Measurement of the concentration of mycotoxins from the UK TDS

The following technical reports will be published with the food safety information sheet. They provide further information on the analytical methods used, tabulate the analytical results and describe the sampling plan used to collect the samples.

Sampling for UK Total Diet Study HallMark Veterinary & Compliance Services.

Final Report TDS Mycotoxin Analysis FS102081, Fera

Executive summary

1. This is the first Total Diet Study (TDS) commissioned by the Food Standards Agency (FSA) to consider the occurrence levels of mycotoxins in the UK diet.

2. The FSA commissioned the TDS to assess the risk to health from dietary exposure to these contaminants and to provide a benchmark for monitoring future trends in occurrence levels.

3. The dietary exposure data for the mycotoxins included in the 2014 TDS have been used by the FSA to provide robust and up to date risk assessments when considering the risk to human health.

4. A TDS is suitable for contaminants which are found in most foods which make up the UK diet. However, mycotoxins have a more limited distribution and not all food categories in the present study were analysed for the full range of mycotoxins.

5. The most frequently detected mycotoxins were deoxynivalenol (DON) and the ergot alkaloids. These were present in all bread and cereal product samples.

6. Of the twelve mycotoxins included in the risk assessment ten did not represent a health concern. These were ochratoxin A (OTA), the ergot alkaloids, fumonisins, moniliformin, patulin, sterigmatocystin, zearalenone, DON (including 3 - Ac DON and 15 - Ac DON), nivalenol, T2, HT2 toxin and neosolaniol (NEO). The estimated dietary exposures were all below health-based guidance values. However, for the aflatoxins (B1, B2, G1, G2) and citrinin there is some uncertainty about the risk to health.

7. Aflatoxins are genotoxic and carcinogenic their presence in food is always undesirable and it is not possible to exclude a food safety concern. Risk management being based on the ALARA principle.

8. For citrinin genotoxicity and carcinogenicity cannot be excluded at the estimated exposure level, because of a lack of available data on these effects. However, it should be noted that the dataset for citrinin was left censored with no numerical values being reported for the food groups analysed in the survey.

Introduction

1. This is the first Total Diet Study (TDS) commissioned by the Food Standards Agency (FSA) to consider mycotoxins in the average UK diet. Previous TDS have focused on the heavy metals/elements and the process contaminant acrylamide. These were also included in the current TDS and the findings are published separately.
2. A TDS typically considers contaminants which are found in most foods making up the diet. However, mycotoxins have a more limited distribution in food. Therefore, the analysis was targeted to the seventeen food groups out of the total of twenty-eight in the TDS, known to be susceptible to mycotoxin contamination.

3. Mycotoxins are produced by moulds which colonise food crops at various growth stages as well as food commodities during storage. They are present in a wide range of foods sourced from around the world. Occurrence is particularly prevalent in countries which experience high temperatures and humidity such as the tropics.

4. Mycotoxins cause adverse health effects in animals and humans. The aflatoxin group are genotoxic and can cause cancer in humans. The European Food Safety Authority (EFSA), European Scientific Committee on Food (SCF) and the Joint FAO/WHO Expert Committee on Food Additives (JECFA) have published scientific opinions on toxicity for zearalenone (ZEN), trichothecenes such as DON (including 3-Ac DON and 15-Ac DON), T2, HT2 & NEO toxins, fumonisins, OTA, aflatoxins, Ergot alkaloids, moniliformin, patulin, sterigmatocystin, nivalenol, citrinin and 4, 15-diacetoxyscirpenol.

5. A guidance value such as a tolerable daily intake (TDI) or tolerable weekly intake (TWI) has been established for many mycotoxins. The TDI or TWI is a health based guideline value for lifetime exposure without adverse effects on human health.

6. To protect consumer health, regulatory controls for: aflatoxin (B1, B2, G1 and G2), OTA, patulin and fusarium toxins in certain foodstuffs, are set out in European Union (EU) legislation, specifically Commission Regulation (EC) No 1881/2006 as amended. It is the responsibility of food businesses to ensure products placed on the market are complaint with the maximum levels set out in the regulation.

The Total Diet Study

1. The TDS is designed to represent the average UK diet and as such can be used to estimate dietary exposures and to identify trends in the levels of contaminants present in food. It is also used to make assessments on the safety and/or nutritional quality of food.

2. For the 2014 TDS, retail samples for one hundred and thirty-eight categories of food were purchased from twenty-four local authority areas. The food categories were classified into twenty-eight food groups.

3. Seventeen food groups known to be susceptible to mycotoxin contamination were analysed for mycotoxins. These food groups were: bread, miscellaneous cereals, offal, oils and fats, eggs, sugars and preserves, potatoes, other vegetables, fresh fruit, fruit products, non-alcoholic beverages, milk, dairy products, nuts, alcoholic drinks, snacks and sandwiches. The contractor’s technical report lists the food categories comprising each food group (Table 1).

4. The food items making up each food category were prepared and cooked according to specified instructions agreed between the FSA and the analytical laboratory Fera (York).

5. The prepared samples were analysed for the following mycotoxins: aflatoxins (B1, B2, G1, G2 and M1), ochratoxin A, fumonisins (B1, B2 and B3), patulin, zearalenone, trichothecenes (deoxynivalenol, 3-acetyldeoxynivalenol, 15-acetyldeoxynivalenol), nivalenol, fusarenon-X, diacetoxyscirpenol, neosolaniol, HT2 toxin and T2 toxin, sterigmatocystin, ergot alkaloids (ergocornine, ergocorninine, ergocristine, ergocristinine, ergocryptine, ergocryptinine, ergometrine, ergometrinine, ergosine, ergosinine, ergotamine and ergotaminine), citrinin, cyclopiazonic acid and moniliformin.
6. Brand names are not reported in the survey because TDS samples are composites of foods of different types from a variety of sources.

7. Consumption data were obtained from the National Diet and Nutrition Survey (NDNS). The age classes considered for the 2014 TDS were: infants (4 to 18 months), toddlers and young children (1.5 to 3 years), young people (4 to 10 years), (11 to 18 years) and adults (19 years and above).

Methodology

Sampling

1. The sampling method is documented in the contractor’s report1. The 2014 TDS comprised one hundred and thirty-eight food categories. Each category consisting of food items sampled from twenty-four UK towns, giving a total of three thousand three hundred and twelve samples. The one hundred and thirty-eight food categories were pooled into twenty-eight food groups. However, the meat substitutes, sandwiches, tap water and bottled water food groups each contained a single food category. The food items for the various food categories were prepared and cooked according to specified instructions agreed between the FSA and the analytical laboratory Fera (York).

Analysis and results

1. Further details of the analytical methods and quality procedures used in the study can be found in the contractor’s technical report2.

2. Both food groups and food categories were analysed for mycotoxins. Where a given mycotoxin was not expected to occur in the food group or category analysis was not undertaken. For example, aflatoxin B1, B2, G1 and G2 were not analysed in dairy and eggs/egg products. Conversely aflatoxin M1 was the only aflatoxin analysed in these foods.

3. The contractor’s analytical report includes the occurrence level results for the various mycotoxins (tables 17 to 30 inclusive).

4. Samples were extracted using solvent and the cleaned-up extracts were analysed using the method that would allow the maximum sensitivity (lowest limit of quantification). In many cases, state of the art LC-MS/MS was used due to the high sensitivity and selectivity of the instrumentation, however HPLC-fluorescence was also used for some analyses where method performance was already well established or better sensitivity could be achieved with this method.

Method development and validation

1. Many methods used were UKAS accredited. Where required, improved extraction and instrumental methods were developed. Validation samples were analysed to establish method repeatability and recovery.

Exposure assessment summary

1. Exposure assessments were carried out using occurrence level data from the TDS for the various mycotoxins21 and consumption data from the Diet and Nutrition Survey of Infants and Young Children (DNSIYC)22and the National Diet and Nutrition Survey rolling programme (NDNS)23,24.

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1 Final Report UK Total Diet Study FS102081 Dec 2014, HallMark Veterinary & Compliance Services
2 Final Report TDS Mycotoxin Analysis FS102081, Dec 2015, Fera, (Stratton J et al)
2. Exposures were assessed for the populations aged 4 – 18 months (infants), 18 months – 3 years (toddlers and young children), 4 – 6 years, 7 – 10 years, 11 – 18 years and 19 + years (adults). Consumption data from the DNSIYC was used for children aged 4 – 18 months and from the NDNS for children aged 18 – 60 months.

3. Possible exposures were calculated from TDS – mycotoxin analysis data for different groups of mycotoxins. Generally, a high proportion of the data were left-censored i.e. non-detects data with concentration data below the limit of detection (LOD) or quantification (LOQ). Thus, the exposure assessments were expressed as the range of lower bound (LB) (where 0 is used as the analytical value) and upper bound (UB) (the limit of detection/quantification is used as the analytical value) values.

4. The high proportion of left censored data may have been due to the poor sensitivity of some of the analytical methods used. Furthermore, several of the food matrices did not have standardised analytical methods and the large recovery corrections required inflated the calculated LOD or LOQ. This increased the upper bound values, leading to an overestimation of dietary exposure. For example, a method for analysing OTA in grape juice or wine was used to analyse a mixed sample of fruit and vegetable juices. The recovery for OTA in this sample was low at just 32%.

**Risk Assessment Summary**

**Aflatoxins (B1, B2, G1 and G2)**

1. Aflatoxins are produced by the *Aspergillus* species of mould that grow in warm, humid conditions, principally *A. flavus*, and *A. parasiticus*.

2. Aflatoxins occur in peanuts (groundnuts) and other edible nuts and their products, dried fruit, spices, cereals and cereal products. Milk and milk products may also be contaminated owing to the consumption of aflatoxin contaminated feed by ruminants.

3. Aflatoxins have been shown to cause cancer of the liver in laboratory animals and to directly damage DNA. They are also considered to cause liver cancer in humans, particularly in developing countries, where high levels of aflatoxins are found in a number of staple foods.

4. EFSA use a Margin of Exposure (MOE) approach to risk characterise aflatoxins. The following is based on the SCF (1994) opinion. The calculated dietary exposures are compared to a value derived from an animal study. For aflatoxins the value used is a BMDL10 value of 170 ng/kg bw/day derived from the animal data. The BMDL10 is the 95th percentile lower confidence limit of the benchmark dose for a 10% increase in the risk of tumours. The EFSA Scientific Committee proposed that a MOE of 10,000 or higher, based on a BMDL10 from an animal study, would be of low concern from a public health point of view.

5. The chronic dietary exposures were calculated using data from the TDS and consumption data from DNSIYC and NDNS. The results from all food samples that were analysed for aflatoxins were below the limit of quantification (LOQ). Therefore, the calculated exposures provided in Table 1 (Annex A) are expressed as lower bound (LB) and upper bound (UB).

6. The highest mean (LB - UB) and 97.5th percentile (LB - UB) dietary exposures for aflatoxin M1 were 0.000 - 0.001 and 0.000 – 0.005 µg/kg bw/day, respectively. These exposures were calculated for the 4 – 18 months age group. Toddlers and young children also had the same mean exposures. The exposures calculated for 11 – 18 year olds and adults were zero.
7. The highest calculated dietary exposures for aflatoxins B1, B2, G1 and G2 were seen for adults. They ranged from 0.000 – 0.011 and 0.000 – 0.031 µg/kg bw/day for mean and 97.5th percentile exposures, respectively.

8. MOEs were calculated for all AF exposures. The MOE approach is normally used for AFB1 and total AF exposures. Total AF results were not provided to the FSA as part of the TDS and due to the inconsistent reporting of the total AFs across the EU it is not certain whether total exposures could be calculated from the data available. Therefore, the MOE approach has been used for individual AFs.

9. The lowest MOEs are 170 and 34. These were calculated for mean and 97.5th percentile exposures, respectively, for aflatoxin M1 in the 4 –18 months age group. The lowest MOEs for aflatoxins B1, B2, G1 and G2 are > 15 and > 5.5 for mean and 97.5th percentile exposures, respectively.

10. Given that aflatoxins are genotoxic and carcinogenic their presence is always undesirable and it is not possible to exclude a safety concern.

Ochratoxin A (OTA)

1. OTA is a mycotoxin produced by several fungal species in the *Penicillium* and *Aspergillus* genera, primarily *Penicillium verrucosum*, *Aspergillus ochraceus* and *Aspergilli* of the section *Nigri*, especially *A. carbonarius*. OTA has been found in a variety of plant products such as cereals and cereal products, coffee beans, beans, pulses, cocoa products, nuts and spices and dried fruit from all over the world. It has also been detected in products such as coffee, wine, beer and grape juice and occurs in kidney, liver and blood from farm animals by transfer from animal feed (EFSA, 2006; EFSA, 2010).

2. The most sensitive and crucial effects of OTA are on the kidneys and these have been observed experimentally in rats and pigs. The extent of the kidney damage is dose- and time-dependent as OTA accumulates in the kidneys. EFSA (2006) established a TWI of up to 120 ng/kg bw based on nephrotoxicity in the pig. The COT agreed that despite its conservative nature this TWI should be used in COT risk assessments.

3. Occurrence levels for a number of mycotoxins, including OTA, were measured in the TDS. Only four food categories contained measurable residues of OTA, the rest were all below the LOQ. OTA was found in the fruit and vegetable juices, dried fruit, herbs and spices and bread (granary, brown) food categories. While the data obtained from the TDS could be used as a qualitative indicator of mycotoxins present in various food categories, it was not possible to use it for a quantitative estimation of dietary exposures.

4. Since analysis of the TDS samples involved a wide range of matrices (some of which had not been routinely examined previously), existing validated methods were adapted/extended to some of the new matrices. For example, the method used for grape juice or wine was used to analyse the composite sample of fruit and vegetable juices. However, the presence of other fruit and vegetables (e.g. orange, carrot) in addition to grape led to some analytical difficulties, with poor recoveries and consequently high results. The low recovery of 32% meant that the large correction inflated the result which will impact on calculated dietary exposures. Given the low recovery, this method is not considered suitable for this food matrix. Also, the reported concentration of 5.62 μg/kg OTA in the fruit and vegetable juice category was much higher than the average based on EFSA survey data (0.55 μg/kg).

5. Furthermore, a range of matrices were included in a single batch in the analysis. This had an impact on recoveries. Usually in a batch, similar matrices are used and a batch average
recovery is applied. In the above OTA example, three very different matrices - dried fruit, herbs and spices were included in the batch with fruit and vegetable juices (each individually spiked). If a batch recovery average which is not specific to the fruit juice matrix and grape juice method is applied, it would give an average recovery of 72% when applied to this sample. This would give a lower result but is not a reliable approach given the differences in the sensitivities of the methods and food matrices encountered in the TDS.

6. A multi-mycotoxin method was used in the analysis for various food groups, which is normally used as a screening tool rather than for quantification. This is reflected in the generally poor recoveries, higher Limits of Quantification/Detection (LOQ/LOD) and when these were corrected for recovery, led to artificially inflated occurrence levels in some cases. This furthers the unsuitability of the TDS data for quantitative exposure estimates.

7. Therefore, occurrence data from the TDS are not suitable for estimating dietary exposures quantitatively. The methods lack suitable sensitivity and are not sufficiently standardised and validated for this purpose.

8. The COT has recently produced a statement on OTA. Due to the limitations of the data outlined in the paragraphs above the COT used data from 2 years of a 4-year FSA retail survey to perform a risk assessment for OTA (FSA, 2010; FSA, 2011). No risk was identified at the levels of OTA measured in the foods tested. (COT, 2018a)

Citrinin

1. Citrinin is a polyketide mycotoxin produced by several species of the genera Aspergillus, Penicillium and Monascus. Citrinin is generally formed after harvest under storage conditions and it occurs mainly in grain but can also occur in other products of plant origin e.g. beans, fruits, fruit and vegetable juices, herbs and spices and in spoiled dairy products. In addition, citrinin is found as an undesirable contaminant in red mould rice (RMR) which is used as a food preservative and colourant in Asian foods. (EFSA, 2012).

2. Repeated dosing toxicity studies all confirmed the nephrotoxicity of citrinin. The status of citrinin as a carcinogen with or without genotoxic potential could not be confirmed given the limitations and uncertainties in the current database. For compounds that may be genotoxic and carcinogenic, EFSA recommends the use of an MOE approach for risk characterisation. However, due to the lack of data on human dietary exposure, an MOE could not be calculated. To give risk managers a tool to evaluate the risk of citrinin in food and feed, EFSA decided to characterise the risk of citrinin based on the available data on nephrotoxicity and determined a level of no concern for nephrotoxicity. EFSA concluded that there would be no concern for nephrotoxicity in humans at an exposure level of 0.2 µg/kg bw/day. Based on the available data, a concern for genotoxicity and carcinogenicity could not be excluded at the level of no concern for nephrotoxicity.

3. The results for all food samples analysed for citrinin were below the limit of quantification (LOQ). Therefore, the exposures provided are lower bound (LB) (zero) and upper bound (UB).

4. The highest UB mean and 97.5th percentile exposures were seen in toddlers and adults which were in the region of 0.019 and 0.043 µg/kg bw/day, respectively (Table 2, Annex A).

5. Mean and 97.5th percentile UB exposures for all age groups are below 0.2 µg/kg bw/day, the exposure level established by EFSA at which there would be no concern for...
nephrotoxicity in humans. However due to lack of available data, a concern for genotoxicity and carcinogenicity cannot be excluded at this level.

**Ergot alkaloids**

1. These mycotoxins are produced by fungi of the *Claviceps* genus, most notably *C. purpurea*, which parasitise the seed heads of living plants (mostly cereals and grasses) at the time of flowering. Ergot is ubiquitous and is more common in seasons with heavy rainfall. Rye and triticale are the most susceptible cereal species because they have open florets. The sclerotia are harvested together with the cereals or grass and can lead to contamination of cereal-based food and feed products. Normally, ergot is easily visible as intact sclerotia. The six most prevalent ergot alkaloids are ergotamine, ergocornine, ergocristine, ergocryptine, ergosine and ergometrine along with their nine stereoisomers.

2. EAs can act on a number of neurotransmitter receptors particularly adrenergic, dopaminergic and serotonergic receptors. The effects of these receptor interactions may be acute or long-term therefore EFSA established an ARfD and a TDI. An overall uncertainty factor of 300 was applied to the BMDL₁₀ of 0.33 mg/kg bw/day, calculated from a 13-week rat feeding study of ergotamine, to produce a group acute reference dose (ARfD) of 1 µg/kg bw for the sum of ergot alkaloids³⁰.

3. Using the same BMDL₁₀ of 0.33 mg/kg bw/day an uncertainty factor of 600 was applied to establish a group TDI of 0.6 µg/kg bw/day for the sum of ergot alkaloids³⁰.

4. All bread samples contained some or all of the 12 ergot alkaloids. Levels found ranged from <1 - 60.1 µg/kg for individual ergot alkaloids in the samples. Brown bread contained a total of 322 µg/kg ergot alkaloids. Ergot alkaloids were also detected in sandwiches, at a similar level to bread samples, and at lower levels in other cereal products such as flour, breakfast cereals, biscuits and pizza. The ergot alkaloids were not detected in branded food drinks, beer, cider or alternatives to milk¹³.

**Acute**

1. A portion size approach was used to calculate acute dietary exposure based on consumption of sandwiches as total ergot alkaloids was detected at a level of 14 µg/kg in the sandwiches group (Tables 5 – 10, Annex A).

2. Adult mean acute exposures were calculated for 1 round of sandwiches or 1 bap with fillings to up to 6 rounds of sandwiches or 6 baps or greater with fillings and exposures ranged from 0.032 – 0.19 µg/kg bw. Mean acute toddler exposures were calculated for 1 round of sandwiches or 1 bap with fillings to up to 4 rounds of sandwiches or 4 baps with fillings and ranged from 0.17 – 0.68 µg/kg bw.

**Chronic**

1. Total ergot alkaloid chronic exposures were calculated using data from the TDS and consumption data from DNSIYC and NDNS. Once the exposures had been calculated the main contributing groups for total ergot alkaloid exposures were “miscellaneous cereals breakfast cereals”, “white sliced bread” and “wholemeal and granary bread”.

2. Mean and 97.5th percentile chronic dietary exposures to total ergot alkaloids were calculated for all age groups. The lowest mean and 97.5th percentile exposures of 0.033 – 0.036 and 0.079 – 0.083 µg/kg bw/day, respectively, were calculated for adults. The highest mean exposures of 0.095 – 0.11 and 0.097 – 0.11µg/kg bw/day were calculated for toddlers and 4 to 6 years old, respectively. The highest 97.5th percentile exposures of 0.23 – 0.24 µg/kg bw/day were calculated for toddlers (Table 11, Annex A).
3. Total ergot alkaloid calculated mean and 97.5\textsuperscript{th} percentile acute exposures for all age groups ranged from 0.032 to 0.68 µg/kg bw, all of which are below the ARfD of 1 µg/kg bw established by EFSA in 2012.

4. Total ergot alkaloid mean and 97.5\textsuperscript{th} percentile chronic exposures for all age groups ranged from 0.033 to 0.24 µg/kg bw/day, all of which are below the TDI of 0.6 µg/kg bw/day.

5. Exposures to total ergot alkaloids at the levels measured in the TDS are unlikely to be of toxicological concern in either the acute or the chronic situation in any age group.

**Fumonisins**

1. Fumonisins are made up of about 15 closely related chemicals; the most common are B1 and B2. They are produced by *Fusarium* species and occur mostly in hot continental climates. Maize is the crop that is most commonly affected. Fumonisins have been related to oesophageal cancer in humans, and to liver and kidney toxicity in animals.

2. In 2012, JECFA derived a provisional maximum tolerable daily intake (PMTDI) of 2 µg/kg bw for fumonisins B1, B2 and B3, alone or in combination, based on a study of liver toxicity in mice\textsuperscript{31}. JECFA evaluated fumonisins again in 2016 and retained the PMTDI\textsuperscript{32}. In 2018, EFSA established a group TDI for fumonisins B1-4 of 1 µg/kg bw, based on increased incidence of megalocytic hepatocytes observed in a chronic study in mice.

3. The results from almost all the food sample groups that were analysed for fumonisins were below the LOD. Chronic exposures were calculated for all age groups using data from the TDS and consumption data from DNSIYC and NDNS. These exposures provided in Table 12, Annex A are LB and UB. FB1 was detected in the sample of “herbs and spices” at a level of 5.53 µg/kg, below the LOQ of 7.15 µg/kg. The highest exposures were calculated for fumonisin B1 and the lowest for fumonisin B3, respectively.

4. The highest mean exposures of 0 – 0.081 µg/kg bw/day were calculated for toddlers and the highest 97.5\textsuperscript{th} percentile exposures of 0.002 – 0.152 were calculated for toddlers and 4 – 6 year olds (Table 12, Annex A).

5. Total fumonisin results were not provided to the FSA as part of the TDS and due to the inconsistent reporting of the total fumonisins across the EU it is not certain whether total exposures could be calculated from the data available. Therefore, comparison to the group TDI and PMTDI of 1 and 2 µg/kg bw, respectively, have been used for individual fumonisins.

6. All calculated mean and 97.5\textsuperscript{th} percentile exposures of FB1, FB2 and FB3 for all age groups are below the TDI and PMTDI of 1 and 2 µg/kg bw, respectively, and are therefore not of toxicological concern.

**Moniliformin (MON)**

1. MON is formed in cereals by a number of *Fusarium* species and *Penicillium melanoconidium*. MON contamination has been found in samples of oats, maize, wheat, triticale and rye.

2. The limited toxicity data available on moniliformin indicated haematotoxic and cardiotoxic effects as the major adverse health effects of MON. EFSA (2018) assessed the toxicological data for moniliformin but were unable to establish an ARfD or TDI because the available information was too limited. Instead they used an MOE approach for both acute and chronic exposures. For acute assessments a NOAEL of 6 mg/kg bw/day for cardiotoxicity from a 28-day study in rats was used to compare with human exposure assessments. For chronic exposure assessments, a reference point of 0.20 mg/kg bw/day...
for haematotoxicity was established from a 28-day feeding study in pigs and was used to compare with human exposures.

3. MON is a very small, charged analyte which is highly soluble in water. The analysis of MON in this TDS was not straightforward, especially due to the number of food matrices analysed. Recovery results were very low, however the methods utilised in the analyses meant that reasonable LOQs could be determined, and if MON had been present in the samples it would have been detected.

4. The results from almost all the food sample groups that were analysed for MON were below the LOD or LOQ. Acute and chronic exposures were calculated for all age groups using data from the TDS and consumption data from DNSIYC and NDNS. The exposures provided in Table 2, Annex A are LB and UB.

Acute

1. The highest mean and 97.5\textsuperscript{th} percentile acute exposures of 0 – 0.22 and 0 – 0.45 µg/kg bw/day, were calculated for toddlers and 4- to 6-year olds, respectively (Table 2, Annex A). The MOE approach adopted by EFSA was used to compare these exposures to the reference point of 6 mg/kg bw/day (6000 µg/kg bw/day). The MOE values for the mean and 97.5\textsuperscript{th} percentile UB exposures were 30000 and 13000, respectively. When EFSA calculated MOEs for mean UB exposures they ranged from 11000 to 73000 and MOEs for 97.5\textsuperscript{th} percentile UB exposures ranged from 4000 to 29000. EFSA concluded that these MOEs were sufficiently large to indicate a low health concern for humans.

2. All calculated acute mean and 97.5\textsuperscript{th} percentile exposures calculated for MON in this TDS produced MOEs greater than the lowest MOE of 4000, considered by EFSA to be of low health concern for humans. Therefore, acute MON exposures would not be considered to be of toxicological concern.

Chronic

1. The highest mean and 97.5\textsuperscript{th} percentile exposures of 0 – 0.084 and 0 – 0.29 µg/kg bw/day, respectively were calculated for 4- to 18-month olds, and toddlers and 4- to 6-year olds, respectively (Table 2, Annex A). The MOE approach adopted by EFSA was used to compare these exposures to the reference point of 0.20 mg/kg bw/day (200 µg/kg bw/day). The MOE values for the mean and 97.5\textsuperscript{th} percentile UB exposures were 2380 and 670, respectively. When EFSA calculated MOEs for mean UB exposures they ranged from 880 to 25000 and MOEs for 97.5\textsuperscript{th} percentile UB exposures ranged from 370 to 4500. EFSA concluded that these MOEs were sufficiently large to indicate a low health concern for humans.

2. All calculated chronic mean and 97.5\textsuperscript{th} percentile exposures calculated for MON in this TDS produced MOEs greater than the lowest MOE of 370, considered by EFSA to be of low health concern for humans. Therefore, chronic MON exposures would not be considered to be of toxicological concern.

Patulin

1. Patulin is produced by the blue mould \textit{Penicillium expansum}. Patulin contamination is mainly found in apple products such as apple juice, although it can occur in other mouldy fruits, grains and foods. Patulin is destroyed by the fermentation process, and is much less prevalent in alcoholic apple beverages, such as cider. Patulin has various toxic effects and can harm the immune system and gastrointestinal tract.

2. In 1995 JECFA established a PMTDI of 0.4 µg/kg bw\textsuperscript{35} based on a NOEL from a 2 year combined reproductive toxicity, long-term toxicity/carcinogenicity study.
3. Patulin exposures were calculated using data from the TDS and consumption data from DNSIYC and NDNS. The results from all food groups that were analysed for patulin were below the LOD. Individual LODs calculated for the samples analysed ranged from 1.7 μg/kg for the mushroom sample to 13.6 μg/kg for the cereal sample.

4. The mean total lower bounds in all age groups for patulin are zero and thus it is not possible to attribute any food group contributing to total exposure.

5. Mean UB exposures ranged from 0.067 – 0.165 μg/kg bw/day for adults and toddlers, respectively. UB exposures for 97.5th percentile consumers ranged from 0.141 – 0.309 μg/kg bw/day for adults and 4 - <18 month olds, respectively (Table 2, Annex A).

6. Mean and 97.5th percentile exposures for all age groups are below the PMTDI of 0.4 μg/kg bw/day.

7. The levels of patulin measured in the food groups in the TDS are not of toxicological concern for any age group.

Sterigmatocystin

1. Sterigmatocystin is produced by more than 50 fungal species, including Aspergillus flavus, A. parasiticus, A. versicolor and A. nidulans, of which A. versicolor is the most common source. Sterigmatocystin is generally produced in storage, rather than in the field, and has been found in grains and grain-based products, green coffee beans, spices, beer, peanuts, crispbread, rye wholemeal, rice, white bread, muesli, chilli and cheese.

2. Sterigmatocystin exhibits genotoxic effects in vitro, in vivo and ex vivo and carcinogenicity of sterigmatocystin has been demonstrated after oral, intraperitoneal, subcutaneous and/or dermal exposure in the animal species tested.

3. Both EFSA and JECFA calculated a BMDL10 of 0.16 mg/kg bw/day to be used as the point of departure, against which to compare human dietary exposures, when using the MOE approach36, 37.

4. Exposures were calculated using data from the TDS and consumption data from DNSIYC and NDNS. Three samples contained sterigmatocystin below the LOQ but above the LOD at levels from 0.46 to 2.17 μg/kg. The mean total lower bounds in all age groups are zero and thus it is not possible to attribute any food group contributing to the total exposure.

5. Mean exposures ranged from 0 – 0.002 to 0 – 0.005 μg/kg bw/day for 4 – 18 month olds and toddlers, respectively. Exposures calculated for 97.5th percentile consumers ranged from 0.001 – 0.006 to 0.001 – 0.011 μg/kg bw/day for 11 – 18 year olds and toddlers, respectively (Table 2, Annex A).

6. MOEs for sterigmatocystin have been calculated based on the fold-difference between the lowest BMDL10 value of 0.16 mg/kg bw/day and the chronic exposures. All values are greater than 10,000.

7. EFSA is of the view that in general an MOE of 10,000 or higher, if it is based on the BMDL10 from an animal study, would be of low concern from a public health point of view25. It is therefore likely that exposures to sterigmatocystin for all age groups are of low concern.

Zearalenone

1. Zearalenone is produced by various Fusarium species and occurs in maize but also in wheat, barley, sorghum and rye in various countries. Generally, the Fusarium species can grow and invade crops in moist cool conditions or cereals post-harvest under poor storage conditions.
2. There is limited evidence of carcinogenicity of zearalenone but it is clastogenic. Zearalenone is oestrogenic and has been shown to exhibit hormonal effects such as infertility in pigs.

3. EFSA established a TDI of 0.25 µg/kg bw/day for zearalenone based on the oestrogenic effects in the pig (the most sensitive species)\(^3\). This TDI is 25,000 times lower than concentrations at which carcinogenic effects would be expected.

4. Exposures were calculated using data from the TDS and consumption data from DNSIYC and NDNS. Five samples contained residues above the LOD but below the LOQ. These levels ranged from 0.57 to 1.92 µg/kg, although these values are not quantitative. The pizza sample contained a level of 16.5 µg/kg and was the highest level measured in all the samples. All levels measured were well below the maximum permitted levels in legislation\(^2\). The snacks group made the main contribution to total zearalenone exposure.

5. Mean exposures ranged from 0.003 – 0.007 to 0.010 – 0.018 µg/kg bw/day for adults and 4 - 6 year olds, respectively. Exposures calculated for 97.5\(^{th}\) percentile consumers ranged from 0.025 – 0.032 to 0.061 – 0.069 µg/kg bw/day for adults and 7 – 10 year olds, respectively (Table 2, Annex A).

Mean and 97.5\(^{th}\) percentile exposures for all age groups were below the group TDI of 0.25 µg/kg bw/day.

The levels of zearalenone present in the food groups in the TDS were not of toxicological concern for any age group.

Deoxynivalenol (DON), 3-acetyldeoxynivalenol (3-Ac-DON) and 15-acetyldeoxynivalenol (15-Ac-DON)

1. DON and its acetylated derivatives are members of the trichothecenes family. DON is a type B trichotheoncne and is produced mainly by the \textit{Fusarium} species. DON, 3-Ac-DON and 15-Ac-DON are produced by the fungi as naturally occurring toxic secondary metabolites. They are predominantly a contaminant in wheat, rice, barley, oats, maize and rye and their products. Contamination may occur prior to harvest or during storage and because DON is, to a certain extent, resistant to thermal processing, can be found in cereals ready to consume.

2. DON has been shown to have effects on the immune system and exhibits reproductive and developmental toxicity in experimental animals. However, both JECFA (2011) and EFSA (2017) considered emesis to be the critical endpoint on which to base an ARfD and a 2-year feeding study in mice was appropriate for the evaluation of the long-term effects. For acute effects, EFSA and JECFA have both established a group ARfD for DON, 3-Ac-DON and 15-Ac-DON of 8 µg/kg bw. JECFA used BMD modelling of the data from 2 pig studies on emesis to calculate the ARfD and EFSA used data from outbreaks of mycotoxicoses in humans. For chronic effects, EFSA and JECFA both established a group TDI of 1 µg/kg bw for DON, 3-Ac-DON and 15-Ac-DON. JECFA reaffirmed its previous TDI based on data from a 2-year feeding study in mice. EFSA also used the 2-year mice feeding study.

**Acute**

1. Exposures were calculated using data from the TDS and consumption data from DNSIYC and NDNS. The derivatives 3-Ac-DON and 15-Ac-DON were not detected in any samples above the LOD. DON was detected in all cereal products, snacks and sandwiches at levels from 11.2 to 166 µg/kg. DON was also seen at concentrations between the LOD and LOQ in herbs and spices, vegetable oil and beer. The recovery of some of the initial analyses was low, therefore the analysis was repeated using the \(^{13}\)C-DON standard. This confirmed the presence of DON in all the samples tested and in most cases the concentration was in
good agreement. The largest variation in data was for the 166 µg/kg value (bread food group), with a repeat analysis value of 79 µg/kg, however this still fell within the range of uncertainty (39%) for this measurement (64 µg/kg). The risk assessment is based on the summed exposure from DON, 3-AC-DON and 15-Ac-DON.

2. The highest mean acute exposures of the sum of DON, 3-AC-DON and 15-Ac-DON were 0.55 – 0.84 µg/kg bw/day calculated for toddlers. The highest LB acute 97.5th percentile exposure was 1.33 µg/kg bw/day in toddlers and the UB was 1.75 µg/kg bw/day. The highest UB 97.5th percentile exposure was in adults and was 1.96 µg/kg bw/day with a corresponding LB exposure of 0.47 µg/kg bw/day (Annex A, Table 3).

3. All calculated acute mean and 97.5th percentile exposures calculated for the sum of DON, 3-Ac-DON and 15-Ac-DON in this TDS were below the ARfD of 8 µg/kg bw/day. Therefore, acute exposures would not be considered to be of toxicological concern.

Chronic

1. The highest mean chronic exposures of the sum of DON, 3-AC-DON and 15-Ac-DON were 0.34 – 0.58 µg/kg bw/day calculated for 4 – 6 year olds. The highest 97.5th percentile exposure was in toddlers and was 0.75 – 1.14 µg/kg bw/day (Annex A, Table 4).

2. All calculated chronic mean exposures calculated for the sum of DON, 3-Ac-DON and 15-Ac-DON in this TDS were below the TDI of 1 µg/kg bw/day and these exposures would not be considered to be of toxicological concern. The highest UB 97.5th percentile exposures exceed the TDI marginally. However, there is only a marginal erosion into the safety factor used to calculate the TDI and in addition the TDI is set over a lifetime and from the age of 7 the UB exposures fall below the TDI. Therefore, chronic exposures would not be considered to be of toxicological concern.

Nivalenol

1. Nivalenol is a type B tricothecene and is produced by the *Fusarium* genus, i.e. *F. crookwellence*, *F. poae*, *F. culmorum* and *F. graminearum*. The *Fusarium* species invade and grow on crops, and may produce nivalenol under moist and cool conditions. Nivalenol is predominantly found in cereal grains and cereal-based products.

2. Generally, trichothecenes are immunotoxic and haematotoxic/myelotoxic. EFSA considered disturbances in white blood cell counts to be the critical effect for risk assessment of nivalenol. A TDI of 1.2 µg/kg bw/day was established, based on haematological disturbances in white blood cell counts observed in rats.

3. EFSA established an ARfD of 14 µg/kg bw based on acute emetic events in mink.

Acute

1. Mean acute exposures ranged from 0.261 to 0.467 µg/kg bw for 11 – 18 year olds and toddlers, respectively. Exposures calculated for 97.5th percentile consumers ranged from 0.656 to 1.161 µg/kg bw for 11 – 18 year olds and 4 – 18 month olds, respectively (Table 3, Annex A).

Chronic

1. Mean nivalenol exposures ranged from 0 – 0.162 to 0 – 0.312 µg/kg bw/day for adults, and toddlers and 4 – 6 year olds, respectively. Exposures calculated for 97.5th percentile consumers ranged from 0 – 0.342 to 0 – 0.695 µg/kg bw/day for 11 – 18 year olds and 4 – 18 month olds, respectively (Table 4, Annex A).
2. All acute exposures for all age groups were below the ARfD and all chronic exposures for all age groups were below the TDI.

3. Exposures calculated from the current levels of nivalenol in food groups measured in the TDS were not of toxicological concern for any age group.

**T2 toxin (T2), HT2 toxin (HT2) and Neosolaniol (NEO)**

1. T2 and HT2 are type A trichothecenes and are produced by a variety of *Fusarium* species (*F. sporotrichoides, F. poae, F. equiseti, F. acuminatum*). They may also be produced by other crop invasive species of *Myrothecium, Cephalosporium, Verticilimonosporum, Trichoderma, Trichothecium* and *Stachybotrys*. *Fusarium* species grow and invade crops and produce the T2 and HT2 toxins under cool, moist conditions prior to harvest. T2 and HT2 are found predominantly in cereal grains (particularly oats) and their products. NEO is a hydrolytic phase I metabolite of T2 and may be formed in fungi and mammals. NEO has been found in some brewed coffee samples, in a sample of cereal-containing baby food and at trace levels in some barley field malt samples (EFSA, 2017a).

2. T2 and HT2 have most recently been evaluated by EFSA. In this Opinion they also evaluated NEO. A group (including T2, HT2 and NEO) ARfD of 0.3 µg/kg bw was established based on an acute oral gavage study in minks. For chronic exposure assessment comparisons, a group TDI of 0.02 µg/kg bw was established based on a 12-week study in rats.

3. Although T2, HT2 and NEO were analysed in various food groups in this TDS, the data were all markedly left censored. All values were below the LOQ and several were below the LOD. While these data could be used as a qualitative indicator of mycotoxins present in various food categories, it is not possible to use them for a quantitative estimation of dietary exposures for the following reasons:

4. Because of the way the TDS is done it can lead to high LOQs which significantly influence the UB values, and consequently the exposure assessment. A multi-mycotoxin method and approach was used in the analysis for the various food groups, which is normally a screening technique rather than a sensitive quantitative analytical method. This is reflected in generally poor recoveries for T2 and HT2. Also, the analysis of the TDS samples involved a wide range of food matrices (some of which have not been routinely examined previously) and so existing validated methods were adapted/extended to some of the new matrices and this may have also impacted on recovery for T2 and HT2. Recoveries ranged from 13 - 140% for T2 and 19 - 100% for HT2. For T2 the LOD ranged from 0.10 – 0.78 µg/kg. The LOQ ranged from 3.58 – 38.9 µg/kg. The LOD for HT2 ranged from 1.00 – 5.39 µg/kg and the LOQ from 4.98 – 26.9 µg/kg. Poor recoveries and higher LOQs/LODs when these are corrected for recovery, led to artificially inflated occurrence levels in some cases.

5. UB exposure estimates resulted in a considerable overestimation of potential exposure. This is not an unfamiliar situation and is routinely encountered in cases where the occurrence data are left-censored. Recently EFSA have published their updated exposure assessment for T2 and HT2. The same problem was documented in their analysis and they have reported that UB estimations were on average fourfold higher than LB estimations.

6. For these reasons, it is not possible to use the T2, HT2 and NEO occurrence data from the TDS for a quantitative estimation of dietary exposure. An exposure assessment cannot be based solely on the calculated UB levels from the sum of LOQs.
7. The COT recently published a statement on T2, HT2 and NEO\(^4\) in the diet of infants and young children aged 1 to 5 years, and in order to carry out acute and chronic exposure assessments, used data from a retail survey of oat-based products (FSA, 2015). Low levels of T2 and HT2 were detected in various products. The FSA survey was commissioned following initial results from the 2014 harvest that showed high levels of T2 and HT2 in oat grains. So, the retail survey was commissioned to estimate exposures in an atypically high exposure scenario. NEO had not been detected in any of the samples analysed in retail surveys, therefore it was not possible to carry out an exposure assessment for this mycotoxin.

8. Acute exposures were below the ARfD and were therefore not of toxicological concern. Mean chronic exposures were below the TDI and were 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 T2 and HT2 in oat grains were particularly high, for a prolonged period. In most years, levels of T2 and HT2 will be much lower than those observed in this harvest. It is therefore unlikely that dietary exposure levels of T2, HT2 or NEO would be of any toxicological concern (COT, 2018b).

Discussion

1. This is the first TDS commissioned by the FSA to consider dietary exposure to mycotoxins. Previous studies have looked at heavy metals/elements and the process contaminant acrylamide. These were also included in the present study with the findings being published separately.

2. Where possible fully established UKAS accredited analytical methods were used to test for mycotoxins. However, these were not suitable for all the food matrices encountered in the TDS. The analytical methods were adapted where necessary, but poor recoveries were reported in some cases e.g. a method for analysing ochratoxin A (OTA) in grape juice or wine was used to analyse a mixed sample of fruit and vegetable juices. The method recovery for OTA in this sample was low at just 32%.

3. A low recovery result can affect the estimation of dietary exposure because of the recovery correction required to determine the analytical result. A large correction can inflate the calculated LOD or LOQ and increase the upper bound value leading to an overestimation of dietary exposure. This is a problem for left censored data where there are few numerical values in the dataset. The occurrence level dataset for this survey had a high proportion of left censored data.

4. Furthermore, some batched samples included very different food matrices which meant that averaging the batch recovery to reduce the correction required was not a suitable approach, because the sensitivity of the analytical method varied with the different food matrices in the batch.

5. The analytical methods were used to detect more than one mycotoxin analyte in a sample. This multi mycotoxin qualitative approach is more readily suitable for rapid screening than the quantification of occurrence levels.

6. To conclude the largely left censored dataset was not readily suitable for estimating dietary exposure. The analytical methods were not sensitive enough or sufficiently standardised and validated for some of the food matrices encountered in the TDS.

7. However, with the caveat to treat the dietary exposure estimates with caution, since they are likely to be an overestimation of exposure they can serve as useful indicators for comparative purposes. For example, where the exposure range estimate was below the health based guidance value it can be concluded that there is unlikely to be a health concern. Conversely, where the exposure range exceeded the guidance value a conclusion cannot be inferred.

8. Three mycotoxins within the scope of the TDS are being re-evaluated by the Committee on Toxicity of Chemicals in Food, Consumer Products and the Environment (COT) and are not included in the risk assessment: fusarenon-X; diacetoxyisoumenol and cyclopiazonic acid. Once the revised toxicological evaluations are completed they will be published on the COT website.

9. Of the twelve mycotoxins included in the risk assessment ten did not represent a health concern. These were: OTA, the ergot alkaloids, fumonisins, moniliformin, patulin, sterigmatocystin, zearalenone, DON, nivalenol and T2, HT2, (NEO) toxins. The estimated dietary exposures were below health based guidance values. For; aflatoxin (B1, B2, G1, G2) and citrinin there is some uncertainty about the risk to health, although it should be noted that the exposure estimates were conservative and should be considered as only indicative of dietary exposure.

10. For aflatoxin (B1, B2, G1, G2) the MOEs calculated for the individual toxins were very low, particularly for the infants and young children age group. The MOE is a ratio which allows comparison of exposure levels in humans with dose levels where adverse effects are observed in animal studies.

11. The health based guidance value for citrinin is based on toxic effects to the kidney. There is a lack of available data on genotoxic or carcinogenic effects. Therefore, although estimated exposures were below the 0.2 μg/kg bw/day set by EFSA for toxic effects to the kidney, adverse health effects such as carcinogenicity cannot be discounted. It is advisable that levels in food are kept as low as reasonably achievable (ALARA).

**Conclusion**

1. Of the twelve mycotoxins included in the risk assessment ten did not represent a health concern. These were: OTA, the ergot alkaloids, fumonisins, moniliformin, patulin, sterigmatocystin, zearalenone, DON (3 - AC DON, 15 - Ac DON), nivalenol, T2, HT2, (NEO) toxin. For these mycotoxins the estimated dietary exposures were below health based guidance values. However, for aflatoxins (B1, B2, G1, G2) and citrinin there remains some uncertainty about the risk to health. For citrinin genotoxicity and carcinogenicity cannot be excluded because of a lack of available data. Although the exposures are likely to be overestimates for aflatoxin (B1, B2, G1, G2) the MOEs calculated for the individual toxins were very low, particularly for the infants and young children age group.

\[5\](https://cot.food.gov.uk/)
Summary of units
Microgram (μg): one thousandth of a milligram (mg)
Milligram (mg): one thousandth of a gram
Kilogram (kg): one thousand grams
Micrograms per kilogram (μg/kg)
Micrograms per kilogram body weight per day (μg/kg bw/day)

Glossary
Limit of detection (LoD)
The lowest concentration at which the analyte can be reliably detected by a particular measurement procedure.

Limit of quantification (LoQ)
The lowest concentration of an analyte that can be determined with acceptable precision and accuracy under the stated conditions of the test.

Lower bound exposure (LB)
The measure of exposure based on a concentration where the analytical result is below the limit of detection and is assumed to have a value of zero.

Upper bound exposure (UB)
The measure of exposure based on a concentration where the analytical result is below the limit of detection and is assumed to have a value equal to the limit of detection or where the analytical result is above the limit of detection but below the limit of quantification is assumed to have a value equal to the limit of quantification.

Upper Level (UL)
The upper level is not a recommended intake level but is the maximum level that can be consumed daily with no appreciable adverse effects to human health.

Health based guidance value (HBGV)
A value derived by dividing a point of departure (a no observed-adverse-effect level (NOAEL), benchmark dose (BMD) or benchmark dose lower confidence limit (BMDL)) by an uncertainty factor to determine a level that can be ingested over a defined time period with no appreciable risk to human health.

Benchmark dose (BMD)
A dose or concentration of a substance that produces a predetermined change in the response rate of an adverse effect. This predetermined change in response is called the benchmark response (BMR). Normally the BMR is for a 5 or 10% change relative to the control group response.
Benchmark dose lower confidence limit (BMDL)

The lower boundary of the confidence interval on the benchmark dose. The BMDL accounts for
the uncertainty in the estimate of the dose–response, due to characteristics of the experimental
design. The BMDL can be used as the point of departure for derivation of a health-based guidance
value or a margin of exposure.

Margin of exposure (MOE)

Ratio of the no observed adverse effect level (NOAEL) or benchmark dose lower
confidence limit (BMDL) for the critical effect to the estimated exposure concentration.

No-observed-adverse-effect level (NOAEL)

The highest concentration of a substance, found by experiment or observation, that causes no
adverse effects on morphology, functional capacity, growth, development or lifespan of the target
organism compared to those of the control organisms.
References


44. EFSA (2013). Scientific Opinion on risks for animal and public health related to the presence of nivalenol in food and feed. Available at: https://www.efsa.europa.eu/en/efsajournal/pub/3262


## Annex A

Exposure tables for mycotoxins measured in the TDS.

### Table 1. TDS Total Dietary Exposure to Aflatoxins and Ochratoxins by UK Population

<table>
<thead>
<tr>
<th>Mycotoxin</th>
<th>4 to 18 month olds</th>
<th>Toddlers (1½ to 3 year-olds)</th>
<th>4 to 6 year-olds</th>
<th>7 to 10 year Olds</th>
<th>11 to 18 year Olds</th>
<th>Adults 19+ year olds</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
</tr>
<tr>
<td>Aflatoxin B1</td>
<td>0.000-0.002</td>
<td>0.000-0.007</td>
<td>0.000-0.012</td>
<td>0.000-0.011</td>
<td>0.000-0.010</td>
<td>0.000-0.009</td>
</tr>
<tr>
<td>Aflatoxin B2</td>
<td>0.000-0.006</td>
<td>0.000-0.004</td>
<td>0.000-0.009</td>
<td>0.000-0.009</td>
<td>0.000-0.007</td>
<td>0.000-0.006</td>
</tr>
<tr>
<td>Aflatoxin G1</td>
<td>0.000-0.002</td>
<td>0.000-0.007</td>
<td>0.000-0.011</td>
<td>0.000-0.011</td>
<td>0.000-0.009</td>
<td>0.000-0.006</td>
</tr>
<tr>
<td>Aflatoxin G2</td>
<td>0.000-0.009</td>
<td>0.000-0.017</td>
<td>0.000-0.017</td>
<td>0.000-0.014</td>
<td>0.000-0.016</td>
<td>0.000-0.011</td>
</tr>
<tr>
<td>Aflatoxin M1</td>
<td>0.000-0.002</td>
<td>0.000-0.017</td>
<td>0.000-0.001</td>
<td>0.000-0.001</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
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<tr>
<td>Ochratoxin A</td>
<td>0.005-0.006</td>
<td>0.044-0.047</td>
<td>0.124-0.126</td>
<td>0.112-0.118</td>
<td>0.076-0.077</td>
<td>0.045-0.046</td>
</tr>
</tbody>
</table>

NB: When a value was described as x <LOQ> y, x was used for the LB and y for the UB
<table>
<thead>
<tr>
<th>Mycotoxin</th>
<th>4 to 18 month olds</th>
<th>Toddlers (1½ to 3 year-olds)</th>
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<tr>
<td></td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
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<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
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<tr>
<td></td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
</tr>
<tr>
<td>Citrinin</td>
<td>0.000-0.010</td>
<td>0.000-0.030</td>
<td>0.000-0.019</td>
<td>0.000-0.043</td>
<td>0.000-0.018</td>
<td>0.000-0.042</td>
</tr>
<tr>
<td>Cyclopiazonic Acid</td>
<td>0.001-0.005</td>
<td>0.005-0.015</td>
<td>0.002-0.009</td>
<td>0.007-0.022</td>
<td>0.002-0.009</td>
<td>0.007-0.020</td>
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<tr>
<td>Moniliformin</td>
<td>0.000-0.084</td>
<td>0.000-0.237</td>
<td>0.000-0.145</td>
<td>0.000-0.289</td>
<td>0.000-0.148</td>
<td>0.000-0.292</td>
</tr>
<tr>
<td>Patulin</td>
<td>0.000-0.122</td>
<td>0.000-0.309</td>
<td>0.000-0.165</td>
<td>0.000-0.308</td>
<td>0.000-0.159</td>
<td>0.000-0.286</td>
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<tr>
<td>Sterigmatocystin</td>
<td>0.000-0.002</td>
<td>0.001-0.008</td>
<td>0.000-0.005</td>
<td>0.001-0.011</td>
<td>0.000-0.004</td>
<td>0.002-0.010</td>
</tr>
<tr>
<td>Zearalenone</td>
<td>0.002-0.008</td>
<td>0.028-0.038</td>
<td>0.007-0.015</td>
<td>0.042-0.053</td>
<td>0.010-0.018</td>
<td>0.050-0.060</td>
</tr>
</tbody>
</table>

NB: When a value was described as x <LOQ> y, x was used for the LB and y for the UB
Table 3. TDS Total Dietary Acute Exposure to 3-Acetyldeoxynivalenol, 15-Acetyldeoxynivalenol, Deoxynivalenol, HT2 toxin, Nivalenol and T2 toxin by UK Population.

<table>
<thead>
<tr>
<th>Mycotoxin</th>
<th>4 to 18 month olds</th>
<th>Toddlers (1½ to 3 year-olds)</th>
<th>4 to 6 year-olds</th>
<th>7 to 10 year Olds</th>
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<th>Adults 19+ year olds</th>
</tr>
</thead>
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<tr>
<td></td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
</tr>
<tr>
<td></td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
</tr>
<tr>
<td>3-Acetyl-Deoxynivalenol</td>
<td>0.001-0.034</td>
<td>0.003-0.086</td>
<td>0.001-0.042</td>
<td>0.003-0.087</td>
<td>0.001-0.041</td>
<td>0.003-0.077</td>
</tr>
<tr>
<td>15-Acetyl-Deoxynivalenol</td>
<td>0.189</td>
<td>0.500</td>
<td>0.243</td>
<td>0.512</td>
<td>0.228</td>
<td>0.458</td>
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<tr>
<td>Deoxynivalenol</td>
<td>0.381-0.401</td>
<td>1.124-1.156</td>
<td>0.554-0.584</td>
<td>1.324-1.351</td>
<td>0.544-0.577</td>
<td>1.167-1.180</td>
</tr>
<tr>
<td>HT2_Toxin</td>
<td>0.017-0.061</td>
<td>0.058-0.190</td>
<td>0.022-0.080</td>
<td>0.057-0.205</td>
<td>0.018-0.066</td>
<td>0.044-0.146</td>
</tr>
<tr>
<td>Nivalenol</td>
<td>0.420</td>
<td>1.161</td>
<td>0.467</td>
<td>0.959</td>
<td>0.459</td>
<td>0.910</td>
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<tr>
<td>T2_Toxin</td>
<td>0.007-0.080</td>
<td>0.023-0.232</td>
<td>0.009-0.097</td>
<td>0.026-0.226</td>
<td>0.007-0.082</td>
<td>0.018-0.166</td>
</tr>
</tbody>
</table>

NB: When a value was described as x <LOQ> y, x was used for the LB and y for the UB
<table>
<thead>
<tr>
<th>Mycotoxin</th>
<th>4 to 18 month olds</th>
<th>Toddlers (1½ to 3 year-olds)</th>
<th>4 to 6 year-olds</th>
<th>7 to 10 year Olds</th>
<th>11 to 18 year Olds</th>
<th>Adults 19+ year olds</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
</tr>
<tr>
<td></td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
</tr>
<tr>
<td>3-Acetyl-Deoxynivalenol</td>
<td>0.000-0.021</td>
<td>0.001-0.056</td>
<td>0.001-0.029</td>
<td>0.002-0.058</td>
<td>0.000-0.029</td>
<td>0.002-0.053</td>
</tr>
<tr>
<td>15-Acetyl-Deoxynivalenol</td>
<td>0.000-0.121</td>
<td>0.000-0.318</td>
<td>0.000-0.165</td>
<td>0.000-0.321</td>
<td>0.000-0.161</td>
<td>0.000-0.300</td>
</tr>
<tr>
<td>Deoxynivalenol</td>
<td>0.228-0.246</td>
<td>0.682-0.722</td>
<td>0.350-0.377</td>
<td>0.788-0.838</td>
<td>0.357-0.387</td>
<td>0.686-0.724</td>
</tr>
<tr>
<td>Diacetoxyscirpenol</td>
<td>0.001-0.030</td>
<td>0.004-0.100</td>
<td>0.001-0.036</td>
<td>0.004-0.092</td>
<td>0.001-0.032</td>
<td>0.003-0.075</td>
</tr>
<tr>
<td>Fusarenon_X</td>
<td>0.001-0.014</td>
<td>0.004-0.037</td>
<td>0.001-0.019</td>
<td>0.005-0.041</td>
<td>0.001-0.019</td>
<td>0.005-0.038</td>
</tr>
<tr>
<td>HT2_Toxin</td>
<td>0.009-0.035</td>
<td>0.031-0.105</td>
<td>0.012-0.048</td>
<td>0.034-0.111</td>
<td>0.010-0.042</td>
<td>0.023-0.079</td>
</tr>
<tr>
<td>Neosolaniol</td>
<td>0.000-0.007</td>
<td>0.001-0.019</td>
<td>0.000-0.010</td>
<td>0.001-0.019</td>
<td>0.000-0.010</td>
<td>0.001-0.019</td>
</tr>
<tr>
<td>Nivalenol</td>
<td>0.000-0.246</td>
<td>0.000-0.695</td>
<td>0.000-0.312</td>
<td>0.000-0.620</td>
<td>0.000-0.312</td>
<td>0.000-0.591</td>
</tr>
<tr>
<td>T2_Toxin</td>
<td>0.004-0.047</td>
<td>0.012-0.133</td>
<td>0.005-0.061</td>
<td>0.012-0.130</td>
<td>0.004-0.055</td>
<td>0.009-0.102</td>
</tr>
</tbody>
</table>

NB: When a value was described as x <LOQ> y, x was used for the LB and y for the UB
UK TDS exposure to total ergot alkaloids from sandwiches

Total ergot alkaloids were detected at a level of 14 µg/kg in the sandwiches group. The NDNS rolling programme does not record consumption of sandwiches. A portion size approach was used for the estimates of UK TDS exposure to total ergot alkaloids from sandwiches. The portions that have been used for adults in the assessment were deduced from the FSA portion size book. The average weight of 1 round of sandwiches (i.e. 2 slices of medium sliced bread with fillings), based on eight popular sandwiches (six made with slices of bread and 2 made with baps/rolls) is 179 g (see Table 5).

Table 5: Average weight of popular sandwiches and baps with fillings

<table>
<thead>
<tr>
<th>1 round of sandwiches comprising 2 medium slices of bread and fillings</th>
<th>Weight (g)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beef, roast &amp; salad</td>
<td>165</td>
</tr>
<tr>
<td>Cheese &amp; pickle</td>
<td>185</td>
</tr>
<tr>
<td>Chicken, roast &amp; salad</td>
<td>205</td>
</tr>
<tr>
<td>Egg, mayonnaise &amp; cress</td>
<td>145</td>
</tr>
<tr>
<td>Ham, cheese &amp; pickle</td>
<td>180</td>
</tr>
<tr>
<td>Tuna mayonnaise</td>
<td>165</td>
</tr>
<tr>
<td><strong>Baps with fillings</strong></td>
<td></td>
</tr>
<tr>
<td>Cheese &amp; pickle</td>
<td>195</td>
</tr>
<tr>
<td>Chicken salad</td>
<td>190</td>
</tr>
<tr>
<td><strong>Average weight of sandwiches and baps (g)</strong></td>
<td>178.8</td>
</tr>
</tbody>
</table>

[Source: FSA portion size book (2002)]

The TDS purchasing instructions for food items comprising the sandwiches food category include:
- White bread or rolls;
- Brown bread or rolls,
- Unspecified sandwiches or rolls.

Tables 6 to 10 present exposure assessments based on the average weights of popular sandwiches and baps (Table 5) with fillings:
Table 6: Consumption of 1 round of sandwiches or 1 bap with fillings and exposure to total ergot alkaloids

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Average consumption (g/kg bw/day)</th>
<th>Average total ergot alkaloids exposure (µg/kg bw/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults (19+ yr. olds)*</td>
<td>2.30</td>
<td>0.032</td>
</tr>
<tr>
<td>Toddlers (1.5 to 3 yr. olds)**</td>
<td>12.18</td>
<td>0.17</td>
</tr>
</tbody>
</table>

Table 7: Consumption of 2 rounds of sandwiches or 2 baps with fillings and exposure to total ergot alkaloids

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Average consumption (g/kg bw/day)</th>
<th>Average total ergot alkaloids exposure (µg/kg bw/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults (19+ yr. olds)*</td>
<td>4.60</td>
<td>0.064</td>
</tr>
<tr>
<td>Toddlers (1.5 to 3 yr. olds)**</td>
<td>24.35</td>
<td>0.34</td>
</tr>
</tbody>
</table>

Table 8: Consumption of 3 rounds of sandwiches or 3 baps with fillings and exposure to total ergot alkaloids

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Consumption (g/kg bw/day)</th>
<th>Total ergot alkaloids exposure (µg/kg bw/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults (19+ yr. olds)*</td>
<td>6.90</td>
<td>0.097</td>
</tr>
<tr>
<td>Toddlers (1.5 to 3 yr. olds)**</td>
<td>36.53</td>
<td>0.51</td>
</tr>
</tbody>
</table>

Table 9: Consumption of 4 rounds of sandwiches or 4 baps with fillings and exposure to a total ergot alkaloids

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Consumption (g/kg bw/day)</th>
<th>Total ergot alkaloids exposure (µg/kg bw/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults (19+ yr. olds)*</td>
<td>9.20</td>
<td>0.13</td>
</tr>
<tr>
<td>Toddlers (1.5 to 3 yr. olds)**</td>
<td>48.71</td>
<td>0.68</td>
</tr>
</tbody>
</table>
Table 10: Consumption of greater - 6 rounds of sandwiches or 6 baps with fillings and exposure to total ergot alkaloids

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Consumption (g/kg bw/day)</th>
<th>Total ergot alkaloids exposure (µg/kg bw/day)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adults (19+ yr. olds)*</td>
<td>13.80</td>
<td>0.19</td>
</tr>
<tr>
<td>Toddlers (1.5 to 3 yr. olds)**</td>
<td>n/a</td>
<td>n/a</td>
</tr>
</tbody>
</table>

* Average bodyweight of adults (19+ year-olds) recorded in years 1 to 6 of NDNS rolling programme = 77.8 kg (Bates et al., 2016)

** Average body weight of toddlers (1.5 to 3-year olds) recorded in years 1 to 6 of NDNS rolling programme = 14.7 kg (Bates et al., 2016)
### Table 11. TDS Total Ergot Alkaloids Chronic Dietary Exposure by UK Population

<table>
<thead>
<tr>
<th>Food Groups</th>
<th>4 to 18 month olds</th>
<th>Toddlers (1½ to 3 year-olds)</th>
<th>4 to 6 year-olds</th>
<th>7 to 10 year Olds</th>
<th>11 to 18 year Olds</th>
<th>Adults 19+ year olds</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
</tr>
<tr>
<td></td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
</tr>
<tr>
<td>Bread - White sliced</td>
<td>0011-0001</td>
<td>0072-0072</td>
<td>0.019-0.019</td>
<td>0.078-0.078</td>
<td>0.005-0.005</td>
<td>0.026-0.027</td>
</tr>
<tr>
<td></td>
<td>0001-0001</td>
<td>0017-0017</td>
<td>0.003-0.003</td>
<td>0.024-0.025</td>
<td>0.033-0.033</td>
<td>0.144-0.144</td>
</tr>
<tr>
<td>Bread - Brown</td>
<td>0001-0001</td>
<td>0011-0011</td>
<td>0.003-0.003</td>
<td>0.043-0.043</td>
<td>0.002-0.002</td>
<td>0.022-0.022</td>
</tr>
<tr>
<td>Bread - Wholemeal and granary</td>
<td>0023-0023</td>
<td>0131-0131</td>
<td>0.034-0.034</td>
<td>0.160-0.160</td>
<td>0.033-0.033</td>
<td>0.144-0.144</td>
</tr>
<tr>
<td>Bread - Other bread</td>
<td>0007-0007</td>
<td>0054-0054</td>
<td>0.010-0.010</td>
<td>0.057-0.057</td>
<td>0.014-0.014</td>
<td>0.079-0.079</td>
</tr>
<tr>
<td>Misc cereals - Flour</td>
<td>0001-0001</td>
<td>0005-0006</td>
<td>0.001-0.001</td>
<td>0.012-0.013</td>
<td>0.001-0.001</td>
<td>0.010-0.011</td>
</tr>
<tr>
<td>---------------------</td>
<td>------------</td>
<td>------------</td>
<td>-------------</td>
<td>-------------</td>
<td>-------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Misc cereals - Buns, cakes and pastries</td>
<td>0001-0001</td>
<td>0009-0012</td>
<td>0.002-0.003</td>
<td>0.011-0.015</td>
<td>0.003-0.004</td>
<td>0.012-0.017</td>
</tr>
<tr>
<td>Misc cereals - Savoury biscuits</td>
<td>0001-0001</td>
<td>0008-0008</td>
<td>0.002-0.002</td>
<td>0.013-0.013</td>
<td>0.001-0.001</td>
<td>0.009-0.009</td>
</tr>
<tr>
<td>Misc cereals - Sweet biscuits</td>
<td>0001-0001</td>
<td>0010-0010</td>
<td>0.003-0.003</td>
<td>0.012-0.013</td>
<td>0.002-0.002</td>
<td>0.011-0.011</td>
</tr>
<tr>
<td>Misc cereals - Chocolate biscuits</td>
<td>0000-0000</td>
<td>0003-0003</td>
<td>0.001-0.001</td>
<td>0.004-0.004</td>
<td>0.001-0.001</td>
<td>0.005-0.006</td>
</tr>
<tr>
<td>Misc cereals - Breakfast cereals</td>
<td>0011-0012</td>
<td>0052-0052</td>
<td>0.012-0.012</td>
<td>0.045-0.046</td>
<td>0.010-0.010</td>
<td>0.036-0.036</td>
</tr>
<tr>
<td>Misc cereals - Rice</td>
<td>0000-0003</td>
<td>0000-0027</td>
<td>0.000-0.003</td>
<td>0.000-0.003</td>
<td>0.000-0.003</td>
<td>0.000-0.003</td>
</tr>
<tr>
<td></td>
<td>0001-0001</td>
<td>0006-0011</td>
<td>0.001-0.012</td>
<td>0.007-0.016</td>
<td>0.001-0.016</td>
<td>0.007-0.012</td>
</tr>
<tr>
<td>----------------------</td>
<td>-----------</td>
<td>-----------</td>
<td>-------------</td>
<td>-------------</td>
<td>-------------</td>
<td>-------------</td>
</tr>
<tr>
<td>Misc cereals -</td>
<td>0002-0006</td>
<td>0012-0035</td>
<td>0.003-0.008</td>
<td>0.011-0.031</td>
<td>0.002-0.005</td>
<td>0.006-0.019</td>
</tr>
<tr>
<td>Other cereal</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>products</td>
<td>0001-0001</td>
<td>0014-0014</td>
<td>0.003-0.003</td>
<td>0.021-0.022</td>
<td>0.004-0.005</td>
<td>0.025-0.026</td>
</tr>
<tr>
<td>Misc cereals -</td>
<td>0000-0000</td>
<td>0000-0000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
</tr>
<tr>
<td>Pasta</td>
<td>0000-0000</td>
<td>0000-0000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
</tr>
<tr>
<td>Alternatives to milk</td>
<td>0000-0000</td>
<td>0000-0000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
</tr>
<tr>
<td>Snacks (not potato</td>
<td>0000-0000</td>
<td>0000-0000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
</tr>
<tr>
<td>based)</td>
<td>0000-0000</td>
<td>0000-0000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
</tr>
<tr>
<td>Beer</td>
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<td>0000-0000</td>
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<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
</tr>
<tr>
<td>Cider</td>
<td>0000-0000</td>
<td>0000-0000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
<td>0.000-0.000</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td><strong>0.062-0.070</strong></td>
<td><strong>0.193-0.203</strong></td>
<td><strong>0.095-0.106</strong></td>
<td><strong>0.225-0.239</strong></td>
<td><strong>0.097-0.106</strong></td>
<td><strong>0.201-0.206</strong></td>
</tr>
</tbody>
</table>

**NB:** When a value was described as x <LOQ> y, x was used for the LB and y for the UB
Table 12. TDS Total Dietary Exposure to Fumonisin by UK Population

<table>
<thead>
<tr>
<th>Mycotoxin</th>
<th>4 to 18 month olds</th>
<th>Toddlers (1½ to 3 year-olds)</th>
<th>4 to 6 year-olds</th>
<th>7 to 10 year Olds</th>
<th>11 to 18 year Olds</th>
<th>Adults 19+ year olds</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
<td>µg/kg bw/d</td>
</tr>
<tr>
<td></td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
<td>Mean LB-UB</td>
<td>97.5 percentile LB-UB</td>
</tr>
<tr>
<td>Fumonisin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B1</td>
<td>0.000-</td>
<td>0.001-</td>
<td>0.000-</td>
<td>0.002-</td>
<td>0.002-</td>
<td>0.001-</td>
</tr>
<tr>
<td></td>
<td>0.057</td>
<td>0.148</td>
<td>0.081</td>
<td>0.152</td>
<td>0.079</td>
<td>0.152</td>
</tr>
<tr>
<td>Fumonisin</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B2</td>
<td>0.000-</td>
<td>0.000-</td>
<td>0.000-</td>
<td>0.000-</td>
<td>0.000-</td>
<td>0.000-</td>
</tr>
<tr>
<td></td>
<td>0.053</td>
<td>0.141</td>
<td>0.077</td>
<td>0.146</td>
<td>0.076</td>
<td>0.143</td>
</tr>
<tr>
<td>Fumonisin</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>B3</td>
<td>0.000-</td>
<td>0.000-</td>
<td>0.000-</td>
<td>0.000-</td>
<td>0.000-</td>
<td>0.000-</td>
</tr>
<tr>
<td></td>
<td>0.046</td>
<td>0.123</td>
<td>0.066</td>
<td>0.124</td>
<td>0.065</td>
<td>0.127</td>
</tr>
</tbody>
</table>

NB: When a value was described as x <LOQ> y, x was used for the LB and y for the UB
References:

