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# Concentrations of organophosphate esters in drinking water from the United Kingdom: Implications for human exposure

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ABSTRACT

Data on the presence of organophosphate esters (OPEs) in drinking water and its significance as a pathway of exposure are limited. In this study, we measure for the first time, concentrations of eight OPEs in 50 UK drinking water samples. Arithmetic mean concentrations of $\sum_8$OPEs were: 6.4 and 11 ng/L in bottled (n = 25) and tap water samples (n = 25), respectively. Concentrations of $\sum_8$OPEs in tap water (mean: 11 ng/L) exceed significantly those in bottled water (mean: 6.4 ng/L) ($p < 0.01$). Moreover, UK tap water is more contaminated with chlorinated, aryl-, and alkyl-OPEs than bottled water. The predominant OPEs detected were: tris (butoxyethyl) phosphate (TBOEP), tris (2-chloroethyl) phosphate (TCEP), and tris(2-chloroisopropyl) phosphate (TCIPP) with arithmetic mean concentrations in the two water sample types ranging between (3.5–3.8 ng/L), (0.60–3.0 ng/L), and (1.02–2.9 ng/L), respectively. Estimated daily intakes (EDIs) (mean and high-end exposure) via drinking water for different sectors of the UK population were: infants (0.93 and 6.4 ng/kg bw/day) > toddlers (0.46 and 3.1 ng/kg bw/day) > children (0.35 and 2.3 ng/kg bw/day) > adults (0.28 and 2.1 ng/kg bw/day). Based on these data, exposure to $\Sigma_8$OPEs via drinking water is much lower than via: food, indoor dust ingestion, inhalation, and dermal uptake for adults and toddlers. Reassuringly, our EDIs were lower than relevant reference dose (RfD) values. However, combining our drinking water ingestion data with exposure via other pathways revealed overall exposure to 2-ethylhexyl diphenyl phosphate (EHDPP) and TCIPP to approach health-based limit values for UK toddlers under a high-end exposure scenario.


## 1. Introduction

Organophosphate esters (OPEs) have been widely used as a flame retardant (FR) in various industrial and consumer products [1]. Other uses include as stabilisers, plasticisers, antifoaming, and wetting agents, as well as additives in lubricants and hydraulic fluids [2,3]. OPEs are used as additive FRs, meaning that they are not chemically bound to consumer products [4], thus these compounds can be released during production, use, and end of life product management by leaching, volatilisation, and abrasion [5]. Global usage of OPEs as a replacement for restricted BFRs has increased rapidly in recent years from 680,000 t in 2015 to 2,800,000 t in 2018 [1,6]. In western Europe, OPEs have been estimated to account for 20% of total FR consumption [3]. Such widespread and substantial use has led to the detection of OPEs in various environmental matrices including: food, water, air, dust, and sediment, as well as in human tissues such as: breast milk, placenta, blood serum, urine, hair, and nails [7–11].

Such evidence of human exposure is of potential concern given reports that OPEs can cause adverse effects. For instance, tri(2-chloroethyl) phosphate (TCEP) has been reported to decrease red blood cell cholinesterase activity, disrupt the thyroid endocrine system, and elicit neurotoxicity; while triphenyl phosphate (TPhP) has been linked to decreased red blood cell cholinesterase activity, along with neurotoxicity, contact allergenic effects, and impaired fertility. Moreover, tributoxyethyl phosphate (TBOEP) was

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associated with decreased red blood cell cholinesterase activity, and triethyl phosphate (TEP) linked with disruption of the thyroid endocrine system [2,3]. Also of concern, both TCEP and tris (1-chloro-2-propyl) phosphate (TCIPP) are potentially carcinogenic [3,12]. The presence of OPEs in human tissues demonstrates that human exposure occurs, but while research to date has highlighted that this arises via: dermal contact, dust ingestion, inhalation, and dietary intake [7]; relatively few studies have examined drinking water as a pathway of exposure to OPEs. Such studies are, hitherto, limited to: China [13–17], the United States [18,19], Spain [20], and South Korea [21,22]. Against this background, the current study reports concentrations of eight OPEs in 52 samples of drinking water collected in the UK, with the primary objective to assess the significance of drinking water as an exposure pathway of the UK population to OPEs. To the best of our knowledge, this is the first study of the occurrence of OPEs in UK drinking water, and as such will form a valuable baseline against which efforts to minimise this occurrence may be evaluated.

## 2. Materials and methods

### 2.1. Chemicals and materials

All chemicals, reagents and materials used in this study are provided as supplementary information (SI) (section 1).

### 2.2. Sample collection and preparation

Fifty drinking water samples comprising: tap water (n = 25), bottled water (n = 25) were collected from three major cities within the West Midlands conurbation of the UK, between July and August 2021. One litre tap water samples were collected from the kitchen of 25 different homes located within different conurbations (Birmingham, Walsall, and Coventry) in the UK West Midlands; while 25 bottled water samples consisted of single 500–1,500 mL polyethylene terephthalate bottles of popular brands purchased from major grocery stores within Birmingham. A detailed description of how drinking water samples were taken is provided in the supplementary information. Following collection, all samples were stored at 4 °C prior to extraction, which was conducted within 24 h of sample collection.

Approximately 200 mL of drinking water were spiked with 10 ng of isotopically-labelled internal (surrogate) standards ($d_{27}$-TnBP and $d_{15}$-TPHP) and extracted using an Oasis® HLB solid phase extraction cartridge (200 mg, 6 $cm^3$; Waters). The cartridges were preconditioned by a sequence of 10 mL each of dichloromethane, methanol, and Milli-Q purified water. After sample loading, cartridges were dried under a gentle nitrogen gas stream, and eluted with 6 mL of dichloromethane. The eluent was concentrated under a gentle stream of nitrogen to incipient dryness. This was reconstituted with 100 μL of iso-octane containing 10 ng of PCB-62 as recovery determination (syringe) standard (RDS). The final sample concentrate was transferred to an amber vial prior to analysis on an Agilent 5975C GC-MS operated in selected ion monitoring electron ionisation mode and fitted with a 30 m DB-5 MS GC column (0.25 mm ID, 0.25 μm film thickness) (Restek, USA). Detailed information on the instrumental analysis conditions is provided as supplementary information (SI, section 2).

### 2.3. Quality assurance and quality control

All laboratory glassware was washed, rinsed with deionised, distilled Milli-Q water, heated at 460 °C for at least 2 h, then rinsed sequentially with hexane, acetone, and dichloromethane prior to use. For all target analytes, the relative standard deviation of the relative response factors (RRFs) in the five calibration standards (0.05–0.75 ng/μL) were below 8%. Two procedural blanks (n = 2) were included for every batch of five samples with only TCEP detected in blanks at an average concentration of 0.10 ± 0.08 ng/L (Table S3). Concentrations of TCEP in each batch of samples were therefore blank-corrected (by subtracting the average concentrations in the two procedural blanks detected with the samples from each batch). In the absence of an appropriate certified reference material, matrix spiked samples (Milli-Q water) (n = 5) containing 10 ng of each target OPE (equivalent to 50 ng/L in the water sample) were analysed to evaluate method performance. Recoveries of our eight target OPEs in spiked samples ranged from 67 to 123% (Table S3). Moreover, recoveries of the two internal standards were 92 ± 17% and 89 ± 11% for $d_{27}$TnBP and $d_{15}$TPHP respectively (Table S3). The limit of detection (LOD) and the limit of quantification (LOQ) were calculated as the amounts of an analyte that yielded signal to noise ratios (S/N) of 3 and 10 respectively based on 11 injections of the lowest concentration calibration standard (0.05 ng/μL, Table S3). For exposure assessment purposes, OPE concentrations below the LOQ were assumed to be present at either zero (lower bound (LB)) or the LOQ (upper bound (UB)).

### 2.4. Statistical analysis

Descriptive and multivariate statistical analyses were performed using IBM SPSS Statistics 28 (USA) for Windows and Microsoft Excel 365. The data were $\log_{10}$ transformed prior to analysis after a Shapiro-Wilk test showed that the data were not normally distributed. Such data transformation is necessary due to the sensitivity of principal component analysis (PCA) to non-uniformly distributed data [23]. A *t*-test was used to investigate significant differences in OPE (chlorinated, aryl and alkyl OPEs) concentrations between tap and bottled water. The Spearman rank correlation coefficient (r) is used to reflect the linear correlation between the analytes as well as the correlation between OPE concentrations in bottled and tap water, while PCA was carried out after fulfilling the condition of data to be normally distributed. PCA was performed to investigate the possible factors driving OPE concentrations in the three drinking water sample types. This was carried out based on varimax orthogonal rotation with eigen-value > 1 after determining the Kaiser-Meyer OIkin (KMO) test that measured sampling adequacy and Bartlett's Test of sphericity was adequate and found to be significant ($p<0.01$) for the variables [23]. The first four principal components (PCs) with (eigen-values > 1) which explained > 75% of the total variance were retained as the most significant components.

### 2.5. Exposure and risk estimation

The estimated daily intake (EDI) expressed as nanograms per kilogram of body weight per day of OPEs via ingestion of drinking water was calculated for infants, toddlers, children, and adults using the following equation (1)

$$EDI = C_{water}\ x\ Ingestion\ rate\ (IR) \quad 1$$

where $C_{water}$ is the concentrations of a given OPE or combination of OPEs in drinking water (ng/L), IR is the average drinking water ingestion rate (L (kg of bw)$^{-1}$day$^{-1}$) which is 0.05 L (kg of bw)$^{-1}$day$^{-1}$ for infants, 0.026 L (kg of bw)$^{-1}$day$^{-1}$ for toddlers, 0.02 L (kg of bw)$^{-1}$day$^{-1}$ for children and 0.016 L (kg of bw)$^{-1}$ day$^{-1}$ for adults [24]. Both a "normal" exposure scenario (assuming the water consumed was contaminated at the arithmetic mean OPE concentration) and a high-end exposure scenario (assuming the water consumed was contaminated at the 95th percentile OPE

concentration) were evaluated for each of the age groups considered.

## 3. Results and discussion

### 3.1. Concentration of OPEs in drinking water

A statistical summary of concentrations of our target OPEs are shown as Table 1. Similar to previous studies [14,17–22,25–29], TCEP was detected in all the studied drinking water samples (detection frequency (DF) = 100%). In contrast, TCIPP, TDCIPP, TPHP and EHDPP displayed varying DFs ranging from 44 to 96% in tap water and 16–92% in bottled water (Table 1). TnBP displayed the lowest DF, ranging from 0% in bottled water to 16% in tap water. With respect to TMTP and TBOEP, DFs were between 28 - 72% and 20–72%, in tap water and bottled water, respectively (Table 1). The higher DFs observed for TCEP, TCIPP, TPHP, and TBOEP may be associated with the wide past and current application of these OPEs in the UK [21,26], as well as their physicochemical properties. Both TCEP and TCIPP have been used widely in: polyurethane carpet backing, furniture foam, and polystyrene building insulation [3]. Meanwhile, TPHP is used as an additive in food packaging materials [30], while TBOEP is widely used in floor polish/wax and as a plasticiser in vinyl plastics and rubber stoppers [3,31]. Moreover, TCEP, TCIPP, and TBOEP have relatively higher water solubilities (exceeding 1000 mg/L at 25 °C) in comparison to other OPEs (Table S1). Average concentrations of target individual OPEs across the two drinking water categories examined (bottled and tap water) were: TBOEP (7.3 ng/L) > TCIPP (3.9 ng/L) > TCEP (3.6 ng/L) > TPHP (1.2 ng/L) > TnBP (0.66 ng/L) > EHDPP (0.39 ng/L) > TMTP (0.25 ng/L) > TDCIPP (0.11 ng/L) accounting for 42%, 23%, 21%, 7%, 4%, 2%, 1%, and 0.7% of $\sum_8$OPEs, respectively. Thus, TCEP and TCIPP were the predominant chlorinated OPEs (Cl-OPEs), TPHP and EHDPP the predominant aryl-OPEs, while TBOEP was the dominant alkyl-OPE (Fig. 1).

Several studies have reported higher concentrations of TCEP, TCIPP, and TBOEP in drinking water samples than in our study; specifically in China [13–17,25,28,29], in Korea [21,22], and the USA [19]. The mean (95th percentile) concentrations of the chlorinated OPEs, TCEP and TCIPP in UK tap water (TCEP: 3.0 (7.3 ng/L), TCIPP: 2.9 (960 ng/L)) and bottled water (TCEP: 0.60 (1.6 ng/L), TCIPP: 1.0 (1.8 ng/L)) in our study; were lower than the average concentrations reported for drinking water from China (TCEP: 38.8 ng/L; TCIPP: 67.0 ng/L) [21], (TCEP: 9.1 ng/L; TCIPP: 6.7 ng/L) (Ding et al., 2015), (TCEP: 18.7 ng/L; TCIPP: 20.0 ng/L) [28], (TCEP: 27.8 ng/L; TCIPP: 218 ng/L) (Huang et al., 2022), in Korea (TCEP: 39.5; TCIPP; 49.4 ng/L) [22], in US (TCEP: 150 ng/L) [18] and TCIPP (40 ng/L) in drinking water from Spain (Rodil et al., 2012) (Table 2). However, our mean concentrations for the aryl OPEs: TPHP (0.64 and 0.59, ng/L) and EHDPP (0.23 and 0.16) in tap and bottled water, respectively (Table 1) were comparable to those reported in China for TPHP (0.14 and 0.28 ng/L) for ambient temperature water and hot water, respectively [17] but lower than the values reported in other studies from China (TPHP: 21.3 ng/L) [28], (TPHP: 1.11 ng/L) (Huang et al., 2022). Meanwhile, our arithmetic mean concentrations of TBOEP (3.5 and 3.8 ng/L) in tap and bottled water were comparable to those reported in Eastern China [14] and below that reported in eight major metropolitan cities in China (26.1 ng/L) [21]. Among our target OPEs, TBOEP was present at the highest average concentration in: bottled water (3.8 ng/L) and tap water (3.5 ng/L) followed by TCEP with a mean concentration of 3.0 ng/L in tap water and 0.60 ng/L in bottled water (Table 1).

### 3.2. Differences in OPE concentrations between drinking water categories

The average $\sum_8$OPEs concentration in tap water (mean = 11 ng/L) exceeded significantly (*t*-test $p < 0.01$) those in bottled water (mean = 6.4 ng/L) (Table 1). Moreover, the average sum of the three chlorinated OPEs (TCEP, TCIPP, and TDCIPP) in tap water (6.0 ng/L) and bottled water (1.7 ng/L) showed a significant difference ($p < 0.05$) (Fig. 1). This indicated that tap water was more contaminated with chlorinated OPEs than bottled water (Fig. 1). The cause of these higher OPE concentrations in tap water is not clear but may possibly be a result of more efficient purification of bottled water. This is consistent with what was reported in China [14,21,28,29], where tap water displayed higher concentrations of chlorinated OPEs than the other drinking water types considered. For the aryl OPEs such as TPHP, EHDPP and TMTP, mean concentrations in tap water (TPHP: 0.64 ng/L; EHDPP: 0.23 ng/L; TMTP: 0.10 ng/L) were slightly higher (but not significantly) than those obtained from bottled water (TPHP: 0.59 ng/L; EHDPP: 0.16 ng/L; TMTP:0.15 ng/L). Moreover, mean concentrations of alkyl-OPEs: TBOEP and TnBP in bottled water (3.8 and <LOD) and tap water (3.5 and 0.65 ng/L), respectively (Fig. 1) were not significantly different.

- Publication year was assumed as the sampling year unless stated otherwise by the study authors

### 3.3. Potential sources of OPEs in UK drinking water

Spearman's correlation coefficients for the OPEs determined in the drinking water samples are presented in Table 3. Spearman's correlation has been used previously for OPE source identification in the environment [32,33]. Most of the OPEs showed a positive correlation with each other. Significant positive correlations were observed between TCEP and: TCIPP ($r = 0.562$, $p < 0.01$), EHDPP

**Table 1**
Statistical summary of OPE concentrations (ng/L) and detection frequency (DF, %) in the three drinking water categories examined in our study.

| | | Tap water (n = 25) | | | Bottled water (n = 25) | | |
|---|---|---|---|---|---|---|---|
| OPE | LOD | DF (%) | Range (mean) | 95th Percentile | DF (%) | Range (mean) | 95th Percentile |
| TCEP | 0.001 | 100 | 0.37–7.6 (3.0) | 7.3 | 100 | 0.12–2.3 (0.60) | 1.6 |
| TCIPP | 0.006 | 96 | <LOD – 9.9 (2.9) | 7.5 | 92 | <LOD – 2.5 (1.02) | 1.8 |
| TDCIPP | 0.004 | 68 | <LOD – 0.40 (0.08) | 0.18 | 36 | <LOD – 0.17 (0.03) | 0.14 |
| TPHP | 0.003 | 84 | <LOD – 2.5 (0.64) | 1.94 | 92 | <LOD – 3.6 (0.59) | 0.98 |
| EHDPP | 0.002 | 44 | <LOD – 2.4 (0.23) | 1.06 | 16 | <LOD – 2.1 (0.16) | 0.83 |
| TnBP | 0.006 | 16 | <LOD – 5.5 (0.65) | 4.2 | 0 | <LOD | <LOD |
| TBOEP | 0.013 | 72 | <LOD – 13 (3.5) | 12 | 72 | <LOD – 11 (3.8) | 8.4 |
| TMTP | 0.004 | 28 | <LOD – 0.50 (0.10) | 0.45 | 20 | <LOD – 1.7 (0.15) | 1.2 |
| $\sum_8$OPEs | | | 0.41–42 (11) | 35 | | 0.15–23 (6.4) | 15 |

LOD: limit of detection.

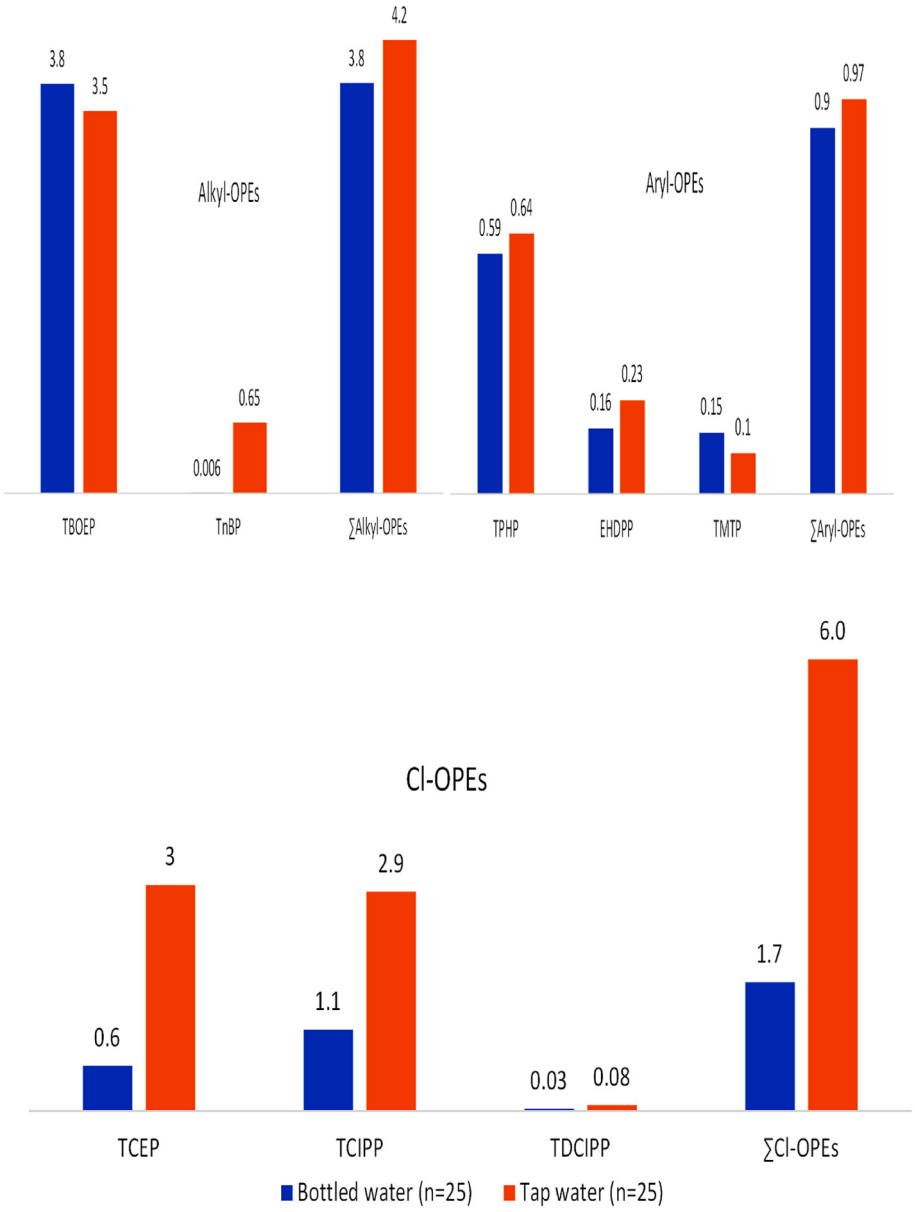


**Fig. 1.** Comparison of arithmetic mean concentrations (ng/L) of different categories of OPEs (chlorinated, aryl and alkyl) in three types of UK drinking water.

($r = 0.458$, $p < 0.01$), and weak but not significant correlations with TDCIPP, TPHP and TnBP ($r = 0.243–0.277$) (Table 3), suggesting that these OPEs likely originate from a common source or shared similar transport mechanism in drinking water [32,34]. This is similar to reports for drinking water in China [26] where TCEP and TCIPP correlated moderately but significantly ($r = 0.40$, $p < 0.05$). Moreover, we also observed a weak positive correlation between TDCIPP and TPHP ($r = 0.323$, $p < 0.05$), EHDPP (0.366, $p < 0.01$), and TMTP ($r = 0.381$, $p < 0.01$), as well as a weak negative correlation between TDCIPP and TnBP ($r = -0.281$, $p < 0.05$) (Table 3). In contrast, TBOEP does not correlate with any other OPE measured, indicating the source(s) of TBOEP differ from those of the other OPEs targeted. These results were also corroborated with PCA results where the initial dimensional reduction and transformation of the data produces four components which explained 79.3% of the data variation (Table S7). The first principal component (PC-1) accounts for 23.8% of the total variation and was heavily loaded on three OPEs: TCEP (0.87), TCIPP (0.74) and EHDPP (0.61) (Table S7). This suggests similar contamination sources of these OPEs, which for the Cl-OPEs likely reflects their widespread use in polyurethane foam (PUF) for furniture, upholstery, and mattresses, and for building insulation [3]. The second component (PC-2) explains 21.1% of the total variance (Table S7) and is driven primarily by a high positive loading of 0.86 for TPHP and a strong negative loading of −0.81 for TnBP. The third component (PC-3) has high positive loadings for TMTP (0.92) and TDCIPP (0.63) and accounted for 18.9% of total variance. Fig. 2 plots the first three PCs. In addition, the fourth component (PC-4) was driven substantially only by TBOEP (0.95) and explained 15.5% of the total variation (Table S7). This is consistent with the results of our correlation analysis and likely reflects the use of TBOEP in floor polish, which is an application distinct from those of our other target OPEs.

### 3.4. Exposure assessment of OPEs via drinking water

To evaluate the potential health risk of OPE exposure via ingestion of UK drinking water, the estimated daily intake (EDI) of OPEs was calculated (Tables S8a–c, Fig. 3). Under a normal

**Table 2**
Comparison of arithmetic mean (unless indicated otherwise) concentrations (ng/L) of OPEs in drinking water measured in this study with mean concentrations reported from other countries.

| Country (Sampling year) | Water type (n) | TCEP | TCIPP | TDCIPP | TPHP | EHDPP | TnBP | TBOEP | TMTP | Reference |
|---|---|---|---|---|---|---|---|---|---|---|
| China (2012) | Bottled water (n = 8) | 0.43 | 6.58 | ND | 6.95 | ND | 1.16 | 31.7 | – | [16] |
| China (2014)[a] | Bottled water (n = 23) | 0.5 | 0.6 | 0.6 | 0.8 | – | 0.1 | 0.2 | – | [14] |
| Korea (2014) | Bottled water (n = 10) | 16.4 | 79.6 | – | 0.99 | – | 4.24 | 64.4 | – | [21] |
| **United Kingdom (2022)** | **Bottled water (n = 25)** | **0.60** | **1.1** | **0.03** | **0.59** | **0.16** | **<0.01** | **3.8** | **0.15** | **This study** |
| China (2014) | Tap water (n = 21) | 48.5 | 43.0 | 5.8 | 1.4 | – | 9.5 | 3.7 | – | [14] |
| China (2014) | Tap water (n = 39) | 14.1 | 20.3 | ND | 5.17 | ND | 3.39 | 3.67 | – | [16] |
| Korea (2014) | Tap water (n = 75) | 25.3 | 10.7 | – | 1.98 | – | 4.29 | 10.7 | – | [21] |
| USA (2016–2017) | Tap water (n = 58) | 0.45 | 11.6 | 4.76 | 3.72 | 0.29 | 2.47 | 10.2 | – | [19] |
| Korea (2017) | Tap water (n = 44) | 39.5 | 49.4 | 2.00 | 23.0 | – | 11.8 | 43.9 | – | [22] |
| China (2020) | Tap water (n = 1) | 0.31 | 8.99 | – | 7.47 | 0.63 | 6.15 | – | – | [26] |
| China (2022) * | Tap water (n = 47) | 18.7 | 20.0 | 3.21 | 21.3 | – | 19.6 | 1.04 | – | [28] |
| **United Kingdom (2022)** | **Tap water (n = 25)** | **3.0** | **2.9** | **0.08** | **0.64** | **0.23** | **0.65** | **3.5** | **0.10** | **This study** |
| China (2014)[a] | Filtered drinking water (n = 17) | 9.1 | 6.7 | 0.5 | 0.2 | – | 0.9 | 0.3 | – | [14] |
| USA (2006–2007)[a] | Drinking Water (n = 15) | 150 | 220 | – | – | – | –[b] | – | – | [18] |
| China (2021)[a] | Drinking water (n = 38) | 27.8 | 218 | NQ | 1.11 | ND | 29.6 | 3.09 | – | [29] |
| Spain (2008)[a] | Drinking water (n = 28) | 5 | 40 | – | – | – | 32 | – | – | [20] |
| Korea (2014) | Drinking water (n = 127) | 38.8 | 67.0 | 4.46 | 2.12 | 0.54 | 3.40 | 26.1 | – | [21] |
| China (2020) | Drinking water (n = 25) | 45.7 | 46.9 | 13.1 | 1.63 | 0.65 | 15.6 | – | 0.62 | [27] |
| China (2014)[a] | Well water (n = 19) | 0.5 | 2.5 | 0.1 | 0.2 | – | 0.2 | 0.2 | – | [14] |
| China (2014)[a] | Barrelled water (n = 19) | 6.9 | 8.0 | 0.5 | 0.2 | – | 0.1 | ND | – | [14] |
| China (2020–2021) | Barrelled water (n = 28) | 0.16 | 0.14 | – | – | – | – | – | – | [17] |
| China (2020–2021 | Normal temperature water (n = 53) | 1.31 | 22.4 | 0.28 | 0.14 | – | 0.34 | – | – | [17] |
| China (2020–2021 | Hot water (n = 53) | 3.74 | 65.0 | 0.50 | 0.28 | – | 0.37 | – | – | [17] |

ND = not detected.
NQ = Not quantified (concentrations above the MLOD but lower that the method limit of quantification (MLOQ) is defined as NQ.
[a] Median value.

**Table 3**
Spearman's correlation coefficients between OPEs in UK drinking water.

| OPE | TCEP | TCIPP | TDCIPP | TPHP | EHDPP | TnBP | TBOEP | TMTP |
|---|---|---|---|---|---|---|---|---|
| **TCEP** | 1.000 | 0.562** | 0.243 | 0.276 | 0.458** | 0.277 | 0.051 | −0.071 |
| **TCIPP** | | 1.000 | 0.198 | 0.069 | 0.278 | 0.235 | −0.132 | −0.086 |
| **TDCIPP** | | | 1.000 | 0.323* | 0.366** | −0.281* | 0.013 | 0.381** |
| **TPHP** | | | | 1.000 | 0.074 | −0.212 | 0.045 | −0.179 |
| **EHDPP** | | | | | 1.000 | 0.181 | 0.047 | 0.211 |
| **TnBP** | | | | | | 1.000 | 0.000 | −0.163 |
| **TBOEP** | | | | | | | 1.000 | −0.147 |
| **TMTP** | | | | | | | | 1.000 |

**. Correlation is significant at the 0.01 level (2-tailed); *. Correlation is significant at the 0.05 level (2-tailed).

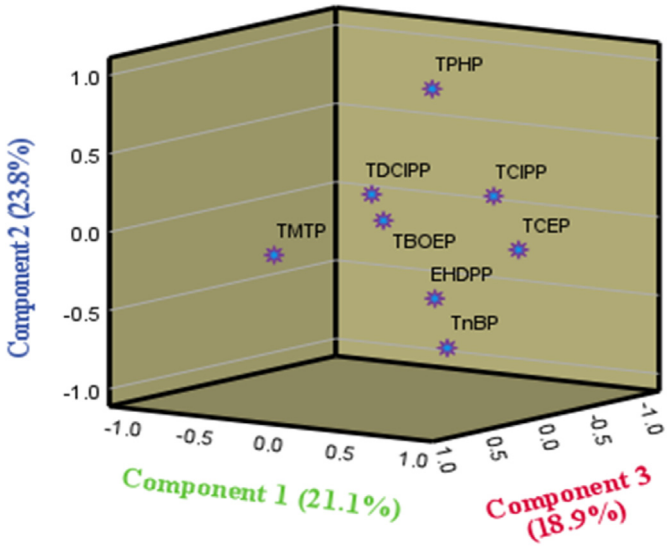


**Fig. 2.** Graphical representation of principal component analysis of OPE concentrations in UK drinking water.

exposure scenario (using mean concentrations), EDIs of $\sum_8$OPEs for infants, toddlers, children, and adults were 0.59, 0.29, 0.22 and 0.18 ng (kg of bw)$^{-1}$ day$^{-1}$ for tap water and 0.34, 0.17, 0.13, and 0.10 ng (kg of bw)$^{-1}$ day$^{-1}$ for bottled water respectively (Table S10). The relative contribution of the individual OPEs to the total EDI via the ingestion of the drinking water for the four age-groups are presented in Fig. S1 and Fig. S2. Daily intakes of $\sum_8$OPEs via tap water ingestion for the four age groups was between 41 and 44% higher than those for bottled water (Tables S9–S10). This concurred with several other studies that reported higher daily intake of $\sum$OPEs for tap water in China (7.07–7.7 ng (kg of bw)$^{-1}$ day$^{-1}$) [14,16,28], and in South Korea (254 ng/person/day) [22]. Our mean EDIs for $\sum_8$OPEs in tap water for adults (0.18 ng (kg of bw)$^{-1}$ day$^{-1}$) and children (0.22 ng (kg of bw)$^{-1}$ day$^{-1}$) were below those reported in drinking water from China [14] for adults (7.07 ng (kg of bw)$^{-1}$ day$^{-1}$) and children (boy: 6.95 ng kg of bw)$^{-1}$ day$^{-1}$; girl: 6.8 ng kg of bw)$^{-1}$ day$^{-1}$, respectively. Table 4 compares our EDIs with those reported for drinking water from other studies, as well as EDIs for other exposure pathways in UK. Our data reveals that infants (mean exposure: 0.93 ng/kg bw/day; high-end exposure: 6.4 ng/kg bw/day) have a higher daily exposure to our target OPEs via drinking water than toddlers

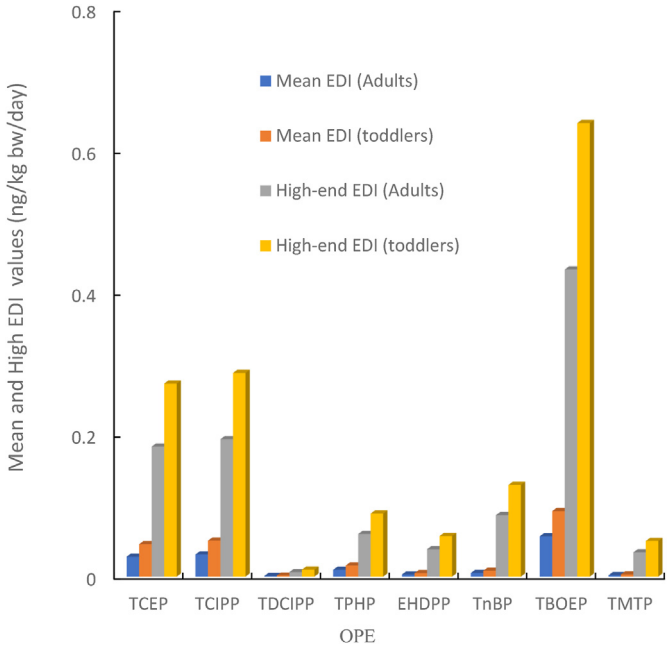


**Fig. 3.** Average and high exposure EDI values (ng/kg bw/day) of individual OPEs via drinking water for adults and toddlers (EDIs plotted represent the average of those obtained for ingestion of tap and bottled water).

(mean: 0.46 ng/kg bw/day; high-end exposure: 3.1 ng/kg bw/day) and children (mean: 0.35 ng/kg bw/day; high-end: 2.3 ng/kg bw/day) with the least exposed being adults (mean: 0.28 ng/kg bw/day; high-end exposure: 2.1 ng/kg bw/day), respectively (Table 5; Table S10). This exposure risk trend is essentially driven by differences between the age groups in the water consumption rates normalised to body weight. The total estimated daily intake of $\sum_8$OPEs attributable to UK drinking water were lower than those reported from different other countries (Table 4). Moreover, the fact that higher EDIs were reported in the drinking water from other countries than the UK, may be because of the small sample size in this study. Therefore, analysis of a greater number of UK drinking water samples is required. We also compared our data for exposure of the UK population to individual OPEs via drinking water with other pathways of human exposure to OPEs. A summary of our drinking water ingestion exposure data in comparison with other exposure pathways is presented in Table 4. This shows that UK population's exposure to OPEs via drinking water for all age groups was below the reported values for other exposure pathways but nonetheless represents an appreciable human exposure pathway, especially for infants, toddlers, and children.

### 3.5. Risk assessment of OPEs via drinking water

To evaluate the health risks of OPEs via drinking water in UK, the $\sum$EDI values for $\sum_8$OPEs for infants, toddlers, children, and adults were compared with different oral reference doses (RfDs) available in the literature [43–47]. The mean and high exposure EDI values for $\sum_8$OPEs for infants (0.93 and 6.4 ng (kg of bw)$^{-1}$ day$^{-1}$), toddlers (0.46 and 3.1 ng (kg of bw)$^{-1}$ day$^{-1}$), children (0.35 and 2.3 ng (kg of bw)$^{-1}$ day$^{-1}$), and adults (0.28 and 2.1 ng (kg of bw)$^{-1}$ day$^{-1}$) (Table 5) were several order of magnitude below the reference dose values provided by Refs. [43–47].

**Table 4**
**Comparison of the estimated daily intake (EDI, ng (kg of bw)$^{-1}$ day$^{-1}$) of OPEs via drinking water for different countries and with other pathways in the UK.**

| Exposure Pathway | Number of OPEs | Country | EDI toddlers | | EDI children | | EDI adults | | Reference |
|---|---|---|---|---|---|---|---|---|---|
| | | | Mean | 95th %ile | Mean | 95th %ile | Mean | 95th %ile | |
| Drinking water ingestion | 8 | United Kingdom | 0.46 | 3.1 | 0.35 | 2.3 | 0.28 | 2.1 | This study |
| Drinking water ingestion[a] | 3 | Korea | 2.55 | 16.5 | 2.10 | 13.6 | 1.81 | 11.8 | [21] |
| Drinking water ingestion | 9 | China | – | – | 6.88 | 11.3 | 6.56 | 10.7 | [14] |
| Drinking water ingestion[a] | 5 | China | – | – | 4.37 | 37.2 | 4.14 | 35.5 | [15] |
| Drinking water ingestion | 11 | China | 1.65 | 114 | 0.65 | 44.6 | 0.93 | 64.5 | [17] |
| Drinking water ingestion | 8 | China | – | – | 3.22 | – | 3.27 | – | [28] |
| Food ingestion | 8 | UK | 420 | 1547 | 155 | 836 | 62.3 | 278 | [35] |
| Dermal absorption | 3 | UK | 15.5–36 | 293 | – | – | 4.1–25.1 | 36.9 | [36,37] |
| Air inhalation | 8–20 | Germany, China, USA and Norway | ND-2.4 | ND | ND-273 | ND-916 | 0.41–170 | ND-570 | [38–41] |
| Dust ingestion[a] | 7 | UK | – | – | 70 | 1740 | 1.3 | 28 | [42] |

Note: ND means not detected.
[a] Median concentrations.

**Table 5**
Estimated daily drinking water ingestion data (mean and – in parentheses - high-exposure scenario (ng/kg bw/day) for OPEs and their corresponding reference doses (RfDs).

| Age group | TCEP | TCIPP | TDCIPP | TPHP | EHDPP | TBOEP | TnBP | TMTP | $\sum$OPEs |
|---|---|---|---|---|---|---|---|---|---|
| Infants (1 to < 3 months) | 0.19 (1.1) | 0.21 (1.2) | 0.01 (0.04) | 0.07 (0.38) | 0.02 (0.24) | 0.39 (2.7) | 0.03 (0.55) | 0.01 (0.22) | 0.93 (6.4) |
| Toddlers (2 to < 3 years) | 0.09 (0.55) | 0.10 (0.58) | 0.003 (0.02) | 0.03 (0.18) | 0.01 (0.12) | 0.19 (1.3) | 0.02 (0.26) | 0.01 (0.10) | 0.46 (3.1) |
| Children (11 to < 16 years) | 0.07 (0.41) | 0.08 (0.44) | 0.002 (0.02) | 0.03 (0.14) | 0.008 (0.09) | 0.15 (0.97) | 0.01 (0.20) | 0.005 (0.08) | 0.35 (2.3) |
| Adults (≥21 years) | 0.06 (0.37) | 0.06 (0.39) | 0.002 (0.01) | 0.02 (0.12) | 0.006 (0.08) | 0.12 (0.87) | 0.011 (0.18) | 0.004 (0.07) | 0.28 (2.1) |
| RfD (ng/kg bw/day)[a] | 7000 | 10000 | 20000 | NA | NA | NA | 10000 | NA | |
| RfD (ng/kg bw/day)[b] | 2200 | 8000 | 1500 | 7000 | 600[d] | 1500 | 2400 | NA | |
| RfD (ng/kg bw/day)[c] | 22000 | 80000 | 15000 | 70000 | NA | 15000 | 24000 | NA | |
| RfD (ng/kg bw/day)[e] | NA | 3600 | NA | NA | NA | NA | NA | NA | |

NA = Not available.
[a] Reference dose (RfD) values of [43].
[b] [44].
[c] [45].
[d] [46].
[e] [47].

To evaluate the overall human exposure to OPEs in UK, the present study examined the combined mean and high-end human exposure to OPEs for adults and toddlers in UK drinking water with exposure data for dust ingestion for adults [42] and toddlers [48], dermal uptake for adults and toddlers [37] – while the high-end exposure estimate for dermal uptake for adults was obtained from Ref. [36], with the recent UK dietary intake exposure data [35]. The available dermal uptake exposure data for only three Cl-OPEs (TCEP, TCIPP and TDCIPP) were used for this combined exposure risk assessment as there is no available data for all other OPEs for all age groups. A further important point for future studies is that there is no UK data for OPEs exposure via air inhalation for all age groups [35]. Based on the available UK OPEs exposure data for the other exposure pathways (dust ingestion, dermal and food ingestion), the combined exposure risk for UK population was evaluated for adults and toddlers for five OPEs (TCEP, TCIPP, TDCIPP, TPHP and EHDPP). For adults, these EDIs for mean and high-end exposure via these four pathways combined were as follows: TCIPP (mean = 26, high-end: 52 = ng/kg bw/day) > EHDPP (14 and 46 ng/kg bw/day) > TPHP (10 and 44 ng/kg bw/day) > TDCIPP (6.8 and 22 ng/kg bw/day) > TCEP (4.1 and 21 ng/kg bw/day) (Table S11). However, for toddlers, the combined EDIs were: TCIPP (mean = 296, high-end = 1340 ng/kg bw/day) > EHDPP (126 and 335 ng/kg bw/day) > TPHP (123 and 309 ng/kg bw/day) > TDCIPP (24 and 123 ng/kg bw/day) > TCEP (17 and 95 ng/kg bw/day) (Table 6 and Table S11).

Our results for the combined exposures for adults and toddlers were still below the reference dose values ($\sum$EDIs <<< RfDs) cited in Table 6 and Table S11 [43–47]. However, for some OPEs, ΣEDIs approached one or more of the RfD values. This may be expressed quantitatively as the hazard quotient (HQ) which represents the ratio of the ΣEDI to the RfD. There is a significant exposure risk for OPEs with HQ $\geq$ 1, a situation where the exposure matches or exceeds the RfD value. As an illustration, Table 6 shows the HQ values for toddlers under the average and high-end exposure scenario, and those adults are provided in Table S11 respectively. While HQ values did not exceed 1 for any of our target OPEs; for toddlers, those for EHDPP were: 0.21 (mean) and 0.56 (high-end) using the RfD value of 600 ng/kg bw/day [46]. Also for toddlers, those for TCIPP ranged from 0.03 (mean) to 0.37 (high-end) depending on which RfD value was used, while a maximum HQ value of 0.08 was obtained for TDCIPP when high-end toddler exposure was compared to an RfD of 1500 ng/kg bw/day [44] (Table S11). This shows that of our target OPEs, exposure risk to UK toddlers is greatest for EHDPP, TCIPP, and to lesser extent TDCIPP (Table 6), and for EHDPP and TCIPP is within an order of magnitude of one or RfD values. It should also be noted that our ΣEDIs do not include inhalation exposure, which will further erode the margin of safety between exposure and the RfD.

## 4. Conclusion

This study provides the first data on concentrations of OPEs in UK drinking water (comprising tap and bottled water). The most abundant OPEs detected in UK tap water were: TCEP (range: 0.37–7.6 ng/L; average: 3.0 ng/L), TCIPP (range: <LOQ – 9.9 ng/L; average: 2.9 ng/L), TPHP (range: <LOQ – 2.5 ng/L; average: 0.64 ng/L), and TBOEP (range: <LOD – 13 ng/L; average: 3.5 ng/L). Our data shows that tap water was the most contaminated with chlorinated OPEs (TCEP and TCIPP), aryl-OPEs (TPHP and EHDPP), and alkyl-OPEs (TBOEP). Comparison of our drinking water data with other exposure pathways to OPEs in the UK, reveals drinking water ingestion poses a lower exposure risk to OPEs than through dermal uptake, food, and dust ingestion. Estimated exposure of the UK population to $\sum_8$OPEs via drinking water was of the order: infants (mean: 0.93; high-end: 6.4 ng/kg bw/day) > toddlers (mean: 0.46; high-end: 3.1 ng/kg bw/day) > children (0.35; high-end: 2.3 ng/kg bw/day) > adults (0.28; high-end: 2.1 ng/kg bw/day). While estimated exposure to OPEs via drinking water fell well below reference dose values; the combined exposure of the UK population via: dermal uptake, drinking water, dust, and food ingestion revealed exposure to be within an order of magnitude of the respective RfDs for: EHDPP and TCIPP for toddlers. Overall, UK drinking water ingestion is an appreciable source of human exposure to OPEs, representing a small but significant addition to overall human exposure.

**Table 6**
Average and high exposure risk estimates (ng/kg bw/day) for toddlers in the UK via dust ingestion, dermal absorption, dietary intake, and water ingestion combined.

| Average exposure estimates | TCEP | TCIPP | TDCIPP | TPHP | EHDPP |
|---|---|---|---|---|---|
| Dust – [48][a] | 3.55 | 262 | 3.06 | 6.13 | 9.65 |
| Food – [35] | 12.5 | 19.4 | 20.5 | 117 | 116 |
| Dermal – [37] | 1.2 | 14.3 | | | |
| Drinking water (this study) | 0.09 | 0.10 | 0.003 | 0.03 | 0.01 |
| **SUM** | **17** | **296** | **24** | **123** | **126** |
| RfD [43] | 7000 | 10000 | 20000 | | |
| RfD [44] | 2200 | 8000 | 1500 | 7000 | 600[b] |
| RfD [45] | 22000 | 80000 | 15000 | 70000 | NA |
| RfD [47] | NA | 3600 | NA | NA | NA |
| HQ based on RfD value provided by [43] | 0.0025 | 0.030 | 0.0012 | – | – |
| HQ based on RfD value provided by [44] | 0.0079 | 0.037 | 0.016 | 0.018 | 0.21[b] |
| HQ based on value provided by [45] | 0.00079 | 0.0037 | 0.0016 | 0.0018 | NA |
| HQ based on RfD value provided by [47] | NA | 0.082 | NA | NA | NA |
| **High-end exposure estimates** | | | | | |
| Dust – [48][a] | 14.2 | 1049 | 12.2 | 24.5 | 38.6 |
| Food – [35] | 41.6 | 71.5 | 74.2 | 284 | 296 |
| Dermal – [36] | 38.6 | 218 | 37 | | |
| Drinking water - (this study) | 0.55 | 0.58 | 0.02 | 0.18 | 0.12 |
| **SUM** | **95** | **1340** | **123** | **309** | **335** |
| HQ based on RfD value provided by [43] | 0.014 | 0.13 | 0.0062 | – | – |
| HQ based on RfD value provided by [44] | 0.043 | 0.17 | 0.082 | 0.044 | 0.56[b] |
| HQ based on RfD value provided by [45] | 0.0043 | 0.017 | 0.0082 | 0.0044 | – |
| HQ based on RfD value provided by [47] | | 0.37[a] | | | |

NA = Not available.
[a] Median EDI values.
[b] [46].

## Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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## Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.emcon.2023.100203.

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