Introduction

1. The Scientific Advisory Committee on Nutrition (SACN) is undertaking a review of scientific evidence that will inform the Government’s dietary recommendations for infants and young children. The SACN is examining the nutritional basis of the advice. The Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) was asked to review the risks of toxicity from chemicals in the diet of infants, most of which has been completed, and young children. The reviews will identify new evidence that has emerged since the Government’s recommendations were formulated, and will appraise that evidence to determine whether the advice should be revised. The recommendations cover diet from birth to age five years.

2. This risk characterisation for tetrabromobisphenol A (TBBPA) makes use of a scientific opinion from EFSA (EFSA 2011). Following a request from the European Commission, the Panel on Contaminants in the Food Chain (CONTAM Panel) was asked to deliver a scientific opinion on TBBPA and its derivatives in food.

3. In the scientific literature reviewed by EFSA, toxicokinetic data for TBBPA was available for the rat but not other experimental animals. TBBPA is readily absorbed from the gastrointestinal tract in rats. Systemic bioavailability of TBBPA as the parent compound is low with most distributed directly to the liver. Glucuronide and sulphate conjugates of TBBPA were identified in bile indicating enterohepatic circulation. The primary route of elimination following $^{14}$C-TBBPA administration was in faeces. The plasma half-life in rats was estimated to be approximately 13 hours. In humans, the half-life of TBBPA-glucuronide in plasma was estimated to be between 48 and 72 hours. Toxicological studies with TBBPA have been carried out using different experimental designs with single or repeated administration during gestation, postnatally or in adulthood. The main target for TBBPA toxicity is thyroid hormone homeostasis. The limited available studies do not indicate reproductive or teratogenic effects of TBBPA. The acute toxicity of TBBPA was reported as very low in rodents with an oral LD50 >50,000 mg/kg in rats and >10,000 mg/kg in mice (ECB 2006).

Previous risk assessments

4. TBBPA has not been evaluated by the Joint FAO/WHO Expert Committee on Food Additives (JECFA). TBBPA and its derivatives are not currently regulated in food by the European Commission. TBBPA has been registered in REACH (Commission Regulation (EC) No. 1907/2006).
5. As TBBPA exposure estimates in infants were lacking, the European Chemicals Bureau (ECB) conducted a chronic risk assessment using the highest concentration of TBBPA detected in breast milk (11 μg/kg fat in breast milk), resulting in an estimated exposure of 24 ng TBBPA /kg b.w./day for breast fed infants during their first 12 months after birth (ECB 2006). By comparing this estimate to a NOAEL of 40 mg/kg/day for the absence of nephrotoxicity in newborn rats (Fukuda et al. 2004), a MOE of $1.7 \times 10^6$ was calculated. The ECB report concluded that this MOE was sufficient to allow for intra- and inter-species differences and that no further information or testing were needed.

6. Based on the European Union (EU) draft risk assessment (later published as ECB 2006), the COT issued a statement on the available toxicological data of TBBPA (COT 2004). The highest oral dose tested in a 90-day rat study and in a two-generation rat reproductive toxicity study of 1,000 mg/kg b.w./day, at which ‘no clear adverse effects were observed’ (MPI research 2002, as cited in ECB 2006 and subsequently published as Cope et al., 2015), was considered to be a NOAEL and used as the basis for deriving a tolerable daily intake (TDI). An uncertainty factor of 100 (for intra- and inter-species variation in toxicokinetics and toxicodynamics) with an additional uncertainty factor of 10 (for the absence of chronic toxicity studies) was applied. Thus, the COT recommended a TDI of 1 mg/kg b.w./day, and concluded that the available data did not raise specific toxicological concerns.

7. It was noted by the ECB in their risk assessment report in TBBPA that one or two Member States expressed concern over the oral absorption of undissolved TBBPA particles, particularly when administered as a suspension at high dose levels (ECB 2006). In the opinion of these Member States, there was some uncertainty as to whether 100 % of the administered dose would be absorbed at these higher dose levels, and consequently whether the dosing of particles in suspension will underestimate the toxicity. However, the majority of Member States agreed with the position of the UK rapporteur that, although this concept is important, the data do not allow a quantitative estimate of oral absorption at such high dose levels to be determined. Therefore, it was agreed to assume that 100 % of an orally administered dose of TBBPA is absorbed.

8. In a scientific opinion from EFSA (2011), no long-term carcinogenicity studies on TBBPA or its derivatives were identified. However, based on the weight of evidence (absence of genotoxicity in vitro, no indications for proliferative changes or cytotoxicity in studies with up to 90 days repeated administration, no immunosuppression except possibly at high doses) the Panel concluded that there are no indications that TBBPA might be carcinogenic. No studies on health effects in humans due to exposure to TBBPA and/or its derivatives were identified in the literature by the Panel.

9. The National Toxicology Program (NTP) published a technical report on the toxicology and long-term carcinogenicity studies of TBBPA (NTP 2014). These studies were performed in the Wistar Han rat and B6C3F1/N mice where they were administered TBBPA (purity of approximately 99 %) in corn oil via gavage 5 days per

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1 Available at: https://cot.food.gov.uk/sites/default/files/cot/cotstatements04tbbpa.pdf
week for 2 years. This dosing regimen was employed to mimic worker exposure. Treated groups received either 250, 500 or 1,000 mg TBBPA/ kg b.w. Control animals received just corn oil by the same method. At the end of the study, tissues from over 40 sites were examined for every animal. In female rats there was ‘clear evidence of carcinogenic activity’, due to significant increases in the incidences of malignant tumours of the uterus in the 500 and 1,000 mg/kg groups (6/50, 11/50, 16/50 and 19/50). In male mice there was ‘some evidence of carcinogenic activity’, due to a significant increase in the incidence of hepatoblastoma in the 250 and 500 mg/kg dose groups (2/50, 11/50, 8/50; incidence data for the 1,000 mg/kg group omitted due to early mortality). It was concluded that TBBPA caused cancers of the uterus in female rats and of the liver in male mice. In addition, TBBPA was found not to be mutagenic in S. typhimurium strains TA98, TA100, TA1535, or TA1537, or E. coli strain WP2 uvrA/pKM101, with or without exogenous metabolic activation. In vivo, no increases in micronucleated erythrocytes were observed in the peripheral blood of male or female B6C3F1/N mice following 3 months of administration of TBBPA by gavage, suggesting that TBBPA did not induce bone marrow toxicity over the dose range tested (10 to 1,000 mg/kg). Several possible mechanisms for the carcinogenicity for TBBPA were discussed which included oxidative damage and disruption of gene regulation and/or hormone signalling.

10. Due to ‘sufficient evidence of carcinogenicity in experimental animals’ and mechanistic evidence, the International Agency for Research on Cancer (IARC) has recently upgraded this flame retardant to group 2A (probably carcinogenic to humans) (IARC 2018). In making its overall evaluation, a majority of the Working Group considered the mechanistic evidence that TBBPA can operate through three key characteristics of carcinogens (modulation of receptor-mediated effects, induction of oxidative stress and immunosuppression) as strong, and that these mechanisms can be operative in humans.

**Background**

11. TBBPA is a brominated flame retardant (BFR) which is incorporated into various consumer and commercial products to improve fire resistance. TBBPA is the BFR with the largest worldwide production volume representing about 60 % of the total BFR market. Although TBBPA is no longer produced in the EU, products containing TBBPA are still imported into the EU from non-EU countries. The IUPAC name for TBBPA is 4,4\'-{(propane-2,2-diyl)bis(2,6-dibromophenol)} and the chemical structure is displayed below.

![Chemical Structure of TBBPA](image)

12. Approximately 90 % of the total use of TBBPA is as a reactive intermediate in the manufacture of epoxy and polycarbonate resins, where it is covalently bound
with the polymer. However a portion of the TBBPA may be unreacted and can leach out of the material. TBBPA is also incorporated additively into materials such as acrylonitrile butadiene styrene (ABS) resins. Here, it is not covalently bound with the polymer, and can leach out into the environment where it has been detected in outdoor and indoor air, domestic dust and biological matrices such as fish and birds.

13. Derivatives of TBBPA exist which are commercially available as flame retardants. These can be used as reactive or additive intermediates in polymer manufacture and, for example, include tetrabromobisphenol A bis(2-hydroxyethyl) ether (TBBPA-bOHEE), tetrabromobisphenol A bisallyl ether (TBBPA-bAE), tetrabromobisphenol A bis(2,3-dibromopropyl ether) (TBBPA-bDiBPrE) and tetrabromobisphenol A bis(glycidyl ether) (TBBPA-bGE).

**Margin of Exposure**

14. In a one-generation reproduction study in Wistar rats dosed with 0, 3, 10, 30, 100, 300, 1000 or 3000 mg TBBPA/kg b.w./day, a BMDL\textsubscript{10} for a decrease in circulating thyroxine (T4; a thyroid hormone) of 16 and 30 mg/kg b.w./day was reported for female and male rats, respectively (van der Ven et al. 2008). For the most sensitive effects, van der Ven et al. 2008 also calculated a BMDL\textsubscript{5} of 0.5 mg/kg b.w./day for increased absolute testes weight and a BMDL\textsubscript{10} of 0.6 mg/kg b.w./day for increased pituitary weight. In contrast, in a 2-generation reproduction study in Sprague-Dawley rats with doses up to 1,000 mg/kg b.w./day, TBBPA had no effects on testes or pituitary weights (MPI research 2002, as cited in ECB 2006 and subsequently published as Cope et al., 2015). Therefore the Panel concluded that the critical effect of TBBPA was on circulating T4 levels in female and male rats (van der Ven et al., 2008) and decided to use the BMDL\textsubscript{10} of 16 mg/kg b.w./day as the reference point for risk characterisation.

15. The Panel identified a number of limitations and uncertainties in the toxicological database which were considered to make the derivation of a health-based guidance value (HBGV) inappropriate for TBBPA. For example, the reported ratios between the benchmark dose (BMD) and its lower and upper confidence limits for several toxicological endpoints were large, indicating considerable uncertainties in the outcome of the BMD modelling. Therefore the Panel used an MOE approach for the risk characterisation of TBBPA.

**Exposure assessment**

16. A call for data on BFRs was issued by EFSA in 2009. EFSA evaluated a total of 652 analytical results for TBBPA in food samples, with no data on TBBPA derivatives. These data were provided by four European countries (Norway, UK, Ireland and Spain) and covered the period from 2003 to 2010. From the 20 aggregated food groups available at the first level of FoodEx (a food classification system developed by EFSA), the dominant food categories were ‘fish and other seafood’ (n = 465), followed by ‘meat and meat products’ (n = 49) and ‘milk and dairy
products’ (n = 40). The analytical results on TBBPA in all food samples from the food groups covered were reported as < LOQ. Therefore the Panel concluded these data were not appropriate to carry out a meaningful dietary exposure assessment for the general population. However the Panel decided to estimate a hypothetical ‘worst-case’ chronic dietary exposure to population groups with potential high exposure to TBBPA. These included breast-fed infants and toddlers (high consumers of cow’s milk). The Panel also estimated chronic exposure from ingestion of dust in homes, classrooms and cars, as this was considered to be another source of exposure for children.

Breast-fed infants

17. TBBPA occurrence data in human milk from three European countries was used to estimate infant exposure via breast-feeding (Table 1). An age of three months was used which corresponds to a body weight of approximately 6.1 kg (mean). An estimated daily consumption of 800 mL/day (mean) or 1,200 mL/day (high) of breast milk with a mean fat content of 3.5 % was used. For infants with mean milk consumption, the daily mean exposures range from 0.28 - 19 ng/kg b.w./day (range <0.18 - 170 ng/kg b.w./day). For infants with high human milk consumption, the daily mean exposures range from 0.41 to 28 ng/kg b.w./day (range <0.28 to 260 ng/kg b.w./day) (EFSA 2011).

**Table 1: Exposure assessment of breast-fed infants to TBBPA across the EU.**

<table>
<thead>
<tr>
<th>European country</th>
<th>Mean milk consumption</th>
<th>High milk consumption</th>
<th>Mean milk consumption</th>
<th>High milk consumption</th>
<th>Reference (occurrence data)</th>
</tr>
</thead>
<tbody>
<tr>
<td>UK</td>
<td>0.28</td>
<td>0.41</td>
<td>&lt;0.18 - 3.0</td>
<td>&lt;0.28 - 4.5</td>
<td>Abdallah &amp; Harrad 2011</td>
</tr>
<tr>
<td>Norway</td>
<td>0.31</td>
<td>0.46</td>
<td>Not given</td>
<td>Not given</td>
<td>Thomsen et al. 2002</td>
</tr>
<tr>
<td>France</td>
<td>19</td>
<td>28</td>
<td>0.28 - 170</td>
<td>0.41 - 260</td>
<td>Cariou et al. 2008</td>
</tr>
</tbody>
</table>

Values rounded to 2 significant figures (SF)

High consumers of cow’s milk (toddlers)

18. The maximum LOQ reported to EFSA for the ‘milk and dairy products’ food group was 0.65 ng/g wet weight. This value was used as the concentration of TBBPA in the ‘liquid milk’ category which were all reported as < LOQ. Together with a daily consumption rate of 85.7 g/kg b.w./day of liquid milk (highest 95th percentile in EFSA database), a hypothetical worst case (i.e. ‘upper bound’) chronic dietary exposure to TBBPA of 55.7 ng/kg b.w./day for toddlers was estimated by the Panel. The Panel noted that this estimation for toddlers should be considered with caution,
as it is derived from limited occurrence data in one food group across four European countries, and therefore may not reflect total dietary exposure across the EU.

Non-dietary exposures (children)

19. Harrad et al. (2010) reported a 95th percentile concentration of TBBPA in dust (from UK classrooms of schools and day care centres) to be 460 ng/g dust. Together with typical and high-end rates of dust ingestion (50 and 200 mg dust/child/day, respectively) and a body weight of 20 kg for children, the Panel estimated exposure to TBBPA via dust ingestion to be 1.2 or 4.6 ng/kg b.w./day, respectively.

UK Total Diet Study

20. Chronic dietary TBBPA exposures were calculated using occurrence data from the UK 2004 Total Diet Study (TDS) (Driffield et al. 2008) and consumption data from the Diet and Nutrition Survey of Infants and Young Children (DNSIYC) (DH 2013, Lennox et al. 2013) and the National Diet and Nutrition Survey rolling programme (NDNS) (Bates et al. 2014 & 2016, Roberts et al. 2018). The results from all 19 food sample groups analysed for TBBPA were below the LOD, which ranged from 0.02 to 0.2 ng/g w.w. for the different food groups. Corresponding human exposures are provided in Table 2, and are expressed as a range of lower bound (LB) and upper bound (UB). The LB was obtained by assigning a value of zero (minimum possible value) to all samples reported as < LOD. The UB was obtained by assigning the numerical value of LOD to values reported as < LOD, as recommended by WHO (2009). Thus, the use of LB and UB estimates are likely to underestimate and overestimate human exposures, respectively.

21. Mean and 97.5th percentile TBBPA chronic exposures for infants aged 4 to 12 months ranged from 0.0 - 3.6 and 0.0 - 13 ng/kg b.w./day, respectively (Table 2). For young children aged 12 to 18 months the mean and 97.5th percentile exposures ranged from 0.0 - 7.3 and 0.0 - 16 ng/kg b.w./day, respectively. Calculated mean and 97.5th percentile dietary exposures for young children aged 18 to 60 months ranged from 0.0 - 6.9 and 0.0 - 16 ng/kg b.w./day, respectively.
Table 2: Estimated chronic TBBPA exposures from the TDS infants and young children aged 4 to 60 months (ng/kg b.w./day)

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Mean (LB, UB)</th>
<th>97.5th percentile (LB, UB)</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 to &lt;6 month-olds (n = 116)</td>
<td>0.0 - 3.2</td>
<td>0.0 - 3.1</td>
</tr>
<tr>
<td>6 to &lt;9 month-olds (n = 606)</td>
<td>0.0 - 12</td>
<td>0.0 - 12</td>
</tr>
<tr>
<td>9 to &lt;12 month-olds (n = 686)</td>
<td>0.0 - 3.6</td>
<td>0.0 - 13</td>
</tr>
<tr>
<td>12 to &lt;15 month-olds (n = 670)</td>
<td>0.0 - 7.3</td>
<td>0.0 - 16</td>
</tr>
<tr>
<td>15 to &lt;18 month-olds (n = 605)</td>
<td>0.0 - 7.3</td>
<td>0.0 - 14</td>
</tr>
<tr>
<td>18 to 24 month-olds (n = 158)</td>
<td>0.0 - 6.9</td>
<td>0.0 - 16</td>
</tr>
<tr>
<td>24 to 60 month-olds (n = 978)</td>
<td>0.0 - 5.2</td>
<td>0.0 - 12</td>
</tr>
</tbody>
</table>

Values rounded to 2 significant figures (SF)
**Risk characterisation**

22. MOEs for TBBPA in Table 3 were calculated by dividing the critical point of departure (a BMDL$_{10}$ of 16 mg/kg b.w./day derived from a one-generation reproduction study in Wistar rats reported by van der Ven *et al.* 2008) by the estimated UK chronic exposures in Table 2.

23. For infants aged 4 to 12 months, the smallest MOE was $\geq 1.2 \times 10^6$ which occurred at the 97.5th percentile for 9 to <12 month-olds. For children aged 12 to 18 months, the smallest MOE was $1.0 \times 10^6$, which occurred at the 97.5th percentile for 12 to <15 month-olds. For young children aged 18 to 60 months, the smallest MOE was $1.0 \times 10^6$, which occurred at the 97.5th percentile for 18 to 24 month-olds.

24. Usually an MOE of 100 is sufficient to cover uncertainties and variability with respect to kinetic and dynamic differences between animal species and humans (factor $4 \times 2.5 = 10$) and within the human population (factor $3.2 \times 3.2 = 10$), and to conclude that there is no health concern. In the case of TBBPA, the Panel noted that an additional factor would be needed to cover deficiencies in the database.

**The RISK21 Matrix**

25. The RISK21 matrix been developed under a program of the Health and Environmental Sciences Institute as a highly visual method of assessing risk using exposure and hazard information and uses the acceptable MOE to derive the visualisation onto which the risk calculation is plotted. Figure 1 shows minimum (LB) and maximum (UB) potential chronic exposure for TBBPA in the diet of infants aged 0 to 12 months and children aged 1 to 5 years. TBBPA is situated in the lower left region of the matrix indicating a low risk of adverse health effects.
<table>
<thead>
<tr>
<th>Calculation of risk</th>
<th>4 to &lt;6 month-olds (n = 116)</th>
<th>6 to &lt;9 month-olds (n = 606)</th>
<th>9 to &lt;12 month-olds (n = 686)</th>
<th>12 to &lt;15 month-olds (n = 670)</th>
<th>15 to &lt;18 month-olds (n = 605)</th>
<th>18 to 24 month-olds (n = 118)</th>
<th>24 to 60 month-olds (n = 688)</th>
</tr>
</thead>
<tbody>
<tr>
<td>MOE</td>
<td>Mean (LB, UB) ≥ 5.0 x 10^6</td>
<td>97.5th percentile (LB, UB) ≥ 1.3 x 10^6</td>
<td>Mean (LB, UB) ≥ 5.2 x 10^6</td>
<td>97.5th percentile (LB, UB) ≥ 1.3 x 10^6</td>
<td>Mean (LB, UB) ≥ 4.4 x 10^6</td>
<td>97.5th percentile (LB, UB) ≥ 1.2 x 10^6</td>
<td>Mean (LB, UB) ≥ 2.2 x 10^6</td>
</tr>
<tr>
<td>% TDI</td>
<td>Mean (LB, UB) ≤ 3.2 x 10^{-4}</td>
<td>97.5th percentile (LB, UB) ≤ 1.2 x 10^{-3}</td>
<td>Mean (LB, UB) ≤ 3.1 x 10^{-4}</td>
<td>97.5th percentile (LB, UB) ≤ 1.2 x 10^{-3}</td>
<td>Mean (LB, UB) ≤ 3.6 x 10^{-4}</td>
<td>97.5th percentile (LB, UB) ≤ 1.3 x 10^{-3}</td>
<td>Mean (LB, UB) ≤ 7.3 x 10^{-4}</td>
</tr>
</tbody>
</table>

Values rounded to 2 significant figures (SF)
26. The Panel conducted a chronic risk characterisation for TBBPA as the parent compound. Based on the magnitude of calculated MOEs (paragraphs 28-30), it was considered unlikely that current dietary exposure to TBBPA, or combined exposure to TBBPA through ingestion of food and dust, raises a health concern for this age group. No data on the occurrence or toxicity of TBBPA derivatives were identified.

Breast-fed infants

27. The Panel noted that data on the levels of TBBPA in human milk are limited and the reported values differ widely. In the study from France (Cariou et al. 2008), higher concentrations were detected, probably due to a hydrolysis step included in the analytical procedure, resulting in hydrolysis of glucuronide and sulphate TBBPA conjugates that might be present in human milk. The Panel considered that hydrolysis of TBBPA conjugates might occur in the human gastrointestinal tract. Therefore the Panel used the result of this study to estimate the exposure of infants to TBBPA via breast-feeding.

28. Based on TBBPA occurrence levels in human milk reported by Cariou et al. 2008, the Panel estimated chronic infant TBBPA exposures to be <0.18 - 170 ng/kg b.w./day for average milk consumption and <0.28 - 260 ng/kg b.w./day for high milk consumption across three European countries (Paragraph 17, Table 1). Subsequently margins of exposure (MOEs) were calculated by dividing the point of departure (a BMDL\textsubscript{10} of 16 mg/kg b.w./day) by the estimated infant exposure to TBBPA. MOEs ranging from $5.3 \times 10^7$ - $9.3 \times 10^4$ and $4.0 \times 10^7$ - $6.2 \times 10^4$, respectively were calculated (Table 4). Based on the magnitude of these MOEs, the Panel concluded that current exposure to infants via consumption of breast milk does not raise a health concern.

Table 4: Calculated margins of exposure (MOE) for estimated exposures of breast-fed infants to TBBPA.

<table>
<thead>
<tr>
<th>European country</th>
<th>MOE for mean daily TBBPA exposures from breast milk</th>
<th>MOE for range of daily TBBPA exposures from breast milk</th>
<th>Reference (occurrence data)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean milk consumption</td>
<td>High milk consumption</td>
<td>Mean milk consumption</td>
</tr>
<tr>
<td>UK</td>
<td>$5.3 \times 10^7$</td>
<td>$4.0 \times 10^7$</td>
<td>$&gt; 5.3 \times 10^6$</td>
</tr>
<tr>
<td>Norway</td>
<td>$5.3 \times 10^7$</td>
<td>$3.2 \times 10^7$</td>
<td>Not given</td>
</tr>
<tr>
<td>France</td>
<td>$8.5 \times 10^5$</td>
<td>$5.7 \times 10^5$</td>
<td>$5.3 \times 10^7$ - $9.3 \times 10^4$</td>
</tr>
</tbody>
</table>

Values rounded to 2 significant figures (SF)
High consumers of cow’s milk (toddlers)

29. The ‘upper bound’ exposure for toddlers through the consumption of cow’s milk was estimated by the Panel to be 55.7 ng TBBPA/kg b.w./day (paragraph 18). Using a BMDL\textsubscript{10} of 16 mg/kg b.w./day identified as the point of departure for the risk characterisation, the resulting MOE is $3 \times 10^5$. Given the magnitude of this MOE, the Panel concluded that despite the uncertainties in the reported occurrence data (i.e. all results reported < LOQ, the limited number of food groups analysed, occurrence data from four European countries, and uncertainty regarding whether TBBPA is present in food as a free or conjugated form), it is unlikely that current dietary exposure to TBBPA raises a health concern for toddlers.

Non-dietary exposures (children)

30. The Panel concluded that the ‘typical’ dust exposure scenario (using a typical dust ingestion rate of 50 mg/child/day and the 95th percentile TBBPA concentration of 460 ng/g dust in UK classrooms of schools and day care centres reported by Harrad \textit{et al.} 2010) provided the most realistic estimate of children’s exposure to TBBPA from ingestion of dust. Using the TBBPA intake resulting from the typical dust exposure scenario (1.2 ng/kg b.w./day) and a BMDL\textsubscript{10} of 16 mg/kg b.w./day identified as the relevant point of departure, an MOE of $1.3 \times 10^7$ was calculated. The Panel concluded that this large MOE indicates children’s exposure to TBBPA from dust does not raise a health concern.

**Conclusions**

31. All estimates of the MOE for chronic dietary TBBPA exposure (based on UK consumption data) exceed the lowest MOE values calculated by EFSA for infants and toddlers concerning exposure through ingestion of breast milk and cow’s milk, respectively. The UK MOE values appear to be adequately protective, and indicate minimal risk from estimated chronic dietary exposures.

**Questions on which the views of the Committee are sought**

32. Members are invited to consider the following questions:

i). Taking into account the potential carcinogenicity of TBBPA, do Members consider the calculated MOEs for UK chronic human exposure to TBBPA to be adequately protective for public health?

ii). Do Members recommend using EFSA’s MOE approach or the COT TDI for future risk assessments?

**Secretariat**
References


NTP (2014). Toxicology studies of tetrabromobisphenol A (CAS No. 79-94-7) in F344/NTac rats and B6C3F1/N mice and toxicology and carcinogenesis studies of tetrabromobisphenol A in wistar han [Crl:WI(Han)] rats and B6C3F1/N mice (gavage studies). Available at: https://ntp.niehs.nih.gov/ntp/htdocs/lt_rpts/tr587_508.pdf


