

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

Discussion paper on the health risk of mycotoxin contamination in oat drinks for 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 health effects of oat drinks in the diets of these age groups.

2. Plant-based drinks are an increasingly popular replacement for dairy and other animal milks. In 2018, 114.5 million litres of plant-based drinks (rice, oat, almond, hazelnut and hemp) were sold in the UK whereas 127.5 million litres of plant-based drinks were sold in 2019. Oat drinks may be consumed due to medical conditions such as lactose or gluten intolerance (pure oats are lactose and gluten free), Cows' milk allergy (caused by casein and/or whey proteins present in cows' milk), or a soy or nut allergy. Additionally, oat drinks may be used to replace dairy milk in vegan diets, for example in beverages and in cooking. Veganism is growing, with 600,000 vegans in the UK in 2019, up from 150,000 in 2014 (Vegan Society, 2016).

3. The UK Government advises that "infant formula is the only suitable alternative to breast milk in the first 12 months of your baby's life. Whole cows' milk can be given as a main drink from the age of 1". Furthermore, "you can give your child unsweetened calcium-fortified milk alternatives, such as soya, almond and oat drinks, from the age of 1 as part of a healthy, balanced diet¹".

Production process of oat drink

4. Oats are often processed in facilities with shared equipment that is also used to process gluten-containing grains such as wheat or barley. This may result in cross-contamination of the oat grains with gluten. However, certified gluten-free oat drink is available to purchase in stores.

5. The oat grain consists of the groat (caryopsis) and the surrounding hull (husk). The hull is indigestible and must be separated and removed in a de-hulling stage. The groats are then stirred in warm water and ground into a slurry. Because starch constitutes the major portion of the oats which poses a problem in preparation of a stable emulsion and during the heat treatment, enzymes are added to break down the starch into smaller components, primarily maltose, which also sweetens the taste of the drink (Sethie *et al.* 2016). The liquid then undergoes filtration to remove any loose shells from the oats. More ingredients are added at this stage (see

¹ <https://www.nhs.uk/conditions/pregnancy-and-baby/understanding-food-groups/>

paragraph 6) before being heat-treated to eliminate pathogens and extend the shelf-life. This may be ultra-heat treatment (where the temperature is increased >135 °C) or pasteurisation (72° C). The oat drink is then packaged for sale.

6. Oat drink is naturally lower in calcium, riboflavin and vitamins B12 and vitamin D than milk, so the addition of these nutrients is necessary in order for the product to be a direct nutritional substitute for dairy milk. Organic oat drinks do not contain added vitamins or minerals. Iron can also be added. Certain brands add sugar to sweeten the taste of the drink. Oat drink often also contains emulsifiers such as canola and rapeseed oil which help keep the water and oats blended together.

7. A number of brands of oat drink are sold in the UK, for example Alpro and Plenish. Because of slight 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

Mycotoxin contamination

8. Under cool and moist conditions, *A. sativa* (the common oat) may be colonised by various species of fungus in the *Fusarium* genus such as *F. langsethiae*. This colonisation is often accompanied by the production of the T-2 mycotoxin. This mycotoxin commonly enters the food chain through contaminated food, mainly cereals. In animals, the T-2 toxin is rapidly metabolised with the HT-2 toxin being a major metabolite. The chemical structures of the T-2 and HT-2 toxins are shown in Figure 1. T-2 and HT-2 toxins inhibit protein synthesis and are highly cytotoxic, causing skin and mucosa erosions and reduction of lymphocytes in exposed animals. The toxicokinetics and toxicity of the T-2 and HT-2 toxins are reviewed in TOX/2018/05 (FSA, 2018). The levels of mycotoxins can be influenced by the processing methods used as outlined in paragraph 5.

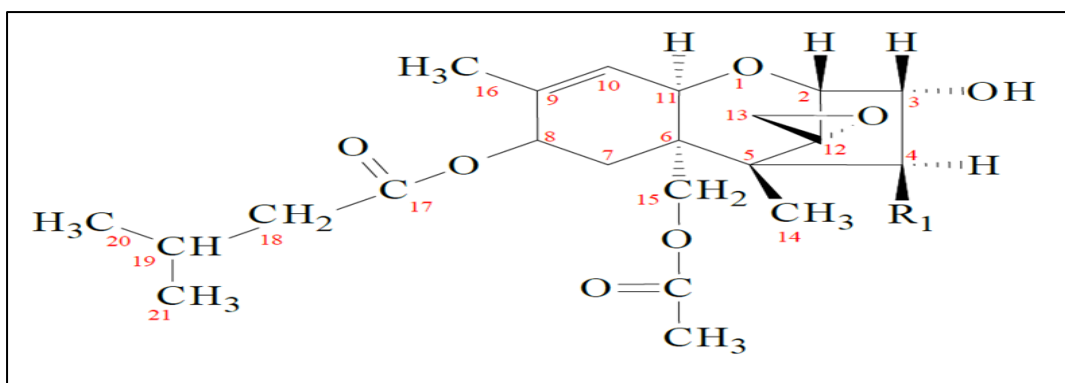


Figure 1: Chemical structure of the T-2 toxin ($R_1 = \text{OAc}$) and HT-2 toxin ($R_1 = \text{OH}$). These toxins are also known as trichothecenes. In aqueous solution, T-2 and HT-2 are stable within a physiological pH range with estimated half-lives of 3.9 and 8.5 years, respectively (Duffy & Reid, 1993). Karlovsky *et al.* (2016) concluded that “most mycotoxins are chemically and thermally stable. While conventional food preparation with temperatures up to 100 °C have little effect on most mycotoxins, higher temperatures used in frying, roasting, toasting, and extrusion might reduce mycotoxin contamination”. Figure taken from EFSA 2011.

9. In 2013, the European Commission published a Recommendation (2013/165/EU) that included indicative levels for the combined concentration of T-2 and HT-2 in various foodstuffs (EC, 2013). For example, the EU indicative level for the combined concentration of HT-2 and T-2 is 1 mg/kg in “unprocessed oats with husk”, and 50 µg/kg in “other cereal milling products”. The Recommendation states that Member States, in conjunction with industry, should continue to monitor these mycotoxins.

Acute Reference Dose

10. 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 BMDL₁₀ 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

11. 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 sub-chronic 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 BMDL₁₀ 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 sub-chronic study).

Previous risk assessments

12. In the 2014 UK oat crop there were findings of higher than usual levels of T-2 and HT-2 in whole oats (FSA, 2013 unpublished data). This led the FSA to commission a survey of oat-based food products expected to be derived from the 2014 UK oat crop. 200 samples intended for human consumption were collected from a broad range of retailers across the UK and included for example porridge oats and oat biscuits, and one sample of oat drink. These samples were analysed for the presence of T-2 and HT-2 toxins (FERA, 2015). The results of this 2015 retail survey showed that when measurement uncertainty was applied, the sum of T-2 and HT-2 toxin levels in all of the samples were below the indicative levels set out in Recommendation 2013/165/EU. The concentration for the sum T-2 and HT-2 toxins in the oat drink sample (Rude Health) was 2.6 µg/kg (lower bound) and 3.6 µg/kg (upper bound), based on the limit of quantification which was 1 µg/kg for both toxins.

13. A UK exposure assessment was conducted to estimate the exposures of infants and young children (ages 4 to 60 months) to the sum of T-2 and HT-2 toxins using occurrence data from all oat-based food products analysed in the 2015 retail survey (FSA, 2018) and consumption data from the Diet and Nutrition Survey of Infants and Young Children (DNSIYC) and the National Diet and Nutrition Survey (NDNS). Mean and 97.5th percentile acute exposures ranged from 0.022-0.032 and 0.056-0.11 µg/kg bw, respectively (across LB-UB estimates and age groups). These were all below the ARfD of 0.3 µg/kg bw and are therefore of no 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 chronic exposures were below the TDI of 0.02 µg/kg bw/day and are therefore of no toxicological concern. The chronic 97.5th percentile chronic exposures, however, ranged from 145-315 % of the TDI. In respect of these chronic exposure estimates, the FSA Secretariat concluded that “whilst an effect on health cannot be entirely excluded it is doubtful that children would be exposed to these levels in most years when there were not the high levels of T-2 and HT-2 observed in this harvest. It is therefore unlikely that T-2 and HT-2 would be of toxicological concern” (FSA, 2018). The COT Committee subsequently “requested a small number of further modifications and the statement would then be cleared by Chair’s action”, however this statement is yet to be published. Although T-2 and HT-2 mycotoxins are regularly found in oats (Pettersson *et al.* 2011), barley, maize and wheat also show frequent contamination (van der Fels-Klerx & Stratakou 2010), so this exposure assessment may underestimate actual exposure to these toxins from the diet.

Occurrence data

14. Oat drink consumed in the UK is made from European-harvested oats (Table 1). 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.

Toxin	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. Adapted from EFSA 2011.

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

15. 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.

16. 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:

$$236 \text{ µg/ kg oats} \times 0.02 = 4.72 \text{ µg/ kg oats} \quad [1]$$

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:

$$(4.72 \text{ µg/ kg} \div 1000 \text{ g}) \times 108 \text{ g} = 0.51 \text{ µg} \quad [2]$$

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).

² <https://ahdb.org.uk/knowledge-library/fusarium-mycotoxins-in-cereals>

Exposure assessment

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

18. Two approaches are hereby taken for estimating UK infant and young children exposures to T-2 and HT-2 from oat drink consumption. These are:

- i. using the estimated concentration of 0.51 µg T-2 and HT-2/ kg oat drink from occurrence data in European-harvested oats (see paragraph 16; Table 3).
- ii. using the analytical result from the 2015 retail survey (FERA, 2015) which reported that the concentration in one oat drink sample was 2.6 µg (LB) and 3.6 µg T-2 and HT-2/ kg oat drink (UB) (see paragraph 12; Table 4).

These occurrence data were combined with acute and chronic consumption data from the DNSIYC (DH, 2013; Lennox *et al.*, 2013) and NDNS (Bates *et al.*, 2014; Bates *et al.*, 2016; Roberts *et al.*, 2018) to estimate the corresponding exposures which are shown in Tables 3 and 4.

19. The DNSIYC survey includes consumption data for infants aged 3 <18 months, whilst the NDNS survey includes the consumption data for ages >18 months. There are some limitations of these consumption data for oat drink:

- A small number of individuals aged 6-18 months (n=4) and young children aged 18 months – 5 years (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 consumption data for cows’ milk were used instead as proxy for oats in the exposure assessment, as a much greater number of individuals are reported to consume it.
- No recipes with oat drink are reported in the DNSIYC and NDNS surveys. For consistency, recipes have therefore been excluded from the cows’ milk consumption data. There are no substantial differences to the magnitude of cows’ milk consumption if recipes are included in this database (data not shown).

Table 3: Estimated acute and chronic exposures to the sum of T-2 and HT-2 from oat drink consumption in infants and young children aged 6 months to 5 years (using an UB concentration of 0.51 µg T-2 and HT-2/ kg oat drink).

Age group	Acute exposure (µg/kg bw/day)*		Chronic exposure (µg/kg bw/day)*	
	Mean	97.5th %ile	Mean	97.5th %ile
6 to <18 month-olds (n = 1826)	0.015	0.050	0.011	0.035
18 month- to 5 year-olds (n = 1053)	0.013	0.036	0.0093	0.028

* Assumes all cows’ milk in diet is replaced with oat drink, but excludes all cow’s milk recipes as well as infant formula. N = number of consumers. Values rounded to 2 significant figures.

Table 4: Estimated acute and chronic exposures to the sum of T-2 and HT-2 from oat drink consumption in infants and young children aged 6 months to 5 years (using 2.6 (LB) and 3.6 (UB) μg T-2 and HT-2/ kg oat drink).

Age group	Acute exposure ($\mu\text{g}/\text{kg}$ bw/day)*		Chronic exposure ($\mu\text{g}/\text{kg}$ bw/day)*	
	Mean	97.5th %ile	Mean	97.5th %ile
6 to <18 month-olds (n = 1826)	0.075-0.10	0.30-0.32	0.056-0.077	0.18-0.25
18 month- to 5 year-olds (n = 1053)	0.067-0.093	0.18-0.25	0.047-0.065	0.14-0.20

* Assumes all cows' milk in diet is replaced with oat drink, but excludes all cow's milk recipes as well as infant formula. N = number of consumers. Values rounded to 2 significant figures.

Risk characterisation

20. The estimated acute and chronic exposures in Tables 3 and 4 ($\mu\text{g}/\text{kg}$ bw/day) were used to calculate the health risks as percentages of the ARfD (0.3 μg T-2 and HT-2/kg bw) and the TDI (0.02 μg T-2 and HT-2/kg bw/day), respectively. These percentages are shown below in Tables 5 and 6.

Table 5: Calculation of acute and chronic risks from estimated dietary exposure to T-2 and HT-2 in oat drink in infants and young children aged 6 months to 5 years (using 0.51 μg T-2 and HT-2/ kg oat drink).

Calculation of health risk	6 to <18 month-olds (n = 1826)		18 month- to <5 year-olds (n = 1053)	
	Mean	97.5 th %ile	Mean	97.5 th %ile
Acute risk (% ARfD)	4.9	15	4.4	12
Chronic risk (% TDI)	55	170	46	140

Values rounded to 2 significant figures. N = number of consumers.

Table 6: Calculation of acute and chronic risks from estimated dietary exposure to T-2 and HT-2 in oat drink in infants and young children aged 6 months to 5 years (using 2.6 (LB) and 3.6 (UB) μg T-2 and HT-2/ kg oat drink).

Calculation of health risk	6 to <18 month-olds (n = 1826) (LB-UB)		18 month- to <5 year-olds (n = 1053) (LB-UB)	
	Mean	97.5 th %ile	Mean	97.5 th %ile
Acute risk (% ARfD)	25-34	76-110	22-31	60-84
Chronic risk (% TDI)	280-390	880-1200	240-330	700-970

Values rounded to 2 significant figures. N = number of consumers.

Conclusions

Risk characterisation using the estimate of 0.51 μg T-2 and HT-2/ kg oat drink:

21. The estimates of acute exposure to T-2 and HT-2 mycotoxins via the consumption of oat drink are below the ARfD for infants and young children for both mean and 97.5th percentile consumption rates. These exposures are likely to be even lower in infants given that the UK Government advises that “infant formula is the only suitable alternative to breast milk in the first 12 months of your baby's life”.

22. Using mean consumption data, estimates of chronic exposure to T-2 and HT-2 mycotoxins via the consumption of oat drink are below the TDI for infants and young children indicating no appreciable health risk.

23. Using 97.5th percentile consumption rates, estimates of chronic exposure exceed the TDI by 70 % for 6 to <18 month-olds, and 40 % for 18 month- to <5 year-olds. These exceedances may indicate a health concern, though this health risk is not expected to be present for the majority of these individuals. Despite these exceedances, these exposure estimates are based on the assumption all dietary cows' milk is replaced with oat drink which is unlikely to be the case for all individuals in these age groups.

Risk characterisation using the analytical result of 2.6 (LB) and 3.6 (UB) μg T-2 and HT-2/ kg oat drink:

24. Estimates of acute exposure to T-2 and HT-2 mycotoxins via the consumption of oat drink are below the ARfD for infants and young children for both mean and 97.5th percentile consumption rates, though for the 6 to <18 month-old age group there is a 10 % upper-bound exceedance of the ARfD at the 97.5th percentile consumption rate.

23. All estimates of chronic exposure to T-2 and HT-2 mycotoxins via the consumption of oat drink exceed the TDI for infants and young children for both mean and 97.5th percentile consumption rates indicating a health risk. Using mean consumption rates, estimates of chronic exposure exceed the TDI by 180-290 % for 6 to <18 month-olds, and 140-230 % for 18 month- to <5 year-olds. Using 97.5th percentile consumption rates, estimates of chronic exposure exceed the TDI by 780-1100 % for 6 to <18 month-olds, and 600-870 % for 18 month- to <5 year-olds.

Questions on which the views of the Committee are sought

25. Members are invited to consider the following questions:

- i. Do Members have any other toxicological concerns concerning oat drink consumption other than exposure to T-2 and HT-2?
- ii. Should the exposure assessment be based on the analytical ($n = 1$) or estimated concentration of T-2 and HT-2 in oat drink?
- iii. For the preferred approach for exposure assessment:
 - a) Are there any other aspects which should be considered for the risk assessment?
 - b) Do Members consider that the corresponding chronic exposure estimates indicate a health concern?

References

Bates B., Lennox A., Prentice A., *et al.* (2014). National Diet and Nutrition Survey (NDNS): results from Years 1 to 4 (combined) of the rolling programme for 2008 and 2009 to 2011 and 2012. Available at:

<https://www.gov.uk/government/statistics/national-diet-and-nutrition-survey-results-from-years-1-to-4-combined-of-the-rolling-programme-for-2008-and-2009-to-2011-and-2012>

Bates B., Cox L., Nicholson S., *et al.* (2016). National Diet and Nutrition Survey Results from Years 5 and 6 (combined) of the Rolling Programme (2012/2013 – 2013/2014). Available at:

<https://www.gov.uk/government/statistics/ndns-results-from-years-5-and-6-combined>

DH (Department of Health) (2013). Diet and Nutrition Survey of Infants and Young Children. Available at: <https://www.gov.uk/government/publications/diet-and-nutrition-survey-of-infants-and-young-children-2011>

Duffy M.J. & Reid R.S. (1993) Measurement of the stability of T-2 toxin in aqueous-solution. *Chemical Research in Toxicology* **6**: 524-529

EC (2013) 2013/165/EU: Commission Recommendation of 27 March 2013 on the presence of T-2 and HT-2 toxin in cereals and cereal products Text with EEA relevance. Available at: <https://eur-lex.europa.eu/legal-content/EN/TXT/?uri=CELEX%3A32013H0165>

EFSA (2011) Scientific Opinion on the risks for animal and public health related to the presence of T-2 and HT-2 toxin in food and feed. *EFSA Journal* 9(12): 2481 Available at: <https://www.efsa.europa.eu/en/efsajournal/pub/2481>

EFSA (2017a) Appropriateness to set a group health based guidance value for T2 and HT2 toxin and its modified forms. *EFSA Journal* 15(1): 4655 Available at: <https://efsa.onlinelibrary.wiley.com/doi/full/10.2903/j.efsa.2017.4655>

FERA (2015) Final report: Retail Survey of T-2 / HT-2 Toxin Levels in Oat Based Products (FS 102126). Available at: <https://fsa-catalogue2.s3.eu-west-2.amazonaws.com/fs102126ferareport.pdf>

FSA (2013) FERA presentation at T-2 and HT-2 toxin Stakeholder Meeting, held Food Standards Agency, Feb 2015.

FSA (2018) Second draft statement of T2-toxin (T2), HT2-toxin (HT-2) and neosolaniol (NEO) in the diet of infants aged 0 to 12 months and children aged 1 to 5 years (TOX/2018/05). Available at: <https://cot.food.gov.uk/sites/default/files/tox2018-05.pdf>

Karlovsy P., Suman M., Berthiller F., *et al.* (2016) Impact of food processing and detoxification treatments on mycotoxin contamination. *Mycotoxin Research* **32**: 179-205

Lennox A, Sommerville J, Ong K, Henderson H, Allen R (2013) Diet and Nutrition Survey of Infants and Young Children. Available at: <https://www.gov.uk/government/publications/diet-and-nutrition-survey-of-infants-and-young-children-2011>

Pettersson H., Brown C., Hauk J., *et al.* (2011) Survey of T-2 and HT-2 toxins by LC-MS/MS in oats and oat products from European oat mills in 2005-2009. *Food Addit Contam Part B*. **4**: 110-115

Rahman S., Sharma A.K., Singh N.D., *et al.* (2014). Clinico-haematological changes in T-2 toxicosis in Wistar rats. *Indian Journal of Veterinary Pathology* **38**: 22-28

Roberts C., Steer T., Maplethorpe N., *et al.* (2018) NDNS: results from years 7 and 8 (combined). Results of the National Diet and Nutrition Survey (NDNS) rolling programme for 2014 to 2015 and 2015 to 2016. Available at: <https://www.gov.uk/government/statistics/ndns-results-from-years-7-and-8-combined>

Schwake-Anduschus C., Langenkämper G., Unbehend G., *et al.* (2010) Occurrence of Fusarium T-2 and HT-2 toxins in oats from cultivar studies in Germany and degradation of the toxins during grain cleaning treatment and food processing. *Food Additives and Contaminants. Part A, Chemistry, Analysis, Control, Exposure & Risk Assessment* **27**: 1253-1260

Sethi S., Tyagi S.K., & Anurag R.K. (2016) Plant-based milk alternatives an emerging segment of functional beverages: a review. *J Food Sci Technol* **53**: 3408-3423

van der Fels-Klerx H.J. & Stratakou I. (2010) T-2 toxin and HT-2 toxin in grain and grain-based commodities in Europe: occurrence, factors affecting occurrence, co-occurrence and toxicological effects. *World Mycotoxin J*. **3**: 349-367

Vegan Society (2019). Available at: <https://www.vegansociety.com/news/media/statistics> (accessed 15/01/2020)

Wu W., Zhou H., Bursian S.J., *et al.* (2016). Emetic responses to T-2 toxin, HT-2 toxin and emetine correspond to plasma elevations of peptide YY3-36 and 5-hydroxytryptamine. *Archives of Toxicology* **90**: 997-1007