hexane (6:94). The extracts were cleaned through a Florisil column and analyzed for 10 PAHs, seven of which are classified by the US Environmental Protection Agency as probable human carcinogens: benz(a)anthracene (BaA), benzo(a)pyrene (BaP), benzo(b)fluoranthene (BbF), benzo(k)fluoranthene (BkF), chrysene (Chr), dibenz(a,h)anthracene (DBaA), and indeno(1,2,3-c,d)pyrene (IdP). The other three PAHs, benzo(ghi)perylene, coronene, and dibenzo(a,e)pyrene, were excluded from analyses due to the small number of samples with detectable concentrations. Chemical analyses were performed with gas chromatography (GC)/mass spectrometry (MS) (Danvers, MA) in selected ion monitoring mode using a DB-5.625 30 m × 0.25 mm ID column (J&W Scientific, Folsom, CA). Confirmation analysis for selected samples was done using the same column under full-scan mass spectral analysis on a second GC/MS instrument. Quantitation was based on five-point calibration curves. Dust samples from 682 cases (98 %) and 513 controls (98 %) were successfully analyzed between September 1999 and September 2001. Extraction from laboratory spiked dust samples (n = 25–27 depending on the specific PAH) was between 87 % (DBaA) and 103 % (BaA). Coefficients of variation calculated for duplicate samples within batches (n = 27 pairs) ranged from 4 (BbF) to 8 % (DBaA).

Geocoding and spatial variables

Geographic coordinates for the majority of residences (89.9 %) were assigned from Garmin GPS12 Personal Navigator (Garmin International, Inc., Olathe, KS) global positioning system readings taken outside the participant's home at time of interview. Geocoding of addresses using a modified Microsoft Visual Basic version 6.0 program (TeleAtlas, Lebanon, NH) to match input addresses to the TeleAtlas MatchMaker SDK Professional version 4.3 street database was done for remaining residences (9.5 %). Eight homes (six cases, two controls) were excluded because they could not be accurately located.

The distance from each residence to the nearest major road and freight route was determined using TeleAtlas Dynamap Transportation version 5.2 (2003). Distance to the nearest railroad was calculated from the National Atlas of the United States database (2005). Locations of industrial combustion facilities that release dioxins and may also release PAHs were obtained from an US Environmental Protection Agency (USEPA) national database [29]. The types of facilities included municipal solid waste incinerators, medical waste incinerators, sewage sludge incinerators, hazardous waste incinerators, cement kilns, and coal-fired electricity-generating plants. We determined the number of facilities within 5 km of each home. The percentage of developed land (≥20 % impervious surface) within 2 km of each residence was calculated using the US Geological Survey 2001 National Land Cover Database, a 30-m resolution land cover database created from Landsat 5 and Landsat 7 satellite imagery [30]. Buffer sizes were selected based on the findings of a previous analysis of polychlorinated biphenyl dust concentrations in this case–control study [31]. Full details of these variables have been described previously [31].

Estimated annual average ambient PAH concentrations at the census tract level, based on US Census 2000 tract assignments, were obtained from the EPA's 1999 National Air Toxics Assessment program (http://www.epa.gov/ttn/atw/nata1999/tables.html). Ambient PAH

concentrations were estimated from the Assessment System for Population Exposure Nationwide (ASPEN) model (http://www.epa.gov/ttnatw01/nata/aspen.html). ASPEN is a computer simulation model used to estimate toxic air pollutant concentrations, which takes into account the rate, location, and height of release, wind speeds and directions, breakdown of the pollutants in the atmosphere, deposition rate, and photochemical transformation into secondary pollutants.

Statistical analysis

Final analyses included 676 cases and 511 controls with known residential location and dust samples analyzed for PAHs. A multiple imputation procedure was applied for instances in which laboratory measurements contained missing data due to concentrations below the limit of detection or when the sample contained other compounds that may have coeluted with the target analyte. Full details of the imputation procedure have been described [26, 32]. Briefly, upper and lower bounds ("intervals") were assigned for each missing datum. Values within each interval were then imputed using Tobit regression assuming a log-normal distribution, which was consistent with the observed distribution of quantified measurements. The imputation procedure was repeated 10 times.

Unconditional logistic regression was used to compute odds ratios (OR) and 95 % confidence intervals (CI) for the associations between NHL and each PAH, summed (total) PAHs, and summed PAHs weighted by their toxic equivalency factors (TEQ). We conducted separate and multinomial logistic regression analyses for NHL subtypes for which we had sufficient cases ($n \geq 25$), grouped according to the World Health Organization classification [33, 34] using the InterLymph consortium guidelines [35, 36]. The NHL subtypes analyzed were diffuse large B cell lymphoma (DLBCL), follicular lymphoma, marginal zone B cell lymphoma, chronic lymphocytic leukemia/small lymphocytic lymphoma (CLL/SLL), all T cell lymphomas, peripheral T cell lymphoma, and lymphoma not otherwise specified (Table 1).

PAH concentrations were categorized into quartiles (≥100 cases) or tertiles (<100 cases) based on the distribution in controls. The linearity of the relationship between PAH concentrations and NHL risk was evaluated by modeling natural log-transformed concentrations as continuous variables. We also evaluated the relationship by modeling risk per 100 ng/g increase in PAH concentration.

All analyses were adjusted for age, sex, and study center. Education (<12, 12–15,>15 years), race (black, white, other), and smoking pack-years (p-y) [non-smoker, <7 p-y, 7–16 p-y, 17–35 p-y, >35 p-y, missing ($n = 714$)] were examined, but did not change OR estimates >10 %, when included individually or together, and were not included in final models. Separate models were fit using the 10 different imputation datasets. The results were combined using the MIANALYZE procedure in SAS version 9.2 (SAS Institute, Inc., Cary, North Carolina) to create a single OR and CI accounting for the variability between the imputed values. In addition, we created models stratified by study center because the measured PAH concentrations in house dust varied greatly between study centers. PAH concentrations in stratified models were categorized based on the distribution among controls within each study center.

As secondary analyses, we evaluated whether home characteristics, demographic variables, and environmental factors were predictive of individual and total PAH concentrations in carpet dust samples of cases and controls. Associations between natural log-transformed PAH concentrations, based on the first imputation, and possible determinants of household PAH concentrations were calculated using multivariate least-squares regression. Results were reported as the percent change in geometric mean (GM) within each level of the individual covariates. Self-reported demographic and home characteristic covariates included: sex, race (black, white, other), education (<12, 12–15, >15), age, smoking pack-years (p-y) (non-smoker, <7 p-y, 7–16 p-y, 17–35 p-y, >35 p-y, missing), case– control status, type of home (single family, other), and the year home was built (<1940, 1940–1969, 1970–1989, >1989). Environmental factors included US Census 2000 block population density (1,000 persons/mile^2), distance to nearest major road and freight route (<100, ≥100 m), distance to nearest railroad (<400, ≥400 m), separate counts of combustion facilities (municipal solid waste incinerators, medical waste incinerators, sewage sludge incinerators, hazardous waste incinerators, cement kilns burning non-hazardous waste, and coal-fired electric generating plants) within 5 km of each home, percentage of developed land (≥20 % impervious surface) within 2 km of each home, and census tract-level estimated ambient PAH concentrations (ng/m^3) from the ASPEN ambient PAH model. All statistical analyses were performed using SAS statistical software version 9.2 (SAS Institute, Inc., Cary, North Carolina). Results were considered statistical significance when two-sided p values were <0.05.

Results

NHL cases and controls in our analyses were similar with respect to study center, gender, race, education level, and smoking status (Table 1). Cases with dust samples were on average slightly younger than controls, with a mean age of 58.9 and 60.3 years ($p = 0.04$), respectively. As described previously [26], participants with dust samples were similar to the study population overall except that they were slightly older and had lived in the interview home longer than participants without dust samples. There was a high degree of correlation between concentrations of individual PAH compounds in the dust samples, with Spearman correlation coefficients among controls ranging from 0.86 to 0.96 (results not shown). The median concentration of total PAHs measured in carpet dust was slightly higher among cases [1,089.0 ng/g, interquartile range (IQR) (603.2–2,624.2)] than controls [1,021.0 ng/g, IQR (530.7–2,950.1)] ($p = 0.04$). Detection rates for the individual PAHs were similar among cases (range 72.9–99.4 %) and controls (range 67.1–100 %) (Supplemental Table 1).

Compared to the lowest quartile of PAH concentration, risks of NHL overall and of the DLBCL and follicular lymphoma subtypes were elevated in the second and third quartiles for many of the PAHs, but not in the highest quartile, indicating a nonlinear relationship (Table 2). The patterns of elevated risk were fairly consistent across the individual PAH compounds, but only a few associations were statistically significant. The associations between tertiles of PAH concentrations and marginal zone lymphoma were not monotonic; many of the ORs were significantly increased for the second tertile of PAH concentration, but there was no association for the highest tertile. Tertiles of PAH concentrations were not associated with CLL/SLL (Table 2). For NHL overall and all the B cell subtypes, there were

no significant associations between PAH concentrations and NHL risk when the natural log of the PAH concentration was analyzed as a continuous variable (Table 2) or for risk per 100 ng/g increase in PAH concentration (not shown).

We observed an increased risk of T cell lymphomas associated with increasing tertiles of PAH exposure (Table 3). Specifically, risk of T cell lymphoma overall was significantly increased among those with the highest tertile of total PAH (OR = 3.04; 95 % CI, 1.09–8.47) and BkF (OR = 3.20; 95 % CI, 1.13–9.11) compared to those with the lowest tertile of the respective residential PAH concentration. Risk was nonsignificantly elevated associated with the highest tertile of concentration of the other PAHs, generally demonstrating a monotonic positive trend with increasing tertiles, although the ORs for a natural log increase in concentrations were not significant. Similar relationships were observed for the subset of peripheral T cell lymphoma cases. Associations between PAHs and NHLs with unspecified subtype followed a pattern similar to NHL overall, but were not significant.

Results for total PAH concentrations and risk of NHL overall were similar when we evaluated risk separately by study center (Supplemental Table 2). Although we had limited power to estimate study center-specific risks by subtype, the patterns were generally similar to those for all centers combined, including for the T cell lymphomas (results not shown).

Determinants of carpet dust PAH

Results for multivariate analyses of the association between total PAH carpet dust concentrations and selected demographic variables, home characteristics, and environmental factors are shown in Table 4. We present results for cases and controls combined because analyses of controls only yielded similar effect estimates (results not shown). Study center was a statistically significant determinant of total PAH concentrations, with the highest adjusted levels found in Detroit (GM = 4,354.7; 95 % CI, 3,269.5–5,799.9), followed by Iowa (GM = 1,374.6; 95 % CI, 995.6–1,898.1), Seattle (GM = 838.0; 95 % CI, 666.2–1,054.0), and Los Angeles (GM = 354.1; 95 % CI, 271.4–462.1). PAH concentrations in homes of white participants were, on average, higher than levels in homes of other races. Participant’s age was positively associated with total PAH concentrations. We observed a 24.2 % lower average total PAH concentration in the homes where the participants had less than 12 years of formal education compared to homes where the participant had 16 or more years of formal education. Adjusted GM measured PAH concentrations did not significantly differ by smoking status or by housing age. Both average annual ambient PAH concentrations, as estimated at the census tract level by the ASPEN model, and the proportion of developed land within 2 km of a residence were positively associated with total PAH concentrations in carpet dust. No significant associations were observed for proximity to major roadways, freight routes, railroads, or industrial combustion facilities (results not shown). Results for center-specific and chemical-specific models were similar (results not shown).

Discussion

In this case–control study, we observed significant positive associations between total PAH and BkF concentrations in residential carpet dust and risk of T cell lymphomas. Although

not statistically significant, risk of T cell lymphomas was also elevated with increasing exposures to each of the six other PAHs analyzed. No significant monotonic exposure–response associations were observed for risk of NHL overall or for B cell lymphomas including DLBCL, follicular lymphoma, marginal zone B cell lymphomas, and CLL/SLL.

Evidence of associations between PAH exposure and NHL risk in human studies is currently limited. In a Canadian cohort study of aluminum reduction plant workers, an industry with potentially high PAH exposures, there was evidence of a positive exposure–response relationship between BaP exposure, assessed via a job exposure matrix (JEM), and NHL risk (p trend <0.01) [12]. Investigations of aluminum reduction plant workers in Quebec observed nonsignificant excesses of NHL incidence and mortality among workers compared to the general population [16]. In regard to PAH exposure, specifically, the authors reported no associations between BaP and risk of NHL incidence or mortality. In addition, no associations between JEM-assessed occupational PAH exposure and NHL risk were observed in a case–control study conducted in Sweden [13] or among a retrospective cohort of workers employed at a California aerospace company [14].

NHL is comprised of a group of related, yet distinct, lymphoid diseases. Studies of cigarette smoking, a major source of PAH exposure in the general population, and NHL have found associations to vary by lymphoma subtype [23, 37–42]. A pooled analysis of 20 case–control studies participating in the InterLymph consortium observed significant positive associations between smoking duration and risks of peripheral T cell, follicular, lymphoplasmacytic lymphoma/Walderstrom macroglobulinemia, and marginal zone lymphomas, but not other subtypes [22]. Morton et al. [21, 22] further describe some additional risk factors that show heterogeneity among major NHL subtypes, although the biological mechanisms are not fully understood. In addition to increased risk of T cell lymphomas overall, we observed an elevated risk of peripheral T cell lymphoma specifically, although there was limited power to detect an association due to a small number of cases. Although no human data are available, T cell lymphomas have been produced in the offspring of mice exposed to dibenzo[a,l]pyrene (DbP) during pregnancy [5, 6]. The potential biological mechanisms for the association of PAH exposure and risk of T cell lymphomas are not fully understood. One possible pathway for carcinogenesis is the bioactivation of DbP via the *CYP1B1* enzyme, which is highly expressed in the thymus where T cells develop [43].

In general, carpet dust PAH concentrations measured in our study were lower than previously reported levels in Durham, NC, USA [44, 45] and Ottawa, Canada [46]. However, measured concentrations in this multicenter study were considerably higher than those measured in the California Childhood Leukemia Study (CCLS), a recent case–control study conducted in the San Francisco Bay area and the Central Valley, USA [47]. The large degree of variability in PAH concentrations reported in these four North American studies is consistent with the substantial geographic variability we observed between our study centers.

Smoking status, which has been associated with household PAH concentrations in other studies [48, 49], was not a significant determinant of PAH concentrations in our study.

However, we had information on the smoking status of the study participant only and not for all household members. Carpet dust in this study may also reflect many sources of PAHs including estimated outdoor air concentrations, which, consistent with our findings, was found to be a significant predictor of house dust PAH concentrations in the CCLS [47].

In the CCLS [47], households in which the mother was Hispanic had lower house dust PAH concentrations than households where the mother was not Hispanic. The authors noted that Hispanic mothers vacuumed carpets more frequently and were less likely to live in an urban area than non-Hispanic mothers. In this study, we did not find an association between PAH concentrations and population density. However, the percent developed land near homes and air concentrations of PAHs were significant predictors indicating that population density alone was not a good proxy for residential exposure. The associations with respondent race and education were unexpected. It is possible that the higher PAH concentrations measured in the households of white participants in our study and the association we observed with education are due to behavioral differences that were not measured.

The large number of participants with dust samples and the use of an exposure measure that is not affected by recall bias are strengths of this study. However, there are limitations to the use of household carpet dust to assess PAH exposures. House dust is not a direct measure of exposure and post-diagnostic samples may not be fully representative of pre-diagnostic dust levels, which were not available due to the case–control design. However, carpet dust can act as a sink for PAHs [24] and therefore may be representative of long-term indoor exposure. In analyses of PAH concentrations in household dust from the CCLS, analytic and within-household temporal variability were approximately 90 % as large as between-household variability [48]. Although the authors observed greater between-household variability for higher molecular weight PAHs, such as those analyzed in our study, these findings point to the potential for misclassification of exposure when using a single carpet dust PAH measurements in epidemiologic studies. Other limitations include the limited statistical power to investigate less common NHL subtypes due to small numbers of cases, incomplete information on smoking status, and the possibility of chance findings due to analyses of multiple PAHs and lymphoma subtypes.

We were unable to explain the non-monotonic risk patterns we observed for NHL overall and for B cell lymphoma subtypes. Although the determinants we investigated did not substantially differ by quartile of measured PAH dust concentration, it is possible dust PAH is a proxy for an unidentified risk factor. However, total PAHs were not significantly correlated with polychlorinated biphenyls or with dioxins and furans measured in a subset of participants with dust (not shown). In addition, the pattern of ORs we observed were consistent across study sites and thus independent of PAH concentration. Our analyses indicate outdoor PAH concentrations are important determinants of indoor levels. The large difference in measured PAH concentrations we observed among our study sites may therefore be due to variability in PAH sources and regional differences in physical and biological degradation patterns. As a result, our findings should be interpreted with caution because dust PAH may be reflective of relative (rank) exposure more than absolute PAH exposure.

The results of our investigation suggest an increased risk of T cell lymphomas associated with residential carpet dust PAH concentrations, including BkF, specifically. Our results are novel and provide additional support for the importance of epidemiologic research on specific histologic subtypes of NHL in order to distinguish potential etiologic heterogeneity. Further evaluation of the associations between PAH exposures and NHL risk by subtype using validated direct exposure metrics is warranted.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

1. U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease. Toxicological profile for polycyclic aromatic hydrocarbons. 1995. Retrieved from http://www.atsdr.cdc.gov/toxprofiles/tp.asp?id=122&tid=25
2. IARC. Polynuclear aromatic compounds. World Health Organization; Lyon: 1983.
3. IARC. Some non-heterocyclic polycyclic aromatic hydrocarbons and some related exposures. World Health Organization; Lyon: 2007.
4. Boffetta P, Jourenkova N, Gustavsson P. Cancer risk from occupational and environmental exposure to polycyclic aromatic hydrocarbons. Cancer Causes Control. 1997; 8:444–472. [PubMed: 9498904]
5. Yu Z, Loehr CV, Fischer KA, et al. In utero exposure of mice to dibenzo a, l pyrene produces lymphoma in the offspring: role of the aryl hydrocarbon receptor. Cancer Res. 2006; 66:755–762. [PubMed: 16424006]
6. Castro DJ, Lohr CV, Fischer KA, Pereira CB, Williams DE. Lymphoma and lung cancer in offspring born to pregnant mice dosed with dibenzo a, l pyrene: the importance of in utero vs. lactational exposure. Toxicol Appl Pharmacol. 2008; 233:454–458. [PubMed: 18848954]
7. Qing WG, Conti CJ, LaBate M, Johnston D, Slaga TJ, MaCleod MC. Induction of mammary cancer and lymphoma by multiple, low oral doses of 7,12-dimethylbenz a anthracene in SENCAR mice. Carcinogenesis. 1997; 18:553–559. [PubMed: 9067556]
8. Zienolddiny S, Ryberg D, Svendsrud DH, et al. Msh2 deficiency increases susceptibility to benzo a pyrene-induced lymphomagenesis. Int J Cancer. 2006; 118:2899–2902. [PubMed: 16381012]
9. Jemal A, Siegel R, Ward E, Murray T, Xu JQ, Thun MJ. Cancer statistics, 2007. CA-Cancer J Clin. 2007; 57:43–66. [PubMed: 17237035]
10. Hartge P, Devesa SS. Quantification of the impact of known risk-factors on time trends in non-Hodgkins-lymphoma incidence. Cancer Res. 1992; 52(Suppl19):5566s–5569s. [PubMed: 1394175]
11. Hartge, P., Wang, SS., Bracci, PM., Devesa, S., Holly, EA. Non-Hodgkin lymphoma. In: Shottenfeld, D., Fraumeni, JF., editors. Cancer epidemiology and prevention. 3rd. Oxford University Press; New York: 2006. p. 898-918.
12. Spinelli JJ, Demers PA, Le ND, et al. Cancer risk in aluminum reduction plant workers (Canada). Cancer Causes Control. 2006; 17:939–948. [PubMed: 16841261]
13. Dryver E, Brandt L, Kauppinen T, Olsson H. Occupational exposures and non-Hodgkin's lymphoma in southern Sweden. Int J Occup Environ Health. 2004; 10:13–21. [PubMed: 15070021]

14. Zhao YX, Krishnadasan A, Kennedy N, Morgenstern H, Ritz B. Estimated effects of solvents and mineral oils on cancer incidence and mortality in a cohort of aerospace workers. Am J Ind Med. 2005; 48:249–258. [PubMed: 16167347]
15. Gibbs GW, Labreche F, Busque MA, Duguay P. Mortality and cancer incidence in aluminum smelter workers: a 5-year update. J Occup Environ Med. 2014; 56(7):739–764. [PubMed: 24988102]
16. Gibbs GW, Labreche F. Cancer risks in aluminum reduction plant workers: a review. J Occup Environ Med. 2014; 56(5 Suppl):S40–S59.
17. Scherer G, Frank S, Riedel K, Meger-Kossien I, Renner T. Biomonitoring of exposure to polycyclic aromatic hydrocarbons of nonoccupationally exposed persons. Cancer Epidemiol Biomarkers Prev. 2000; 9:373–380. [PubMed: 10794481]
18. Troy JD, Hartge P, Weissfeld JL, et al. Associations between anthropometry, cigarette smoking, alcohol consumption, and non-Hodgkin lymphoma in the prostate, lung, colorectal, and ovarian cancer screening trial. Am J Epidemiol. 2010; 171:1270–1281. [PubMed: 20494998]
19. Schollkopf C, Smedby KE, Hjalgrim H, et al. Cigarette smoking and risk of non-Hodgkin's lymphoma—a population-based case–control study. Cancer Epidemiol Biomarkers Prev. 2005; 14:1791–1796. [PubMed: 16030118]
20. Lim U, Morton LM, Subar AF, et al. Alcohol, smoking, and body size in relation to incident Hodgkin's and non-Hodgkin's lymphoma risk. Am J Epidemiol. 2007; 166:697–708. [PubMed: 17596266]
21. Morton LM, Wang SS, Cozen W, et al. Etiologic heterogeneity among non-Hodgkin lymphoma subtypes. Blood. 2008; 112:5150–5160. [PubMed: 18796628]
22. Morton LM, Slager SL, Cerhan JR, et al. Etiologic heterogeneity among non-Hodgkin lymphoma subtypes: the InterLymph non-Hodgkin lymphoma subtypes project. J Natl Cancer Inst Monogr. 2014; 2014:130–144. [PubMed: 25174034]
23. Morton LM, Hartge P, Holford TR, et al. Cigarette smoking and risk of non-Hodgkin lymphoma: a pooled analysis from the international lymphoma epidemiology consortium (InterLymph). Cancer Epidemiol Biomarkers Prev. 2005; 14:925–933. [PubMed: 15824165]
24. Roberts, JW., Wallace, LA., Camann, DP., et al. Monitoring and reducing exposure of infants to pollutants in house dust. In: Whitacre, DM., editor. Reviews of environmental contamination and toxicology. Vol. 201. Springer; New York: 2009. p. 1-39.
25. Chatterjee N, Hartge P, Cerhan JR, et al. Risk of non-Hodgkin's lymphoma and family history of lymphatic, hematologic, and other cancers. Cancer Epidemiol Biomarkers Prev. 2004; 13:1415–1421. [PubMed: 15342441]
26. Colt JS, Severson RK, Lubin J, et al. Organochlorines in carpet dust and non-Hodgkin lymphoma. Epidemiology. 2005; 16:516–525. [PubMed: 15951670]
27. De Roos AJ, Davis S, Colt JS, et al. Residential proximity to industrial facilities and risk of non-Hodgkin lymphoma. Environ Res. 2010; 110:70–78. [PubMed: 19840879]
28. Colt JS, Lubin J, Camann D, et al. Comparison of pesticide levels in carpet dust and self-reported pest treatment practices in four US sites. J Expo Anal Environ Epidemiol. 2004; 14:74–83. [PubMed: 14726946]
29. U.S. EPA. An inventory of sources and environmental releases of dioxin-like compounds in the United States for the years 1987, 1995, and 2000. 2006
30. Homer C, Dewitz J, Fry J, et al. Completion of the 2001 national land cover database for the conterminous United States. Photogramm Eng Remote Sens. 2007; 73:337–341.
31. DellaValle CT, Wheeler DC, Deziel NC, et al. Environmental determinants of polychlorinated biphenyl concentrations in residential carpet dust. Environ Sci Technol. 2013; 47:10405–10414. [PubMed: 23952055]
32. Lubin JH, Colt JS, Camann D, et al. Epidemiologic evaluation of measurement data in the presence of detection limits. Environ Health Perspect. 2004; 112:1691–1696. [PubMed: 15579415]
33. Jaffe, ES., Harris, NL., Stein, H., Vardiman, JW. World Health Organization classification of tumours of haematopoietic and lymphoid tissues. IARC Press; Lyon: 2001.
34. Swerdlow, SH., Campo, E., Harris, NL., et al. World Health Organization classification of tumours of haematopoietic and lymphoid tissues. 4th. IARC Press; Lyon: 2008.

35. Morton LM, Turner JJ, Cerhan JR, et al. Proposed classification of lymphoid neoplasms for epidemiologic research from the Pathology Working Group of the International Lymphoma Epidemiology Consortium (InterLymph). Blood. 2007; 110:695–708. [PubMed: 17389762]
36. Turner JJ, Morton LM, Linet MS, et al. InterLymph hierarchical classification of lymphoid neoplasms for epidemiologic research based on the WHO classification (2008): update and future directions. Blood. 2010; 116:E90–E98. [PubMed: 20699439]
37. Morton LM, Holford TR, Leaderer B, et al. Cigarette smoking and risk of non-Hodgkin lymphoma subtypes among women. Br J Cancer. 2003; 89:2087–2092. [PubMed: 14647142]
38. Stagnaro E, Tumino R, Parodi S, et al. Non-Hodgkins lymphoma and type of tobacco smoke. Cancer Epidemiol Biomarkers Prev. 2004; 13:431–437. [PubMed: 15006920]
39. Stagnaro E, Ramazzotti V, Crosignani P, et al. Smoking and hematolymphopoietic malignancies. Cancer Causes Control. 2001; 12:325–334. [PubMed: 11456228]
40. Parker AS, Cerhan JR, Dick F, et al. Smoking and risk of non-Hodgkin lymphoma subtypes in a cohort of older women. Leuk Lymphoma. 2000; 37:341–349. [PubMed: 10752985]
41. Herrinton LJ, Friedman GD. Cigarette smoking and risk of non-Hodgkin's lymphoma subtypes. Cancer Epidemiol Biomarkers Prev. 1998; 7:25–28. [PubMed: 9456239]
42. Peach HG, Barnett NE. Critical review of epidemiological studies of the association between smoking and non-Hodgkin's lymphoma. Hematol Oncol. 2001; 19:67–80. [PubMed: 11438976]
43. Choudhary D, Jansson I, Stoilov I, Sarfarazi M, Schenkman JB. Expression patterns of mouse and human CYP orthologs (families 1–4) during development and in different adult tissues. Arch Biochem Biophys. 2005; 436:50–61. [PubMed: 15752708]
44. Chuang JC, Callahan PJ, Menton RG, Gordon SM, Lewis RG, Wilson NK. Monitoring methods for polycyclic aromatic-hydrocarbons and their distribution in-house dust and track-in soil. Environ Sci Technol. 1995; 29:494–500. [PubMed: 22201397]
45. Lewis RG, Fortune CR, Willis RD, Camann DE, Antley JT. Distribution of pesticides and polycyclic aromatic hydrocarbons in house dust as a function of particle size. Environ Health Perspect. 1999; 107:721–726. [PubMed: 10464072]
46. Maertens RM, Yang XF, Zhu JP, Gagne RW, Douglas GR, White PA. Mutagenic and carcinogenic hazards of settled house dust I: polycyclic aromatic hydrocarbon content and excess lifetime cancer risk from preschool exposure. Environ Sci Technol. 2008; 42:1747–1753. [PubMed: 18441830]
47. Whitehead T, Metayer C, Gunier RB, et al. Determinants of polycyclic aromatic hydrocarbon levels in house dust. J Expo Sci Environ Epidemiol. 2011; 21:123–132. [PubMed: 20040932]
48. Whitehead TP, Metayer C, Petreas M, Does M, Buffler PA, Rappaport SM. Polycyclic aromatic hydrocarbons in residential dust: sources of variability. Environ Health Perspect. 2013; 121:543–550. [PubMed: 23461863]
49. Hoh E, Hunt RN, Quintana PJE, et al. Environmental tobacco smoke as a source of polycyclic aromatic hydrocarbons in settled household dust. Environ Sci Technol. 2012; 46:4174–4183. [PubMed: 22397504]

Table 1

Characteristics of NHL cases (n = 676) and frequency-matched controls (n = 511)[a]

	Cases (%)	Controls (%)
Histology		
DLBCL	208 (17.5)	
Marginal zone lymphoma	61 (5.1)	
Burkitt lymphoma/leukemia	8 (0.7)	
CLL/SLL	68 (5.7)	
Follicular lymphoma	157 (13.2)	
Lymphoplasmarytic lymphoma	19 (1.6)	
Mantle cell lymphoma	25 (2.1)	
Peripheral T cell lymphomas	25 (2.1)	
Mycosis fungoides	15 (1.3)	
Not otherwise specified	90 (7.6)	
Study center		
Detroit	127 (18.8)	77 (15.1)
Iowa	195 (28.9)	147 (28.8)
Los Angeles	169 (25.0)	126 (24.7)
Seattle	185 (27.4)	161 (31.5)
Mean age (*SD*)	58.9 (11.3)	60.3 (11.0)
Gender		
Male	366 (54.1)	271 (53.0)
Female	310 (45.9)	240 (47.0)
Race		
White	597 (88.3)	442 (86.5)
Black	42 (6.2)	45 (8.8)
Other/unknown	37 (5.5)	24 (4.7)
Education		
<12 years	59 (8.7)	48 (9.4)
12–15 years	436 (64.5)	311 (60.9)
16+ years	181 (26.8)	152 (29.8)
Smoking status[b]		
Never	125 (18.5)	105 (20.6)
Former	91 (13.5)	91 (17.8)
Current	38 (5.6)	23 (4.5)
Missing/N/A	422 (62.4)	292 (57.1)

DLBCL diffuse large B cell lymphoma, *CLL/SLL* chronic lymphocytic leukemia/small lymphocytic lymphoma

[a]Cases and controls with dust samples analyzed for polycyclic aromatic hydrocarbons and known location of sampled home

[b]Due to split questionnaire design, smoking status was only assessed among a subset of participants

Table 2

Associations between total and individual PAHs and NHL overall and by major B cell subtypes

PAH Quartiles (ng/g dust)	All NHLs		DLBCL		Follicular		PAH Tertiles (ng/g dust)	Marginal zone		CLL/SLL	
	Cases/controls	OR[a] (95% CI)	Cases/controls	OR[a] (95% CI)	Cases/controls	OR[a] (95% CI)		Cases/controls	OR[a] (95% CI)	Cases/controls	OR[a] (95% CI)
Total PAH[b]							*Total PAH*[b]				
0–530.7	139/127	1.00	45/127	1.00	33/127	1.00	0–658.2	16/170	1.00	21/170	1.00
530.8–1,021.0	184/128	1.34 (0.96–1.87)	51/128	1.15 (0.71–1.85)	41/128	1.30 (0.77–2.21)	658.3–1,873.8	33/170	2.53 (1.29–4.94)	24/170	1.15 (0.61–2.19)
1,021.1–2,950.0	199/128	1.40 (1.00–1.97)	61/128	1.30 (0.81–2.10)	47/128	1.36 (0.81–2.30)	≥1,873.9	12/171	0.99 (0.38–2.60)	23/171	1.03 (0.49–2.15)
≥2,950.1	154/128	0.85 (0.56–1.29)	51/128	0.86 (0.47–1.57)	36/128	0.86 (0.45–1.63)					
Continuous[c]		0.94 (0.85–1.05)		0.96 (0.83–1.11)		0.93 (0.80–1.10)	Continuous[c]		1.02 (0.79–1.30)		0.90 (0.71–1.13)
TEQ[d]							*TEQ*[d]				
0–160.9	133/127	1.00	46/127	1.00		1.00	0–211.4	15/170	1.00	20/170	1.00
161.0–351.2	180/128	1.39 (0.99–1.95)	49/128	1.14 (0.71–1.85)	26/127	1.86 (1.07–3.22)	211.5–640.7	36/171	2.40 (1.25–4.62)	22/171	1.12 (0.58–2.16)
351.3–1,026.5	207/129	1.53 (1.10–2.15)	59/129	1.31 (0.81–2.10)	44/128	1.95 (1.13–3.37)	≥640.8	10/170	0.68 (0.23–1.96)	26/170	1.35 (0.66–2.76)
≥1,026.6	156/127	0.95 (0.63–1.44)	54/127	1.01 (0.57–1.78)	51/129	1.16 (0.60–2.23)					
Continuous[c]		0.98 (0.89–1.08)		0.86 (0.70–1.06)	36/127	0.97 (0.83–1.12)	Continuous[c]		1.02 (0.81–1.29)		1.09 (0.84–1.40)
Benz(a)anthracene							*Benz(a)anthracene*				
0–56.0	145/127	1.00	50/127	1.00	32/127	1.00	0–70.4	17/170	1.00	22/170	1.00
56.1–113.0	171/127	1.23 (0.88–1.72)	45/127	0.95 (0.58–1.53)	40/127	1.31 (0.77–2.24)	70.5–210.9	32/170	2.59 (1.32–5.09)	24/170	1.04 (0.55–1.97)
113.1–347.9	200/128	1.38 (0.98–1.95)	58/128	1.14 (0.70–1.85)	47/128	1.38 (0.80–2.37)	≥211.0	12/171	1.08 (0.41–2.83)	22/171	0.88 (0.41–1.86)
≥348.0	160/129	0.91 (0.61–1.38)	55/129	0.91 (0.51–1.61)	38/129	0.97 (0.52–1.84)					
Continuous[c]		0.96 (0.87–1.06)		0.98 (0.85–1.12)		0.94 (0.81–1.10)	Continuous[c]		1.04 (0.82–1.30)		0.92 (0.73–1.15)
Benzo(a)pyrene							*Benzo(a)pyrene*				
0–61.6	147/128	1.00	51/128	1.00	33/128	1.00	0–78.5	21/170	1.00	24/170	1.00
61.7–121.0	148/126	1.02 (0.73–1.43)	42/126	0.84 (0.52–1.36)	33/126	1.02 (0.59–1.75)	78.6–239.9	26/170	1.38 (0.73–2.61)	21/170	0.85 (0.45–1.61)
121.1–391.9	223/129	1.48 (1.07–2.06)	59/129	1.11 (0.70–1.76)	56/129	1.59 (0.95–2.64)	≥240.0	14/171	0.92 (0.39–2.20)	23/171	0.89 (0.43–1.82)
≥392.0	158/128	0.88 (0.59–1.33)	56/128	0.92 (0.53–1.61)	35/128	0.85 (0.45–1.61)					
Continuous[c]		0.99 (0.90–1.09)		1.01 (0.89–1.14)		0.97 (0.84–1.12)	Continuous[c]		1.02 (0.82–1.27)		0.98 (0.80–1.19)
Benzo(b)fluoranthene							*Benzo(b)fluoranthene*				
0–123.0	134/126	1.00	41/126	1.00	29/126	1.00	0–152.0	17/170	1.00	23/170	1.00

PAH Quartiles (ng/g dust)	All NHLs		DLBCL		Follicular		PAH Tertiles (ng/g dust)	Marginal zone		CLL/SLL	
	Cases/controls	OR[a] (95% CI)	Cases/controls	OR[a] (95% CI)	Cases/controls	OR[a] (95% CI)		Cases/controls	OR[a] (95% CI)	Cases/controls	OR[a] (95% CI)
123.1–247.0	183/128	1.38 (0.99–1.93)	56/128	1.42 (0.88–2.30)	41/128	1.48 (0.86–2.54)	152.1–499.9	33/170	2.46 (1.27–4.71)	22/170	0.90 (0.48–1.71)
247.1–797.9	211/129	1.53 (1.09–2.15)	63/129	1.45 (0.89–2.38)	53/129	1.70 (0.99–2.90)	≥500.0	11/171	0.84 (0.31–2.28)	23/171	0.91 (0.43–1.90)
≥798.0	148/128	0.82 (0.54–1.26)	48/128	0.85 (0.45–1.57)	34/128	0.90 (0.46–1.76)					
Continuous[c]		0.94 (0.85–1.04)		0.96 (0.84–1.11)		0.95 (0.82–1.11)	Continuous[c]		1.00 (0.78–1.26)		0.89 (0.72–1.12)
Benzo(k)fluoranthene							*Benzo(k)fluoranthene*				
0–42.1	130/128	1.00	40/128	1.00	30/128	1.00	0–56.5	13/170	1.00	25/170	1.00
42.2–88.6	183/129	1.46 (1.04–2.04)	48/129	1.31 (0.80–2.15)	43/129	1.52 (0.89–2.59)	56.6–156.9	33/170	3.35 (1.65–6.83)	20/170	0.73 (0.38–1.39)
88.7–264.9	208/125	1.66 (1.18–2.34)	68/125	1.88 (1.15–3.08)	49/125	1.65 (0.96–2.83)	≥157.0	15/171	2.03 (0.80–5.16)	23/171	0.80 (0.39–1.67)
≥265.0	155/129	0.94 (0.62–1.43)	52/129	1.09 (0.60–2.00)	35/129	0.91 (0.47–1.76)					
Continuous[c]		0.99 (0.90–1.09)		1.03 (0.90–1.17)		0.97 (0.84–1.12)	Continuous[c]		1.07 (0.86–1.34)		0.91 (0.74–1.12)
Chrysene							*Chrysene*				
0–128.0	137/126	1.00	44/126	1.00	30/126	1.00	0–167.0	19/170	1.00	23/170	1.00
128.1–247.0	193/129	1.38 (0.99–1.92)	58/129	1.24 (0.78–1.98)	45/129	1.47 (0.87–2.50)	167.1–422.9	26/170	1.59 (0.83–3.07)	24/170	1.03 (0.55–1.91)
247.1–673.9	195/128	1.38 (0.99–1.94)	59/128	1.27 (0.78–2.05)	44/128	1.39 (0.81–2.39)	≥423.0	16/171	1.23 (0.53–2.82)	21/171	0.80 (0.38–1.68)
≥674.0	151/128	0.85 (0.56–1.27)	47/128	0.76 (0.42–1.36)	38/128	1.05 (0.57–1.96)					
Continuous[c]		0.92 (0.82–1.02)		0.93 (0.80–1.08)		0.92 (0.78–1.09)	Continuous[c]		1.02 (0.80–1.31)		0.85 (0.67–1.09)
Dibenz(a,h)anthracene							*Dibenz(a,h)anthracene*				
0–12.4	123/127	1.00	42/127	1.00	28/127	1.00	0–21.4	15/171	1.00	23/171	1.00
12.5–34.0	209/128	1.74 (1.24–2.42)	59/128	1.52 (0.94–2.44)	49/128	1.82 (1.07–3.10)	21.5–59.9	34/169	2.20 (1.14–4.24)	20/169	0.90 (0.47–1.72)
34.1–91.1	192/129	1.52 (1.08–2.13)	57/129	1.39 (0.86–2.25)	43/129	1.51 (0.88–2.61)	≥60.0	12/171	0.91 (0.35–2.38)	25/171	1.11 (0.55–2.23)
≥91.2	152/127	0.94 (0.62–1.43)	50/127	0.93 (0.51–1.68)	37/127	1.07 (0.56–2.04)					
Continuous[c]		1.00 (0.92–1.09)		1.02 (0.91v1.15)		1.01 (0.89–1.15)	Continuous[c]		1.03 (0.85–1.26)		0.98 (0.83–1.17)
Indeno(1,2,3-c,d)pyrene							*Indeno(1,2,3-c,d)pyrene*				
0–70.4	141/127	1.00	45/127	1.00	34/127	1.00	0–88.9	13/170	1.00	22/170	1.00
70.5–149.0	185/128	1.34 (0.96–1.86)	58/128	1.40 (0.88–2.25)	40/128	1.26 (0.74–2.13)	89.0–270.9	38/170	3.06 (1.54–6.08)	24/170	1.10 (0.58–2.07)
149.1–450.9	201/128	1.39 (0.99–1.94)	60/128	1.29 (0.80–2.08)	48/128	1.38 (0.82–2.31)	≥271.0	10/171	0.79 (0.27–2.33)	22/171	0.91 (0.43–1.93)
≥451.0	149/128	0.78 (0.51–1.19)	45/128	0.68 (0.37–1.27)	35/128	0.79 (0.41–1.51)					
Continuous[c]		0.95 (0.86–1.04)		0.96 (0.84–1.10)		0.92 (0.80–1.06)	Continuous[c]		0.99 (0.79–1.23)		0.93 (0.75–1.14)

[a]Models adjusted for age, sex, and study center

[b] Total PAH = benz(a)anthracene + benzo(a)pyrene + benzo(b)fluoranthene + benzo(k)fluoranthene + chrysene + dibenz(a,h)anthracene + indeno(1,2,3-c,d)pyrene

[c] OR for one ln(PAH) increase in concentration (ng/g dust)

[d] TEQ = sum of benz(a)anthracene + benzo(a)pyrene + benzo(b)fluoranthene + benzo(k)fluoranthene + chrysene + dibenz(a,h)anthracene + indeno(1,2,3-c,d)pyrene, each weighted by their toxic equivalency factor (TEF)

Table 3

Associations between total and individual PAHs and T cell lymphomas and NHL not otherwise specified

PAH Tertiles (ng/g dust)	All T cell lymphomas		Peripheral T cell		NOS	
	Cases/controls	OR[a] (95 % CI)	Cases/controls	OR[a] (95 % CI)	Cases/controls	OR[a] (95 % CI)
Total PAH[b]						
0–658.2	7/170	1.00	4/170	1.00	27/170	1.00
658.3–1,873.8	13/170	1.95 (0.74–5.12)	9/170	2.58 (0.75–8.84)	41/170	1.66 (0.95–2.89)
≥1,873.9	21/171	3.04 (1.09–8.47)	12/171	3.59 (0.94–13.67)	22/171	0.51 (0.22–1.17)
Continuous[c]		1.03 (0.79–1.35)		1.06 (0.76–1.48)		0.82 (0.66–1.02)
TEQ[d]						
0–211.4	7/170	1.00	4/170	1.00	28/170	1.00
211.5–640.7	15/171	2.20 (0.86–5.64)	10/171	2.69 (0.81–8.96)	36/171	1.28 (0.74–2.23)
≥640.8	19/170	2.43 (0.85–6.90)	11/170	2.96 (0.76–11.48)	26/170	0.69 (0.32–1.48)
Continuous[c]		1.09 (0.84–1.40)		1.09 (0.80–1.49)		0.86 (0.71–1.06)
Benz(a)anthracene						
0–70.4	9/170	1.00	6/170	1.00	29/170	1.00
70.5–210.9	13/170	1.55 (0.62–3.85)	8/170	1.59 (0.51–4.93)	35/170	1.39 (0.79–2.46)
≥211.0	19/171	1.98 (0.73–5.41)	11/171	2.09 (0.59–7.34)	26/171	0.76 (0.36–1.63)
Continuous[c]		1.06 (0.82–1.37)		1.07 (0.78–1.46)		0.85 (0.69–1.04)
Benzo(a)pyrene						
0–78.5	8/170	1.00	6/170	1.00	28/170	1.00
78.6–239.9	14/170	1.84 (0.74–4.61)	8/170	1.52 (0.50–4.63)	37/170	1.42 (0.81–2.47)
≥240.0	19/171	2.15 (0.78–5.94)	11/171	1.99 (0.57–6.91)	25/171	0.68 (0.32–1.48)
Continuous[c]		1.04 (0.82–1.33)		1.09 (0.80–1.46)		0.91 (0.75–1.09)
Benzo(b)fluoranthene						
0–152.0	9/170	1.00	6/170	1.00	25/170	1.00
152.1–499.9	15/170	1.80 (0.75–4.30)	9/170	1.72 (0.58–5.06)	43/170	1.91 (1.09–3.34)
≥500.0	17/171	1.47 (0.52–4.11)	10/171	1.62 (0.45–5.81)	22/171	0.59 (0.25–1.37)
Continuous[c]		0.99 (0.76–1.29)		1.03 (0.74–1.43)		0.83 (0.68–1.02)
Benzo(k)fluoranthene						

PAH Tertiles (ng/g dust)	All T cell lymphomas		Peripheral T cell		NOS	
	Cases/controls	OR[a] (95 % CI)	Cases/controls	OR[a] (95 % CI)	Cases/controls	OR[a] (95 % CI)
0–56.5	7/170	1.00	5/170	1.00	28/170	1.00
56.6–156.9	13/170	2.07 (0.78–5.48)	8/170	1.97 (0.60–6.41)	35/170	1.39 (0.79–2.43)
≥157.0	21/171	3.20 (1.13–9.11)	12/171	3.10 (0.85–11.39)	27/171	0.81 (0.38–1.74)
Continuous[c]		1.06 (0.83–1.36)		1.09 (0.80–1.48)		0.89 (0.73–1.07)
Chrysene						
0–167.0	7/170	1.00	4/170	1.00	29/170	1.00
167.1–422.9	14/170	2.02 (0.78–5.20)	10/170	2.64 (0.80–8.76)	40/170	1.44 (0.84–2.47)
≥423.0	20/171	2.68 (0.97–7.42)	11/171	2.83 (0.75–10.73)	21/171	0.43 (0.19–0.98)
Continuous[c]		1.02 (0.77–1.35)		1.04 (0.73–1.47)		0.77 (0.62–0.97)
Dibenz(a,h)anthracene						
0–21.4	7/171	1.00	4/171	1.00	27/171	1.00
21.5–59.9	15/169	2.27 (0.89–5.83)	10/169	2.78 (0.83–9.31)	39/169	1.42 (0.82–2.46)
≥60.0	19/171	2.32 (0.83–6.53)	11/171	2.74 (0.72–10.39)	24/171	0.60 (0.27–1.30)
Continuous[c]		1.12 (0.90–1.41)		1.10 (0.83–1.45)		0.86 (0.74–1.02)
Indeno(1,2,3-c,d)pyrene						
0–88.9	6/170	1.00	3/170	1.00	27/170	1.00
89.0–270.9	17/170	3.13 (1.18–8.32)	11/170	4.41 (1.17–16.62)	34/170	1.29 (0.73–2.27)
≥271.0	18/171	2.53 (0.83–7.73)	11/171	4.07 (0.92–17.91)	29/171	0.93 (0.45–1.92)
Continuous[c]		1.08 (0.84–1.38)		1.12 (0.83–1.51)		0.90 (0.75–1.09)

[a] Models adjusted for age, sex, and study center

[b] Total PAH = benz(a)anthracene + benzo(a)pyrene + benzo(b)fluoranthene + benzo(k)fluoranthene + chrysene + dibenz(a,h)anthracene + indeno(1,2,3-c,d)pyrene

[c] OR per one ln(PAH) increase in concentration (ng/g dust)

[d] TEQ = sum of benz(a)anthracene + benzo(a)pyrene + benzo(b)fluoranthene + benzo(k)fluoranthene + chrysene + dibenz(a,h)anthracene + indeno(1,2,3-c,d)pyrene, each weighted by their toxic equivalency factor (TEF)

Table 4

Multivariate analysis of determinants of natural log-transformed total PAH levels in residential carpet dust (n = 1,187)

	β[a]	% Change in geometric mean[a]	Adjusted geometric mean[a] (ng/g dust)	*p* value
Study center				
Detroit	1.65	420.7	4,354.7 (3,269.5–5,799.9)	<0.01
Iowa	0.50	64.0	1,374.6 (995.6–1,898.1)	<0.01
Los Angeles	−0.86	−57.7	354.1 (271.4–462.1)	<0.01
Seattle	Reference		838.0 (666.2–1,054.0)	
Gender				
Male	0.03	3.3	1,173.6 (953.8–1,443.8)	0.61
Female	Reference		1,135.7 (921.4–1,400.0)	
Case status				
Control	0.09	9.2	1,206.6 (979.9–1,485.8)	0.17
Case	Reference		1,104.6 (897.1–1,359.9)	
Race				
Black	0.02	1.6	1,034.8 (773.8–1,383.8)	0.93
White	0.36	43.3	1,459.6 (1,237.9–1,720.9)	0.02
Other/unknown	Reference		1,018.7 (738.8–1,404.7)	
Age	0.01	0.6		0.03
Education				
<12 years	−0.28	−24.2	995.6 (760.5–1,303.4)	0.03
12–15 years	−0.11	−10.4	1,176.6 (962.9–1,437.8)	0.14
16+ years	Reference		1,313.4 (1,052.4)	
Smoking status				
Never	−0.02	−1.8	1,156.8 (917.7–1,458.1)	0.91
Former	0.01	0.8	1,186.8 (931.9–1,511.6)	0.96
Current	Reference		1,177.6 (846.3–1,638.3)	
Missing	−0.07	−6.7	1,098.8 (915.4–1,319.0)	0.64
Home type				
Single family	Reference		1,172.3 (957.6–1,435.1)	
other/missing	−0.03	−3.0	1,136.9 (902.7–1,432.0)	0.73
Year home built				
1990–1999	0.04	3.6	1,231.9 (1,009.0–1,503.9)	0.70
1970–1989	0.07	7.7	1,280.1 (1,021.3–1,604.6)	0.52
1940–1969	0.14	15.2	1,367.9 (1,125.3–1,666.4)	0.11
Before 1940	Reference		1,188.6 (968.0–1,459.3)	
Unknown	−0.40	−32.8	799.0 (432.5–1,476.3)	0.21
Census tract[b] *(ppsm/10,000)*	−0.02	−2.0		0.94
ln [*ambient total PAHs (ng/m*^{3}*)*][c]	0.02	2.3		<0.01
% Developed land[d] *(2 km)*	0.01	0.5		<0.01

	β[a]	% Change in geometric mean[a]	Adjusted geometric mean[a] (ng/g dust)	*p* value
Distance to freight route[e]				
<100 m	0.01	0.7	1,125.6 (864.5–1,465.6)	0.95
≥100 m	Reference		1,117.2 (846.8–1,473.9)	
Distance to major roads[e]				
<100 m	0.08	8.5	1,168.4 (824.8–1,655.1)	0.62
≥100 m	Reference		1,076.3 (854.0–1,356.7)	
Distance to railroads[f]				
<400 m	−0.16	−15.0	1,034.0 (762.5–1,402.2)	0.17
≥400 m	Reference		1,216.2 (962.4–1,537.0)	

[a]Fully adjusted model includes all variables listed in Table plus number of industrial facilities within 5 km of the home that release dioxins/furan (cement kilns burning non-hazardous waste, coal-fired electric plants, hazardous waste incinerators, medical waste incinerators, municipal solid waste incinerators, and sewage sludge incinerators) (results were not significant and are not shown)

[b]United States Census 2000

[c]Annual average ambient PAH concentration estimated at the census tract level from the USEPA Assessment System for Population Exposure Nationwide model

[d]Percentage of developed land (≥20 % impervious surface) as defined by the US Geological Survey 2001 National Land Cover Database within 2 km of a home

[e]Major roads and freight routes were located using TeleAtlas Dynamap Transportation version 5.2 (2003)

[f]Railways were located using the National Atlas of the United States database (2005)