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Risk Assessment of Dermal Exposure to Tris(2-chloroethyl) Phosphate and Tris(1,3-dichloroisopropyl) Phosphate via Contact with Indoor Dust

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How To Cite: Hashim, B.B.; Al-Omran, L.S.; Stubbings, W.A.; et al. Risk Assessment of Dermal Exposure to Tris(2-chloroethyl) Phosphate and Tris(1,3-dichloroisopropyl) Phosphate via Contact with Indoor Dust. *Environmental Contamination: Causes and Solutions* **2026**, *2*(1), 1. https://doi.org/10.53941/eccs.2026.100001

Received: 29 October 2025
Revised: 29 December 2025
Accepted: 2 January 2026
Published: 21 January 2026

Abstract: Dermal uptake from indoor dust—especially within residential and vehicular environments—constitutes a potentially important exposure route. This study aimed to estimate daily intakes (EDIs) via dermal contact of tris(2-chloroethyl) phosphate (TCEP) and tris(1,3-dichloroisopropyl) phosphate (TDCIPP) using the existing dust concentration data from our previous studies that were designed to investigate oral ingestion exposure to such pollutants in indoor dust from residences and private vehicles in Iraq. Non-carcinogenic (non-CR) and carcinogenic risk (CR) assessments using both dermal and oral ingestion pathways were also determined. Under mean exposure conditions, the EDI values via dermal contact for both compounds were highest in toddlers, followed by professional taxi drivers and then adults, with values ranging from 0.011 to 0.215 ng/kg bw/day. For home dust, corresponding values ranged between 0.036 and 1.48 ng/kg bw/day. Dermal exposure was identified as the second most important pathway, contributing 35% and 32% of total TCEP exposure via home dust, while dermal exposure via contact with car dust contributed 18% and 20% of total TDCIPP exposure, for adults and toddlers, respectively. Hazard index (HI) values were orders of magnitude lower than the reference value (<1), suggesting minimal non-CR health risk. While most CR values were below 1×10^{-6}, high-end exposure scenarios slightly exceeded the threshold for TDCIPP. This study provides the first comprehensive dermal exposure assessment for TCEP and TDCIPP in Iraq, and emphasises the need to consider dermal exposure in future risk evaluations.

Keywords: TCEP; TDCIPP; indoor dust; dermal exposure; health risk assessment

1. Introduction

Organophosphate esters (OPEs) are synthetic chemicals extensively applied as flame retardants and plasticisers in a wide range of products, including polyurethane foams, textiles, furniture, and construction materials [1,2]. As semi-volatile organic compounds and non-covalently bound chemicals, OPEs can be gradually released into the environment through various mechanisms, including abrasion, leaching, and volatilisation [3,4]. They have been reported in many environmental matrices, such as air [5], drinking water [6], sediments [7], indoor dust [8], and human biological samples such as urine and blood [9].

Tris(2-chloroethyl) phosphate (TCEP) and tris(1,3-dichloroisopropyl) phosphate (TDCIPP) are two well-known chlorinated organophosphate esters (Cl-OPEs) that have raised significant concerns associated with risks to human health. Both TCEP and TDCIPP are classified as animal carcinogens under California's Proposition 65

[10,11]. In 2019, the US EPA (United States Environmental Protection Agency) classified TCEP as one of 20 high-priority chemicals under the Toxic Substances Control Act (TSCA), and in 2024, the final risk evaluation for TCEP was released. TCEP mainly poses risks of kidney cancer and reproductive system disorders, endocrine and thyroid disruption, hepatic toxicity, and developmental impacts [12]. For TDCIPP, no TSCA risk evaluation has been initiated to date; however, TDCIPP constitutes a multifaceted health risk [3]. Several studies have indicated that exposure to TDCIPP may result in possible reproductive harm [13], respiratory toxicity [14], renal damage [15], DNA damage [16], and impacts on the functional activity of pancreatic beta cells [17].

The above adverse health consequences have resulted in the cessation of TCEP production for foam applications in Canada and the EU [18,19] and the decline of production in the USA [12]. However, TCEP continues to be manufactured and extensively used in China, with annual production levels reaching tens of thousands of tonnes [20]. It is still used in rigid and flexible foam (building insulation, furniture, and car seats), polymers and plastics (PVC, polyester resins, epoxy coatings, and cellulose plastics), as well as paints and coatings (fire-resistant paints, varnishes, and adhesives) [21]. TDCIPP remains a standard additive in flexible polyurethane foam (FPUF) and associated coatings, and has been classified as a high-production-volume chemical. Nevertheless, limited information exists regarding TDCPP manufacture in developing countries [3].

Human exposure to TCEP and TDCIPP can occur via inhalation, ingestion, and dermal contact [22]. As a non-dietary exposure source, dust ingestion has been estimated to be the dominant exposure pathway, particularly for young children [8,23,24]. However, results of both in vitro research and biomonitoring studies provide strong evidence that dermal uptake from dust may surpass both ingestion and inhalation. Via various indoor environments, Balasch et al. (2023) reported that the contribution of dermal exposure to total exposure was the highest [25]. Several other studies have also shown that dermal absorption via dust results in a more substantial intake of OPEs than inhalation and dust ingestion [26–28]. Nevertheless, other studies reported that while dust ingestion was the dominant pathway for toddlers, dermal absorption was the dominant pathway for adults [29].

The estimated daily intake of environmental contaminants requires information on the time that people spend in the specific microenvironment. Evidence suggests that people spend approximately 80–90% of their time indoors, predominantly within residential settings [30]. This implies the significant impact of homes on human exposure assessment. However, numerous studies investigating Cl-OPEs in indoor dust reported that concentrations of TCEP and TDCIPP in vehicles were an order of magnitude higher than those of other microenvironments [29,31,32]. This evidence suggests dust in vehicles may represent an important pathway of human exposure to such chemicals [33,34], particularly for professional drivers who spend long periods inside the vehicles. However, few studies have documented dermal exposure to Cl-OPEs via car dust in the Middle East [35–37] and around the world [24,29,32,38]. Thus, it is crucial to conduct further studies to quantify dermal exposure of individuals in professional occupations, as well as for the general population.

Building on this context, our study aims to provide an assessment of daily exposure to TCEP and TDCIPP via dermal contact using the existing dust concentration data from our previous studies [39,40] to improve upon prior investigations of such pollutants in indoor dust from residences and private vehicles in Iraq. Additionally, the study aimed to assess both carcinogenic and non-carcinogenic health hazards and to provide a more comprehensive assessment of human exposure to indoor dust from homes and cars.

2. Methods

2.1. Study Design and Data Sources

Data on TCEP and TDCIPP concentrations were obtained from our previous studies, which aimed to estimate the daily intake of eight OPEs and three Cl-OPEs through indoor dust ingestion in Basrah, Iraq. House dust samples (n = 40) were collected in 2019 from elevated surfaces and floors of the living room [39], while car dust samples (n = 24) were collected in 2021 from urban areas from the same city. According to the country of manufacture, cars were classified into four groups: Korean (n = 10), Chinese (n = 5), Japanese (n = 5), and USA (n = 4) [40].

TCEP and TDCIPP were detected in all house dust samples from both elevated surface areas (e.g., tables, chairs) and floors in the living room of the sampled houses, as well as in car dust. Table S1 presents a statistical summary of TCEP and TDCIPP concentrations (ng/g) in home and car dust samples.

2.2. Dermal Exposure Assessment

This study estimated daily dermal intake (EDI dermal) of TCEP and TDCIPP for adults, toddlers, and professional drivers using dust concentration data from the above studies [39,40]. EDI dermal (ng/kg bw/day) was calculated using Equation (1) [41–43].

$$\text{EDI dermal} = \frac{C \times ESA \times DA \times AF \times FT}{bw \times 1000} \quad (1)$$

where C is the concentration of TCEP or TDCIPP in dust (ng/g), and bw is body weight (kg). ESA is the exposed body surface area (cm^2) available for contact with indoor dust, including the hands, arms, and lower legs in adults and the corresponding limb surface areas in toddlers, and DA is the mass of dust that sticks to skin per unit area during contact ($mg \cdot cm^{-2}$). Without well-established regulatory in vivo dermal absorption coefficients for individual organophosphate esters, researchers adopted AF values from experimental and exposure studies, which represent conservative estimates commonly used in human exposure assessments. Chemical-specific dermal absorption fractions of 0.28 for TCEP and 0.13 for TDCIPP, indicating that TCEP has greater skin permeability compared to TDCIPP [42–45]. For time-activity fractions, it was assumed that adults and toddlers spend about 63.8% and 86.1% of their time indoors at home, respectively. The general population was estimated to spend 4.1% of their time inside vehicles, while professional drivers were estimated to spend 27.9% of their time inside vehicles, assuming 6–7 h/day driving [23,24,36,41]. Parameter values used in the equation are listed in Table S2. Three exposure scenarios were considered: (1) Low-end exposure, assuming the Cl-OPE concentration in the dust absorbed was at the 5th percentile; (2) "typical" or mean exposure, whereby the Cl-OPE concentration was assumed to be at the median level; and (3) High-end exposure in which Cl-OPE concentrations were taken to equal the 95th percentile [44].

2.3. Health Risk Assessment

A health risk assessment was conducted to evaluate potential non-carcinogenic and carcinogenic hazards associated with exposure. The non-carcinogenic risk (non-CR) posed by TCEP and TDCIPP is quantified by a hazard quotient (HQ) and a hazard index (HI) computed using Equations (2)–(4) [25,38].

$$\text{HQ dermal} = \frac{\text{EDI dermal}}{\text{RfD}} \quad (2)$$

$$\text{HQ ingestion} = \frac{\text{EDI ingestion}}{\text{RfD}} \quad (3)$$

$$\text{HI} = \sum_{i=1}^{n} \text{HQi} \quad (4)$$

Here, RfD (ng/kg/day) is the reference dose, defined as 2200 ng/kg/day for TCEP and 1500 ng/kg/day for TDCIPP [45,46]. An HQ ≤ 1 suggests that adverse health effects are unlikely, while HQ > 1 may indicate a potential risk [12,47].

Carcinogenic risk (CR), representing the probability of developing cancer from exposure, was calculated using Equation (5) [24,27].

$$CR = EDI \times SF \quad (5)$$

where SF is the slope factor (0.02 and 0.13 mg/kg-day for TCEP and TDCIPP, respectively). It should be noted that both RfD and SF values are estimated for oral exposure [48,49]. Carcinogenic risks greater than 1×10^{-4} are considered elevated, those below 1×10^{-6} are negligible, and risks between these values represent moderate concern [24,27].

3. Results and Discussion

3.1. Estimated Daily Intake of TCEP and TDCIPP via Dermal Absorption

Estimated daily intake via dermal absorption was assessed for adults, toddlers, and professional taxi drivers using Equation (1). Table 1 lists estimated daily intakes (EDIs) of TCEP and TDCIPP via dermal contact with home and car dust for the three groups of the Iraqi population.

Table 1. Estimated daily intakes (EDIs) (ng/kg bw/day) of TCEP and TDCIPP for adults, toddlers, and Professional-drivers via indoor dust using the three exposure scenarios (low-end, mean and high-end).

Scenario	Car		Home	
	TCEP	TDCIPP	TCEP	TDCIPP
		Adults		
Low-end	0.005	0.002	0.046	0.010
Mean	0.017	0.011	0.129	0.036
High-end	0.068	0.115	0.389	0.105
		Toddlers		
Low-end	0.060	0.030	0.254	0.070
Mean	0.215	0.139	1.48	0.422
High-end	0.870	1.48	3.59	1.08
		Professional drivers		
Low-end	0.032	0.016		
Mean	0.114	0.073		
High-end	0.460	0.783		

The results showed that dermal EDI values for both compounds followed the order: toddlers > taxi drivers > adults. The mean daily intake of TCEP in car dust samples ranged between 0.017 and 0.215 ng/kg bw/day, whereas that of TDCIPP ranged between 0.011 and 0.139 ng/kg bw/day. In the high-end exposure scenario, the EDI dermal of TCEP ranged between 0.068 and 0.870 ng/kg bw/day, whereas that of TDCIPP ranged between 0.115 and 1.48 ng/kg bw/day. The EDI via dermal exposure for toddlers exceeded that for adults and professional drivers by factors of 2 and 13, respectively. This pattern likely reflects the smaller body mass of toddlers, which increases dose per unit weight, and the extended time taxi drivers spend inside vehicles. For home dust, mean daily dermal intakes were 0.129 and 0.036 ng/kg bw/day for adults and 1.48 and 0.422 ng/kg bw/day for toddlers for TCEP and TDCIPP, respectively. As shown in Figure 1, toddlers experienced 9–13 times higher dermal exposure than adults in both microenvironments.

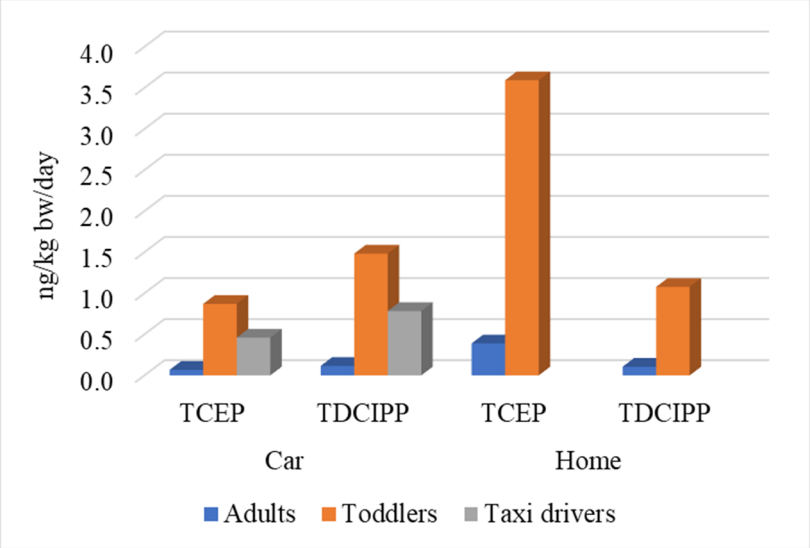

Figure 1. Estimated daily intakes of TCEP and TDCIPP via dust dermal contact with home and car dust for adults, toddlers, and taxi drivers under the high-end exposure scenario.

The distribution profiles of EDIs via dermal exposure differed between compounds in the two microenvironments studied. Dermal exposure to TCEP was highest via home dust, while TDCIPP exposure was highest from car dust. The EDI home/car ratios for TCEP were 5.8 (adults) and 4.1 (toddlers), whereas the car/home ratios for TDCIPP were 1.1 and 1.4, respectively. These results suggest that dermal exposure to TCEP from residential dust contributed substantially to overall non-dietary intake, while dermal exposure to TDCIPP from vehicles represents an important source. Although people often spend less time in vehicles, the elevated TDCIPP concentrations in automobile dust represent a considerable exposure hazard. This phenomenon is probably associated with the distinct applications of these flame retardants: TCEP is predominantly found in

furniture and fabrics, whereas TDCIPP is extensively utilised in car seat foams and similar interior materials. Our results are consistent with those in Australia, Germany, the UK, and Colombia [29,31].

3.2. Variation in Dermal Exposure by Vehicle Manufacturer

Although the dataset is relatively small, it offers valuable insights into how the country of vehicle manufacture influences exposure levels. Table 2 illustrates the EDI mean dermal results for TCEP and TDCIPP via car dust absorption, based on the country of manufacture. For TCEP, our results revealed that human exposure was in the order of Korean > Japanese > Chinese > USA. In contrast, for TDCIPP, the human exposure was in the order of USA > Japanese > Chinese > Korean cars. These differences likely reflect variations in flame-retardant formulations and usage practices across manufacturers. Figure S1 illustrates high-end EDI dermal values (95th percentile) for the three population groups across vehicle types.

Table 2. The EDI dermal results for TCEP and TDCIPP via car dust absorption for toddlers and professional drivers, based on the country of manufacture.

		Adults		
	Chinese	Japanese	Korean	USA
TCEP	0.040	0.045	0.134	0.020
TDCIPP	0.028	0.107	0.023	0.181
		Toddlers		
	Chinese	Japanese	Korean	USA
TCEP	0.513	0.576	1.723	0.262
TDCIPP	0.365	1.376	0.293	2.336
		Professional-drivers		
	Chinese	Japanese	Korean	USA
TCEP	0.271	0.304	0.910	0.138
TDCIPP	0.193	0.727	0.155	1.234

3.3. Total Estimated Daily Intake and Risk Assessment

Human exposure to dust occurs primarily via non-dietary routes such as ingestion, dermal absorption, and inhalation. Nonetheless, ingestion and cutaneous absorption have been identified as the primary pathways [31]. Thus, in the current study, total estimated daily intake (EDI-total) was obtained as a sum of EDI via dust ingestion [39,40] and dermal absorption for adults and toddlers using the median and 95th percentile concentrations of TCEP and TDCIPP in home and car dust samples. We followed the scenario, excluding dust from offices that have been used previously [31]. Table 3 presents the calculated values of the total EDI for TCEP and TDCIPP, while Figure 2 shows the mean contributions of EDI ingestion and dermal exposure to the total EDI for both adults and toddlers.

Table 3. Total estimated daily intake (EDI total) of TCEP and TDCIPP via dust ingestion and dermal contact for adults and toddlers under mean and high-end exposure scenarios.

	EDI Ingestion	**EDI Dermal**	**EDI Total**
		Adults/Mean exposure	
TCEP	0.23	0.15	0.37
TDCIPP	0.15	0.05	0.20
		Toddlers/Mean exposure	
TCEP	2.94	1.69	4.64
TDCIPP	2.10	0.56	2.66
		Adults/High-end exposure	
TCEP	2.12	0.46	2.58
TDCIPP	2.20	0.22	2.42
		Toddlers/High-end exposure	
TCEP	15.5	4.5	20.0
TDCIPP	19.2	2.6	21.8

EDI total values ranged between 0.37 and 20.0 ng/kg bw/day for TCEP and between 0.20 and 21.8 ng/kg bw/day for TDCIPP under the mean and high-end exposure scenarios, respectively. Under the mean scenario, estimated daily intakes via both exposure pathways and the EDI total for TCEP exceeded those for TDCIPP.

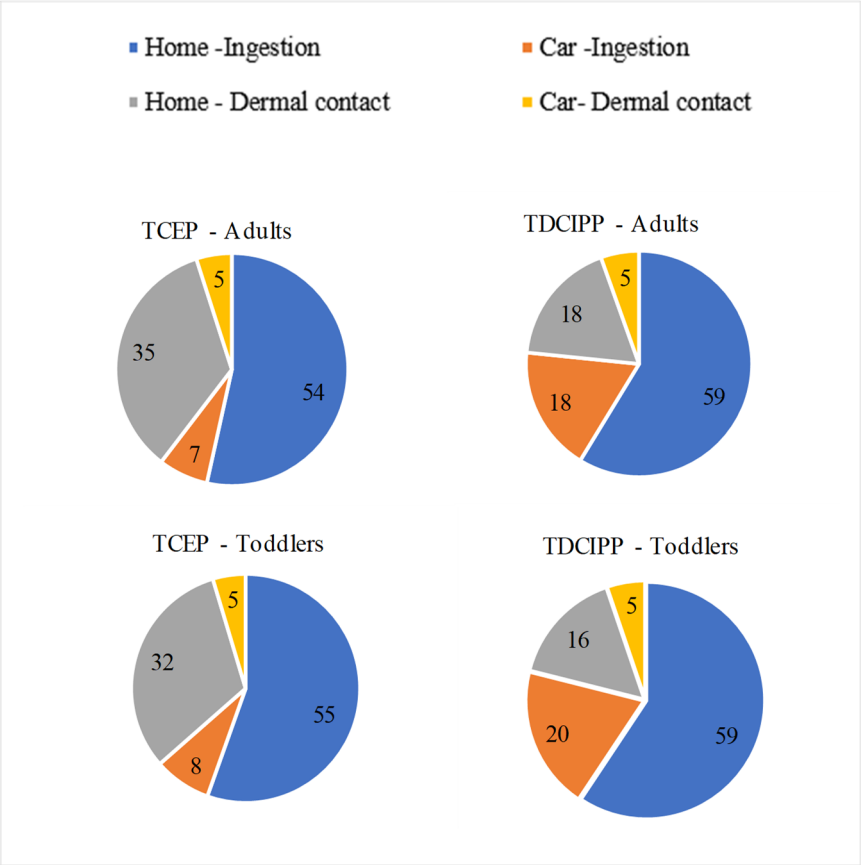

Figure 2. Relative contribution of the estimated daily intakes via ingestion and dermal absorption of TCEP and TDCIPP to the EDI Total in home and car microenvironment for adults and toddlers.

The most substantial non-dietary exposure pathway for both adults and toddlers was home dust ingestion, which accounted for more than 50% of the total estimated daily intake of TCEP and TDCIPP. For TCEP, dermal uptake via household dust constituted the second most significant exposure pathway, accounting for 35% and 32% of total exposure for adults and toddlers, respectively. For TDCIPP, dermal contact with car dust was the secondary exposure pathway, which contributed 20% of the total exposure. We observed comparable levels of dermal exposure to TDCIPP in adults, with both car and house dust contributing around 18%. These results align with previous studies, which have reported that while dust ingestion is the primary exposure route, dermal absorption constitutes an important secondary human exposure pathway to Cl-OPEs [8,24,29].

3.4. Non-Carcinogenic and Carcinogenic Risk Assessments

From the obtained EDI values, the associated risk of non-carcinogenic (non-CR) effects, represented by hazard quotient (HQ) and hazard index (HI), and the carcinogenic risk (CR) for TCEP and TDCIPP were determined using Equations (2)-(5). Table 4 provides a summary of the HQ, HI, and CR values for the two exposure pathways based on mean and high-end exposure scenarios.

Table 4. Hazard quotient (HQ), hazard index (HI), and carcinogenic (CR) values of TCEP and TDCIPP via dust ingestion and dermal absorption for adults and toddlers, under mean and high-end exposure scenarios.

	HQ-Ingestion	CR-Ingestion	HQ-Dermal	CR-Dermal	HI Total-per Chemical	CR-Total-per Chemical
			Adults /Mean exposure			
TCEP	1.03×10^{-4}	4.5×10^{-9}	6.64×10^{-5}	2.92×10^{-9}	1.69×10^{-4}	7.44×10^{-9}
TDCIPP	1.00×10^{-4}	2.0×10^{-8}	3.13×10^{-5}	6.11×10^{-9}	1.31×10^{-4}	2.61×10^{-8}
			Toddlers/Mean exposure			
TCEP	1.34×10^{-3}	5.9×10^{-8}	7.69×10^{-4}	3.38×10^{-8}	2.11×10^{-3}	9.27×10^{-8}
TDCIPP	1.40×10^{-3}	2.7×10^{-7}	3.74×10^{-4}	7.29×10^{-8}	1.77×10^{-3}	3.46×10^{-7}
			Adults/High-end exposure			
TCEP	9.64×10^{-4}	4.2×10^{-8}	2.08×10^{-4}	9.14×10^{-9}	1.17×10^{-3}	5.16×10^{-8}
TDCIPP	1.47×10^{-3}	2.9×10^{-7}	1.47×10^{-4}	2.86×10^{-8}	1.61×10^{-3}	3.15×10^{-7}
			Toddlers/High-end exposure			
TCEP	7.05×10^{-3}	3.1×10^{-7}	2.03×10^{-3}	8.92×10^{-8}	9.07×10^{-3}	4.00×10^{-7}
TDCIPP	1.28×10^{-2}	2.5×10^{-6}	1.71×10^{-3}	3.33×10^{-7}	1.45×10^{-2}	2.83×10^{-6}

Our results revealed that the estimated HI values (total HQ via ingestion and dermal contact) for adults and toddlers under the mean and high-end exposure scenarios were between 10^{-4} and 10^{-2}. HI values were several orders of magnitude below 1, indicating that exposure to TCEP and TDCIPP via dust ingestion and dermal contact represents minimal noncancer risk.

Regarding carcinogenic risk, the mean estimated CR values ranged between 10^{-9} and 10^{-7}, which were lower than the carcinogenic risk limit (10^{-6}). This suggests that the carcinogenic risk from exposure to TCEP and TDCIPP for both adults and toddlers via dust ingestion and dermal contact was almost negligible under average exposure conditions. However, under high-end exposure conditions, the cancer risk (CR) associated with TDCIPP exposure reached 2.5×10^{-6}, marginally exceeding the US EPA risk management range of 1×10^{-6} to 10^{-4} [12]. These elevated exposure conditions may warrant attention. However, the excess that was recognised in the high-end exposure scenario represents a precautionary condition rather than typical population exposure. Similar trends have been reported in other studies [24,29]. Generally, CR values greater than 1×10^{-4} are considered high, values less than 1×10^{-6} are considered negligible, and those that fall between these thresholds indicate low-to-moderate concern [12]. The total CR values observed in this study were comparable to those reported for hotel dust in Saudi Arabia [27].

4. Conclusions

This study provides the first assessment of dermal exposure to TCEP and TDCIPP through indoor dust in Iraq. It extends previous investigations by ingestion and provides a comprehensive risk assessment for human health, including both carcinogenic and non-carcinogenic results. From different population groups, our findings showed that the estimated daily intakes via dermal contact for the professional drivers were 7 times higher than those for regular adults, implying the importance of the occupational assessments. The results indicated that ingestion exposure to Cl-OPEs from indoor dust contributes significantly to overall human intake. However, TDCIPP via dermal contact with car dust was the second largest contributor to the total EDI, particularly for toddlers. The non-carcinogenic and carcinogenic risks of exposure to TCEP and TDCIPP through ingestion and dermal contact were low for both adult and toddler populations. Further studies are recommended to assess food and water ingestion, air inhalation, and dermal exposure via direct contact with Cl-OPE containing materials like fabrics.

Supplementary Materials

The additional data and information can be downloaded at: https://media.sciltp.com/articles/others/260121135524357 8/ECCS-25100128-SM-final.pdf. Figure S1: High-end dermal EDI values (95th percentile) for the three population groups across vehicle types. Table S1: Statistical summary of TCEP and TDCIPP concentrations (ng/g) in home and car dust samples. Table S2: Parameters used in the equation 1 for EDI dermal.

Author Contributions

B.B.H.: conceptualisation, study design, and writing—original draft. L.S.A.-O.: conceptualisation, supervision, methodology, data curation, and writing—review and editing. W.A.S.: Writing—review and editing, validation, software, and resources. S.H.: Investigation, validation, visualisation, and writing—review and editing. All authors have read and agreed to the published version of the manuscript.

Funding

This research received no external funding.

Institutional Review Board Statement

Institutional Review Board approval was not required for this study.

Informed Consent Statement

Informed consent was not applicable to this study.

Data Availability Statement

The data are available from the corresponding author upon reasonable request.

Acknowledgments

This study was supported by the University of Basrah, Iraq.

Conflicts of Interest

The authors declare no conflict of interest. Given the role of Stuart Harrad as Editor-in-Chief of the journal and Layla Salih Al-Omran as a member of the Editorial Board, had no involvement in the peer review of this paper and no access to information regarding its peer-review process. Full responsibility for the editorial process of this paper was delegated to another editor of the journal.

Use of AI and AI-Assisted Technologies

No AI tools were utilized for this paper.

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