# ORGANOPHOSPHORUS FLAME RETARDANTS AND PLASTICIZERS IN DUST FROM AUTOMOBILES, HOMES, OFFICES AND UNIVERSITY CLASSROOMS: IMPLICATIONS FOR PERSONAL EXPOSURE VIA INADVERTENT DUST INGESTION

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## Introduction

Organophophate esters are used in a variety of applications. The chlorinated alkylphosphates such as tris(1-chloro-2-propyl) phosphate (TCPP), tris(1,3-dichloro-2-propyl) phosphate (TDCPP) and tris(2-chloroethyl) phosphate (TCEP) are used mainly as phosphorus flame retardants (PFRs) in polyurethane foams,[1] electronic equipment, textiles, plastic and building materials,[2] whilst the non-derivatized organophosphates such as triphenyl phosphates (TPP) are majorly used as plasticizers, lubricants and to regulate pore sizes such as in concrete. They are sometimes used as substitute flame retardants for the halogenated compounds, e.g., TPP in electronic devices.[1] Because PFRs are not covalently bound to the materials but are used as additives, they leach from products into the environment.[3] Several toxic effects have been associated with PFRs. Studies have reported that TDCPP is mutagenic, carcinogenic in rats and humans, and a moderate hazard for reproductive and developmental effects. Similarly, TCEP is reportedly carcinogenic for animals, neurotoxic to rats and mice, induces adverse reproductive effects in rats, and hemolytic and reproductive effects such as reduced fertility, longer estrous cycle length, reduced sperm motility and density in humans. TCEP was associated with the acute death of dogs after ingestion of car seat cushions containing enormous amounts of TCEP.[4] PFRs have been detected in indoor dust of various microenvironments in several locations worldwide.[5] Much attention has been engrossed recently on the significance of indoor dust as a pathway of human exposure to PFRs. The spontaneous relationship between dust and human body burdens is strongly implied by the correlation of PFRs in household dusts and human semen quality and hormone levels.[6] Nothing is known on the production, use, distribution and fate of PFRs in South Africa. Furthermore, despite the increasing proof of the significant implications of indoor dusts for human exposure to PFRs, attempts to link indoor contaminants with probable source items has had limited success. A dearth of information also exists for human exposure and pathways to PFRs. To breach these gaps, the aim of the present study was to investigate indoor contamination of four PFRs (TCEP, TCPP, TDCPP and TPP) in multiple indoor environments in South Africa. We also aimed to establish the relationships of various household products and the concentrations of PFRs in dust in order to identify possible sources of PFRs in the indoor environment. Finally, we estimated the exposure magnitude of PFRs among different population groups, utilizing various exposure scenarios.

## Materials and methods

Pure standards of TCEP, TCPP, TDCPP and TPP were obtained from Sigma Aldrich, South Africa. The standard reference material (SRM 2585: Organic contaminants in house dust) was purchased from the National Institute of Standards and Technology (NIST), Gaithsburg, USA. Anhydrous sodium sulfate was from Associated Chemical Enterprises (ACE), Johannesburg, South Africa. A Rtx®-1614 fused silica (5% diphenyl 95% dimethyl polysiloxane) capillary column was obtained as a generous gift from Restek Corporation, Bellefonte, PA, USA. All solvents were high performance liquid chromatography grade obtained from Sigma Aldrich, South Africa.

### *Sampling*

A total of 50 dust samples were collected from homes, $n$ = 10, university students' computer laboratories, $n$ = 12, and university staff offices, $n$ = 9, between August and October 2012 in Durban, South Africa. Similarly, dust samples, $n$ = 19, were collected between January and March 2013, from personal and previously-owned automobiles available for resale. The previously-owned automobile dust samples were collected from a dealership in Durban, South Africa. All automobiles from the dealership had been through a thorough cleaning

process on arrival at the dealership prior to resale. Similarly, personal automobiles sampled had not undergone any form of cleaning for at least three days prior to sampling.

***Recovery Experiment***

Recoveries for PFRs in dust were determined from spiked anhydrous sodium sulfate with different spike concentrations. Samples were left to stand for at least 21 days at -10 °C. Spiked samples were extracted and cleaned-up following the procedure for real samples.

***GC-EI/MS Analysis***

An Agilent 6890 GC fitted with a Restek Rtx®-1614 fused silica (5% diphenyl, 95% dimethyl polysiloxane) capillary column (15 m × 250 µm × 0.1 µm) coupled to an Agilent 5973N series mass spectrometer was used for the separation, detection and quantitation of all PFRs. Injections were made in the pulsed splitless mode with the injector temperature set at 250 ºC. The injection volume was 2 µL. The GC oven temperature programme started at 90 ºC (held for 2 mins), then increased at 20 ºC $min^{-1}$ to 270 ºC and held for 1 min and finally ramped at 10 ºC to 290 ºC and held for a minute. Helium was used as the carrier gas at a flow rate of 1.2 mL $min^{-1}$ and a constant linear velocity of 37 cm $s^{-1}$. For the MS, the ion source and transfer line temperatures were 230 ºC and 350 ºC, respectively; and the ionization energy was 70 eV. PFR mass spectra were obtained in full scan mode to select prominent ions for SIM. The following ions were selected: 205, 249 for TCEP; 201, 277 for TCPP; 321, 381 for TDCPP and 326, 325 for TPP, for the SIM mode utilized for quantitation of PFRs.

***Statistics***

Data were log normally transformed and PCA analysis was carried out with SIMCA version 13.0 software. Descriptive statistics such as sum, mean, median, minimum, maximum, *t*-test and analysis of variance (ANOVA) were calculated by using Microsoft Excel® 2010. Limits of detection (LOD) and quantitation (LOQ) were estimated following Thomsen *et al.*[8] Samples below the detection limit were treated as zero throughout the statistical analysis.

**Results and discussion**

All four PFRs were sufficiently resolved and separated on the Rtx®-1614 capillary column.

Instrument response to the calibration standards was linear for all PFRs with $r^2 > 0.99$. Limits of detections were 0.56 ng $g^{-1}$, 50.65 ng $g^{-1}$, 91.4 ng $g^{-1}$ and 100.2 ng $g^{-1}$ for TCEP, TCPP, TDCPP and TPP respectively. Spiked recoveries ($n = 4$) for each concentration ranged from 86-122.4% for TCEP, 74.1-98.6% for TCPP, 94.4-135.3% for TDCPP and 101.4-107.9% for TPP.

TCEP, TCPP and TPP were detected in all the samples from all microenvironments; however, TDCPP was absent in one of the automobile and one of the computer laboratory samples but otherwise present in the remainder of the samples. Figs 1a – d show the percentage contributions of each PFR to the total PFR contamination of dust samples from the different microenvironments.

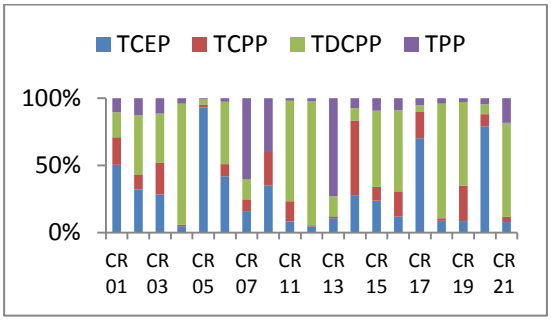


Fig 1a: Percentage contribution of individual organophosphate compounds in dust collected from automobiles in South Africa.

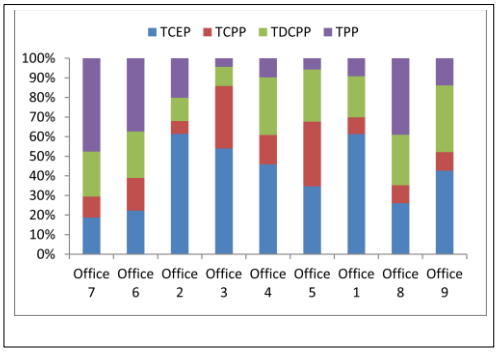


Fig 1b: Percentage contribution of individual organophosphate compounds in dust collected from offices in South Africa.

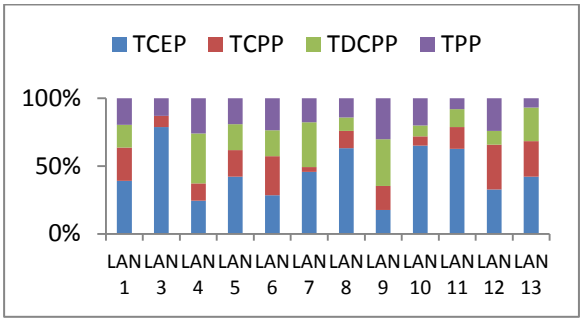


Fig 1c: Percentage contribution of individual organophosphate compounds in dust collected from university computer laboratories in South Africa.

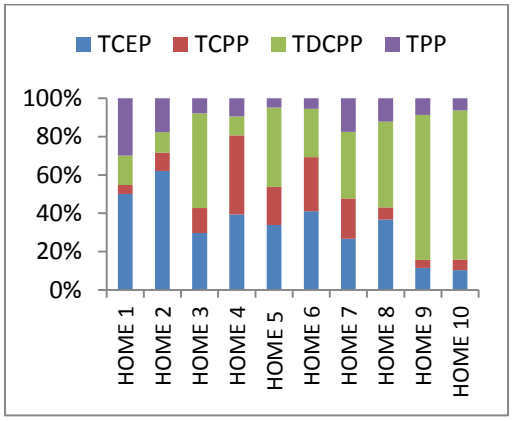


Fig 1d: Percentage contribution of individual organophosphate compounds in dust collected from homes in South Africa.

There were wide variations in the profiles of PFRs in automobiles from the same manufacturer with the exception of those from Toyota which showed similar profiles. Similarly, there were wide variations in the profiles of PFRs in automobiles manufactured between 2005 and 2012; whilst those manufactured prior to 2004 showed a close range. This implies large volume usage of PFRs in automobiles since the ban in 2004 of commercial pentaBDE in soft and rigid polyurethane foams used in automobile cushions, seats and high impact polystyrenes (HIPs). Similar to data from the USA,[9] Germany[10] and the Netherlands,[3] automobiles contain the bulk of PFRs compared to the other microenvironments.

TDCPP and TPP concentrations showed significant correlations in all microenvironments, suggesting source similarities. The ∑PFR in homes strongly correlated (r = 0.86) with the number of electronic devices and also correlated strongly (r = 0.90) with foams and furniture, suggesting that these PFRs emanated from these household products.

The concentrations of TCEP and TCPP showed strong correlations in automobiles (r = 0.995) and in homes (r = 0.82). This relationship could suggest a possibile gradual replacement of TCEP and TCPP in various consumer products in South Africa.

By utilizing the exposure factors in a previous study,[7] TDCPP appeared to be of great threat to the South African population particularly for young children (toddlers). The worse-case exposure scenario (utilizing the 95$^{th}$ percentile contaminant concentrations) showed that toddlers ingest as high as 26978.40 ng day$^{-1}$ TDCPP and 34777.8 ng day$^{-1}$ ∑PFRs through inadvertent dust ingestion. This dosage is at par with the toxicological reference dose of TDCPP.[5]

Generally, the results of this study reveal that current human exposure to PFRs via inadvertent indoor dust ingestion poses a risk to the South African general population and particularly to young children. We recommend that other exposure pathways such as inhalation, dietary and dermal contact should be studied in order to estimate total human intake of PFRs. Furthermore, studies on bioaccessibility and bioavailability of PFRs are strongly recommended for accurate risk assessment of PFRs.

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