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Predictors of Tris(1,3-dichloro-2-propyl) phosphate Metabolite in the Urine of Office Workers

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Abstract

Tris(1,3-dichloro-2-propyl) phosphate (TDCPP) is a flame retardant widely used in furniture containing polyurethane foam. It is a carcinogen, endocrine disruptor, and potentially neurotoxic. Our objectives were to characterize exposure of adult office workers (n=29) to TDCPP by measuring its primary metabolite, bis(1,3-dichloro-2-propyl) phosphate (BDCPP), in their urine; measuring TDCPP in dust from their homes; offices and vehicles; and assessing possible predictors of exposure. We identified TDCPP in 99% of dust (GM=4.43 µg/g) and BDCPP in 100% of urine samples (GM=408 pg/mL). Concentrations of TDCPP in dust were significantly higher in vehicles (GM=12.5 µg/g) and offices (GM=6.06 µg/g) than in dust from the main living area (GM=4.21 µg/g) or bedrooms (GM=1.40 µg/g) of worker homes. Urinary BDCPP concentrations among participants who worked in a new office building were 26% of those who worked in older buildings (p=0.01). We found some evidence of a positive trend between urinary BDCPP and TDCPP in office dust that was not observed in the other microenvironments and may be related to the timing of urine sample collection during the afternoon of a workday. Overall our findings suggest that exposure to TDCPP in the work environment is one of the contributors to the personal exposure for office workers. Further research is needed to confirm specific exposure sources (e.g., polyurethane foam), determine the importance of exposure in other microenvironments such as homes and vehicles, and address the inhalation and dermal exposure pathways.

Keywords

BDCPP; chlorinated tris; dust; human exposure; indoor environment; office; organophosphate flame retardant; TDCPP; urine metabolite

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Appendix A. Supplementary data

Supplementary data to this article can be found online at.

1. Introduction

Tris(1,3-dichloro-2-propyl) phosphate (TDCPP) is a semivolatile flame retardant that was removed from use in children's pajamas in the early 1980s due to concerns regarding mutagenicity (Gold et al. 1978; Lynn et al. 1981; Soderlund et al. 1985). Today it is widely used at percent levels as an additive to polyurethane foam, a material commonly found in upholstered sofas, chairs, vehicles, and many baby products (Stapleton et al. 2009, 2011). Similar to other additive flame retardants, it may escape products via volatilization or physical breakdown of polymers (Webster et al. 2009). TDCPP is found in household dust, where it can likely lead to human exposure (Allen et al. 2008a; Meeker and Stapleton 2010; Watkins et al. 2011; Wu et al. 2007). Potential health effects remain a concern as an increased incidence of testicular tumors has been found in rats (Freudenthal and Henrich 2000). In 2011, TDCPP was added to the Proposition 65 list of chemicals known by the State of California to cause cancer (CalEPA 2011a). In vitro studies suggest TDCPP may be nephro- and neurotoxic and increased concentrations in household dust were associated with decreased semen quality and reduced free thyroxine in Boston-area men, suggesting possible effects on fertility and thyroid function (Dishaw et al. 2011; Meeker and Stapleton 2010; Soderlund et al. 1985).

Animal studies show TDCPP is readily absorbed through both the skin and gastrointestinal tract (Nomeir et al. 1981). Following rapid distribution through the body, TDCPP is quickly metabolized to bis(1,3-dichloro-2-propyl) phosphate (BDCPP) and excreted primarily in urine (Lynn et al. 1981; Nomeir et al. 1981). In vitro investigation of human metabolism of TDCPP is consistent with rodent studies, yielding BDCPP as the primary metabolite (Cooper and Stapleton 2011). The half-life of TDCPP in rats ranges from 1.5–5.4 hours depending on the tissue (Nomeir et al. 1981). The half-life in humans is not known but is likely to be similarly short.

The nature and extent of human exposure to TDCPP is not well understood, but it has been found at high concentrations and detection frequencies in household dust (van der Veen and de Boer 2012). Similar to polybrominated diphenyl ether (PBDE) flame retardants, hand-to-mouth behavior may be an important route of exposure (Watkins et al. 2011). Methods were recently developed for measuring BDCPP in human urine; BDCPP has no known source other than as a metabolite of TDCPP (Cooper et al. 2011). The objectives of our study were therefore to 1) characterize human exposure by measuring urinary BDCPP in a cross-sectional sample of adult office workers, 2) measure TDCPP in dust from their microenvironments (homes, vehicles and offices), and 3) assess possible predictors of exposure.

2. Methods

2.1. Study Design

We recruited a convenience sample of 31 adults who worked and lived in the Boston, Massachusetts (USA) area. The study population consisted of 26 female and 5 male healthy non-smokers. The population was 90% white, had a median age of 49 years, and worked at least 18 hours per week in an enclosed office (excluding cubicles). From January through March 2009, we collected dust from each participant's microenvironments (office, vehicle, bedroom and main living area [MLA]) and administered a questionnaire within the course of one week. Three to six months later (June – July 2009) we collected a spot urine sample from 29 of the 31 participants. The urine samples were collected to supplement a study designed to evaluate office worker exposure to PBDEs (Watkins et al. 2011). The Boston University Medical Center Institutional Review Board approved the study protocol, and all participants gave their informed consent.

Participant offices were located in seven different buildings and categorized into three groups. Building A (6 offices) was constructed approximately one year before the study began and therefore had a modern HVAC system, sealed windows, new carpets and newly purchased polyurethane foam office chairs in all offices. Building B (17 offices) was partially renovated one year before the study began. These renovations included some new carpeting, but new furniture was not purchased. The five 'Other' buildings (8 offices) were not known to have undergone recent renovation. All offices had at least one office chair and all but one of the 31 offices were carpeted (Other category).

2.2 Dust samples

We collected dust samples using cellulose extraction thimbles (Whatman Inc., Piscataway, NJ) as previously described (Allen et al. 2008b). Each microenvironment was vacuumed for approximately 10 min. In the office, MLA, and bedroom, we vacuumed the entire floor surface area of the room, including accessible floor space under desks as well as the tops of immovable furniture. In two of the homes (a shared apartment and a studio) we collected dust from only one microenvironment. In vehicles, we vacuumed the entire accessible surface area of the front and back seats. Only 20 participants owned a vehicle. We measured and recorded the surface area, temperature, relative humidity, and other characteristics of each microenvironment such as the presence of carpeting.

After sample collection, each thimble was wrapped in aluminum foil, sealed in a polyurethane bag and stored at room temperature until processed. We collected dust field blanks by vacuuming sodium sulfate powder (as a surrogate for dust) placed on clean aluminum foil. Dust samples were sieved to collect particles $< 500 \mu\text{m}$ in size, placed in clean amber glass jars, and stored at -20°C . Dust samples ($\sim 300 \text{ mg}$) were analyzed for TDCPP using gas chromatography/mass spectrometry operated in electron-capture negative-ionization mode as previously described (Stapleton et al. 2009). For quality assurance purposes, we quantified TDCPP in laboratory blanks ($n=3$) and ran three samples in duplicate. Laboratory blanks consisted of 5.0 g of sodium sulfate extracted alongside the samples. TDCPP measured in laboratory blanks ($4.56 \pm 1.13 \text{ ng/g}$) was lower than in field blanks ($31.7 \pm 11.7 \text{ ng/g}$). The method detection limit (MDL) of 35.0 ng/g was calculated using three times the standard deviation of the field blanks. The relative standard deviation of duplicate samples ranged from 5.5 to 20%. TDCPP could not be quantified in one office and one vehicle dust sample due to a chromatographic interference with the TDCPP peak.

2.3 Urine samples

A single urine sample was collected in a sterile polypropylene cup from each participant during the afternoon (1–4 pm) of a workday (Tuesday–Friday). Participants were asked for the time of their last urination and if there had been any changes in the office, home, or vehicle since the dust sampling. One urine field blank sample was collected by transferring deionized water from one sterile polypropylene cup to another during sampling. TDCPP was not detected in the field blank. A refractometer (Atago, Bellevue, WA, USA) was used to measure the specific gravity (SG) of each sample within an hour of collection. Samples were stored at -80°C .

BDCPP was measured in urine samples using mixed-mode anion exchange solid phase extraction and a mass-labeled internal standard ($\text{D}_{10}\text{-BDCPP}$) with analysis by atmospheric pressure chemical ionization liquid chromatography tandem mass spectrometry as previously described (Cooper et al. 2011). For quality assurance purposes, we quantified BDCPP in laboratory blanks ($n=3$; 5 mL of deionized water) and ran five samples in duplicate. Recoveries of the internal standard, $\text{D}_{10}\text{-BDCPP}$, averaged $99 \pm 19\%$. BDCPP was present at very low levels in laboratory blanks ($1.78 \pm 1.25 \text{ pg/mL}$). The MDL of 3.75

pg/mL was calculated using three times the standard deviation of the laboratory blanks. The relative standard deviation of duplicate samples averaged 7.3%. Urinary creatinine was measured using an enzymatic assay (Diazme, Poway, CA, USA) with a colorimetric endpoint analyzed on a plate reader (FLUOStar Optima).

2.4 Questionnaire

We administered a questionnaire to collect information on demographics and personal habits such as time spent in the office and vehicle per week, as well as the average number of times per day each participant washed their hands. Information on hand-washing was collected as < 2 times/day, 2–4 times/day, 4–6 times/day, or ≥ 6 times/day. For data analysis, we collapsed the four categories into two: <6 times/day (low hand-washers) and ≥ 6 times/day (high hand-washers).

2.5 Data Analysis

We corrected concentrations of BDCPP in urine and TDCPP in dust by subtracting the mean of laboratory and field blanks, respectively. Sample concentrations <MDL were substituted with a value of MDL/2. To account for urinary output, concentrations of BDCPP in urine were normalized to urine SG and creatinine (Cooper et al. 2011); the two were highly correlated ($r_s=0.94$, $p=0.001$). The SG-adjusted data was used in all analyses.

Concentrations of BDCPP in urine and TDCPP in dust were not normally distributed (Shapiro-Wilk test, $p<0.05$), but were approximately log-normal. Accordingly, we natural log-transformed data prior to analysis and report the geometric mean (GM) and geometric standard deviation (GSD). We used non-parametric tests including the Wilcoxon rank sum test for comparison across categories and Spearman's rank order (r_s) correlation coefficients to examine associations between continuous variables. We used linear regression models to determine predictors of In BDCPP concentrations in urine and In TDCPP in dust. To aid interpretation, results of the regression analysis (β -coefficients) were exponentiated (e^β) to provide the multiplicative change in outcome (See Supplementary Data for details). Data points with Cook's distance exceeding $4/n$ (where n = number of samples) were considered influential. Potential confounding was evaluated as greater than a 10% change in effect estimate. All statistical analyses were performed using SAS (version 9.1; SAS Institute Inc., Cary, NC) with statistical significance defined as $\alpha = 0.05$.

3. Results

3.1 Analyte Concentrations in Dust and Urine

We detected TDCPP in 99% of dust samples with concentrations ranging from <0.03 to 326 $\mu\text{g/g}$ with a GM of 4.43 $\mu\text{g/g}$ (Table 1). GM concentrations of TDCPP in dust from vehicles (GM=12.5 $\mu\text{g/g}$) and offices (GM=6.06 $\mu\text{g/g}$) were significantly higher than in dust from the MLA (GM=4.21 $\mu\text{g/g}$) or bedroom (GM=1.40 $\mu\text{g/g}$). GM concentrations of TDCPP in dust from MLAs were significantly higher than in dust from bedrooms, although concentrations in these paired samples were positively correlated ($r_s=0.63$, $p<0.001$) (Table A.1). Office dust concentrations were uncorrelated with concentrations in dust from homes or vehicles. Vehicle dust concentrations were weakly but non-significantly correlated with MLA and bedroom dust concentrations. The concentration of TDCPP in dust from the one non-carpeted office (0.06 $\mu\text{g/g}$) was much lower than other offices.

We detected BDCPP in 100% of urine samples. SG-adjusted concentrations ranged from 62.1 to 1760 pg/mL with a GM of 408 pg/mL (Table 1).

3.2 Predictors of TDCPP in Dust

Linear regression of carpeted offices showed that TDCPP in dust from Building A was only 30% of concentrations in the Other buildings ($p=0.04$) and non-significantly lower than in Building B (Figure 1a, Table 2). Concentrations of TDCPP in vehicle dust were not associated with year of manufacture (Figure A.1), make, model or seat type (leather/upholstery). Home and office dust concentrations were not associated with room characteristics such as time and area vacuumed, temperature or humidity. None of the homes had wall-to-wall carpeting, and the presence of rugs was not associated with concentrations of TDCPP in house dust (data not shown).

3.3 Predictors of BDCPP in Urine

Office building was a strong, statistically significant predictor of urinary BDCPP (Table 3). The GM concentration of urinary BDCPP among Building A workers was only 26% of the GM concentration among workers in the Other office buildings ($p=0.01$) and 30% of the GM concentration among Building B workers ($p=0.01$) (Figure 1b, Table 3). Urinary BDCPP was not significantly different between workers in Building B and workers in the Other office buildings.

Other predictors that were not statistically significant, but were suggestive of a relationship with urinary BDCPP included concentrations of TDCPP in dust from worker offices (but not homes or vehicles), participant age, and hand-washing frequency (Table 3). Results of linear regression analysis using three categories of TDCPP concentrations in office dust suggested a 13% and 44% increase in average urinary BDCPP concentration for participants with medium and high concentrations of TDCPP in their office dust compared to those with low concentrations ($p=0.79$ and $p=0.43$, respectively).

Examined continuously, we found some evidence for a positive trend between urinary BDCPP and TDCPP in office dust that was not observed for dust from other microenvironments (Figures 2 and A.2). Restricting the analysis to carpeted offices ($n=28$) revealed a weak, but non-significant correlation between urinary BDCPP and TDCPP in office dust ($r_s=0.33$, $p=0.09$). We evaluated this relationship for reported changes to the office environment. One participant had begun using an air purifier in the office (P1), another had recently purchased new office chairs (P2), and a third had recently changed offices on the same floor (P3). P1 was identified as an influential point using Cooks Distance, but not P2 or P3. Removal of P1 from the analysis of carpeted offices resulted in a stronger correlation between urinary BDCPP and TDCPP in office dust ($r_s=0.45$, $p=0.02$) and also strengthened results from the linear regression (Table A.2).

The relationship between urinary BDCPP and TDCPP in office dust may be influenced by hand-washing frequency. Although statistical power to evaluate this factor was limited by a small sample size we observed that low hand-washers (<6 times/day) with high concentrations of TDCPP in their office dust (>7.6 $\mu\text{g/g}$) had the highest concentrations of urinary BDCPP (GM=599 pg/mL) compared to the other combinations of dust and hand-washing categories. The GM urinary BDCPP concentration in this high dust, low hand-washing group was approximately twice that found in the low dust, high hand-washing category (GM=297 pg/mL; $p=0.11$) (Figure 3).

We evaluated potential confounding of the relationship between urinary BDCPP and office building using multivariate linear regression and found no evidence of confounding by participant age, hand-washing, or TDCPP in office dust (Tables A.3–A.5). The coefficients for dust were reduced when included in a multivariate model with office building, suggesting that the TDCPP in office dust may be an intermediate variable between building and urinary BDCPP. As concentrations of TDCPP in dust from offices were uncorrelated

with dust from homes or vehicles, exposure in the latter microenvironments should not confound associations between TDCPP in office dust and urinary BDCPP.

Other factors such as time since last urination, sex, age, body mass index, hours spent in office and vehicle per week, and commuting type were evaluated but not found to be predictors of urinary BDCPP (data not shown).

4. DISCUSSION

This is the first study to utilize urinary BDCPP as a biomarker of TDCPP exposure in office workers and to characterize TDCPP in dust from multiple microenvironments. Our results suggest that TDCPP is pervasive in the indoor environment, that its metabolite BDCPP is ubiquitous in office worker urine, and that the office environment may contribute to human exposure.

We detected TDCPP in 99% of dust samples from participant microenvironments, demonstrating the widespread presence of this flame retardant in the indoor environment. The major source of TDCPP in our dust samples is likely upholstered furniture and other products containing polyurethane foam (e.g., office chairs, vehicle seats, couches) (Stapleton et al. 2009, 2011). Concentrations of TDCPP in dust from participant homes were similar to those found in dust from home vacuum cleaner bags in a previous Boston-area study (n=50, GM=1.89 µg/g) (Stapleton et al. 2009). The high concentrations we observed in dust from offices may reflect requirements by the City of Boston that office furniture meet California fire retardant standards, a rule that is not required of residential furniture in Boston (Boston Fire Department 1995). The high concentrations of TDCPP in dust from participant vehicles may also reflect fire standards specific to automobiles, or may reflect differences in sample collection for vehicles compared to the other microenvironments. Higher average concentrations of TDCPP in participant MLAs compared to bedrooms are consistent with expectations, based on the distribution of sources within the home. MLAs typically contain upholstered furniture such as couches and chairs that may have flame retardant treated polyurethane foam, whereas bedrooms likely contain fewer of these items; standard U.S. mattresses typically use barrier technology rather than additive flame retardants to meet fire standards (CPSC 2006a). The moderate correlation observed between concentrations of TDCPP in dust from the MLAs and bedrooms of participant homes may reflect transport within the home and is consistent with previous observations for PBDEs (Allen et al. 2008b; Watkins et al. 2012). We hypothesized that newer vehicles would have higher concentrations of TDCPP in dust compared to older vehicles due to the phase out of PentaBDE since 2004; however, we found no trend with vehicle age, suggesting that TDCPP has been used as a flame retardant in this application since at least 1995.

We identified BDCPP in all urine samples and at concentrations similar to those found in a small pilot study conducted elsewhere in the U.S. (n=9, GM=410 pg/mL) (Cooper et al. 2011). The office environment was our strongest predictor of urinary BDCPP. Working in a new office building (Building A) was associated with significantly lower urinary BDCPP, as well as lower concentrations of TDCPP in office dust. The association we observed between urinary BDCPP and concentrations of TDCPP in office dust (but not with dust from other microenvironments) further supports this finding. Exclusion of the one uncarpeted office and the office with a recently installed air purifier (PI) greatly strengthened the observed relationship between urinary BDCPP and TDCPP in office dust. Air cleaners have been shown to trap flame retardants, including TDCPP (Takigami et al. 2009). The air purifier was placed in the PI office after the dust sampling but before the urine sampling, thus may have reduced the worker's exposure. However, more research on the effect of air purifiers

on exposure is needed. It is unclear why dust concentrations of TDCPP in the single, uncarpeted (tile floor) office were extremely low.

There are multiple characteristics of Building A that may have contributed to decreased worker exposure. For example, Building A may contain fewer sources of TDCPP. The majority of upholstered furniture (mainly office chairs) in Building A offices was purchased new within the year prior to our study. The low concentrations of TDCPP in Building A offices suggest that the new furniture did not contain TDCPP or that it had not yet had sufficient time to migrate out of the products. Other differences between buildings may also explain our results; possibilities include HVAC systems or cleaning methods. More research is needed to determine factors that influence TDCPP concentrations in dust in relation to building contents and characteristics.

The association we observed between urinary BDCPP and concentrations of TDCPP in office dust (but not with dust from other microenvironments) was likely influenced by collection of urine samples during the afternoon (1–4 pm) of a workday (Tuesday–Friday), coupled with relatively quick metabolism of TDCPP and urinary excretion of its metabolite. Interestingly, a previous study of PBDEs in this same population found concentrations of PentaBDE in dust from the home (MLA and bedroom) were significantly correlated with concentrations of PentaBDE in serum, whereas concentrations in dust from the office were not (Watkins et al. 2012). This contrast may be due to the more persistent nature of PentaBDE congeners in the body, activity pattern differences by microenvironment, and/or potential differences in exposure route contribution. For example, studies suggest the primary exposure route for PBDEs in the U.S. is related to dust ingestion whereas dermal or inhalation exposure to TDCPP may also be important (CPSC 2006b; Hughes et al. 2001; Watkins et al. 2011).

Similar to PBDEs, our results suggest that frequent hand-washing may decrease personal exposure to TDCPP (Watkins et al. 2011). However, this association was comparatively weaker for TDCPP and not statistically significant, possibly due to the differences in exposure routes previously discussed.

We used an uncertain central exposure factor for adults (30 mg/day) recommended by the U.S. Environmental Protection Agency (USEPA 2011) and the GM TDCPP concentration in office dust to estimate dust ingestion of TDCPP in our population as roughly 0.2 µg/day. This yields 0.1 µg/day of BDCPP assuming complete absorption and metabolism of TDCPP to BDCPP (Cooper and Stapleton 2011) (Table A.6). Based on the GM BDCPP concentration in office worker urine (unadjusted for SG) and the daily output range for adults, we estimate total daily urinary output in our population to be about 0.1–0.4 µg/day. Assuming steady state, these rough estimates of input and output match well given the uncertainties. We also assume concentrations of TDCPP in office dust remained relatively constant over the 3–6 months between dust and urine sampling. This is likely a fair assumption, as average concentrations of PBDEs in house dust were previously found to not differ significantly over an 8 month period when there was little change in room furnishings (Allen et al. 2008b).

Strengths of our study include the collection of an exposure biomarker along with dust from multiple microenvironments. Limitations include a relatively small sample size, a lack of data on dietary intake of TDCPP, a several-month delay between collection of dust and urine, and the use of a single urine sample per person. No data are available on BDCPP concentrations in urine over time or differential biotransformation capacity between individuals. The delay between urine and dust collection may be important for TDCPP, which is believed to have a short half-life in humans. However, the expected random error

introduced by such delays, as well as interindividual variability, would tend to bias results toward the null. Spot urine samples were collected at work during the afternoon. Collection at other times may better reflect exposure in other microenvironments. We were unable to assess the influence of TDCPP inhalation on exposure. Estimates of the vapor pressure of TDCPP vary greatly, but fall within the semivolatile range (CalEPA 2011b; FFRP 2005; Wescher and Nazaroff 2008). Concentrations of TDCPP in dust and air are therefore likely correlated, and may explain some of our results. More research on inhalation exposure to TDCPP is needed. We were unable to examine specific sources of TDCPP within each office (i.e., concentrations/presence in furniture) as predictors of concentrations in dust or personal exposure. Urinary BDCPP concentrations may be higher than reported due to the use of plastic collection cups (Cooper et al. 2011). Finally, we used a convenience sample of participants. While this would not affect the internal validity of our findings, caution is needed in generalizing results to other populations.

Overall our findings suggest that exposure to TDCPP in the work environment is one of the contributors to the personal exposure for office workers. The positive trend between urinary BDCPP and TDCPP in office dust suggests a potential pathway of exposure. Further research is needed to confirm specific exposure sources (e.g., polyurethane foam), determine the importance of exposure in other microenvironments such as homes and vehicles, and address the inhalation and dermal exposure pathways.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

BDCPP	bis(1,3-dichloro-2-propyl) phosphate
PBDE	polybrominated diphenyl ether
SG	specific gravity
TDCPP	tris(1,3-dichloro-2-propyl) phosphate

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Highlights

- We identified TDCPP, an organophosphate flame retardant, in 99% of dust samples from offices, homes and vehicles.
- We identified the primary metabolite of TDCPP in 100% of urine samples from adult office workers.
- Participants who worked in a new office building had significantly lower concentrations of urinary metabolite than those who worked in older office buildings.
- The average concentration of TDCPP in dust was significantly lower in the new office building compared to the older office building categories.
- Timing of urine sample collection during the afternoon of a workday was likely important to these findings.

Figure 1.

Workers with offices in Building A had lower GM concentrations of: a) TDCPP in office dust and b) urinary BDCPP. Box plots show GM and quartiles. ¹One non-carpeted office ($0.06 \mu\text{g/g}$ TDCPP in dust) was excluded from the Other category.

Figure 2. Relationship between TDCPP in dust from worker offices and BDCPP in urine ($r_s=0.24$, $p=0.21$). $\blacklozenge$ Carpeted offices ($r_s=0.33$, $p=0.09$); $\diamond$ Uncarpeted office. P1–P3: Participants who reported changes in their office environments between the dust and urine sampling events.

Figure 3. GM concentrations of urinary BDCPP stratified by categories of TDCPP in office dust (low 7.6 $\mu\text{g/g}$ and high > 7.6 $\mu\text{g/g}$) and hand-washing frequency (low < 6 times/day and high 6 times/day).

Table 1

Measurements of target compounds in office worker urine and dust.

	% Detect (n)	GM (GSD)	Range
BDCPP in Urine ^a (pg/mL)	100% (29)	408 (2.6)	62.1 – 1760
TDCPP in Dust (µg/g)	99% (110)	4.43 (4.5)	<0.03 – 326
<i>Bedroom</i>	100% (29)	1.40 [*] (2.5)	0.27 – 18.2
<i>Main Living Area</i>	100% (31)	4.21 [*] (2.7)	0.56 – 30.6
<i>Office</i>	100% (30)	6.06 [*] (5.4)	0.06 – 72.0
<i>Vehicle</i>	95% (20)	12.5 [*] (6.5)	<0.03 – 326

* Contrasts between microenvironments were all significantly different (p<0.05, Wilcoxon rank sum test) except for office vs. vehicle.

^a Adjusted for specific gravity

Table 2

Predictors of TDCPP in dust from carpeted offices (n=30).

Office Building	n	GM (µg/g)	Multiplicative Increase ^a (p-value)
Building A	6	4.55	0.30 (0.04) *
Building B	17	8.59	0.56 (0.22)
Other	7	15.41	Reference

* Statistically significant at the $\alpha=0.05$ level.

^a Multiplicative increase in dust concentration relative to the reference group.

Table 3

Predictors of BDCPP in office worker urine (n=29).

Predictor	n	GM (pg/mL)	Multiplicative Increase ^a (p-value)
Office Building			
Building A	6	152	0.26 (0.01) *
Building B	16	503	0.85 (0.68)
Other	7	590	Reference
TDCPP in office dust			
High (> 10 µg/g)	10	497	1.44 (0.43)
Medium (5.3 to 10 µg/g)	10	390	1.13 (0.79)
Low (< 5.30 µg/g)	9	345	Reference
Age			
57	9	286	0.47 (0.09)
38 to 56	10	376	0.61 (0.26)
37	10	611	Reference
Hand-washings/day			
6	12	293	0.57 (0.12)
<6	17	515	Reference

* Statistically significant at the $\alpha=0.05$ level. Building A is also significantly lower than Building B ($p=0.01$)

^a Multiplicative increase in urinary metabolite relative to the reference group.