TOX/2020/33

COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

Overarching discussion paper on consumption of plant-based drinks in children aged 6 months to 5 years of age.

Introduction

1. The Department of Health and Social Care (DHSC), Public Health England (PHE) and the FSA are receiving an increasing number of enquiries regarding the use of plant-based drinks in the diets of infants and young children. Therefore, we are asking the COT to consider the potential adverse health effects of soya, almond and oat drinks consumed in the diets of these age groups.

2. Currently the UK government advises that parents should only use infant formula as an alternative to breast milk in the first 12 months of a baby's life. It is also advised that whole cows' milk and unsweetened calcium-fortified milk alternatives, such as soya, almond and oat drinks can be given to children from the age of 1 as a part of a healthy, balanced diet (NHS, 2018)

3. Soya drinks are a popular alternative to dairy products and their use is becoming more widespread. Soya products contain phytoestrogens (also known as isoflavones) which have been shown to produce some reproductive and developmental changes in animal studies.

4. Almond drinks have a lower nutritional value than soya or oat drinks, however they are recommended as an alternative in cases where children refuse soya and oat drinks. The mycotoxin, aflatoxin B1 was identified as a common chemical contaminant in almonds which could be potentially transferred to almond drinks. Aflatoxin B1 is genotoxic and carcinogenic so its maximum levels set by the EU are established using the "as low as reasonably achievable (ALARA)" principle. Moreover, almonds contain cyanogenic glycosides which, once macerated, may interact with the enzyme ß-glucosidase. This enzyme hydrolyses the cyanogenic glycosides and can yield hydrogen cyanide, benzaldehyde, glucose and ketone. The quantity of cyanogenic glycosides present in almond drinks is uncertain, but low levels of cyanide have been detected on analysis. Exposure to large amounts of the hydrogen cyanide component can lead to convulsions, loss of consciousness, dizziness, weakness, mental confusion and heart failure.

5. Similarly, oat drinks are proposed as an alternative to soya milk for children following plant based or dairy- free diets. Oats can be contaminated with the mycotoxins T-2 and HT-2, DON and OTA. EFSA considered the safety of T-2 and HT-2 in 2017, where health-based guidance values were established for emetic effects following acute exposure, and for immune- and hepatotoxicity effects for chronic exposure. For OTA, EFSA (2020) established a MOE approach for neoplastic and non neoplastic effects in the kidney, while a group TDI was established for the sum of DON, 3-Ac-DON, 15-Ac-DON and DON-3-glucoside based on reduced body weight gain in animals (EFSA, 2017b).

6. Soya, almond and oat drinks were previously considered by the COT as separate papers TOX/2019/71 for soya, TOX /2020/16 for almonds and TOX/2020/03 for oats. The main challenge in the assessment of the safety of these drinks is the lack of information regarding dietary intakes for children following dairy-free or plant-based diets. At the suggestion of the Scientific Advisory on Nutrition (SACN) Secretariat, the exposures have been revisited, using information from several sources including the British Nutrition Foundation (2019), the First Steps Nutrition Trust Eating Well: vegan infants and under 5s (2020), Vegan Society Food tips for vegan children (2017) and Public Health England's (PHE) published Example menus for Early Years Settings in England (EYS) (2017). The above sources provided guidance for frequency and portion sizes for children under 5 and, considering the lack of consumption information for the groups of interest they were deemed the most representative scenarios for the diet of children following dairy-free or plant-based diets.

7. The Committee are asked to consider the information presented and address the questions posed at the end of each Annex.

Annexes to this paper

- **Annex A: Follow up discussion paper on the potential risks from soya drink consumption in children aged 6 months to 5 years of age.**

  - **Annex A1: Detailed assumptions for the consumption of soya-based products by infants and children aged 6 months to 5 years.**

- **Annex B: Follow up discussion paper on the potential risks from almond drink consumption in children aged 6 months to 5 years of age.**

- **Annex C: Follow up discussion paper on the potential risks from the consumption of oat drinks for children aged 6 months to 5 years of age**

Secretariat

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Annex A to TOX/2020/33

COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

Discussion paper on soya drink consumption in children aged 6 months to 5 years of age.

Background

1. Soya drinks are a popular alternative to dairy products and their use is becoming more widespread. In 2018, 95.6 million litres of soya drinks were sold in the UK amounting to sales of £135.6 million pounds. They are commonly consumed by all sectors of the population including those wishing to avoid dairy products and by individuals with an intolerance to lactose or another component of milk or who follow a plant-based diet.

2. Soya products contain phytoestrogens (also known as isoflavones) which have been shown to produce some reproduction and developmental changes in animal studies, although human epidemiological studies have not reported comparable effects[1,2]. A paper on soya drinks was considered at the December 2019 meeting (TOX/2019/71). In the current paper, the exposure calculations have been revised based on a number of resources that offer recommendations for the diet of children following a plant- based diet. These recommendations aim to achieve a well-balanced, nutritious diet and they were used to provide an indication of more realistic exposures to isoflavones from the diet.

3. In the lay summary statement from 2013, the Committee on Toxicity concluded that:

"Evidence from the few relevant epidemiological studies does not suggest important impacts of soya-based formula on later reproductive health in humans, although some studies have raised the possibility of subtle effects of uncertain clinical significance. However, animal studies where exposure to isoflavones was at levels similar to those reported in infants exclusively fed soya-based infant formula indicate some developmental and reproductive changes. There is thus some uncertainty about the safety of soya-based formula.

There is no scientific basis for a change in the current government advice that there is no substantive medical need for, nor health benefit arising from, the use of soya-

[1] Report available at: https://cot.food.gov.uk/sites/default/files/cot/phytoreport0503.pdf
[2] Statement available at https://cot.food.gov.uk/sites/default/files/cot/cotstaphytos.pdf

based infant formula and it should only be used in exceptional circumstances to ensure adequate nutrition."

4. The levels of phytoestrogens in soya-based infant formula have been found to range from 18 - 46.7 mg/L while the levels in soya drinks have been found to be around 100 mg/L (BDA, 2017). Since the Department of Health and Social Care (DHSC), Public Health England (PHE) and the FSA are receiving an increasing number of enquiries regarding the use of plant-based drinks in the diets of infants and young children, the COT considered the potential adverse health effects of soya drinks consumed in the diets of children aged 6 months to 5 years given that the levels of phytoestrogens in these drinks are greater than those found in soya-based infant formula. To aid comparison with the diet more generally, the Secretariat have also carried out an exposure assessment for the contribution other soya-based products such as soya alternatives to other dairy products or meat to the diet.

5. Currently the UK government advises that parents should only use infant formula as an alternative to breast milk in the first 12 months of a baby's life as the main milk drink. It is also advised that whole cows' milk and unsweetened calcium-fortified milk alternatives, such as soya, almond and oat drinks can be given to children from the age of 1 as a part of a healthy, balanced diet. Soya formula should only be introduced to the diet from the age of 6 months if it has been recommended or prescribed by a health visitor or GP.

Toxicity

6. As noted above, soya products contain phytoestrogens which have been shown to produce developmental and reproductive changes in animal studies, although similar effects have not been reliably observed in human epidemiological studies.

7. The COT has considered the safety of phytoestrogens previously (2003, 2013). In 2013, the Committee concluded that there was no scientific basis for a change in the current government advice that there is no substantive medical need for, nor health benefit arising from, the use of soya-based infant formula and it should be used only in exceptional circumstances in this age group, to ensure adequate nutrition. It was also concluded that, based on the evidence of some developmental and reproductive effects observed in animal studies along with evidence from human studies raising the possibility of subtle effects of uncertain clinical significance, there was some uncertainty about the safety of soya-based formula.

8. More recently a discussion paper was presented to the Committee (TOX/2019/71) presenting new information available since the 2013 evaluation. This included new human and animal studies. In summary, the new animal studies did not add significantly to the overall database. A variety of effects were reported in human studies including a significantly higher degree of methylation in vaginal epithelial

cells compared to those receiving cows' milk formula (Harlid et al, 2017), higher vaginal-cell Maturation Index, slower decrease in uterine volume, girls' estradiol trajectories and larger decrease for boys' breast-bud diameter (Adgent et al, 2018), a possible increase in height, BMI/age, height/age and weight/age of the girls, associated with variations in fat-free mass (Duitama et al, 2018) and earlier adjusted median age at the onset of puberty as well as age at pubarche (Segovia-Siapco et al, 2018), some with unknown long term implications (Harlid et al, 2017).

9. COT concluded that it was not possible to determine, on the basis of the available data, whether sensitivity to phytoestrogens varied among different age groups.

Exposure to phytoestrogens

Occurrence in foods

10. Table 1 below contains information on the isoflavone content of a variety of soya-based foods (data adapted from Kuhnle et al, 2009). The isoflavone content of soya-based foods and beverages is highly variable and these figures are a guide only.

Food product	Concentration of phytoestrogens (µg/100g)
Soya milk, unsweetened	6028
Burger, soya based	4430
Sausage, TVP/Soya	3994
Tofu, microwaved	10619
Yogurt, soya	8286
Mince, savoury, soya TVP based (high Soya content)	28758
Soya ice cream	13494
Soya mince granules, cooked	20850
Cheddar-like soya cheese[3]	6700

Table 1: Data on the soya phytoestrogen content of a range of foods (adapted from data from Kuhnle et al, 2009)

Exposure

11. Potential exposures to isoflavones have been calculated using the isoflavone levels provided above. An initial estimate was performed using food consumption estimates taken from the UK Dietary and Nutrition Survey of Infants and Young Children (DNSIYC) (DH, 2013) for children up to 18 months of age and those

[3] Data on soya-based cheese taken from the Linus Pauling Institute, 2004 as no equivalent UK data were available.

between 18 months and 5 years from the National Diet and Nutrition Survey (NDNS) (Bates et al, 2014; 2016; Roberts et al, 2018).

12. Due to the limited information on the consumption of the foods of interest by these age groups, the chronic exposure estimates for isoflavones in children between 6 months and 5 years of age were calculated assuming that a) milk, yogurt and cheese are replaced with soya-based alternatives; b) all dairy is replaced with soya alternatives and c) meat is replaced with soya alternatives plus isoflavone contribution from tofu, vegetables and bread; these were the scenarios discussed in TOX/2019/71. However, it was considered that these approaches might not have been representative of the actual exposures to isoflavones in the diets of those wishing to avoid dairy products, individuals with an intolerance to lactose or another component of milk or following a plant-based diet.

13. To refine the exposures, several sources of information were considered including the British Nutrition Foundation (2019), the First Steps Nutrition Trust Eating Well: vegan infants and under 5s (2020), Vegan Society Food tips for vegan children (2017) and Public Health England's (PHE) published Example menus for Early Years Settings in England (EYS) (2017). The above sources provided guidance for frequency and portion sizes for children under 5 and, considering the lack of consumption information for the groups of interest they were deemed the most representative scenarios for the diet of children following dairy-free or plant-based diets. The latter group is important when considering aggregate exposures to isoflavones from soya in the diet as their diets may be likely to consist of multiple sources of soya-based products in order to meet the nutritional requirement usually provided by meat and dairy.

14. In summary, the assumptions for each age group are presented in Table 2. These are representative of a worst-case scenario and would make allowance for consumption of commodities such as soya cheese, for which the available information for incorporation in the diet is limited. Based on the available information tofu and soya meat alternatives (including burgers and sausages) are used interchangeably to replace meat derived protein in children following a plant-based diet. As a worst-case scenario, the commodity with the highest concentration of isoflavones (soya mince) was selected for calculations. More information on the assumptions used can be found in Annex A1.

Table 2: Summary assumptions for intake of soya products for children aged 6 months to 5 years.

Age (months)	Commodity	Intake	Reference
6-≥12	Soya-based formula 18-46.7 mg/L	400mL/d	PHE- EYS (2017), Vegan Society (2017), First steps Nutrition Trust (2020)
6-≥12	Soya Milk	200mL/d	PHE- EYS (2017), Vegan Society (2017), First steps

			Nutrition Trust (2020)
6-≥12	Meat alternatives	20g/d	PHE-EYS (2017), First steps Nutrition Trust (2020)
6-≥12	Soya Yoghurt	50g/d	First steps Nutrition Trust (2018,2020)
12-≥24	Soya Milk	300-500mL/d	PHE- EYS (2017), Vegan Society (2016), First steps Nutrition Trust- Eating Well (2020)
12-≥24	Meat alternatives	60g/d	British Nutrition Foundation (2019), PHE-EYS (2017) First steps Nutrition Trust: Good food choices and portion sizes for 1-4year olds (2018)
12-≥24	Soya Yoghurt	50g	First Steps Nutrition Trust- Eating well (2020)
24≥48	Soya Milk	300-500mL/d	PHE- EYS (2017), Vegan Society (2017), First steps Nutrition Trust- Eating Well (2020)
12-≥24	Meat alternatives	75g/d	British Nutrition Foundation (2019), PHE-EYS (2017) First steps Nutrition Trust: Good food choices and portion sizes for 1-4year olds (2018)
12-≥24	Soya Yoghurt	50g/d	First Steps Nutrition Trust- Eating well (2020)
48≥60	Soya Milk	300-500mL	PHE- EYS (2017), Vegan Society (2017), First steps

			Nutrition Trust-Eating Well (2020)
48≥60	Meat alternatives	135g/d	British Nutrition Foundation (2019), PHE-EYS (2017) First steps Nutrition Trust: Good food choices and portion sizes for 1-4year olds (2018)
48≥60	Soya Yoghurt	50g/d	First Steps Nutrition Trust-Eating well (2020)

15. Table 4 below presents the exposures to isoflavones based on the scenario presented above, the mean bodyweights in Table 3 (DH, 2013; Bates et al, 2014; 2016; Roberts et al, 2018) and using the concentrations reported in Table 1.

Table 3: Average bodyweights

Age (months)	Mean bw in kg
6-<12	9.2
12-≤18	10.89
18-<24	12.1
24-<48	15.2
48-<60	18.4

Table 4: Exposure to isoflavones from soya products for children aged 6 months to 5 years.

Age (months)	Commodity	Exposure (µg/kg bw/d)
6-<12	Soya Based Formula	780 - 2000
6-<12	Soya Milk	1300
6-<12	Meat alternatives	625
6-<12	Soya Yoghurt	450

12-≤18	Soya Milk	1700 - 2800
12-≤18	Meat alternatives	1600
12-≤18	Soya Yoghurt	380
18-<24	Soya Milk	1500 - 2500
18-<24	Meat alternatives	1400
18-<24	Soya Yoghurt	342
24<48	Soya Milk	1200 – 2000
24<48	Meat alternatives	1400
24<48	Soya Yoghurt	270
48<60	Soya Milk	980 – 1600
48<60	Meat alternatives	2100
48<60	Soya Yoghurt	225

*Rounded to 2 SF

16. These exposures are indicative of the average diet of a child following a plant-based diet based on the dietary recommendations from the multiple sources described. However, lacking accurate consumption information, it is likely that these exposures would vary in practice, for example if a child consumes soya-based ice-cream as dessert on occasion.

17. Furthermore, isoflavones are present in other food commodities. These were considered to account for aggregate isoflavone exposure from the diet. Table 5 presents isoflavone exposures from background diet, based on food consumption estimates taken from the UK Dietary and Nutrition Survey of Infants and Young Children (DNSIYC) (DH, 2013) for children up to 18 months of age and those between 18 months and 5 years from the National Diet and Nutrition Survey (NDNS) (Bates et al, 2014; 2016; Roberts et al, 2018). Overall, the background diet does not significantly contribute to isoflavone intakes, considering exposure from soya products alone.

Table 5: Overall isoflavone exposure from background diet (µg/kg bw per day)

Age (months)	mean	maximum	97.5th percentile
6-<12	17	1400	67
12-≤18	26	600	73
18-<24	24	93	52
24-<48	26	480	66
48-<60	29	57	430

*Rounded to 2SF

18. For reference, the exposures to isoflavones from soya-based infant formula for infants aged 0-6 months are given in Table 6. This is based on information available from the Vegan Society[4] that: "If breastfeeding is not an option, infant formula is recommended. Soya-based infant formula can be fed to vegan infants when breastfeeding is not an option, but please speak to your health visitor or doctor before using it."

Table 6: Estimated total isoflavone exposure (mg/kg bw/day) of infants fed exclusively soya-based infant formula.

Isoflavone level in mg aglycone equivalents/L	Age in months (consumption)	Age in months (consumption)	Age in months (consumption)	Age in months (consumption)
	0-4 (800mL)*	0-4 (1200 mL)*	>4-6 (800 mL)*	>4-6 (1200mL)*
Soya-based formula 18-46.7 mg/L	2.4-6.3	3.7-9.5	1.8-4.8	2.8-7.2

Risk characterisation and conclusions

19. There is a large body of literature which demonstrates the adverse effects of soya phytoestrogens on reproductive endpoints in animal studies at levels varying from 1.6-500 mg/kg bw/day (COT, 2003), although many of these studies used administration via the intravenous or subcutaneous route. It is generally accepted that soya-rich diets in animal models have a detrimental effect on reproductive systems which may be wholly or partly irreversible depending on the timing of exposure. It is also widely recognised that the levels of phytoestrogens found to cause these reproductive effects are similar to those consumed in a soya-rich western diet. What is not currently understood is the relative susceptibility of animals and humans to these effects. Many of the available studies consider total phytoestrogen consumption, therefore the pattern of any adverse effects produced may be affected by the composition of phytoestrogens in different foods.

20. Furthermore, based on the available information it was not possible to determine whether sensitivity to phytoestrogens varied among the age groups of concern; it was similarly not possible to determine whether the level of concern differed between age groups.

21. The exposure assessment has been revised as per the request of SACN Secretariat to consider smaller groups within the wider age range of 6 months to 5

[4] https://www.vegansociety.com/resources/nutrition-and-health/life-stages/under-fives

years of age, for consistency with previous work. As previously mentioned, it was proposed that to revise the exposures using sources of information that provide nutritional guidance to high consumers of these foods (i.e. children following a plant-based diet) would be more suitable to use.

22. Previously, in TOX/2019/71 the exposures were calculated for the age groups of 6-18 months and 18 months- 5 years of age. As the latter groups do not align with the current evaluations direct comparisons cannot be made. The estimated exposures for milk (excluding formula) calculated previously ranged from 1296.7 to 7351.8 µg/kg bw/d and from 1095.3 to 6313.5 µg/kg bw/d for 6-18 months and 18 months to 5 years respectively assuming cow's milk was replaced with soya alternatives. For yoghurt the exposures ranged from 287 to 2387.6 µg/kg bw/d (6-18 months) and from 210.6 to 1368.2 µg/kg bw/d (18 months – 5 years) assuming dairy yoghurt was replaced with soya alternatives. In that paper, an estimation was given for cheese, again assuming dairy cheese was replaced with soya alternatives. The estimated exposures were 39.7-303 µg/kg bw/d (6-18 months) and 42.1 - 316.9 µg/kg bw/d (18 months to 5 years).

23. Although in the current evaluation it was not possible to perform an individual exposure assessment for this foodstuff, due to the limited nutritional advice available for soya-based cheese, the assumptions outlined in Annex A1 allow for isoflavone exposure from this commodity. In TOX/2019/71 an estimated exposure from all dairy was also presented, assuming these foods were replaced with soya alternatives. The exposures ranged from 1196.3 to 7454.2 µg/kg bw/d (6-18 months) and 1178.6 to 6313.5 µg/kg bw/d for 18 months to 5 years of age. Finally, the contribution to isoflavone exposure from meat alternatives (also taking into account tofu, sweet potato and bread) was calculated previously. The exposures ranged from 135-1791 µg/kg bw/d for 6-18 months and from 1-10 µg/kg bw/d for 18 months to 5 years.

24. Generally, for all scenarios presented above the respective refined estimates fall within the previously calculated range of exposures except for meat alternatives. The contribution to isoflavones exposure in the diet using the dietary recommendations for children following a plant-based diet are significantly higher than those calculated using animal derived meat as a proxy for soya-based alternatives for the age groups of 18-24 months, 24-48 months and 48-60 months. Based on the new estimates, exposures from these foods will range from 1600 µg/kg bw/d (12-18 months) to 2100 µg/kg bw/d (48-60 months) as opposed to the previously estimated 1-10 µg/kg bw/d for 18 months to 5 years. Despite the most conservative approach being used in the calculations presented in the current paper (the commodity with the highest concentration of isoflavones was used for exposure estimates) exposure is still significantly higher than previously presented, even allowing some variation from foodstuffs with lower isoflavone concentrations being consumed instead.

25. Overall, the background diet does not significantly contribute to isoflavone intakes, considering exposure from soya products alone. Exposure estimates for isoflavone exposure from soya-based infant formula for infants aged 0-6 months were also presented and it can be concluded that soya formula contributes significantly to isoflavone exposure in these age groups. This is important to

consider, given that advice for vegan infants is that “soya-based infant formula can be fed to vegan infants when breastfeeding is not an option, but please speak to your health visitor or doctor before using it[4]”

26. It is these uncertainties which prompted the COT to conclude that soya-based infant formula should not be generally consumed unless under exceptional circumstances. Based on the information previously presented (TOX/2019/71) the COT confirmed that current advice for children aged from 0-12 months, that soy formula should be used only in exceptional circumstances still stands and there are also potential concerns for children up to 5 years of age consuming soy drinks.

Questions for the Committee:

Members are asked to review the evidence available and consider the following questions:

i) Do the Committee think that intakes of phytoestrogens from consumption of soya drinks may be of concern in children aged 6 months to 5 years of age?
ii) Do the Committee have concerns over other soya-based products in the diets of children aged 6 months to 5 years of age based on the information provided?
iii) Do the Committee consider it necessary to issue additional advice aimed at consumers?
iv) Do the Committee have any other comments?

Annex A1

Detailed assumptions for the consumption of soya-based products by infants and children aged 6 months to 5 years.

1. Despite the lack of consumption information for the commodities of interest for these age groups, multiple sources are available that offer recommendations for the diet of children following a plant- based diet. These recommendations aim to achieve a well-balanced, nutritious diet and they were used to provide an indication of more realistic exposures to isoflavones from the diet.

2. The approach used to establish a consumption scenario was to review the available information and select the most conservative recommendations as a representation of a worst-case scenario.

3. For **milk** the recommendations are uniform across the sources of information. For infants aged **6 to 12 months**. In this age group it is recommended that the main drink should be breastmilk. However, if not possible infant formula should be the main milk drink. For children following a plant-based diet, information from the Vegan society suggests that :Soya-based infant formula can be fed to vegan infants when breastfeeding is not an option, but please speak to your health visitor or doctor before using it." It is recommended that children in this age group should be given at least 4 milk feeds of infant formula (100mL portions) in addition to milk being used in cooking. Unsweetened calcium-fortified soya can be used in cooking for infants from 6 months of age (First Steps Nutrition Trust-Eating well, 2020). An allowance of 2 extra portions of 100 mL of unsweetened soya milk was made based on information from the Eating well: vegan infants and under 5s where sample recipes include unsweetened soya milk used with cereal from the age of 7 months and information from the Vegan society for milk to be mixed in mashed foods (Vegan Society, 2017). Apart from use in cereals these would also account for use as a drink and consumption of soya-based cheese. **Above the age of 1 year**, unsweetened soya milk is suggested as a main milk drink (Vegan Society, 2017; PHE-EYS, 2017; First Steps Nutrition Trust,2018; 2020). Consistently across these sources the recommendation is for at least 300mL of milk per day. Unsweetened calcium-fortified soya milk is proposed as an effective alternative to cow's milk in terms of nutritional value. Similarly, to the younger age groups an allowance of an extra 200mL was made to account for soya milk used in cooking (including with cereals) and consumption of soya-based cheese (PHE, 2017; First Steps Nutrition Trust, 2018; 2020, Food Safety Authority of Ireland, 2020).

4. A daily portion of **yoghurt** was incorporated in the consumption assumptions as, across sources it forms part of the dietary recommendations/sample recipes from weaning onwards. It was also considered that while some children may not be having a yogurt every day, they may have some other kind of soya-based sweet

product/snack, this would allow for variations between children. A portion size of 50g was used (First Steps Nutrition Trust, 2020)

5. For **meat alternatives** soya-based alternatives (such as mince, burgers, sausages) and tofu are used interchangeably to replace animal meat in recipes. Since the concentration of isoflavones is higher in soya mince it was assumed that it would be the main source of protein as a worst-case scenario. For the age group of **6-12 months** an assumption of 20g/d was made for meat alternatives. This was based on information provided from several sources: Portion information was derived from recipes in the Eating well guide. This was up to 30g of soya mince per portion. (First Steps Nutrition Trust-Eating well, 2020). Furthermore, PHE's Example menus for early years settings in England (2017) also includes sample 5-day menus for children aged 1-4 years old, which have also been adapted to infants aged 7-12 months. Based on the information provided from the sample menus, soya or tofu meat alternatives were incorporated in the diet on average 3 days in the 5-day calendar. The resulting exposure assuming three, 30g portions over a 5-day week would be 18g/d, which was rounded to 20g/d to allow for higher consumers. For the **rest of the age groups**, the British Nutrition Foundation suggests 3 portions of protein foods for vegetarian/vegan children (British Nutrition Foundation, 2019). Similarly to above, an average portion size of :20g was assumed for children aged 12 to 24 month, 25g for children aged 24-48 months, 45g for children aged 48-60 months based on information from First step Nutrition's trust Good food choices and portion sizes for 1-4 year olds (2018) and the Eating well: vegan infants and under 5s guide (2020). The respective consumption based on 3 daily portions were: 60g/d for children aged 12 to 24 month, 75g/d for children aged 24-48 months and 135g/d for children aged 48-60 months.

6. With regards to the **background diet** food consumption estimates taken from the UK Dietary and Nutrition Survey of Infants and Young Children (DNSIYC) (DH, 2013) for children up to 18 months of age and those between 18 months and 5 years from the National Diet and Nutrition Survey (NDNS) (Bates et al, 2014; 2016; Roberts et al, 2018) were used and occurrence data from Kuhnle et al. (2009). The selection of the commodities of interest was based on a soya content of ≥5%. Where more than one types of food were available (e.g. bread) the commodity with the highest phytoestrogen content was used.

7. Table 7 summarises the commodities considered for the background diet calculations as well as the isoflavone concentrations reported for these.

Food	Phytoestrogen calculation (µg/g)
Soya flour	1247.27
Soya beans, cooked	175.56
Muesli	51.56
Brazil nuts	8.87
Bread	7.47

Dates, dried	5.99
Apricot, dried	4.43
Peanuts	4.27
Chickpeas, cooked	4.20
Figs, fresh	3.89
Prunes, raw	3.63
Pomegranate	3.04
Chestnut, cooked	2.83
Sweet Potato, cooked	2.51
Blackberry, cooked	2.21
Porridge oats (quick cook)	1.94
Dates, fresh	1.92
Cashew nuts	1.82
Walnuts	1.75
Runner Bean, cooked	1.56
Asparagus, cooked	1.54
Plum, cooked	1.52
Whole wheat flakes	1.38
Figs, dried	1.29
Carrots, raw and cooked	1.25
Almonds	1.12
Kiwi, raw	1.11
Blackcurrant, all	1.09
Prunes, cooked	1.08

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COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

Second discussion paper on the potential risks from almond drink consumption in children aged 6 months to 5 years of age.

Background

1. The Department of Health and Social Care (DHSC), Public Health England (PHE) and the FSA are receiving an increasing number of enquiries regarding the use of plant-based drinks in the diets of infants and young children. Therefore, we are asking the COT to consider the potential health effects of almond drinks in the diets of these age groups.

2. Currently the UK government advises that parents should only use infant formula as an alternative to breast milk in the first 12 months of a baby's life. It is also advised that whole cows' milk and unsweetened calcium-fortified milk alternatives, such as soya, almond and oat drinks can be given to children from the age of 1 as a part of a healthy, balanced diet (NHS, 2018)

3. A first discussion paper on the potential risks from almond drink consumption in children aged 6 months to 5 years of age was presented to the COT in March 2020.[5]

4. The Committee had agreed that more information on the likelihood of bitter almond contamination of almond drinks and what precautions were taken by manufacturers to prevent their entry to the supply chain should be investigated. Members had also agreed that aflatoxin exposure from almond drinks were a potential concern as there were uncertainties. Overall, it was agreed that further work needed to be done to refine the aflatoxin exposure assessment.

Occurrence data

5. Eleven drinks containing raw vegetables and fruit, flax seeds, whole apples with seeds, raw almond drink and pasteurised almond drink were analysed for total cyanide. Total cyanide levels of 9.6, 41,134 and 272 µg/L were detected in smoothies containing almond. The two smoothies with the highest levels also contained flaxseed which is also high in cyanide-containing molecules (Baker *et al*, 2018).

6. There is a lack of data for total cyanide in almond drinks alone. Therefore, occurrence data of total cyanide in sweet and bitter almonds were taken from

[5] https://cot.food.gov.uk/sites/default/files/tox202017almonddrinksseconddraft.pdf

EFSA's database which comprises of analytical samples from across the European Union (Table 1).

Table 1. Total cyanide concentration of almonds (µg/kg)*

Food commodity	Number of samples	Mean LB	Mean UB
Sweet almond	35	4500	4500
Bitter almond	3	1437000	1437000

LB: lower bound; UB: upper bound. EFSA 2019.
*Converted from mg/kg to µg/kg

7. There were no data for aflatoxin concentrations in almond drinks. However, some data exist for aflatoxin concentrations in almond nits. Forty nut samples (shelled almonds, pistachios, hazelnuts, peanuts and walnuts) taken from an Algerian market were tested for aflatoxigenic fungi and aflatoxin contamination. Out of the 40 nut samples, 8 were almond nut samples, all of which tested positive for aflatoxin B1 (AFB1) with levels ranging from 1.65 - 4.00 µg/kg, the mean was reported to be 2.12 µg/kg AFB1 (Riba *et al*, 2018). The sample size and the origin of the almonds is unlikely to be representative of AFB1 levels in almond nuts. Therefore, this concentration will not be considered further.

8. Twenty-one samples of Portuguese almond nuts were tested for aflatoxigenic fungi and aflatoxin contamination. Out of the 21 almond nut samples, AFB1 was detected in 1 sample at a concentration of 4.97 µg/kg. As AFB1 was only detected in one almond nut sample, this sample is unlikely to be representative of AFB1 levels in almond nuts. Therefore, this concentration will not be considered further (Rodrigues *et al*, 2012)

9. A total of 80 almond kernel samples (50 roasted and 30 raw almonds) taken randomly from supermarkets and small retail shops in Turkey were tested for aflatoxin contamination. Out of the 80 raw almond nut samples, AFB1 was detected in 12 samples ranging from 0.118 to 0.508 µg/kg. The sample size and the origin of the almonds is unlikely to be representative of AFB1 levels in almond nuts (Kanek *et al*, 2019).

10. EU Maximum levels for aflatoxins have been set for almonds that are used for direct human consumption or for use as an ingredient in food. The levels are currently set at 8 µg/kg/AFB1 (almonds ready to eat) and 12 µg/kg/AFB1 (almonds for further processing) (Official Journal of the European Union, 2006).

Cyanogenic glycosides

11. Cyanogenic glycosides in bitter apricot kernels were previously reviewed by COT in 2006 where a nominal acute reference dose of 5 µg/kg was established. More recently, cyanogenic glycosides in raw apricot kernels. were reviewed by EFSA (EFSA, 2016). An acute reference dose (ARfD) of 20 µg/kg body weight was established for cyanide. In the most recent EFSA opinion (EFSA, 2019),

the panel concluded that the ARfD of 20 µg/kg body weight is applicable for acute effects of cyanide regardless the dietary source.

12. Once the almond is macerated the cyanogenic glycosides interact with ß-glucosidase. This enzyme hydrolyses the cyanogenic glycosides and can yield hydrogen cyanide, benzaldehyde, glucose and ketones (Haque, 2002). The quantity of intact cyanogenic glycoside present in almond drinks is unknown.

13. Exposure to large amounts of cyanide can lead to convulsions, loss of consciousness, dizziness, weakness, mental confusion and heart failure (Burns *et al*, 2012). The Committee had previously agreed that if only sweet almonds were used in the preparation of almond drinks, then there would be no health concern. However, Members had agreed that more information on the likelihood of bitter almond contamination of almond drinks and what precautions were taken by manufacturers to prevent their entry to the supply chain should be investigated.

14. Bitter almonds are not used in almond drinks because of the flavour profile. Most manufacturers do not want a flavour profile which differs significantly from dairy– the use of bitter almonds would impart an excessively almond-flavoured product which would more resemble marzipan than milk. It should be noted that the almonds produced in Californian commercial orchards are only sweet varieties; there are no bitter varieties grown in California which is a major supplier of almonds to Europe[6]. Additionally, information provided by an almond drink manufacturer has also confirmed that only sweet almonds are used in their drinks.[7]

Exposure assessment for cyanogenic glycosides

15. The sweet almond mean upper bound and lower bound concentration of 4500 µg/kg (EFSA, 2019) was used to estimate the amount of cyanide that could be present in 1 litre of almond drink assuming 6% (w/w) almond nuts in the drink. The value of 270 µg/kg was calculated. A similar value has not been calculated for bitter almonds as almond drink is highly unlikely to be made from this type of almond. The value of 6% has been assumed because information publicly available for almond drinks produced by the manufacturer Rude Health ranges from 1% to 6% almond content. Rude Health produce a variety of almond drink products, some of which contain only spring water and almonds, whilst others contain other ingredients such as oil and rice milk, hence the differing percentage almond content between products[8]. Whilst the manufacturer has provided an approximate value of around 90% dilution of almond pulp with water via correspondence with the FSA[9], it was considered more realistic to use the publicly available information on the packaging of these products as there has been no clarification of the ingredients in the

[6] Personal communication. Almond Board of California

[7] Personal communication from almond drink company.

[8] Rude Health (2020) Available at: https://rudehealth.com/foods-and-drinks-london-based-brand/

[9] Personal communication from Rude Health

remaining 10%. Thus, it has been assumed that this may contain other ingredients as described for Rude Health almond drink products above.

16. No almond drink consumers in infants between the ages of 6 to 18 months were reported in the DNSIYC survey. A small number of almond drink consumers between the ages of 18 months to 5 years (n=4) were reported in the NDNS survey. The sweet almond mean LB and UB cyanide exposures estimated for these consumers (n=4) of almond drink ranged from 0.08 – 0.12 µg/kg bw/day. These estimates are uncertain as they are based on only 4 consumers of almond drink. As mentioned previously, due to the limited information on the consumption of almond drinks by these age groups, the chronic exposure estimates for cyanide exposure in children between 6 months and 5 years of age were originally calculated assuming that all milk was replaced with almond-based alternatives. To refine these exposures several sources of information were considered; British Nutrition Foundation (2019), the First Steps Nutrition Trust Eating Well: vegan infants and under 5s (2020), Vegan Society Food tips for vegan children (2017) and Public Health England's (PHE) published Example menus for Early Years Settings in England (EYS) (2017). The above sources provided guidance for frequency and portion sizes for children under 5 and considering the lack of consumption information for the groups of interest they were deemed the most representative scenarios for the diet of children following dairy-free or plant-based diets. It must be noted that the recommendations from these sources were for soya-alternatives to milk; these have been used in the absence of representative consumption information for almond drinks. However, it should be noted that soya-milk has been recommended due to its similar nutritional value and monetary cost to cow's milk, so it is likely that children following dairy-free of plant-based diets may not be consuming as much almond drink as others might consume soya drinks. Hence, these are conservative assumptions but considered a more relevant proxy than cow's milk. Additionally, it should be further noted that children under 1 year are not recommended to consume dairy alternatives as their main milk drink, the assumption made here for the 6 – 12 month age group was that up to 200 ml of almond milk may be used in cooking infant foods.

17. Table 2 gives the acute exposure estimates for total cyanide in children between the ages of 6 months and 5 years, with the assumption that all soya milk would be replaced with almond drink in a diet. Potential acute cyanide exposures were calculated using the estimated total cyanide concentration of 4500 µg/kg (sweet almond) combined with the consumption assumptions described in paragraph 16.

Table 2: The acute exposure estimates of the total cyanide in children between the ages of 6 months and 5 years, with an assumption that all soya milk would be replaced with almond drink containing 270 µg/kg CN in the diet (µg/kg bw/day*)

Age group (months)	Sweet almond 270 µg/kg CN	Sweet almond 270 µg/kg CN
6 ≤12	5.9[10]	--
12 ≤18	7.4	12
18 < 24	6.7	11
24 <48	5.3	8.9
48 <60	4.4	7.3

*rounded to 2s.f

Aflatoxin B1 (AFB1) in almonds

18. Aflatoxins are produced as a result of fungal contamination with *Aspergillus flavus* and *A. parasiticus* moulds under warm and humid conditions in tree nuts such as almonds. The degree of contamination is dependent on temperature, humidity, soil and storage conditions.

19. Aflatoxins have been previously reviewed by the Scientific Committee for Food (SCF) in 1996, the European Food Safety Authority (EFSA) in 2007 and the Joint FAO/WHO Expert Committee on Food Additives (JECFA) in 1998, 2001 (AFM1) and 2018.

20. In the most recent EFSA opinion (EFSA, 2020) the panel considered the chronic endpoint of liver carcinogenicity in a rat performed in a study by Wogan *et al*, 1974 to be the most sensitive and adequate study for dose response modelling.

21. Groups of male Fisher rats were administered diets containing 0, 1, 5, 15, 50, or 100 µg/kg diet of AFB1 (purity >95%) until clinical deterioration of animals was observed, at which time all survivors in that treatment group were killed. EFSA converted the dietary concentrations of AFB1 into daily intakes assuming that an average adult male rat consumed 40 g diet per kg body weight per day. EFSA also adjusted the daily intake to 104 weeks in order to compensate for the shorter study duration in some of the AFB1 groups. In the modelling of the results from the Wogan et al. (1974) study the highest dose was omitted because this dose resulted in a 100% tumour incidence. Using model averaging, the $BMDL_{10}$ was 0.4 µg/kg bw per day.

[10] One value presented based on a consumption of 200 mL

22. AFB1 was identified as a common chemical component in almonds which could be potentially transferred to almond drinks.

23. AFB1 is considered to be genotoxic and carcinogenic so its maximum permitted levels set by the EU are set at a level which is based on the "as low as reasonably achievable" (ALARA) principle. The AFB1 maximum levels set by the EU are 8 µg/kg for ready to eat almonds and 12 µg/kg for almonds that require further processing (Official Journal of the European Union, 2006). There are few data on the distribution of levels below the maximum level. The Rapid Alert for Food and Feed (RASFF Portal)[11] is a tool that provides information on public health warnings issues by food safety authorities and food companies. It also provides the latest information on food recall notices. Between December 2019 to June 2020, there were two reported incidences whereby levels of AFB1 were detected in almond nuts (from EU member states) according to the RASFF Portal. A RASFF alert notification[12] was raised by the German authority for almonds that had originated from Spain, whereby an AFB1 level of 37.5 µg/kg was reported[13]. In the second incident, a RASFF information notification[14] was raised by the Dutch authority for almonds that had originated from the United States of America, whereby an AFB1 level of 2.9 µg/kg was reported.[15] However, the almond nut industry has programmes and procedures in place to ensure that almond nuts that exceed the set AFB1 EU maximum levels are rejected (Almond board of California, 2016).

24. The almond drink industry has also confirmed that their almond nut suppliers test for aflatoxin levels to ensure that levels comply with the set EU maximum AFB1 levels. The almond drink production starts off with ingredients in reception/storage and water. The whole almonds are shelled, blanched, lightly roasted or unroasted and ground to produce a creamy almond paste. The almond paste is blended in water and other ingredients and nutrients are added to create the almond drinks.[16]

Exposure assessment for aflatoxin

25. Previously an exposure assessment was performed using aflatoxin concentrations from the Riba *et al* paper along with cows' milk consumption as a proxy, i.e. assuming that cows' milk will be replaced with almond milk. The Committee had agreed that aflatoxin exposure from almond drinks were a potential

[11] European Commision (2020) – Food and Feed Safety Alerts. Available at: https://ec.europa.eu/food/safety/rasff_en

[12] RASFF Alert: are sent when a food or feed presenting a serious health risk is on the market when rapid action is required.

[13] EC RASFF Portal. Available at: https://webgate.ec.europa.eu/rasff-window/portal/?event=notificationDetail&NOTIF_REFERENCE=2020.1392

[14] RASFF information: are used when a risk has been identified about food or feed places on the market, but the other members do not have to take rapid action.

[15] EC RASFF Portal. Available at: https://webgate.ec.europa.eu/rasff-window/portal/?event=notificationDetail&NOTIF_REFERENCE=2020.1148

[16] Personal communication from an almond drink company

concern as there were uncertainties. Overall, it was agreed that further work needed to be done to refine the aflatoxin exposure assessment.

26. No relevant data on aflatoxin contamination in almond drink were identified. Although some analytical data are available (see paragraphs 7-9), these are from small sample sizes and are not sufficiently representative to use to assess AFB1 exposure from almonds. However, maximum levels for aflatoxins were set by the European Union (EU) for almonds that are ready to eat and that require further processing. The aflatoxin B1 maximum levels (MLs) set by the EU are 8 µg/kg for ready to eat almonds and 12 µg/kg for almonds that require further processing (Official Journal of the European Union, 2006). The maximum level of 8 µg/kg set for ready to eat almonds was used to estimate the amount of AFB1 that could be present in 1 litre of almond drink assuming 6% (w/w) almond nuts in the drink, as described in paragraph 15. The value of 0.48 µg/kg aflatoxin was calculated.

27. The maximum level of 12 µg/kg set for almonds that require further processing was used to estimate the amount of AFB1 that would be present in 1 litre of almond drink assuming 6% (w/w) almond nuts in the drink (Official Journal of the European Union, 2006). The value of 0.72 µg/kg aflatoxin was calculated.

28. As described in paragraph 16 there were very few or no almond milk consumers reported in the NDNS and DNSIYC surveys. Therefore, the exposure assessment for AFB1 also used soya milk recommendations as a proxy with the same assumptions and uncertainties described for cyanogenic glycosides. Table 3 gives the chronic exposure estimates for AFB1 in children between the ages of 6 months to 60 months, with the assumption that soya milk has been replaced with almond drink in the diet of a plant-based child or those that avoid dairy. Potential chronic AFB1 exposures were calculated using the estimated concentrations of 0.48 and 0.72 µg/kg combined with soya milk consumption data for children aged 6 – 60 months, respectively.

Table 3: Estimated chronic AFB1 exposure of children aged 6 months to 60 months based on an assumption that all soya milk would be replaced with almond drink in diet (excluding infant formula) (using estimated aflatoxin concentrations in almond drink). (µg/kg bw/day)*

Age group (months)	0.48 µg/kg AFB1 – almonds ready to eat exposure range	0.48 µg/kg AFB1 – almonds ready to eat exposure range	0.72 µg/kg AFB1 – almonds for further processing exposure range	0.72 µg/kg AFB1 – almonds for further processing exposure range
6 < 12	0.010	---	0.016	---

12 ≤ 18	0.013	0.022	0.020	0.033
18 < 24	0.012	0.020	0.018	0.030
24 < 48	0.0095	0.016	0.014	0.024
48 < 60	0.0078	0.013	0.012	0.020

*rounded to 2 SF

29. There are very few data available on contaminants in almond drinks or consumption. The exposure assessment for aflatoxin is based on a worst-case scenario as it is assumed that almond drink will be consumed in the same way as soya milk since there are very few almond drink consumers.

30. The fate of aflatoxins during the processing of contaminated almonds was reported by (Zivoli et al, 2014). Four kg of shelled almonds purchased from a local market in Italy were inoculated with aflatoxin B1 and B2 strains. The blanching and peeling of almonds did not reduce aflatoxin levels. Peeled contaminated almond nuts were roasted for 30 – 120 minutes at 120°C and 150°C, respectively. Roasting and peeling almonds reduced aflatoxin levels by up to 84% as seen in table 4. Based on this data it appears that the roasting of almond nuts during the manufacturing process can further reduce aflatoxin levels.

Table 4. Effect of roasting on AFB1 in contaminated almonds

	Level (µg/kg)	Mean reduction (%)
Initial level	5558.7 ±4091.3	
120°C, 30 min	5258.6 ±2708.9	5
120°C, 60 min	2295.4 ±1376.6	58
120°C, 120 min	2571.4 ±458.4	54
150°C, 30 min	2500.4 ±974.8	55
150°C, 60 min	1465.8 ±1006.1	74
150°C, 120 min	907.1 ±611.9	84

Background diet

31. In the most recent EFSA opinion on aflatoxins (EFSA, 2020) the panel calculated the chronic dietary exposures for aflatoxins from a total diet as seen in Table 5. These exposure values give a good estimation on levels of AFB1 exposure that could come from a background diet. However, these age groups do not align fully with those used in the exposure assessments performed for this paper and, the consumption and occurrence data are from multiple European sources including the UK. Furthermore, the EFSA exposure assessment included dairy products so therefore did not consider plant-based children or those avoiding dairy for various reasons, it may also not include almond drinks. Therefore, we would expect these

figures to be lower for the background diet of children following a plant-based or dairy-free diet in the UK.

Table 5: EFSA mean and high chronic exposure to AFB1 (µg/kg bw per day)*

Age group		Minimum	Median	Maximum
Infants (<12 months old)	LB	0.000080	0.00018	0.00060
	UB	0.00058	0.0020	0.0049
Toddlers (≥12 months to < 36 months	LB	0.00043	0.00064	0.0011
	UB	0.0032	0.0054	0.0070
Other children (≥36 months to < 10 years old)	LB	0.00047	0.00076	0.0018
	UB	0.0035	0.0050	0.0061

AFB1: aflatoxin B1; bw:body weight; LB: lower bound;
*Converted from ng/kg/bw/day to µg/kg bw per day and rounded to 2s.f.

Risk characterisation

Cyanogenic glycosides

Acute reference dose (ARfD)

32. In the most recent EFSA opinion (EFSA, 2019) the panel concluded that the ARfD of 20 µg/kg body weight was applicable for the acute effects of cyanide regardless the dietary source. No chronic health-based guidance has been established for cyanide.

33. The estimated acute exposures in table 2 (µg/kg bw/day) were used to calculate the health risks as percentages of the ARfD (20 µg/kg body weight) These percentages are shown below in Table 6. All estimated exposures are below the ARfD.

Table 6: Calculation of acute risk following total cyanide exposure of children aged 6 months to 5 years based on an assumption that all soya milk would be replaced with almond drink in diet (excluding infant formula) (using estimated mean LB and UB concentration from the sweet almond). (% ARfD)*

Age group (months)	Sweet almond (270 µg/kg HCN)	Sweet almond (270 µg/kg HCN)
6 < 12	29[17]	--
12 ≤18	37	62
18 < 24	33	56
24 < 48	27	44
48 < 60	22	37

*rounded to 2 SF

Aflatoxin

Margin of Exposure (MOE)

34. The EFSA Scientific Committee stated that an MOE of 10,000 or higher, if based on the $BMDL_{10}$ from a carcinogenicity study, would be of low concern from a public health point of view (EFSA, 2005).

35. A margin of exposure (MOE) calculation can be carried out, as below:
MOE = ($BMDL_{10}$ (µg per kg per day) / Exposure value (µg/kg bw/day))

36. Estimated AFB1 MOEs for infants and young children aged 6 months to 5 years were calculated using a $BMDL_{10}$ of 0.4 µg/kg bw per day (EFSA, 2020) and the estimated aflatoxin chronic exposures (Table 7). Based on the assumptions used these are below the MOE of 10,000.

[17] One value presented based on a consumption of 200 mL

Table 7: Estimated MOEs following AFB1 exposure of children aged 6 months to 5 years based on an assumption that all soya milk would be replaced with almond drink in diet (excluding infant formula) (using estimated mean and maximal AFB1 concentrations). *

Age group (months)	0.48 µg/kg AFB1 – almonds ready to eat exposure range	0.48 µg/kg AFB1 – almonds ready to eat exposure range	0.72 µg/kg AFB1 – almonds for further processing exposure range	0.72 µg/kg AFB1 – almonds for further processing exposure range
6 < 12	40[18]	--	25[7]	--
12 ≤ 18	31	18	20	12
18 < 24	33	20	22	13
24 <48	29	25	28	17
48 < 60	33	31	33	20

*rounded to 2SF

Conclusions

37. There are very few data available on contaminants in almond drinks or consumption.

38. The risk assessment for cyanide is based on a worst-case exposure scenario as it is used soya milk recommendations as a proxy that almond drink would be consumed in the same way. Although the use of bitter almonds in almond milk drinks cannot be completely ruled out, based on industry information, it seems highly unlikely that these will be used in the manufacture of these products.

39. Estimates of acute exposure to cyanide assuming sweet almonds were used to make the almond drink do not exceed the ARfd for infants and young children aged 6 months to 60 months.

40. The risk for cyanide is based on a worse-case scenario for sweet almonds used in almond drinks. Although, in practice, bitter almonds would not be deliberately used in almond drinks as they would be unpalatable and impart a strong ‘marzipan’ flavour to the drink. However, contamination with bitter kernels or the presence of

[18] One value presented based on a consumption of 200 mL

almond kernels or the presence of almond kernels could occur but data on the level of cross-contamination with bitter almonds are not available.

41. As mentioned in paragraph 30 the almond production includes either the roasting or non-roasting of whole almonds. Although the roasting of almond nuts could further reduce AFB1 levels as demonstrated in the Zivoli *et al*, 2014, no data on whether roasted or unroasted almonds are used has been provided by industry to confirm if AFB1 levels are lower in almond drinks. Furthermore, as mentioned in paragraph 15, information publicly available for almond drinks produced by Rude Health ranges from 1% to 6% almond content, which indicates that AFB1 levels may be lower depending on the almond percentage in the almond drink.

42. The risk assessment for AFB1 assumes a high level of AFB1 as the EU maximum levels were used (Official Journal of the European Union, 2006). It should be noted that the exposure assessment uses a conservative approach as it uses AFB1 MLs (almond nuts) and the nutritional recommendations that are made for soya milk. AFB1 is considered to be a genotoxic carcinogen so the MLs set by the EU are set at a level that is "as low as reasonable possible", so it is likely that the actual levels will vary and may be potentially lower than the set MLs. Some analytical data were available (see paragraphs 7-9) and while these were not representative enough to use in the exposure assessment they indicated that where AFB1 was detected, the reported levels were in the range 0.118 to 4.97 µg/kg. The published analytical data does not show samples exceeding the MRL. The RASSF data shows that the MRL can be exceeded on occasion. Actual data on AFB1 levels from almond drinks are not available and it is unclear how processing during manufacturing might further reduce AFB1 levels in almond drinks. It is also unclear how much of an overestimate will occur from assuming all almonds are at the MRL but in the absence of data on the actual distribution of AFB1 levels in almonds it provides an upper value to the potential exposure. The potential for some drinks to contain almonds with higher AFB1 levels than the MRL is expected to be low given the monitoring by growers.

43. The calculated MOEs are significantly lower than 10,000 (Table 6), which have the potential to be a health concern. It has also been assumed that almond milk would be the primary source of milk in the diet of children following diary free or plant-based diets. However, it must be considered that as soya-milk has been recommended as a good alternative due to its similar nutritional and monetary value to cow's milk, so it is likely that children following dairy-free of plant-based diets may not be consuming as much almond drink as others might consume soya drinks, because of its lower nutritional value and higher cost (First Steps Nutrition Trust, 2020). The Scientific Committee of the Food Safety Authority of Ireland (2020) stated that the use of almond milk in children aged 1 to 5 years old in Ireland is not recommended, as its nutritionally inadequate. As such, the consumption should be lower than assumed and should occur in combination with other, more nutritious sources, such as soya milk. It is thus likely that potential exposures to AFB1 from the diet would be much lower than one assuming all drinks are almond milk at the MRL and the MOE, whilst still of concern, higher than estimated, this exposure is likely to be of limited duration and the risk is likely to be low.

44. Since the exposure assessments for both cyanide and AFB1 are based on estimates of levels of consumption and occurrence of contaminants, further data would be required to refine these estimates in a meaningful way.

Questions to the Committee

i) Do the Committee consider that, based on the available data, intakes of aflatoxins from the consumption of almond drink are of concern to the health of children ages 6 months to 5 years of age?

ii) Do the Committee consider that, based on the available data, intakes of cyanide from the consumption of almond are of concern to the health of children ages 6 months to 5 years of age if sweet almonds are used in almond drinks?

iii) Taking into account the potential contaminants, Do the Committee consider the current government advice that these drinks can be introduced from 1 year of age to be appropriate?

Secretariat

June 2020

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Annex C to TOX/2020/33

COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT

Follow up discussion paper on the potential risks from the consumption of oat drinks for children aged 6 months to 5 years of age

Background

1. The Department of Health and Social Care (DHSC), Public Health England (PHE) and the FSA are receiving an increasing number of enquiries regarding the use of plant-based drinks in the diets of infants and young children. Therefore, we are asking the COT to consider the potential health effects of oat drinks in the diets of these age groups.

2. A first discussion paper on oat drinks was presented to the COT in January 2020[1]. It was considered that the analytical data concerning the concentration of HT-2 and T-2 derived from one sample of oat drink was unlikely to be representative for the UK given the lack of surveillance data and variability of contamination levels in the UK oat harvest. Furthermore, it was noted that there was no indication that mycotoxin concentrations in UK oats would be substantially different to those reported in other European countries by EFSA. The Committee agreed that follow-up work should 1) assess the contribution of mycotoxin exposure from oat drinks in relation to exposure from total oats in the general diet, and 2) estimate the amount of oat drink one would need to consume relative to the rest of the diet to approach the HBGV.

3. The COT has previously assessed HT-2 and T-2 in in the diet of infants aged 0 to 12 months and children aged 1 to 5 years (COT, 2018). The outcomes of this assessment are described in further detail in paragraph 24. In addition to HT-2 and T-2[19], other mycotoxins have been detected in oats and have appeared on the Rapid Alert System for Food and Feed (RASFF) portal, for example:

- Deoxynivalenol (DON) (948 µg/kg) in gluten-free oats from the UK[20]
- DON (1461 µg/kg) in gluten-free oat flakes from the UK[21]

[19] https://webgate.ec.europa.eu/rasff-window/portal/?event=notificationDetail&NOTIF_REFERENCE=2017.0737

[20] https://webgate.ec.europa.eu/rasff-window/portal/?event=notificationDetail&NOTIF_REFERENCE=2018.3508

[21] https://webgate.ec.europa.eu/rasff-window/portal/?event=notificationDetail&NOTIF_REFERENCE=2018.3506

- Ochratoxin A (OTA) (69 µg/kg) in organic oats from the Czech Republic[22]

4. Subsequently, the scientific literature was assessed to determine the potential for these mycotoxins to appear in oat drinks and the potential exposures that could occur.

5. A number of brands of oat drink are sold in the UK, for example Alpro and Plenish. Because of differences in their production processes, there are slight differences in the oat content of these drinks (Table 1). The mean average oat content across the brands of oat drink listed in Table 1 is 108 grams of oats per litre of oat drink.

Table 1: Oat content of the brands of oat drink sold in the UK.

Brand of oat drink	**Oat content (g/ 100 mL)***	**Country of origin of oats**
Alpro	9.8	Unspecified (Europe)
Oatley	10	Sweden
Rude Health	11	Unspecified
Plenish	11**	Unspecified
Provamel	12**	Unspecified (Europe)

* Data taken from https://www.olivemagazine.com/guides/best-oat-milk-taste-test/
** Organic oats

Toxicology

HT-2 and T-2

Acute Reference Dose

6. In 2017, the EFSA Panel on Contaminants in the Food Chain (CONTAM) established an acute reference dose (ARfD) of 0.3 µg for T-2 and HT-2/kg bw, based on acute emetic events in mink (EFSA, 2017a; Wu *et al*., 2016). Using a $BMDL_{10}$ of 2.97 µg/kg bw for T-2 and HT-2 based on their emetic effects, and the application of an uncertainty factor of 10 for intraspecies differences, an ARfD of 0.3 (rounded from 0.297) µg T-2 and HT-2/kg bw was established. An interspecies uncertainty factor was not included because humans were not considered to be more sensitive to this endpoint than mink.

Tolerable Daily Intake

7. In addition, the CONTAM Panel established a tolerable daily intake (TDI) for T-2 and HT-2 of 0.02 µg/kg body weight (bw) per day based on a new 90-day

[22] https://webgate.ec.europa.eu/rasff-window/portal/?event=notificationDetail&NOTIF_REFERENCE=2018.2393

subchronic toxicity study in rats that confirmed that immune- and haematotoxicity are the critical effects of T-2 (EFSA, 2017a; Rahman *et al*., 2014). The Panel used decreases in leukocytes counts as the critical endpoint to derive a $BMDL_{10}$ of 3.3 µg T-2/kg bw. Based on rapid metabolism of T-2 to HT-2 and structural similarities, this value was used as a reference point for establishing a TDI for both T-2 and HT-2. An uncertainty factor of 200 was used (x 10 for interspecies differences, x 10 for intraspecies variation and x 2 since it was a subchronic study).

OTA

8. OTA is genotoxic both *in vitro* and *in vivo*, though the mechanisms of genotoxicity are unclear (EFSA, 2020). Direct and indirect genotoxic and non-genotoxic modes of action might each contribute to tumour formation. Since recent studies have raised uncertainty regarding the mode of action for kidney carcinogenicity, EFSA (2020) concluded that it was inappropriate to establish a health-based guidance value (HBGV) and agreed to apply a margin of exposure (MOE) approach.

9. For the characterisation of non-neoplastic effects, EFSA (2020) used a BMDL10 of 4.73 µg/kg bw/day (calculated from kidney lesions observed in pigs). For characterisation of neoplastic effects, EFSA (2020) used a BMDL10 of 14.5 µg/kg bw/day (calculated from kidney tumours seen in rats). In this paper, both of these BMDLs are used, separately, to calculate margins of exposure (MOEs) to assess the health risk of exposure to OTA.

10. For neoplastic effects, an MOE of ≥10,000 would indicate low concern. This MOE was derived following EFSA guidance for substances that are both genotoxic and carcinogenic. The Opinion states that “In the interpretation of the MOE for the neoplastic risks, the Panel considered that the MOE of 10,000 for substances that are genotoxic and carcinogenic could be particularly conservative in this case because the evidence for a direct interaction of OTA with the DNA is inconclusive.” For characterisation of chronic non-neoplastic effects, an MOE of ≥ 200 was considered as being of low health concern. This MOE was derived by applying a default uncertainty factor (UF) of 100 for intra- and interspecies toxicokinetic and toxicodynamic differences combined with an additional UF of 2 to account for extrapolation of a 3-month study in pigs to a chronic situation in that species (EFSA, 2020).

DON

11. A group-TDI for the sum of DON, 3-Ac-DON, 15-Ac-DON and DON-3-glucoside of 1 µg/kg bw/day has been used by EFSA (2017b). This group TDI is based on reduced bodyweight gain in mice. The CONTAM Panel identified vomiting as critical acute effect in humans. To assess acute human health risk, epidemiological data from mycotoxicoses were assessed and a group-ARfD of 8 µg/kg bw per eating occasion was calculated (EFSA, 2017b). In 2020, the health risks from exposure to DON in the diets of infants and young children aged 0-5 years

old, as part of the Addendum to the Overarching Statement on the potential risks from contaminants in the diet of infants aged 0 to 12 months and children aged 1 to 5 years (COT, 2020).

Estimation of T-2 and HT-2 concentration in oat drink

12. Oat drink consumed in the UK is made from European-harvested oats. Therefore, occurrence data of T-2 and HT-2 in oats were taken from EFSA's database which comprises of samples of unprocessed oats taken from across the European Union (Table 2).

Table 2: Concentrations of T-2 toxin, HT-2 toxin and the sum of the T-2 and HT-2 toxins in unprocessed oat grains.

Mycotoxin	**N**	**LC**	**LB/UB**	**Concentration (µg/kg)**			
				Mean	**P50**	**P75**	**P95**
T-2	1453	26%	LB	68	18	65	302
			UB	69	19	65	302
HT-2	1412	15%	LB	168	40	157	722
			UB	169	40	157	722
Sum of T-2 & HT-2	1422	26%	LB	234	58	224	981
			UB	236	60	225	981

N: number of samples; LC: left censored data (percentage of analytical results below the LOD or LOQ); LB: lower-bound; UB: upper-bound; P50: 50th percentile; P75: 75th percentile; P95: 95th percentile. Taken from EFSA 2011.

13. Schwake-Anduschus *et al.* (2010) demonstrated that T-2 and HT-2 toxins are mostly attached to the outer hull of oat grains, as the "de-hulling of oats led to a T-2/ HT-2 reduction of 98 % in the mean, where reduction varied between 93.8 % and 100 %". This substantial reduction in the concentration of T-2 and HT-2 by de-hulling oat grains is recognised by EFSA and the Agriculture and Horticulture Development Board (AHDB). For example, the "normal cleaning and dehulling during mill processing can reduce these levels by 80-95 %" (EFSA, 2011). Furthermore, on their website, the AHDB states that "there is good evidence that at least 90 % of mycotoxins are removed during dehulling"[2]. These results explain why studies in the UK have shown high levels of mycotoxins in oats at harvest but generally low concentrations in consumer products.

14. Using the upper bound mean concentration of 236 µg/ kg for the sum of T-2 and HT-2 toxins in unprocessed oats (Table 2) and a mean concentration reduction of 98 % after de-hulling as reported by Schwake-Anduschus *et al.* 2010:

236 µg/ kg oats x 0.02 = 4.72 µg T-2/HT-2 /kg oats [1]

Thus 4.72 µg of T-2 and HT-2 would be expected to remain in 1 kg of groats after cleaning and de-hulling.

To produce one litre of oat drink, 108 grams of processed oats are required (using data from Table 1, and 1 kg/L oat drink density). The sum of T-2 and HT-2 toxin expected to be present in 108 g of processed oats is:

(4.72 µg/ kg ÷ 1000 g) x 108 g = 0.51 µg [2]

Thus, 0.51 µg of T-2 and HT-2 is estimated to be present in 1 kg of oat drink. This mean concentration was used for a UK exposure assessment (see below).

Estimation of DON concentration in oat drink

15. Oat drink consumed in the UK is made from European-harvested oats. Therefore, occurrence data of DON in oats were taken from EFSA's database which comprises of samples of unprocessed oats taken from across the European Union. EFSA (2013) have reported the mean concentration of DON in oat grains intended for human consumption across European countries as being 209 µg/kg (n = 203).

16. Scudamore *et al.* (2007) demonstrated that DON is mostly attached to the outer hull of oat grains and quoted a mean reduction of 88.2% if DON in dehulled oats.

17. Using a concentration of 209 µg/ kg for DON in unprocessed oats and a mean concentration reduction of 88.2 % after de-hulling as reported by Scudamore *et al.* (2007):

209 µg/ kg oats - (0.882 x 209) = 24.66µg/ kg oats

Thus 24.66 µg of DON would be expected to remain in 1 kg of groats after cleaning and de-hulling.

To produce one litre of oat drink, 108 grams of processed oats are required (using data from Table 1, and 1 kg/L oat drink density). The amount of DON expected to be present in 108 g of processed oats is:

(24.66µg/ kg ÷ 1000 g) x 108 g = 2.66 µg

Thus, 2.66 µg of DON is estimated to be present in 1 kg of oat drink. This mean concentration was used for a UK exposure assessment (see paragraph 26)

Estimation of OTA concentration in oat drink

18. Oat drink consumed in the UK is made from European-harvested oats. Therefore, occurrence data of OTA in oats were taken from JECFA's database which comprises of samples of unprocessed oats taken from across the European Union. JECFA (2001) have reported maximum concentrations of OTA measured in processed oats intended for human consumption across European countries which ranges from 0.05 - 56.6 µg/kg. The value of 56.6 µg/kg has been used because it is considered more representative and was found in 'oat kernels' which have been 'de-hulled'. This was the maximum value for these 'oat kernels' and has been used to give an estimate of worst-case scenario in the exposure assessment.

19. To produce one litre of oat drink, 108 grams of processed oats are required (using data from Table 1, and 1 kg/L oat drink density). The amount of OTA expected to be present in 108 g of processed oats is:

(56.6µg/ kg ÷ 1000 g) x 108 g = 6.11µg

Thus, 6.11 µg of OTA is estimated to be present in 1 kg of oat drink. This mean concentration was used for a UK exposure assessment (see paragraph 32)

Exposure assessment

20. The "sum of T-2 and HT-2" was used for estimating exposure which is consistent with what has been done previously for other mycotoxins.

Exposure to HT-2 and T-2 from oat drinks in the UK

21. To estimate UK infant and young children exposures to T-2 and HT-2 from oat drink consumption, the estimated concentration of 0.51 µg T-2 and HT-2/ kg oat drink from occurrence data in European-harvested oats was used (see paragraph 10).

22. Consumption data from the DNSIYC survey (DH, 2013; Lennox *et al*., 2013; for infants aged 3 ≤ 18 months) and the NDNS survey (Bates *et al.,* 2014; Bates *et al*., 2016; Roberts *et al.,* 2018; for ages >18 months) were initially assessed for the exposure assessment for 6 to 60-month olds. However, a small number of individuals aged 6-18 months (n=4) and young children aged 18 - 60 months (n=6) were reported to consume oat drink in these surveys, so these data are unlikely to be representative for this age group in the UK. Therefore, in this chronic exposure assessment, consumption assumptions for soya drink are used as a proxy for oat drink (Table 3).

23. Due to the limited information on the consumption of the commodities of interest by these age groups, the chronic exposure estimates for HT-2 and T-2 in

children between 6 months and 5 years of age were calculated assuming that all cow's milk in the diet (including recipes) is replaced by soya milk. It was considered that this approach may not be representative of the actual exposures to HT-2 and T-2 in the diets of those wishing to avoid dairy products, individuals with an intolerance to lactose or another component of milk or following a plant-based diet.

24. To refine the exposures several sources of information were considered; the First Steps Nutrition Trust Eating Well: vegan infants and under 5s (2020), Vegan Society Food tips for vegan children (2017) and Public Health England's (PHE) published Example menus for Early Years Settings (EYS) (2017). The above sources provided guidance for frequency and portion sizes for children under 5 and considering the lack of consumption information for the groups of interest they were deemed the most representative scenarios for the diet of children following dairy-free or plant-based diets. It must be noted that the recommendations from these sources were for soya-alternatives to milk, these have been used in absence of representative consumption information for oat drinks. However, it must be considered that soya-milk has been recommended due to its similar nutritional and monetary value to cow's milk, so it is likely that children following dairy-free or plant-based diets may not be consuming as much oat drink as others might consume soya drinks. Hence, these are conservative assumptions but considered a more relevant proxy than cow's milk. Additionally, it should be noted that children under 1 year are not recommended to consume dairy alternatives as their main milk drink, the assumption made here for the 6 – 12 month age group was that up to 200 ml of oat drink may be used in cooking infant food. Soya drink consumption for those wishing to avoid dairy products, individuals with an intolerance to lactose or another component of milk or those following a plant-based diet is likely to be greater than for the general public, therefore leading to exposure estimates that may be overestimated when considering the general population.

Table 3: Estimated chronic exposure to the sum of HT-2 and T-2 from consumption of oat drink for 6 to 60-month olds in the UK (**ng/kg b.w./day**)**

Age group	**Minimum***	**Maximum***
6 to < 12 months	11	---
12 to ≤ 18 months	14	23
18 to < 24 months	13	21
24 to < 48 months	10	17
48 to < 60 months	8.3	13

* Uses soya drink consumption rates (minimum & maximum) from a review of recommendations derived from the First Steps Nutrition Trust, Vegan Society and Public Health England as a proxy for oat drink consumption. Values rounded to 2 s.f.

** Uses estimated concentration of 0.51 µg T-2 & HT-2/ kg oat drink. Does not consider exposure to HT-2 and T-2 from other food sources.

Amount of oat drink required to exceed HBGV for HT-2 and T-2

25. The amount of oat drink one would need to consume to approach the TDI was estimated (Table 4). This calculation does not take into account background dietary exposure to HT-2 and T-2. Although EFSA (2017) have reported background dietary exposure levels to the HT-2 and T-2 toxins, these data were not used because 1) their assessment is for the general diet (which includes dairy products, processed oats and possibly oat drinks) and thus does not consider children following a plant-based diet or avoid dairy, and 2) the age groups reported by EFSA are not aligned with the age groups reported in Table 3.

Table 4: Estimated quantity of oat drink required in **L** or **Kg*** to exceed the HBGV for HT-2 and T-2 mycotoxins for 6 to 60-month olds in the UK

Age group	**ARfD**	**TDI**
6 to < 12 months	5.4	0.36
12 to ≤ 18 months	6.4	0.43
18 to < 24 months	7.1	0.47
24 to < 48 months	8.9	0.60
48 to < 60 months	11	0.72

* Uses soya drink consumption rates (minimum & maximum) from a review of recommendations derived from the First Steps Nutrition Trust, Vegan Society and Public Health England as a proxy for oat drink consumption. Uses estimated concentration of 0.51 µg T-2 & HT-2/ kg oat drink. Does not consider exposure to HT-2 and T-2 from other food sources. Values rounded to 2 s.f.

Dietary exposure to HT-2 and T-2 from processed oats in the general diet in the UK

26. The Minutes from the January COT meeting stated a requirement for the FSA to 'assess the contribution of mycotoxin exposure from oat drinks in relation to exposure from total oats in the general diet'. Whilst exposure is assessed below in Table 5, it is noted that the HT-2 and T-2 toxins are not exclusively found in oats; these mycotoxins are also found in similar concentrations in other grains such as wheat and barley (EFSA, 2017). As such the background exposure from the total diet has been considered in the next section (paragraph 28) which takes into account grains such as wheat and barley.

27. For this exposure assessment for 6 to 60-month olds, consumption data from the DNSIYC survey (DH, 2013; Lennox *et al.,* 2013; for infants aged 3 <18 months) and the NDNS survey (Bates *et al.,* 2014; Bates *et al.,* 2016; Roberts *et al*., 2018; for ages >18 months) were used. An estimated concentration of 4.72 µg HT-2 & T-2/ kg processed oats was used (see paragraph 14).

Table 5: Estimated acute and chronic exposure to the sum of HT-2 and T-2 mycotoxins from consumption of processed oats in the general diet for 6 to 60-month olds in the UK.

Age group	**Number of consumers**	**Acute exposure (ng/kg bw/day)***		**Chronic exposure (ng/kg bw/day)***	
		Mean	**97.5th %ile**	**Mean**	**97.5th %ile**
6 to < 12 months	632	5.2	19	2.4	9.0
12 to ≤ 18 months	713	6.1	20	2.8	11
18 to < 24 months	89	5.4	17	2.4	9.9
24 to < 48 months	347	4.8	15	2.1	7.6
48 to < 60 months	151	4.1	11	1.9	5.8

* Recipes were used for this assessment with oat content > 5% and 'oat based alternatives to milk' were removed from the assessment. The assumption was made that all oats consumed were processed, and had a concentration of 4.72 µg HT-2 & T-2/ kg processed oats. Values rounded to 2 s.f.

Exposure to HT-2 and T-2 from oat drinks and processed oats in the general diet (combined) in the UK

Table 6: Estimates of chronic exposure to the sum of HT-2 and T-2 mycotoxins from consumption of oat drinks and processed oats in the general diet combined for 6 to 60-month olds in the UK **(ng/kg b.w./day)***

Age group	**Minimum**	**Maximum**
6 to < 12 months	24	--**
12 to ≤ 18 months	17	26
18 to < 24 months	15	23
24 to < 48 months	12	19
48 to < 60 months	10	15

*The mean chronic exposure to HT-2 and T-2 from processed oats in the general diet has been summed with the minimum and maximum exposure from oat drinks. The assumption was made that all oats consumed were processed, and had a concentration of 4.72 µg HT-2 & T-2/ kg processed oats. Uses estimated concentration of 0.51 µg T-2 & HT-2/ kg oat drink. Values rounded to 2 s.f.
** One value presented based on a consumption of 200 mL

Background dietary exposure to HT-2 and T-2 in the general diet

28. HT-2 and T-2 mycotoxins are not exclusively found in oats. For example, these mycotoxins also occur in bread, pasta, and breakfast cereals. EFSA (2017) have collated occurrence data from various foodstuffs across Europe and estimated total exposure to HT-2 and T-2 mycotoxins in the general diet (which includes dairy products, processed oats and possibly oat drinks). It was possible to distinguish the UK data from this EFSA dataset as it was presented in an annex (see table 7).These estimates may overestimate the background diet exposure of UK children following a plant-based diet, or those avoiding dairy because it considers dairy products which they are unlikely to consume and oats (possibly including oat drinks) which have been considered separately here.

Table 7: Summary statistics of the chronic dietary exposure to the sum of T-2 and HT-2 toxins in the general diet for young children in the UK(EFSA, 2017) **(ng/kg b.w. per day)***

		HT-2		**T-2**	
Age group**		**Mean**	**95th Percentile**	**Mean**	**95th Percentile**
Infants (<12 months old)	**LB**	11	33	4.3	12
	UB	34	79	24	54
Toddlers (≥12 months to < 18 months	**LB**	17	39	7.1	16
	UB	49	91	34	64
Toddlers (≥18 months to < 36 months	**LB**	14	34	6.8	14
	UB	47	87	34	62
Other children (≥36 months to < 10 years old)	**LB**	12	26	6.1	12
	UB	42	71	31	50

* Data taken from EFSA (2017). Values rounded to 2 s.f. Data has been lifted from Appendix G.3 for individual T2 and HT2 toxins so could not be summed
** Age groups do not align with those presented for 6 to 60-month olds in Tables 3-6.

Exposure to DON from oat drinks in the UK

29. To estimate UK infant and young children exposures to DON from oat drink consumption, the estimated concentration of 2.66 µg DON/ kg oat drink from occurrence data in European-harvested oats was used (see paragraph 15).

30. As described in paragraphs 22 – 24 for exposure to T-2 and HT-2 from oat drinks in the UK, there was no representative consumption data for oat drinks from the DNSIYC survey (DH, 2013; Lennox et al., 2013; for infants aged 3 ≤ 18 months) and the NDNS survey (Bates et al., 2014; Bates et al., 2016; Roberts et al., 2018; for ages >18 months). Therefore, in this chronic exposure assessment, consumption assumptions for soya drink are used as a proxy for oat drink (Table 8). The assumptions are described in paragraph 24.

Table 8**:** Estimated chronic exposure to DON from consumption of oat drink for 6 to 60-month olds in the UK **(ng/kg b.w./day)****

Age group	**Minimum***	**Maximum***
6 to < 12 months	58	---
12 to ≤ 18 months	73	120
18 to < 24 months	66	110
24 to < 48 months	53	88
48 to < 60 months	43	72

* Uses soya drink consumption rates (minimum & maximum) from a review of recommendations derived from the First Steps Nutrition Trust, Vegan Society and Public Health England as a proxy for oat drink consumption. Values rounded to 2 s.f.
** Uses estimated concentration of 2.66 µg DON/ kg oat drink. Does not consider exposure to DON from other food sources.

Amount of oat drink required to exceed HBGV for DON

31. The amount of oat drink one would need to consume to approach the TDI and ARfD was estimated (Table 9). This calculation does not take into account background dietary exposure to DON. Although EFSA (2017) have reported background dietary exposure levels to DON, these data were not used because 1) their assessment is for the general diet (which includes dairy products, processed oats and possibly oat drinks) and thus does not consider children following a plant-based diet or those avoiding dairy, and 2) the age groups reported by EFSA are not aligned with the age groups reported in Table 9.

Table 9: Estimated quantity of oat drink required to exceed the HBGV for DON for 6 to 60-month olds in the UK **(L)***

Age group	**ARfD**	**TDI**
6 to < 12 months	28	3.5
12 to ≤ 18 months	33	4.1
18 to < 24 months	36	4.5
24 to < 48 months	46	5.7
48 to < 60 months	55	6.9

* Uses soya drink consumption rates (minimum & maximum) from a review of recommendations derived from the First Steps Nutrition Trust, Vegan Society and Public Health England as a proxy for oat drink consumption. Uses estimated concentration of 2.66 µg DON/ kg oat drink. Does not consider exposure to DON from other food sources. Values rounded to 2 s.f.

Dietary exposure to DON from processed oats in the general diet in the UK

32. For this exposure assessment for 6 to 60-month olds, consumption data from the DNSIYC survey (DH, 2013; Lennox *et al.,* 2013; for infants aged 3 <18 months) and the NDNS survey (Bates *et al.,* 2014; Bates *et al.,* 2016; Roberts *et al*., 2018; for ages >18 months) were used. An estimated concentration of 24.66 µg DON/ kg processed oats was used (see paragraph 17).

Table 10: Estimated acute and chronic exposure to DON from consumption of processed oats in the general diet for 6 to 60-month olds in the UK.

Age group	**Number of consumers**	**Acute exposure (ng/kg bw/day)***		**Chronic exposure (ng/kg bw/day)***	
		Mean	**97.5th %ile**	**Mean**	**97.5th %ile**
6 to < 12 months	632	27	100	12	47
12 to ≤ 18 months	713	32	100	15	60
18 to < 24 months	89	28	87	12	52
24 to < 48 months	347	25	80	11	40
48 to < 60 months	151	21	56	10	30

* Recipes were used for this assessment with oat content > 5% and 'oat based alternatives to milk' were removed from the assessment. The assumption was made that all oats consumed were processed, and had a concentration of 24.66 µg DON/ kg processed oats. Values rounded to 2 s.f.

Exposure to DON from oat drinks and processed oats in the general diet (combined) in the UK

Table 11: Estimates of chronic exposure to DON from consumption of oat drinks and processed oats in the general diet combined for 6 to 60-month olds in the UK **(ng/kg b.w./day)***

Age group	**Minimum**	**Maximum**
6 to < 12 months	70	--**
12 to ≤ 18 months	88	140
18 to < 24 months	78	120
24 to < 48 months	64	99
48 to < 60 months	53	82

*The mean chronic exposure to DON from processed oats in the general diet has been summed with the minimum and maximum exposure from oat drinks. The assumption was made that all oats consumed were processed, and had a concentration of 24.66 µg DON/ kg processed oats. Uses estimated concentration of 2.66 µg DON/ kg oat drink. Values rounded to 2 s.f.
** One value presented based on a consumption of 200 mL

Background dietary exposure to DON in the general diet

33. DON is not exclusively found in oats. For example, these mycotoxins also occur in other grains. EFSA (2017) have collated occurrence data from various foodstuffs across Europe and estimated total acute exposure to DON in the general diet (which includes dairy products, processed oats and possibly oat drinks). It was possible to distinguish the UK data from this EFSA dataset as it was presented in the annex (see table 12). These estimates may overestimate background diet exposure of UK children following a plant-based diet, or those avoiding dairy because it considers dairy products which they are unlikely to consume and oats (possibly including oat drinks) which have been considered separately here.

Table 12: Summary statistics of the chronic dietary exposure to DON in the general diet for young children in the UK (EFSA, 2017) **(ng/kg b.w. per day)***

Age group**		**Mean dietary exposure**	**95th percentile diary exposure**
Infants (<12 months old)	**LB**	400	1000
	MB	700	1600
	UB	100	2200
Toddlers (≥12 months to < 18 months	**LB**	800	1500
	MB	1200	2100
	UB	1600	2700
Toddlers (≥18 months to < 36 months	**LB**	800	1500
	MB	1200	2000
	UB	1600	2700
Other children (≥36 months to < 10 years old)	**LB**	800	1300
	MB	1000	1700
	UB	1300	2200

* Data taken from EFSA (2017). Values rounded to 2 s.f. Lifted from Appendix F, table F.1
** Age groups do not align with those presented for 6 to 60-month olds in Tables 8-11

Exposure to OTA from oat drinks in the UK

34. To estimate UK infant and young children exposures to OTA from oat drink consumption, the estimated concentration of 6.11 µg OTA/ kg oat drink from occurrence data in European-harvested oats was used (see paragraph 19). This estimated concentration was derived from the maximum concentration value and has been used to give an estimate of worst-case scenario in the exposure assessment.

35. As described in paragraphs 22– 24 for exposure to T-2 and HT-2 from oat drinks in the UK, there was no representative consumption data for oat drinks from the DNSIYC survey (DH, 2013; Lennox *et al*., 2013; for infants aged 3 ≤ 18 months) and the NDNS survey (Bates *et al.,* 2014; Bates *et al*., 2016; Roberts *et al.,* 2018; for ages >18 months). Therefore, in this chronic exposure assessment, consumption assumptions for soya drink are used as a proxy for oat drink (Table 13). The assumptions are described in paragraph 24.

Table 13: Estimated chronic exposure to OTA from consumption of oat drink for 6 to 60-month olds in the UK **(ng/kg b.w./day)****

Age group	**Minimum***	**Maximum***
6 to < 12 months	130	---
12 to ≤ 18 months	170	280
18 to < 24 months	150	250
24 to < 48 months	120	200
48 to < 60 months	100	170

* Uses soya drink consumption rates (minimum & maximum) from a review of recommendations derived from the First Steps Nutrition Trust, Vegan Society and Public Health England as a proxy for oat drink consumption. Values rounded to 2 s.f.
** Uses estimated concentration of 6.11 µg OTA/ kg oat drink. Does not consider exposure to OTA from other food sources.

36. The 2020 EFSA risk assessment of OTA in food states that using the HBGV is not suitable and proposed an approach using the MOE approach (EFSA, 2020).Therefore calculations for the amount of oat drink required to exceed the HBGV could not be calculated for OTA.

Dietary exposure to OTA from processed oats in the general diet in the UK

37. For this exposure assessment for 6 to 60-month olds, consumption data from the DNSIYC survey (DH, 2013; Lennox *et al.,* 2013; for infants aged 3 <18 months) and the NDNS survey (Bates *et al.,* 2014; Bates *et al.,* 2016; Roberts *et al*., 2018; for ages >18 months) were used. An estimated concentration of 56.6 µg OTA/ kg processed oats was used (see paragraph 18).

Table 14: Estimated acute and chronic exposure to OTA from consumption of processed oats in the general diet for 6 to 60-month olds in the UK.

Age group	**Number of consumers**	**Acute exposure (ng/kg bw/day)***		**Chronic exposure (ng/kg bw/day)***	
		Mean	**97.5th %ile**	**Mean**	**97.5th %ile**
6 to < 12 months	632	62	230	28	110
12 to ≤ 18 months	713	73	240	34	140
18 to < 24 months	89	65	200	29	120
24 to < 48 months	347	58	180	25	91
48 to < 60 months	151	49	130	23	69

* Recipes were used for this assessment with oat content > 5% and 'oat based alternatives to milk' were removed from the assessment. The assumption was made that all oats consumed were processed, and had a concentration of 56.6 µg OTA/ kg processed oats. Values rounded to 2 s.f.

Exposure to OTA from oat drinks and processed oats in the general diet (combined) in the UK

Table 15: Estimates of chronic exposure to OTA from consumption of oat drinks and processed oats in the general diet combined for 6 to 60-month olds in the UK (**ng/kg b.w./day**)*

Age group	**Minimum**	**Maximum**
6 to < 12 months	160	--**
12 to ≤ 18 months	200	310
18 to < 24 months	180	280
24 to < 48 months	150	230
48 to < 60 months	120	190

*The mean chronic exposure to DON from processed oats in the general diet has been summed with the minimum and maximum exposure from oat drinks. The assumption was made that all oats consumed were processed, and had a concentration of 56.6 µg OTA/ kg processed oats. Uses estimated concentration of 6.11 µg OTA/ kg oat drink. Values rounded to 2 s.f.
** One value presented based on a consumption of 200 mL

Background dietary exposure to OTA in the general diet

38. OTA is not exclusively found in oats. For example, these mycotoxins also occur in other grains. EFSA (2020) have collated occurrence data from various foodstuffs across Europe and estimated total chronic exposure to OTA in the general diet (which includes dairy products, processed oats and possibly oat drinks). It was possible to distinguish the UK data from this EFSA dataset as it was presented in annex (see table 16 below). These estimates may overestimate background diet exposure of UK children following a plant-based diet, or those avoiding dairy because it considers dairy products which they are unlikely to consume.

Table 16: Summary statistics of the chronic dietary exposure to OTA in the general diet for young children in the UK (EFSA, 2020) (**ng/kg b.w. per day**)*

Age group**		**Mean dietary exposure**	**95th percentile diary exposure**
Infants (<12 months old)	**LB**	1.9	6.1
	UB	6.2	14
Toddlers (≥12 months to < 18 months	**LB**	4.5	10
	UB	11	20
Toddlers (≥18 months to < 36 months	**LB**	5.4	10
	UB	12	21
Other children (≥36 months to < 10 years old)	**LB**	4.8	9.3
	UB	10.8	19

* Data taken from EFSA (2020). Values rounded to 2 s.f.
** Age groups do not align with those presented for 6 to 60-month olds in Tables 13-15

Risk characterisation

T-2 and HT-2

39. As shown in Table 4, substantial quantities of oat drink would be required to exceed the ARfD for HT-2 and T-2 toxins in a single exposure event therefore the risk from acute consumption is very low.

40. The chronic health risk for 6 to 60-month olds was calculated as a percentage of the TDI, using the estimate of chronic exposure to HT-2 and T-2 toxins from consumption of oat drink (Table 17) and processed oats in the general diet (Table 18).

Table 17: Chronic health risk calculated from estimated exposure to the sum of HT-2 and T-2 from consumption of oat drink for 6 to 60-month olds in the UK (**% TDI**)*

Age group	**Minimum**	**Maximum**
6 to < 12 months	55	---
12 to ≤ 18 months	70	120
18 to < 24 months	65	110
24 to < 48 months	50	85
48 to < 60 months	42	65

* Uses soya drink consumption rates (minimum & maximum) derived from the First Steps Nutrition Trust, Vegan Society and Public Health England as a proxy for oat drink consumption. Uses estimated concentration of 0.51 µg T-2 & HT-2/ kg oat drink, and TDI of 0.02 µg T-2 & HT-2 /kg b.w. Values rounded to 2 s.f.

Table 18: Chronic health risk calculated from estimated chronic exposure to the sum of HT-2 and T-2 mycotoxins from consumption of processed oats in the UK general diet for 6 to 60-month olds in the UK **(%TDI)***

Age group	**Mean**
6 to < 12 months	12
12 to ≤ 18 months	14
18 to < 24 months	12
24 to < 48 months	11
48 to < 60 months	9.5

* Recipes were used for this assessment with oat content > 5% and 'oat based alternatives to milk' were removed from the assessment. The assumption was made that all oats consumed were processed, and had a concentration of 4.72 µg HT-2 & T-2/ kg processed oats. Uses the TDI of 0.02 µg T-2 & HT-2 /kg b.w. Values rounded to 2 s.f.

41. There are minor exceedances of the TDI for the 12 to ≤ 18 and 18 to < 24 month-old age groups. However, these estimates are considered conservative for children on a plant-based or dairy-free diet as consumption of oat drinks is likely to be lower for all age groups as, based on the existing advice from the Vegan society, First Steps Nutrition Trust and PHE, soya is the main recommended alternative to milk drink. For children who are not following plant-based or dairy free diets, cow's milk is the main recommended milk drink. Therefore, all age groups are likely to be consuming less oat drink than estimated here.

42. As shown in Table 18, estimated exposure to HT-2 and T-2 from processed oats in the general diet is only a small fraction of the TDI.

43. The chronic health risk for 6 to 60-month olds was also calculated using the estimate of chronic exposure to HT-2 and T-2 toxins from consumption of oat drink and processed oats in the general diet combined, and is presented as a percentage of the TDI (Table 19).

Table 19: Chronic health risk calculated from estimated exposure to the sum of HT-2 and T-2 mycotoxins from consumption of processed oats in the general diet and oat drinks combined for 6 to 60-month olds in the UK **(% TDI)***

Age group	**Minimum**	**Maximum**
6 to < 12 months	120	--**
12 to ≤ 18 months	85	130
18 to < 24 months	75	120
24 to < 48 months	60	95
48 to < 60 months	50	75

*The mean chronic exposure to processed oats has been summed with the minimum and maximum exposure to oat drinks. The assumption was made that all oats consumed were processed and had a concentration of 4.72 µg HT-2 & T-2/ kg processed oats. Uses estimated concentration of 0.51 µg T-2 & HT-2/ kg oat drink. Uses the TDI of 0.02 µg T-2 & HT-2 /kg b.w. Values rounded to 2 s.f.
** One value presented based on a consumption of 200 mL

44. The chronic health risk for young children was also calculated using the chronic exposure estimates for sum of HT-2 and T-2 in the background diet in the UK as reported by EFSA (2017). These exposure estimates are presented as percentages of the TDI in Table 20.

Table 20: Chronic health risk calculated from UK dietary exposures to the T-2 and HT-2 toxins in the general diet of young children (EFSA, 2017**) (% TDI)***

		HT-2		**T-2**	
Age group**		**Mean**	**95th Percentile**	**Mean**	**95th Percentile**
Infants (<12 months old)	**LB**	56	160	21	61
	UB	170	400	120	270
Toddlers (≥12 months to < 18 months	**LB**	84	190	35	78
	UB	240	450	170	320
Toddlers (≥18 months to < 36 months	**LB**	72	170	34	70

	UB	240	430	170	310
Other children (≥36 months to < 10 years old)	**LB**	59	130	31	59
	UB	210	360	150	250

* Data taken from EFSA (2017) to calculate chronic health risk. Uses the TDI of 0.02 µg T-2 & HT-2 /kg b.w. for the health risk calculation. Values rounded to 2 s.f. Data has been lifted from Appendix G.3 for individual T2 and HT2 toxins so could not be summed

** Age groups do not align with those presented for 6 to 60-month olds in Tables 3-6.

45. The data shown in Table 20 take into account exposure to HT-2 and T-2 from processed oats in the general diet in addition to other major dietary sources. In their exposure assessment, EFSA noted that foods within the 'Grains and Grain-based products' category had the highest levels of the sum of HT-2 and T-2 mycotoxins, specifically 'Grains for human consumption' and 'Breakfast cereals', and in particular oat-containing commodities within these groups (EFSA, 2017). However, it can be seen that a substantial amount of 'left-censored' data is included in their assessment, leading to some overestimation of the upper-bound estimates of exposure. In addition, these age groups do not align fully with those used in the exposure assessments performed for this paper and, the consumption and occurrence data are from multiple European sources including the UK. Furthermore, the EFSA exposure assessment included dairy products so therefore did not consider children on plant-based diets or those avoiding dairy for various reasons, and it may also include oat drinks. Therefore, we would expect these figures to be lower for the background diet of children following a plant-based or dairy-free diet in the UK.

46. The COT has previously assessed HT-2 and T-2 in in the diet of infants aged 0 to 12 months and children aged 1 to 5 years (COT, 2018). Acute and chronic exposures were calculated for the sum of T-2 and HT-2 using occurrence data from a retail survey of oat-based products commissioned by the FSA in 2015 and consumption data from NDNS and DNSIYC. Exposures in 0 to 4-month old infants are negligible as infants in this age range are unlikely to consume solid foods, including oat-based products. Mean and 97.5th percentile acute exposures ranged from 0.022 – 0.032 and 0.056 – 0.11 µg/kg bw, respectively. These were all below the ARfD of 0.3 µg/kg bw and are therefore not of toxicological concern. Mean and 97.5th percentile chronic exposures were calculated and ranged from 0.0099 – 0.014 and 0.029 – 0.063 µg/kg bw/day, respectively. All the mean exposures were below the TDI of 0.02 µg/kg bw and are therefore not of toxicological concern. The chronic 97.5th percentile exposures ranged from 145 – 315% of the EFSA TDI. Whilst an effect on health cannot be entirely excluded it is doubtful that children would be regularly exposed to these levels, which were measured in a year in which levels of

T-2/HT-2 in oat grains were particularly high, for a prolonged period. In most years, levels of T-2 and HT-2 will be much lower than those observed in this harvest. It is therefore unlikely that dietary exposure levels of T-2 and HT-2 would be of any toxicological concern in infants and young children.

OTA

47. The MOE values calculated from estimated exposure to OTA from consumption of oat drink for 6 to 60-month olds in the UK (shown in Table 22) are all below 10,000 for neoplastic effects based on a BMDL of 14.5 µg/kg. This would indicate a possible health concern in the case of genotoxicity. For non-neoplastic effects an MOE of ≥200 would indicate low concern. As seen in Table 22, the MOEs for non-neoplastic effects based on a BMDL10 of 4.73 µg/kg are below 200 indicating a potential health concern.

Table 21: Chronic health risk calculated from estimated exposure to OTA from chronic dietary exposure to the OTA in the general diet for young children in the UK (EFSA, 2020) (calculated as margins of exposure**)***

Age group	**MOE (using BMDL10 of 4.73 µg/kg bw/day)****	**MOE (using BMDL10 of 14.5 µg/kg bw/day)****
Infants (<12 months old)	760-2500	2300-7600
Toddlers (≥12 months to < 18 months	430-1100	1300-3200
Toddlers (≥18 months to < 36 months	390-880	1200-2700
Other children (≥36 months to < 10 years old)	440-990	1300-3000

* Derived from exposure data taken from EFSA (2020). Values rounded to 2 s.f.

** Age groups do not align with those presented for 6 to 60-month olds in Tables 13-15.

Table 22: Chronic health risk calculated from estimated exposure to OTA from consumption of oat drink for 6 to 60-month olds in the UK (calculated as margins of exposure)*****

Age group	**MOE (using BMDL10 of 4.73 µg/kg bw/day)****	**MOE (using BMDL10 of 14.5 µg/kg bw/day)****
6 to < 12 months	36 - ***	112 - ***
12 to ≤ 18 months	17-28	52-85
18 to < 24 months	19-32	58-97
24 to < 48 months	24-39	73-121
48 to < 60 months	28-47	87-145

* Uses soya drink consumption rates (minimum & maximum) from a review of recommendations derived from the First Steps Nutrition Trust, Vegan Society and Public Health England as a proxy for oat drink consumption. Values rounded to 2 s.f.

** Uses estimated concentration of 6.11 µg OTA/ kg oat drink. Does not consider exposure to OTA from other food sources. Range of MOEs comes from the minimum and maximum soya drink consumption rates.

*** One value presented based on a consumption of 200 mL

Table 23: Chronic health risk calculated from estimated exposure to OTA from consumption of processed oats in the general diet for 6 to 60-month olds in the UK (calculated as margins of exposure)*****

Age group	**MOE (using BMDL10 of 4.73 µg/kg bw/day)****	**MOE (using BMDL10 of 14.5 µg/kg bw/day)****
6 to < 12 months	43-170	130-520
12 to ≤ 18 months	34-140	100-430
18 to < 24 months	39-160	120-500
24 to < 48 months	52-190	160-580
48 to < 60 months	69-210	210-630

* Uses maximum analytical concentration of 56.6 µg OTA/ kg processed oats. Does not consider exposure to OTA from other food sources. Range of MOEs comes from the mean and 97.5th percentile consumption rates. Values rounded to 2 s.f.

Table 24: Chronic health risk calculated from estimated exposure to OTA from consumption of oat drinks and processed oats in the general diet combined for 6 to 60-month olds in the UK (calculated as margins of exposure)*

Age group	**MOE (using BMDL10 of 4.73 µg/kg bw/day)****	**MOE (using BMDL10 of 14.5 µg/kg bw/day)****
6 to < 12 months	30- ---**	90- ---**
12 to ≤ 18 months	15-24	47-73
18 to < 24 months	17-26	52-81
24 to < 48 months	21-32	63-97
48 to < 60 months	25-39	76-120

*The mean chronic exposure to OTA from processed oats in the general diet has been summed with estimated exposure from oat drinks which uses minimum and maximum soya milk consumption rates. The assumption was made that all oats consumed were processed, and had a concentration of 56.6 µg OTA/ kg processed oats. Uses estimated concentration of 6.11 µg OTA/ kg oat drink. Values rounded to 2 s.f.
** One value presented based on a consumption of 200 mL

48. In the EFSA assessment, for non-neoplastic effects, the MOEs ranged from 7391 (lowest minimum LB exposure across national consumption surveys) to 266 (highest maximum UB exposure across national consumption surveys) for the mean exposure estimates, and from 1971 (lowest minimum LB) to 92 (highest maximum UB exposure) for the 95th percentile exposure estimates across dietary surveys and

age groups. The MOEs that were lower than 200 were for the 95th percentile exposures at maximum LB in the age group of 'Infants' and at maximum UB in the age group of 'Infants', 'Toddlers' and 'Other children' indicating a possible health concern for these age groups. MOEs in breastfed infants were all above 200 indicating a low health concern. The CONTAM Panel concluded that these MOEs indicate a possible health concern for non-neoplastic effects of OTA for high consumers in the young age groups. For neoplastic effects, the MOE values ranged from 22,656 (lowest minimum LB exposure) to 815 (highest maximum UB exposure) for the mean exposure estimates, and from 6,042 (lowest minimum LB exposure) to 281 (highest maximum UB exposure) for the 95th percentile exposure estimates across dietary surveys and age groups. The Panel concluded that for neoplastic effects in most of the surveys, in particular for high consumers and for breastfed infants in all scenarios the MOEs were below 10,000 and thus indicate a possible health concern for these consumer groups. In the interpretation of the MOE for the neoplastic risks, the Panel considered that the MOE of10,000 for substances that are directly genotoxic and carcinogenic may be too conservative in this case because the evidence for a direct interaction of OTA with the DNA is inconclusive.

DON

49. As seen from Table 25 below, consumption of oat drinks represents only a small fraction of the TDI for DON.

Table 25: Chronic health risk calculated from estimated exposure to DON from consumption of oat drink for 6 to 60-month olds in the UK (**% TDI**)*

Age group	**Minimum**	**Maximum**
6 to < 12 months	5.8	--**
12 to ≤ 18 months	7.3	12
18 to < 24 months	6.6	11
24 to < 48 months	5.3	8.8
48 to < 60 months	4.3	7.2

*The mean chronic exposure to processed oats has been summed with the minimum and maximum exposure to oat drinks. The assumption was made that all oats consumed were processed, and had a concentration of 24.66 µg DON/ kg processed oats. Uses estimated concentration of 2.66 µg DON/ kg oat drink. Uses the TDI of 1 µg DON /kg b.w. Values rounded to 2 s.f.

** One value presented based on a consumption of 200 Ml

Table 26: Chronic health risk calculated from estimated exposure to DON from consumption of processed oats in the general diet for 6 to 60-month olds in the UK **(% TDI)***

Age group	**Chronic health risk***	
	Mean	**97.5th %ile**
6 to < 12 months	1.2	4.7
12 to ≤ 18 months	1.5	6.0
18 to < 24 months	1.2	5.2
24 to < 48 months	1.1	4.0
48 to < 60 months	1.0	3.0

*Uses mean and 97.5th percentile consumption rates. Recipes were used for this assessment with oat content > 5% and 'oat-based alternatives to milk' were removed from the assessment. The assumption was made that all oats consumed were processed, and had a concentration of 24.66 µg DON/ kg processed oats. Values rounded to 2 s.f. Uses the TDI of 1 µg DON /kg b.w.

Table 27 Chronic health risk calculated from estimated exposure to DON from consumption of oat drinks and processed oats in the general diet combined for 6 to 60-month olds in the UK **(% TDI)***

Age group	**Minimum**	**Maximum**
6 to < 12 months	7.0	--**
12 to ≤ 18 months	8.8	14
18 to < 24 months	7.8	12
24 to < 48 months	6.4	9.9
48 to < 60 months	5.3	8.2

*The mean chronic exposure to DON from processed oats in the general diet has been summed with estimated exposure from oat drinks which uses minimum and maximum soya milk consumption rates. The assumption was made that all oats consumed were processed, and had a concentration of 24.66 µg DON/ kg processed oats. Uses estimated concentration of 2.66 µg DON/ kg oat drink. Values rounded to 2 s.f. Uses the TDI of 1 µg DON /kg b.w.
** One value presented based on a consumption of 200 mL

Table 28: Chronic health risk calculated from UK dietary exposures to DON in the general diet for young children in the UK (EFSA, 2017) **(%TDI)***

Age group**		**Mean dietary exposure**	**95th percentile diary exposure**
Infants (<12 months old)	**LB**	40	100
	MB	70	160
	UB	110	220
Toddlers (≥12 months to < 18 months	**LB**	80	150

	MB	120	210
	UB	160	270
Toddlers (≥18 months to < 36 months	**LB**	80	150
	MB	120	200
	UB	160	270
Other children (≥36 months to < 10 years old)	**LB**	80	130
	MB	100	170
	UB	130	220

* Data taken from EFSA (2017). Values rounded to 2 s.f. Lifted from Appendix F, table F.1. Uses the TDI of 1 µg DON /kg b.w.
** Age groups do not align with those presented for 6 to 60-month olds in Tables 8-11

50. The estimated exposures to DON from consumption of oat drink for 6 to 60-month olds in the UK (shown in Table 27) are below the TDI and are thus of no health concern. In the Addendum to the Overarching statement on the potential risks from contaminants in the diet of infants aged 0 to 12 months and children aged 1 to 5 years (COT, 2020), the acute and chronic exposures were calculated using data from the TDS; measurements were performed for DON, 3-AC-DON and 15-AC-DON, no measurements were available for 3-DON-glycoside. 3-Ac-DON and 15-Ac-DON were not detected in any samples above the limit of detection (LOD). A combined concentration for the sum of 15-Ac-DON, 3-Ac-DON and DON was not provided to the FSA as part of the TDS, thus the sum used in the exposure assessment was estimated by summing the individual concentrations of all three forms.

51. Mean and 97.5th percentile acute exposures to 15-Ac-DON, 3-Ac-DON and DON and the sum of all three forms were below the group ARfD of 8.0 µg/kg bw, for all age groups and are therefore not of toxicological concern for infants and young children aged 0 to 5 years old.

52. Mean and 97.5th percentile chronic exposures to 15-Ac-DON, 3-Ac-DON and DON were below the TDI of 1.0 µg/kg bw, for all age groups and were therefore not of toxicological concern. All mean and 97.5th percentile chronic exposures to the sum of all three forms were below the TDI, except the 97.5th percentile UB exposure in children > 12 months of age, which were at or up to 1.3-fold the TDI (see Table 28). This was considered unlikely to be of toxicological concern. It was noted that the sum of all forms was not based on individual measured values but on summing the respective averages of the concentrations provided. Therefore, exposure estimates might have been conservative (COT, 2020)

Conclusions

53. Two approaches have been taken to assess exposure to HT-2 and T-2 mycotoxins for 6 to 60-month olds consuming oat drink. Firstly, assessment of exposure to HT-2 and T-2 from oat drinks as well as from consumption of oats from other sources in the diet. These have been considered both individually and aggregately for their contribution to the TDI. Secondly, the amount of oat drink required to exceed the HBGVs was estimated.

54. In terms of acute exposure to HT-2, T-2, and DON, it can be seen from Tables 4 and 9 that vast amounts of oat drink are required to exceed the ARfD, thus acute exposure to HT-2 and T-2 and DON from consumption of oat drink is of low risk. Acute exposure for OTA has not been assessed in the same way because EFSA has stated that the use of an HBGV for OTA would not be appropriate (an MOE approach was used instead).

55. As noted previously, there are several sources of uncertainty in this risk characterisation relating to the use of soya milk consumption data and the concentrations in oat drink. Therefore, the MOEs should be interpreted with these uncertainties in mind. The maximum reported concentration of OTA in “oat kernels” (i.e. groats) was used, however reported exposures ranged from 0.05 - 56.6 µg/kg for processed oats ready for human consumption. Furthermore, it is unclear whether further processing during oat drink manufacturing could further reduce OTA levels. As already mentioned the intake assumptions for oat drinks are based on recommendations on soya, which is indicated as the preferable dairy milk alternative. It is therefore likely that the consumption could be lower than assumed and should occur in combination with other, more nutritious alternatives, such as soya milk. It is thus likely that exposures would be much lower, and any potential exceedances will be occasionally and of very short duration. It should also be noted that based on EFSA’s recent evaluation, for neoplastic effects the MOEs are lower than 10,000 across most age groups. They also noted that the MOE of 10,000 for substances that are directly genotoxic and carcinogenic may not be appropriate in this case because the evidence for a direct interaction of OTA with the DNA is inconclusive. They therefore considered the uncertainty in the assessment to be high and noted that it is likely that the risk was being overestimated.

56. In terms of chronic dietary exposure to DON, it can be seen that estimated exposure from consumption of oat drinks (Table 25), processed oats in the general diet (Table 26), and also combined exposure from both sources (Table 27) are below the TDI for young children indicating no health concern.

57. It can be seen from the health risk calculations that oat drinks contribute significantly to dietary mycotoxin exposure compared to the contribution from oats and the background diet; and that consumption of oat drinks might lead to minor exceedances of the TDI. However, the assumptions are very conservative, because

soya drink consumption for those wishing to avoid dairy products, individuals with an intolerance to lactose or another component of milk or those following a plant-based diet is likely to be greater than for the general public, leading to exposure estimates that may be overestimated when considering the general population. It should also be noted that the TDI sets safe limits for exposures over lifetime, however these minor exceedances will be transient in nature given the short duration of exposure for these age groups as children grow up.

58. There are several limitations of the risk calculations using EFSA's background diet (shown in Table 20). The background diet includes dairy and oats and thus their estimated exposure to HT-2 and T2 may therefore be an overestimation for children following a plant based or dairy free diet. Although this is not UK data alone, it nevertheless provides an estimation of exposure to these mycotoxins from the total diet versus a high consumer of oat drinks. In 2018, the COT has previously assessed HT-2 and T-2 in in the diet of infants aged 0 to 12 months and children aged 1 to 5 years, and the Committee concluded here that it is unlikely that chronic dietary exposure levels of T-2 and HT-2 would be of any toxicological concern in infants and young children (COT, 2018).

Questions to the Committee

59. Members are asked to review the evidence available and consider the following questions:

i) Do the Committee think that intakes of HT-2 and T-2 from consumption of oat drinks may be of concern in children aged 6 months to 5 years of age?
ii) Do the Committee think that intakes of DON from consumption of oat drinks may be of concern in children aged 6 months to 5 years of age?
iii) Do the Committee think that intakes of OTA from consumption of oat drinks may be of concern in children aged 6 months to 5 years of age?
iv) Do the Committee have any other comments?

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