Ministry for Primary Industries
Manatū Ahu Matua

The New Zealand Mycotoxin Surveillance Program 06-14 Report Series

FW14019 Dietary exposure to ochratoxin A and trichothecene mycotoxins: Risk estimates and proportionality of exposure source.

MPI Technical Report – Paper No: 2016/29

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ISBN No: 978-1-77665-297-6 (online)
ISSN No: 2253-3923 (online)

July 2014

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Scientific Interpretive Summary

This SIS is prepared by MPI risk assessors to provide context to the following report for MPI risk managers and external readers

The New Zealand Mycotoxin Surveillance Program 06-14 Report Series

FW14019 Dietary exposure to ochratoxin A and trichothecene mycotoxins: Risk estimates and proportionality of exposure source.

These reports are the outputs of MPIs ongoing mycotoxin surveillance programme. The nine reports form a series detailing the research undertaken over the last eight years to characterise and quantify the risk to the New Zealand public through the presence of mycotoxins in the food supply.

The nine reports are:

- Risk Profile: Mycotoxin in Foods 2006
- Aflatoxins in Maize Products 2008
- Aflatoxins and Ochratoxin A in Dried Fruits and Spices 2009
- Aflatoxins in Nuts and Nut Products 2010
- Dietary Exposure to Aflatoxins 2011
- Ochratoxin A in Cereal Products, Wine, Beer and Coffee 2011
- Trichothecene Mycotoxins in Cereal Products 2014
- Dietary Exposure to Ochratoxin A and Trichothecene Mycotoxins 2014
- Risk Profile: Mycotoxin in Foods 2014

<u>Dietary Exposure to Ochratoxin A and Trichothecene Mycotoxins 2014</u>

Exposure assessment of Trichothecene levels and Ochratoxin A levels, which had been surveyed in 2 previous reports, were undertaken for adults and children.

The methodology of the assessment is highly detailed and very robust.

Total exposures for Ochratoxin A and the surveyed Trichothecenes fell well below the TDIs for the respective mycotoxins, the highest was to 5-6 year olds and constituted below 25% of the TDI for DON and NIV.

Acute exposure assessment indicated a probability of 0.003% of a child exceeding the acute reference dose in a single sitting and a negligible probability for adults.

New Zealand dietary exposure to Trichothecenes and Ochratoxin A is on the lower end of the range seen internationally and does not constitute a public health concern.

DIETARY EXPOSURE TO OCHRATOXIN A AND TRICHOTHECENE MYCOTOXINS: RISK ESTIMATES AND PROPORTIONALITY OF EXPOSURE SOURCE

Client Report FW14019

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DIETARY EXPOSURE TO OCHRATOXIN A AND TRICHOTHECENE MYCOTOXINS: RISK ESTIMATES AND PROPORTIONALITY OF EXPOSURE SOURCE

Prepared for Ministry for Primary Industries
under project CFS/13/02, Dietary exposure to mycotoxins (trichothecene and ochratoxin A): Risk estimates and proportionality of exposure source,
as part of overall contract for scientific services

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Peter Cressey

July 2014

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ACKNOWLEDGEMENTS

The author wishes to acknowledge the Ministry of Health as owner of the copyright and funders of the 2008-2009 Adult Nutrition Survey and the 2002 National Children's Nutrition Survey and to thank them for access to food consumption information (24-hour dietary recall and qualitative food frequency questionnaire) from these surveys.

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SUMMARY

Mycotoxins are toxic secondary metabolites of fungi that may be present in food due to infection of crops or stored foods. The current study considered dietary exposure to ochratoxin A (OTA) and the trichothecene mycotoxins. OTA is a potent nephrotoxin, while the trichothecene mycotoxins are able to inhibit protein synthesis, resulting in a range of adverse health effects, including immunotoxicity and gastrointestinal effects.

Information on the prevalence and concentration of these mycotoxins in foods available in New Zealand was collated and combined with information on consumption of these foods and the body weights of consumers to give estimates of dietary exposure. Estimates were derived for a range of age-gender groups. Due to the high proportion of left-censored ('not detected') results for the analysis of mycotoxins in foods, exposure estimates were determined as a range (lower-upper bound).

Foods available for consumption in New Zealand are frequently contaminated with OTA and/or trichothecene mycotoxins. However, dietary exposure to these contaminants appears to be at the low end of the range seen internationally and exposures are well within health-based exposure limits.

Mean OTA exposures range from 0.8-3.2 ng/kg body weight/day for 5-6-year-old children to 0.3-1.0 ng/kg body weight/day for adult females. The corresponding 95th percentile dietary exposure estimates are 2.0-6.7 and 0.7-2.1 ng/kg body weight/day, respectively. The lowest tolerable intake is 100 ng/kg body weight/week or 14.3 ng/kg body weight/day, derived by the Joint FAO/WHO Expert Committee on Food Additives (JECFA). These 95th percentile exposure estimates are based on single-day food consumption records and use of statistical techniques, to determine the distribution of long-term usual exposures, results in even lower estimates of 95th percentile dietary exposure. These estimates suggest that current levels of exposure to OTA by New Zealanders are of low public health concern.

Exposure to OTA is mainly through consumption of cereal products, particularly bread and pasta/noodles. Coffee is also a significant contributor to dietary OTA exposure for adult consumers.

Of the trichothecene mycotoxins, only deoxynivalenol (DON) and nivalenol (NIV) were detected frequently in foods available in New Zealand. Occasional detections of T-2 toxin, 15-acetyldeoxynivalenol and diacetoxyscirpenol did not provide sufficient data for exposure assessment.

Mean exposures to DON and NIV were highest for the 5-6-year-old child group, with exposures of 76-77 and 22-24 ng/kg body weight/day, respectively. The lowest mean exposures to DON and NIV were in the adult female group (16.6-17.1 and 3.6-4.2 ng/kg body weight/day, respectively). All exposure estimates (mean and 95th percentile) were less than 25% of the respective tolerable intakes (1000 and 1200 ng/kg body weight/day for DON and NIV, respectively) and are of low public health concern.

Assessments of acute dietary exposure were also carried out for DON. For children, there was a very low probability (0.003%) of daily exposure exceeding the acute reference dose

(ARfD), while the probability of an adult exceeding the ARfD was so low that it was not measurable.

Bread and pasta/noodles were the major contributors to dietary DON exposure, with snack foods also a major contributor for children and beer a significant contributor for adult males. Dietary exposure to NIV was even more strongly dominated by the contribution from bread, with biscuits also contributing.

Quantification of uncertainty associated with the inputs to the exposure assessment, due to measurement and sampling, produced credible intervals for exposure estimates that were still well within health-based exposure limits.

1 INTRODUCTION

1.1 Mycotoxin Surveillance Programme (MSP)

The Mycotoxin Surveillance Programme (MSP) involves investigation of food safety issues associated with mycotoxins in the New Zealand food supply.

As with other activities of the Ministry for Primary Industries (MPI), activities in this area are directed on the basis of risk. A risk profile of mycotoxins in the New Zealand food supply (Cressey and Thomson, 2006) is viewed as a starting point for this process. The risk profile identified a number of issues to be investigated or clarified. Priority mycotoxin issues for New Zealand were identified, relating to aflatoxins (AF), ochratoxin A (OTA) and trichothecene mycotoxins. The work programme for AF was completed with a risk assessment (Cressey, 2011), using results from earlier survey projects (Cressey and Jones, 2008; 2009; 2010). Survey work on priority foods for OTA and the trichothecene mycotoxins has been completed (Cressey and Jones, 2009; 2011; Cressey *et al.*, 2014).

Risk assessment of OTA and the trichothecene mycotoxins will complete the initially identified mycotoxin priorities for New Zealand.

1.2 OTA

1.2.1 Hazard identification

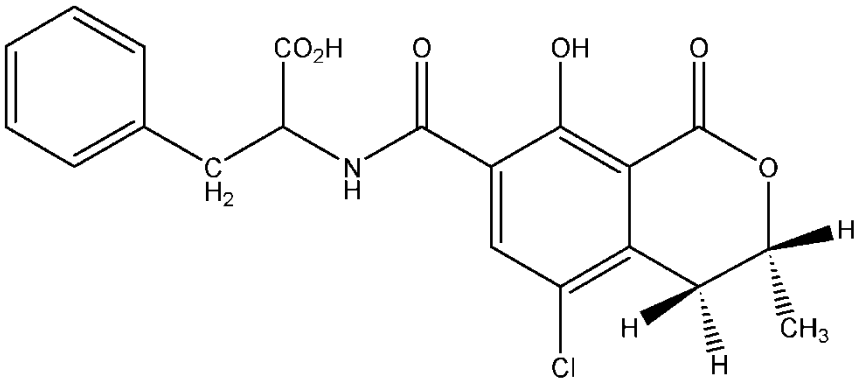
OTA has been reported to be produced by *Aspergillus ochraceus* and a related *Aspergillus* species, *A. carbonarius*, as well as some isolates of *A. niger*, and by *Penicillium verrucosum* (EFSA, 2006; Samson *et al.*, 2004). However, it has since been concluded that the species originally identified as *A. ochraceus* is actually *A. westerdijkiae* and that this species and *A. steynii* are the major contributors of OTA to the human diet (Frisvad *et al.*, 2004; JECFA, 2008). It was suggested that the name, *A. ochraceus*, is likely to persist and should be considered to include all three species (JECFA, 2008). Some strains previously classified as *P. verrucosum* have been reclassified as *P. nordicum*, but this species is considered to be a minor source of OTA in foods, compared to *P. verrucosum* (JECFA, 2008)

These organisms differ in their geographical distribution and ecological niche, in the commodities affected, and the point at which they are likely to infect commodities.

1.2.2 Structure and nomenclature

OTA contains a chlorinated isocoumarin moiety linked via a carboxyl group to L-phenylalanine. Ochratoxin B (a dechlorinated analogue of OTA) and ochratoxin C (an ethyl ester of OTA) have also been detected, but OTA is by far the major contaminant found in crop plants (Walker, 1999).

Figure 1: Structure of OTA



1.2.3 Occurrence

P. verrucosum is a cool climate organism (0-31˚C, optimum 20˚C), occurring only in cool temperate regions, and is able to grow at water activities as low as 0.8 (Pitt and Hocking, 1997). It is the source of OTA formation in stored cereals and cereal products in European countries and Canada. Due to the high stability of OTA this can also result in the presence of OTA in animals fed contaminated cereals. *P. verrucosum* does not occur in the tropics (JECFA, 2008).

A. ochraceus grows at moderate temperatures (8-37˚C, optimum 24-31˚C) and at water activities above 0.8 (optimum 0.95-0.99) (Pitt and Hocking, 1997). It occurs occasionally on a wide range of stored food products, but is seldom the cause of substantial concentrations of OTA (JECFA, 2008).

A. carbonarius grows at high temperatures (max 40˚C, optimum 32-35˚C) and is associated with maturing fruit, particularly grapes. It is the major source of OTA in fresh grapes, dried vine fruits and wine (JECFA, 2008).

OTA contamination is principally found in cereals, but can also occur in coffee, cocoa, nuts, dried vine fruits, grape juice and wine, beer, and pork and pork products made from animals fed OTA contaminated feed (Walker, 1999).

Sayer and Lauren (1991) did not report isolation of any of these OTA-forming fungal species from New Zealand grain sampled at harvest. However, no subsequent surveys have been conducted and no information was found on occurrence of these fungal species on stored grain in New Zealand. Similarly, no information was found on *Aspergillus* infection of grapes or other fruits in New Zealand.

1.3 Trichothecene Mycotoxins

1.3.1 Hazard identification

The trichothecene mycotoxins are a family of approximately 150 structurally-related compounds produced by fungi of the genera *Fusarium, Cephalosporium, Myrothecium,*

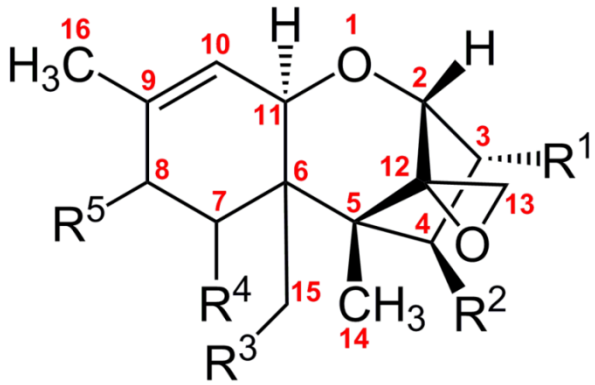
Stachybotrys, *Trichoderma* and others. Trichothecene mycotoxins of significance in food are produced by *Fusarium* species, including *F. poae, F. sporotrichioides, F. acuminatum, F. graminearum, F. culmorum, F. crookwellense, F. avenaceum* and *F. equiseti* (Council for Agriculture and Technology, 2003). The toxins in this group that have received the most attention are deoxynivalenol (DON), nivalenol (NIV), T-2 toxin (T2) and HT-2 toxin (HT2), with lesser attention paid to diacetoxyscirpenol (DAS) and other trichothecene toxins. Focus on these toxins has been due to the fact that they are the major toxins formed in foods and/or there is evidence for their involvement in human disease. Trichothecenes have been reported in cereal grain crops worldwide (Schothorst and Van Egmond, 2004).

1.3.2 Structure and nomenclature

The trichothecenes are sesquiterpenoids possessing a tetracyclic 12,13-epoxytrichothecene skeleton. They can be conveniently divided into four categories according to similarity of functional groups. The first class is characterised by a functional group other than a ketone at C-8 (type A) and include T2 and HT2, DAS and neosolaniol (NEO). The second category of trichothecenes usually has a carbonyl function at C-8 (type B), typified by DON and NIV. The third category is characterised by a second epoxide group at C-7,8 or C-9,10 (type C), and the fourth contains a macrocyclic ring system between C-4 and C-15 with two ester linkages (type D). Type C and type D trichothecenes are not normally associated with food.

Structural summaries for the A and B trichothecene mycotoxins are shown in Figure 2.

Figure 2: Structure of type A and B trichothecenes



Type A: T2 (R_1 = OH, R_2 = OAc, R_3 = OAc, R_4 = H, R_5 = $OCOCH_2CH(CH_3)_2$), HT2 (R_1 = OH, R_2 = OH, R_3 = OAc, R_4 = H, R_5 = $OCOCH_2CH(CH_3)_2$), DAS (R_1 = OH, R_2 = OAc, R_3 = Ac, R_4 = H, R_5 = H), NEO (R_1 = OH, R_2 = OAc, R_3 = OAc, R_4 = H, R_5 = OH)

Type B: DON (R_1 = OH, R_2 = H, R_3 = OH, R_4 = OH, R_5 = O), NIV (R_1 = OH, R_2 = OH, R_3 = OH, R_4 = OH, R_5 = O), 3-acetylDON (R_1 = OAc, R_2 = H, R_3 = OH, R_4 = OH, R_5 = O), 15-acetylDON (R_1 = OH, R_2 = H, R_3 = OAc, R_4 = OH, R_5 = O), DON-3-glucoside (R_1 = $OC_6H_{11}O_5$), R_2 = H, R_3 = OH, R_4 = OH, R_5 = O), Fusarenon X (R_1 = OH, R_2 = OAc, R_3 = OH, R_4 = OH, R_5 = O)

4.1.2 Occurrence

Type A trichothecenes (T2, HT2) are frequently associated with *F. tricintum, F. poae, F. sporotrichioides, F. acuminatum, F. equiseti* and *F. semitectum* (WHO, 1990). Trichothecene

formation by these fungal species has been reported in Europe and North America and occasionally in Asia, but not in Africa or Australia (Council for Agriculture and Technology, 2003). Type B trichothecenes (DON, NIV) are frequently associated with *F. graminearum* and *F. culmorum* (WHO, 1990). Trichothecene formation by these species, particularly *F. graminearum* appears to be almost universal (Council for Agriculture and Technology, 2003).

Table 1 summarises information on *Fusarium* species occurring and production of trichothecene mycotoxins in New Zealand crops.

Table 1: Trichothecene production by *Fusarium* species in New Zealand crops

Crop	Fungal species ('>' indicates order of detection frequency)	Trichothecenes detected	Study reference
Maize (Manawatu)	*F. graminearum* *F. culmorum* *F. subglutinans* *F. acuminatum*	T2, DON, DAS detected, but no details of which species produced which mycotoxins	(Hussein *et al.*, 1987)
Maize (Waikato)	*F. graminearum* >*F. semitectum* > *F. crookwellense*[1]	No analyses carried out for mycotoxins	(Sayer, 1991)
Wheat (Waikato) Wheat (East Coast) Wheat (Manawatu) Wheat (South Island) Barley (Waikato) Barley (East Coast) Barley (Manawatu) Barley (South Island) Oats (East Coast) Oats (South Island) Maize (North Island)	*F. graminearum* >*F. avenaceum, F. crookwellense, F. poae* *F. culmorum* > *F. poae* *F. graminearum, F. culmorum* > *F. avenaceum, F. crookwellense, F. poae* *F. avenaceum* > *F. poae, F. culmorum* *F. graminearum* >*F. avenaceum, F. crookwellense, F. poae* *F. avenaceum, F. culmorum, F, equiseti,*> *F. poae* *F. poae* > *F. avenaceum, F. crookwellense, F. culmorum, F. graminearum* *F. avenaceum, F. poae* > *F. culmorum, F. graminearum* *F. poae* > *F. avenaceum* *F. avenaceum* > *F. culmorum* > *F. poae* *F. graminearum* > *F. crookwellense* > *F. semitectum*	No analyses carried out for mycotoxins	(Sayer and Lauren, 1991)
Maize, wheat, barley, oats (all New Zealand)	*F. graminearum* *F. culmorum* *F. avenaceum* *F. crookwellense* *F. poae* *F. semitectum* *F. equiseti* *F. tricinctum*	NIV, DON NIV, (DAS)[2] NIV, (DAS)[2] DAS	(Lauren *et al.*, 1992)
Wheat, barley (North Island)	*F. graminearum* > *F. avenaceum* > *F. poae* > *F. crookwellense* > *F. culmorum*	No analyses carried out for mycotoxins	(Cromey *et al.*, 2001)

Crop	Fungal species ('>' indicates order of detection frequency)	Trichothecenes detected	Study reference
Wheat, barley (South Island	*F. avenaceum* > *F. culmorum* > *F. graminearum* > *F crookwellense*	No analyses carried out for mycotoxins	(Cromey *et al.*, 2001)
Maize (Manawatu)	*F. graminearum* > *F. culmorum* > *F. acuminatum, F. subglutinans*	No analyses carried out for mycotoxins	(Hussein *et al.*, 2003)

T2 = T-2 toxin, DON = deoxynivalenol, NIV = nivalenol, DAS = diacetoxyscirpenol

[1] Order given here is for field maize. In stored maize the proportion of *F. semitectum* was greater than *F. graminearum.*

[2] Detections of DAS were infrequent in comparison to detections of NIV

While there are differences between different studies, *F. graminearum* appears to be the *Fusarium* species that most commonly infects grain crops in the North Island. *F. graminearum* is associated with production of Type B trichothecenes (NIV, DON). Monds *et al.* (2005) examined the mycotoxin-producing potential of a number of *F. graminearum* isolates from New Zealand grains and found that the isolates either produced NIV or DON or neither, but rarely both NIV and DON in significant amounts.

South Island crops are more likely to be infected with *F. avenaceum*. Bosch *et al*. (1989) demonstrated significant rodent toxicity in extracts from *F. avenaceum* and found high levels of moniliformin in these extracts and a haemorrhagic factor (wortmannin) in one extract. There have been no reports of significant trichothecene production by *F. avenaceum* isolates in New Zealand. *F. avenaceum* is the most common *Fusarium* species infecting crops in Northern Europe and has been reported to produce the mycotoxins moniliformin, beauvericin and enniatins (Morrison *et al.*, 2002).

Other species common in New Zealand grain are known trichothecene producers, including *F. culmorum* (DON, NIV), *F. poae* (HT2, T2, DAS, NIV), and *F. crookwellense* (NIV, DAS).

The study of Hussein *et al*. (1987) is unique in New Zealand in reporting detection of T2. Although T2 has been looked for, it has not been detected in subsequent studies.

1.4 Current Project

While information on the prevalence and concentrations of mycotoxin contamination in foods is useful, estimation of the risks associated with mycotoxin contamination in different foods requires combination of this information with food consumption information to provide estimates of dietary exposure. The current project has three objectives:

- To estimate dietary mycotoxin (OTA and trichothecene mycotoxins) exposure in New Zealand, including estimates of the distribution of exposure;
- To compare estimates of exposure to existing health-based exposure limits; and
- To determine the proportionality of different dietary sources of mycotoxin (OTA and trichothecene mycotoxins) to the overall risk.

2 METHODS, RATIONALES AND ASSUMPTIONS

For dietary exposure to chemicals, exposure can be defined as:

$$E_i = \Sigma \frac{Q_{i,k} \, x \, C_{i,k}}{bw_i}$$

Where E_i is the exposure of individual i to some chemical at some specified point in time, $Q_{i,k}$ is the amount of food k consumed by individual i, $C_{i,k}$ is the concentration of the chemical of interest in food k consumed by individual i and bw_i is the body weight of individual i. For deterministic (point) estimates of exposure these parameters (concentration, food consumption and body weight) are represented by population averages or selected percentiles. For dietary modelling, food consumption and body weight will be represented by actual reported values for an individual on one particular day or on several days, depending on the structure of the dietary survey.

2.1 Concentration Data

2.1.1 OTA

2.1.1.1 Sources of concentration data

Recent data are available on the concentration of OTA in foods consumed in New Zealand (Cressey and Jones, 2009; 2011). In addition to these recent studies, an older study (Stanton, 2000) and some unpublished analytical results from 2007 (Darren Saunders, ESR, personal communication), were available for New Zealand foods. Where the analyses from the older and the unpublished study related to relevant foods, these data have also been used in the current study. The exception was for data related to OTA in dried fruit, where there is evidence that the OTA content of these products has decreased in the last 10-15 years, due to initiatives in producer countries. Details of the OTA survey data used in the current study are included in Appendix 1.

Analyses carried out for coffee analysed the beverage as consumed (prepared with hot water) (Cressey and Jones, 2011). However, it became apparent that the food consumption records used for the exposure assessment often reported instant coffee in terms of the dry coffee powder, separate from the added hot water. An earlier New Zealand study included analysis of 15 dry instant or soluble coffee samples, with a concentration range of 0.3-3.5 µg/kg and a mean of 1.36 µg/kg (Stanton, 2000). As this study is now quite old, reference to the scientific literature was carried out to identify other studies on the OTA content of instant coffee. Details of relevant studies are summarised in Table 2.

Table 2: Ochratoxin A (OTA) in instant coffee

Country	Sample description	Samples positive for OTA/total samples	OTA concentration range (µg/kg)	OTA mean concentration (µg/kg)	Reference
Argentina	Soluble coffee	17/22	0.22-13.66	1.99	(Vanesa and Ana, 2013)
Brazil	Instant coffee	81/82	0.17-6.29	1.24	(de Almeida *et al.*, 2007)
Canada	Instant coffee	20/30	<0.1-3.1	1.06	(Lombaert *et al.*, 2002)
Italy	Instant coffee	42/44	0.32-6.40	1.27	(Vecchio *et al.*, 2012)
Japan	Instant coffee	5/7	0.16-1.1	-	(Tabata *et al.*, 2008)
United Kingdom	Soluble retail coffee products	64/80	0.1-8.0	1.0	(MAFF, 1995)
Weighted mean				1.22[1]	

[1] The weighted mean excludes the study of Tabata *et al.*, for which no mean concentration value was reported

Given the reasonable consistency of mean values for the OTA content of instant coffee across studies and countries, the mean value of 1.36 µg/kg from the earlier New Zealand study was used as the concentration value for instant coffee in the current exposure assessment. Given the frequent detection of OTA in instant coffee in the studies summarised in Table 2, a conservative position was adopted and it was assumed that OTA would always be present in instant coffee consumed in New Zealand.

2.1.1.2 Reporting of OTA concentrations. Chemical entity reported

Unlike some other mycotoxins, both measurement of ochratoxin in food and its toxicological assessment relate to OTA only. OTB has been reported to occur at much lower concentrations than OTA in foods and appears to be considerably less toxic than OTA (EFSA, 2006).

2.1.1.3 Use of OTA concentration data in exposure assessment

Exposure to OTA is of concern due to nephrotoxicity and in laboratory animals has been shown to be dose dependent, but also dependent on the duration of exposure (EFSA, 2006). In this context, the parameter of interest is the chronic, habitual/usual level of exposure. In the absence of more detailed information, it must be assumed that individuals within the population will be exposed to the complete distribution of OTA concentrations in a particular food over time. Therefore, the most appropriate parameter of the distribution of OTA concentrations for calculation of chronic exposure is the mean or expected value. This is consistent with the conclusions of the Joint FAO/WHO Expert Committee on Food Additives (JECFA) and the European Food Safety Authority (EFSA) (EFSA, 2006; JECFA, 2008).

Where the number of data points is small, as is the case for the OTA concentration of many of the foods included in the current project, the arithmetic mean can be very sensitive to single extreme values. In order to assess the impact of this in the current context, exposure calculations were compared based on arithmetic and geometric mean concentration values.

Exposure estimates based on geometric mean concentration values were 5-8% lower than estimates based on arithmetic mean concentration values. It was concluded that these differences were not large in the context of the current exercise. Exposure estimates reported in this study are those based on arithmetic means.

2.1.2 Trichothecene mycotoxins

2.1.2.1 Sources of concentration data

While a number of studies have examined the presence of trichothecene mycotoxins in harvested grains in New Zealand (see Table 1), only three studies were identified that determined the concentration of these toxins in consumer foods:

- A study of 124 maize-containing foods for the presence of DON and NIV (and zearalenone) conducted in 1995 (Lauren and Veitch, 1996). The limits of detection for both DON and NIV were 50 µg/kg.
- A small study by a visiting student at ESR of DON and NIV in 27 grain-derived foods (Eva Kosanic, 2009, unpublished). The limits of detection were in the range 12-70 µg/kg, depending on the matrix and the toxin.
- A survey, conducted under MPI's Mycotoxin Surveillance Programme, of trichothecene mycotoxins (DON, 3ADON, 15ADON, NIV, FX, T2, HT2, DAS and NEO) in 200 samples of cereal-based foods (Cressey *et al.*, 2014). Limits of detection were in the range 0.4-21 µg/kg, depending on the toxin and the matrix.

The study of Lauren and Veitch is now rather old and the limits of detection were considerably higher than is currently achievable. The student study also achieved much higher limits of detection than those reported by Cressey *et al*. (2014). For these reasons, the data from Cressey *et al*. (2014) were used exclusively in the current exposure assessment.

The survey data used contained two subsets of data; 176 composite samples retained from the 2009 New Zealand Total Diet Study (Vannoort and Thomson, 2011) and 24 single samples obtained from Christchurch during March 2014. While the sampling regime for the two sets of data were different, inclusion or exclusion of concentration data from the additional 24 samples was found to have very little impact on estimates of dietary exposure and data from these samples were included in the current study.

Details of the trichothecene mycotoxin survey data used in the current study and previous surveys are included in Appendix 1.

2.1.2.2 Reporting of trichothecene mycotoxins concentrations. Chemical entity reported

Studies in recent years have highlighted that trichothecene mycotoxins may be extensively metabolised by the producing fungi or in plants to produced so-called masked forms of the toxins. Glucosides of the major type A trichothecenes (T2, HT2, DAS, NEO) have been reported in naturally or artificially infected cereals (De Angelis *et al.*, 2012; Lattanzio *et al.*, 2012; Nakagawa *et al.*, 2012; 2013; Veprikova *et al.*, 2012). Triol and tetraol metabolites of T2 have been reported in naturally contaminated cereal samples, although this has not been reported in any other studies (Gottschalk *et al.*, 2009).

Similarly, glucosides of NIV and fusarenon X (FX) have recently been reported, with the authors estimating that levels of the glucoside were greater than 15% of those of the parent toxin (Nakagawa *et al.*, 2011).

At this stage there is insufficient information to incorporate estimates for the contribution of these masked metabolites into exposure assessments.

By far the best characterised masked trichothecenes are metabolites of DON. The most prominent of these are the acetylated derivatives, 3-acetylDON and 15-acetylDON (3ADON and 15ADON) and a glucoside, DON-3-glucoside (DON-3-G). 3ADON and 15ADON appear to be fungal metabolites, while DON-3-G appears to be a plant metabolite and may be associated with *Fusarium* head blight (FHB) resistance in some cereal varieties (Berthiller *et al.*, 2013).

DON-3-G has been shown to release the aglycone (DON) under simulated human intestinal conditions (Berthiller *et al.*, 2011; Dall'Erta *et al.*, 2013) and in *in vivo* rat studies (Nagl *et al.*, 2012). However, other studies simulating human digestion showed little production of DON from DON-3-G (De Nijs *et al.*, 2012). An *in vivo* study in a human volunteer did not detect intact DON-3-G or 3ADON in urinary output (Warth *et al.*, 2013). However, concentrations of these metabolites were low in the test diet.

Rapid hydrolysis of 3ADON and 15ADON in rat stomach has been demonstrated (Veršilovskis *et al.*, 2012).

On the basis of currently available information, it appears prudent to assume that 3ADON, 15ADON and DON-3-G present in food samples will be converted quantitatively to DON in the human digestive tract and should be considered as part of 'total DON'. Exposure estimates will be calculated in terms of DON and total DON. This is consistent with the approach taken in a recent European assessment of dietary DON exposure.

A recent consolidation of European data concluded that 3ADON and 15ADON were detected less frequently than DON and at lower concentrations (EFSA, 2013a). On average, the contribution of 3ADON to total DON was less than 2% for lower bound estimates (not detected = zero) and 13-20% for upper bound estimates (not detected = limit of detection). 15ADON contributed 10-15% to total DON for both lower and upper bound estimates. Few data were provided on DON-3-G. However, it almost always co-occurred with DON and on average represented 5.6% of the lower bound sum of DON and DON-3-G. Table 3 summarises other literature information on the relative concentrations of the various forms of DON in foods.

Table 3: Concentrations of DON and 3ADON, 15ADON and DON-3-G in foods

Country	Food	Mean concentration (µg/kg)				Reference
		DON	3ADON	15ADON	DON-3-G	
Belgium[1]	Maize	2036	305	334	340	(De Boevre *et al.*, 2012b)
	Wheat	58.5	17	-	18	
	Oats	46	79	20	51	
	Bread	43	35	18	27	
	Cornflakes	207	38	17	25	
Belgium[1]	Fibre-enriched bread	34	14	9	34	(De Boevre *et al.*, 2012a)
	Bran-enriched bread	25	16	7	21	
	Cornflakes	44	31	10	13	
	Popcorn	49	30	26	33	
	Oatmeal	18	45	7	28	
Cameroon	Maize	171	NA	NA	16	(Abia *et al.*, 2013)
Czech Republic[1]	White flour products	125	NA	NA	15	(Malachova *et al.*, 2011)
	Mixed flour products	139	NA	NA	19	
	Breakfast cereals	189	NA	NA	35	
	Snacks	124	NA	NA	32	
	Flours	103	NA	NA	15	
Denmark[1]	Winter wheat[3]	4312	NA	NA	587	(Rasmussen *et al.*, 2012)
	Spring wheat[3]	2386	NA	NA	497	
	Triticale[3]	6908	NA	NA	1838	
	Winter wheat[4]	1536	NA	NA	191	
	Triticale[4]	737	NA	NA	109	
	Oats[4]	1241	102	NA	224	
Korea[1]	Rice	23.8	4.7	4.3	NA	(Ok *et al.*, 2011)
	Glutinous rice	18.2	0.0	2.9	NA	
	Brown rice	20.9	5.0	2.0	NA	
	Barley	23.8	2.8	3.7	NA	
	Mixed grains	43.7	4.7	18.5	NA	
	Corn	114.0	4.1	21.7	NA	
	Wheat	46.1	3.3	0.0	NA	
	Wheat flour	35.4	4.3	3.6	NA	
	Breakfast cereals	32.3	5.8	2.7	NA	
England	Oats	<10	<10	NA	NA	(Scudamore *et al.*, 2007)
		15	<10	NA	NA	
		20	<10	NA	NA	
		<10	<10	NA	NA	
		298	40	NA	NA	
		<10	<10	NA	NA	
		26	<10	NA	NA	
		1230	60	NA	NA	
		731	52	NA	NA	
		20	<10	NA	NA	
		253	16	NA	NA	
		<10	<10	NA	NA	
Germany[1]	Wheat products	57	0.57	0.90	NA	(Gottschalk *et al.*, 2009)
	Rye products	28	0.39	0.73	NA	
	Oat products	2.8	0.43	0.11	NA	

Country	Food	Mean concentration (µg/kg)				Reference
		DON	3ADON	15ADON	DON-3-G	
Italy	Cereal biscuits	132	NA	NA	12	(Suman *et al.*, 2013)
	Cocoa biscuits	90	NA	NA	<LOQ	
	Minicake	165	NA	NA	<LOQ	
	Crackers	240	NA	NA	21	
	Wholemeal crackers	310	NA	NA	30	
	Bread	289	NA	NA	19	
	Wholemeal bread	358	NA	NA	28	
Norway[2]	Barley	150 (636)	17.8 (141)	NA	67.8 (270)	(Uhlig *et al.*, 2013)
	Oats	1070 (7230)	128 (1380)	NA	252 (2580)	
	Wheat	383 (1400)	14.0 (49.5)	NA	56.4 (152)	
Serbia[1]	Wheat	260	NA	NA	44	(Škrbić *et al.*, 2011)
		108	NA	NA	<1	
		89	NA	NA	24	
		54	NA	NA	9	
		93	NA	NA	42	
		89	NA	NA	<1	
USA	Whole wheat	5.23	NA	NA	0.38	(Simsek *et al.*, 2012)

NA = not available/not reported, DON = deoxynivalenol, 3ADON = 3-acetyldeoxynivalenol, 15ADON = 15-acetyldeoxynivalenol, DON-3-G = deoxynivalenol-3-glucoside

[1] Mean of positive results

[2] Median (maximum)

[3] Artificially infected

[4] Naturally infected

Molecular weights of DON and its metabolites are:

- DON 296.3
- 3ADON 338.4
- 15ADON 338.4
- DON-3-G 458.5

The analytical method used to generate trichothecene mycotoxin concentration data for the current study was insufficiently sensitive to quantify DON-3-G or 3ADON in any of the samples analysed (Cressey *et al.*, 2014). 15ADON was only quantified in one sample of flavoured snack food. The information in Table 3 does not suggest the conjugates of DON are present in a predictable ratio to DON, even within a particular matrix and a particular geographical location. No extra adjustment was made to exposure estimates for DON to account for the probable, but undetected, presence of its conjugated forms. However, the conjugated forms were considered when interpreting the proximity of exposure estimate to health-based exposure limits, such as Tolerable Daily Intakes (TDIs).

2.1.2.3 Use of trichothecene mycotoxin concentration data in exposure assessment

Trichothecene mycotoxins have been implicated in both acute and chronic adverse health effects. The recent JECFA assessment of public health risks associated with DON derived both a Provisional Maximum Tolerable Daily Intake (PMTDI) and an Acute Reference Dose (ARfD), to allow assessment of both chronic and acute exposure estimates (JECFA, 2011). An exposure assessment for DON carried out for European countries also derived both chronic and acute estimates of exposure (EFSA, 2013a).

Due to data limitations, both JECFA and EFSA used a 24-hour day as the period of acute exposure, rather than a single consumption event. EFSA used all available food concentration data for DON and carried out the acute exposure assessment stochastically. JECFA calculated acute exposure using high percentile (97.5th percentile) daily food consumption estimates and 'a high concentration of DON'. The high DON concentrations were taken as the highest mean value from a review of occurrence data.

In the current study, arithmetic mean trichothecene mycotoxin concentrations for foods were used for chronic exposure estimates. As for OTA, the impact of using geometric, rather than arithmetic, mean concentrations was assessed. Geometric mean concentrations resulted in estimates of dietary exposure that were approximately 40% lower than the estimates derived using arithmetic means. However, arithmetic means are most commonly used for chronic exposure assessments internationally and, in this case, result in a more conservative reference point for assessing risk. All relevant New Zealand concentration data were used for acute exposure assessment, with concentration values drawn at random for each estimate of exposure.

2.1.3 Treatment of 'not detected' (left censored) data

Left censorship refers to the situation where the distribution of observed results is truncated at the left hand end due to the limitations of measurement technologies. The data set for OTA in New Zealand foods contains a high proportion of left-censored (non-detected) data. This may include both true zero and true very low concentration data.

A previous New Zealand mycotoxin (aflatoxin) exposure study applied statistical techniques to the determination of mean values for left-censored data sets (Cressey, 2011). However, these techniques (maximum likelihood, regression on order statistic, Kaplan-Meier) proved to be unsatisfactory for small data sets (less than 50 data points), with a high proportion of non-detects. These characteristics apply equally to the OTA and trichothecene mycotoxins concentration data sets.

As for the earlier aflatoxin exposure assessment, it was decided to use the WHO GEMS/Food conventions for left censored data sets (WHO GEMS/Food-Euro, 1995), specifically:

- When 60% or less of data are censored, the mean was calculated using a value of half the limit of detection for values below the limit of detection; and
- When more than 60% of data are censored two estimated of the mean are calculated; one assuming that all values less than the limit of detection are true zero values (lower bound) and one assuming that all values less than the limit of detection are true non-detects with values equal to the limit of detection (upper bound)

Adoption of these conventions means that all estimates of dietary exposure will be represented by an interval, rather than a single value.

2.2 Food Consumption Information

2.2.1 National Nutrition Survey (NNS) records

Periodic national nutrition surveys (NNSs) are carried out in New Zealand. The most recent are the 2008-2009 Adult Nutrition Survey (2009ANS) covering adult New Zealanders, aged 15 years and over (University of Otago and Ministry of Health, 2011) and the 2002 National Children's Nutrition Survey (2002CNS) covering New Zealand children aged 5-14 years (Ministry of Health, 2003).

These two surveys contain include 24-hour dietary recall records (24HDR). These include a complete listing of all foods consumed by an individual during one 24-hour period. Days of the week and time of year are randomised across the survey to avoid bias due to these factors. The 2009ANS contains 24HDR records for 4,721 respondents and the 02CNS contains 24HDR records for 3,275 respondents.

2.2.1.1 Mapping of NNS foods to mycotoxin-containing foods

The NNSs contain almost 11,000 unique food descriptors. In order to estimate the mycotoxin concentration of each of these foods it is necessary to map the foods for which mycotoxin concentrations are available to the list of unique NNS food descriptors. Three situations arise:

- The food for which mycotoxin concentration information is available is sufficiently similar to the NNS food descriptor to allow direct application of the determined mycotoxin concentration;
- The NNS food is unrelated to any food for which mycotoxin concentration information is available and is unlikely to contain the mycotoxin(s) of interest. Such foods are assumed to have a mycotoxin concentration of zero; or
- The NNS food is similar to or contains (as part of a recipe) one of the foods for which mycotoxin concentration information is available.

The bulk of the effort in mapping relates to the third situation. Appendix 2 outlines the methodology used to determine the amount of mycotoxin-containing food in a recipe, while Appendix 3 identifies the range of foods and recipes that were identified as needing to be mapped to the list of mycotoxin-containing foods.

In addition to these processes it was necessary to apply a standard set of assumptions to the mapping process. These included:

- OTA were assumed not to be present in dried fruits other than those for which OTA concentration information was available (e.g. dried apple);
- If no suitable recipe information was available, but a food was known or strongly suspected of containing a particular spice, it was assumed that the spice content of recipes was 0.5%. This figure was based on examination of a range of spice-containing recipes. While the exact recipe is a secret, it was assumed that the coating of Kentucky Fried Chicken included paprika. This assumption is based on a consensus of internet guesses and conjecture.[1]

[1] http://wiki.answers.com/Q/What_are_the_original_11_herbs_and_spices_used_in_Kentucky_Fried_Chicken#slice=4&article=What_are_the_original_11_herbs_and_spices_used_in_Kentucky_Fried_Chicken. Accessed 9 May 2014

2.3 Chronic Dietary Exposure Assessment

The food mapping was used to assign a mean mycotoxin concentration to all instances of consumption of relevant foods reported in the 24HDR components of the 2002CNS and 2009ANS. Concentration values were multiplied by the food consumption amount and summed for each NNS respondent to give 3725 (2002CNS) and 4721 (2009ANS) individual estimates of daily dietary mycotoxin exposure. Body weight information was not collected for all NNS respondents and resulting set of exposure estimates had to be 'cleaned' to remove estimates for respondents for whom no body weight information was available. The remaining exposure estimates were divided by the respondent's body weight. All calculations were carried out using Microsoft Excel.

2.3.1 Estimation of usual dietary exposure to mycotoxins

While the 24HDR records provide a very good record of the food intake and resultant exposure to mycotoxins by an individual on a particular day, this is not the same as the individual's habitual long-term (usual) food intake and may include consumption of foods rarely eaten by the individual or exclude foods commonly eaten by the individual. This will mean that any exposure estimate based on 24HDR records may not be a true representation of habitual exposure for an individual. While the mean of exposures derived in this manner are likely to be good estimates of the true mean, it is expected that the variability in dietary exposure derived from 24HDR records will be greater than the true population habitual exposure variability, as it will include both between person variability (inter-person) and within person variability (intra-person) (Dodd *et al.*, 2006; Hoffmann *et al.*, 2002; Nusser *et al.*, 1996). Between-person variability is the parameter of interest for risk assessment associated with chronic exposure, as is the case for OTA and the trichothecene mycotoxins.

For the 2009ANS and 02CNS, 24HDR dietary information was collected on a second day for approximately 15% of respondents. These duplicate days can be used to estimate intra-person variability and correct the overall estimate of exposure variability to only represent inter-person variability (Dodd *et al.*, 2006; Hoffmann *et al.*, 2002; Nusser *et al.*, 1996).

However, the correction process does not work well when the dataset contains a high proportion of zero exposure days. This was a significant issue for the earlier work on aflatoxin exposure, as aflatoxins have not been detected in dietary staples in New Zealand. Due to the presence of OTA and trichothecene mycotoxins in dietary staples, such as bread, this is less of an issue for these mycotoxins and, wherever possible, percentiles of the dietary exposure were corrected for intra-person variability using PC-SIDE software.[2]

2.4 Acute Dietary Exposure Assessment

Acute (single day) exposure estimates were only determined for DON. Estimates were made using a probabilistic simulation model, similar to that recently used for acute exposure assessment of DON in the EU (EFSA, 2013a). Acute dietary exposure assessments were carried out for all respondents in either the 2009ANS or 2002CNS, rather than subdividing into the age-gender groups used for chronic dietary exposure assessment. This approach was

[2] http://www.side.stat.iastate.edu/pc-side.php PC-SIDE software for intake distribution estimation. Iowa State University. Accessed 7 November 2013

taken so that the maximum amount of information on variation in dietary patterns was retained.

The model carried out the following steps at each iteration or cycle of the model:

- A respondent was drawn at random from the relevant NNS (2002CNS or 2009ANS);
- For each potentially DON-containing food consumed by that respondent, a DON concentration was drawn randomly from survey results for that food (Cressey *et al.*, 2014);
- The consumption amount for the food was multiplied by the selected concentration and summed to give a 24-hour exposure estimate; and
- The exposure estimate was divided by the respondent's body weight.

The model was constructed using the Excel add-in @Risk (Palisades Corporation, Ithaca, New York, USA). The model used upper bound estimates of DON concentration for food samples in which DON was not detected. The model was run for 100,000 iterations.

2.5 Quantification of Uncertainty

Inputs to the exposure assessment will have a degree of uncertainty associated with them (Cullen and Frey, 1999). In some cases techniques exist to allow quantification of this uncertainty, allowing the definition of credible intervals around output parameters of the exposure assessment. For the current exercise, three sources of uncertainty were assessed:

- Mycotoxin concentration measurement uncertainty;
- Mycotoxin survey sampling uncertainty; and
- National nutrition survey sampling uncertainty.

2.5.1 Mycotoxin measurement and sampling uncertainty

Measurement uncertainty can be viewed as made up of two components:

- A fixed uncertainty associated with 'near zero' measurements. This uncertainty is usually expressed in terms of a limit of detection.
- A variable uncertainty associated with quantifiable values. This uncertainty is usually expressed in terms of a coefficient of variation, where the uncertainty is proportional to the measured value.

These two components of uncertainty have been incorporated into a model for use in analytical chemistry (Rocke and Lorenzato, 1995). This model can be expressed as:

$$x = \mu e^{\eta} + \varepsilon \qquad (1)$$

Where x is the measured value, μ is the true value, and η and ε are the variable and near zero (fixed) analytical uncertainties. The uncertainty terms are assumed to be normally distributed with means equal to zero and variances σ_η^2 and σ_ε^2. The method coefficient of variation and the limit of detection were used to derive estimates for σ_η and σ_ε, respectively.

The uncertainty introduced by the (often) relatively small (<50) number of samples that are collected and analysed was quantified according to the method of Vannoort *et al.* (2013). This approach uses classical statistics to estimate the uncertainty in an estimate of the mean of a distribution, based on the mean and standard deviation of a sample from that population. The approach uses the Student's *t* distribution and is explained in more detail below.

For each of the foods sampled the *n* concentration values can be summarised in terms of the sample mean, μ, and the sample standard error of the mean, $s_{\bar{X}}$;

$$s_{\bar{X}} = \frac{sample\ standard\ deviation}{\sqrt{sample\ size}} = \frac{s}{\sqrt{n}} \quad (2)$$

The standard error represents the expected spread of possible values for the true mean concentration in a food type given the samples taken and the number of samples. A distribution of possible values of the mean concentration, $\bar{X}^*$, can be calculated from the *t* distribution, t^*,

$$\bar{X}^* = t^*.s_{\bar{X}} + \mu \ , \quad (3)$$

where the shape of the *t* distribution is also dependent on the sample size.

Simulation analysis (@Risk, 100,000 iterations) was used to determine the impact of measurement and sampling uncertainty on mean lower and upper bound estimates of the concentration of mycotoxins in surveyed foods. Simulation outputs were used to derive an uncertainty distribution for each mean mycotoxin concentration. The use of distributions symmetric around zero in the simulation means that occasionally iterations will generate negative estimates of the mycotoxin concentration. The simulation outputs were truncated to exclude such negative values.

The concentration uncertainty distributions derived were modelled as normal distributions, with mean equal to the deterministic mean and standard deviation equal to the standard deviation of the simulated uncertainty distribution. The uncertainty distributions for concentration values were then used to assess the impact of measurement and sampling uncertainty on uncertainty in mean and percentile estimates of dietary mycotoxin exposure. However, given the complexity of the latter model, it was only feasible to run simulations for a relative small number of iterations (n = 100). Replicate runs of 100 iterations were run for some scenarios and demonstrated that this number of iterations was sufficient to achieve convergence and stability in summary statistics of exposure.

2.5.2 Sampling uncertainty

Dietary modelling exposure estimates are based on responses provided by participants in the 2009ANS and 2002CNS. These participants represent a sample of the New Zealand population and estimates of dietary exposure to mycotoxins, based on their responses, will include uncertainty associated with this sampling process.

Sampling uncertainty in exposure estimates was quantified using a non-parametric bootstrap method (Efron and Tibshirani, 1986). For a data set of *n* samples, (x_1, x_2, ….x_n), it is possible to create B bootstrap samples, (x_1^*, x_2^*, ….x_n^*), where each x_i^* is a random sample, with replacement, from the original *n* samples. For each of the B bootstrap samples the statistic of interest (e.g. mean) can then be calculated. The distribution of the B estimates of the statistic represents the bootstrap estimate of uncertainty in that statistic.

Each of the bootstrap samples must be the same size as the original sample. Caution should be exercised in applying this method for small samples. However, the nutrition surveys contain sufficient participants and corresponding estimates of exposure that this is not an issue. While no definitive rules exist, it is generally considered that B=50-200 is sufficient to gain a good estimate of uncertainty. In the current study, 10,000 bootstrap samples were generated to ensure stability of the uncertainty estimates.

2.6 Risk Assessment

Two general approaches to risk assessment of mycotoxins are taken in the current study:

- Comparison with health-based exposure limits. Exposure estimates are compared to tolerable intakes. Risk is usually expressed in terms of the percentage of the health-based exposure limits, with lower percentages representing lower levels of risk. For acute exposure assessment, risk is expressed in terms of the probability of exceeding the health-based exposure limit (ARfD).
- Determination of margin of exposure (MoE). MoE approaches determine a ratio (MoE) between estimated exposure and a toxicological reference point or 'point of departure' (Dybing *et al.*, 2008). The selected point of departure is expressed in terms of a benchmark response (BMR) (Davis *et al.*, 2011; Dybing *et al.*, 2008). For example, a BMR of 10 would be a point on the dose response curve that equated to a 10% increase in response over background levels, the benchmark dose (BMD). The point of comparison is usually defined as the lower 95th percentile confidence limit of the BMD, the BMDL. While there is currently no standardisation of BMRs, $BMDL_{10}$ is gaining some support as a standard, as this level of excess response can be calculated with acceptable confidence from animal studies with 50 animals per dose group (EFSA, 2005; Wignall *et al.*, 2014). For consistency, the current study has used $BMDL_{10}$ benchmark doses, whenever these are available. The EFSA Scientific Committee expressed an opinion that "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 view and might be considered as a low priority for risk management actions" (EFSA, 2005).

2.6.1 OTA

2.6.1.1 Tolerable intake

JECFA reassessed OTA at their 68th meeting (JECFA, 2008). After considering a number of new toxicological studies, the committee confirmed their previous assessment of minimal renal changes in the pig, at 8 µg/kg body weight/day, as the critical effect for risk assessment. JECFA were particularly concerned with elucidating the mechanism by which OTA causes renal tumours. A number of genotoxic and non-genotoxic mechanism have been proposed and evidence supporting these was reviewed. JECFA concluded that a number of non-genotoxic mechanisms may be contributing to tumour formation. JECFA confirmed the existing Provisional Tolerable Weekly Intake (PTWI) of 100 ng/kg body weight/week.

EFSA reviewed recent toxicological information on OTA, but concluded that the information was not relevant to the overall assessment of risk and confirmed their earlier Tolerable Weekly Intake (TWI) of 120 ng/kg body weight/week (EFSA, 2010).

A Canadian risk assessment highlighted limitations in the 90-day pig study used as the basis for the JECFA PTWI and the EFSA TWI (Kuiper-Goodman *et al.*, 2010). They derived a Negligible Cancer Risk Intake (NCRI) of 4 ng/kg body weight/day (28 ng/kg body weight/week), derived from tumour incidence in rats and using an excess cancer risk level of 1:100,000. Given that tumour formation in pigs is believed to be caused through non-genotoxic mechanisms, there are some questions around the appropriateness of this NCRI and it was not used as a point of comparison in the current study.

2.6.1.2 Benchmark doses

For OTA, BMDs have been determined (Muri *et al.*, 2009a). BMDs were calculated at the 1% and 5% levels (doses causing a 1 or 5% increase in the incidence of kidney adenomas or kidney carcinomas in rats). Associated BMD_{01} or BMD_{05} and BMDL (lower 95th percentile limit of the respect BMD estimate) for these endpoints and by rat gender are shown in Table 4.

Table 4: Benchmark doses for OTA

Endpoint	Benchmark response level (%)	Gender	BMD (µg/kg body weight/day)	BMDL (µg/kg body weight/day)
Kidney adenoma	1	M	24	17
	1	F	135	79
	5	M	86	68
	5	F	198	142
Kidney carcinoma	1	M	46	43
	1	F	67	62
	5	M	53	50
	5	F	88	78

M = male, F = female, BMD = benchmark dose, BMDL = benchmark dose lower 95th percentile confidence limit

JECFA also calculated benchmark doses for OTA, based on total renal tumour incidence in rats (JECFA, 2008). Depending on the model used, BMD_{10} values were in the range 18-32 µg/kg body weight/day and $BMDL_{10}$ were in the range 15-25 µg/kg body weight/day. The lowest $BMDL_{10}$ (15 µg/kg body weight/day) was used for MoE calculations for OTA in the current study.

JECFA noted that the 'points of departure' calculated by this method were higher than the Lowest Observed Effect Level (LOEL) used to derive the PTWI (8 µg/kg body weight/day).

2.6.2 Trichothecene mycotoxins

2.6.2.1 Tolerable intake

DON

JECFA established a Provisional Maximum Tolerable Daily Intake (PMTDI) for DON of 1 µg/kg body weight/day (1000 ng/kg body weight/day), based on a no observed effect level (NOEL) of 100 µg/kg body weight/day in a 2-year mouse feeding study (JECFA, 2001a). JECFA have subsequently confirmed the PMTDI and converted it to a group PMTDI, as the Committee considered 3ADON and 15ADON to be as toxic as DON (JECFA, 2011). At that time, they concluded that there was insufficient evidence to included DON-3-G in the group PMTDI.

JECFA also established a group ARfD, for DON and its acetylated forms, of 8 µg/kg body weight, based on emesis in pigs (JECFA, 2011). Recent acute exposure assessments carried out by EFSA used the JECFA ARfD (EFSA, 2013a).

The EU Scientific Committee on Food establishing a temporary TDI (t-TDI) for DON of 1 µg/kg body weight/day (Scientific Committee on Food, 1999). This t-TDI was subsequently upgraded to a full TDI (Scientific Committee on Food, 2002). The same TDI has also been derived by other authorities (Food Safety Commission of Japan, 2010).

NIV

The EU Scientific Committee on Food noted that only lowest observed adverse effect levels (LOAELs) were available from long-term studies on NIV and used a lowest LOAEL of 0.7 mg/kg body weight/day, for growth retardation and leucopenia in mice, and a safety of 1000 to derive a temporary TDI (t-TDI) for NIV of 0.7 µg/kg body weight/day (Scientific Committee on Food, 2000). This t-TDI was subsequently confirmed (Scientific Committee on Food, 2002). EFSA considered NIV again in 2013 and established a TDI of 1.2 µg/kg body weight/day (1200 ng/kg body weight/day), based on decreases in white blood cell (WBC) counts in rats receiving NIV for 90 days (EFSA, 2013b).

A Dutch study derived a t-TDI for nivalenol of 0.7 µg/kg body weight/day (Pronk *et al.*, 2002). The Food Safety Commission of Japan derived a lower TDI of 0.4 µg/kg body weight/day by applying a safety factor of 1000 to the LOAEL from the 90 day rat study mentioned above (Food Safety Commission of Japan, 2010).

The EFSA TDI was used as the point of comparison for the current study.

T2/HT2

JECFA concluded that the toxic effects of T2 and HT2 could not be differentiated and included HT2 in the PMTDI established for T2 (JECFA, 2001b). JECFA concluded that the single long term study available was not suitable for establishing a tolerable intake and based their assessment on critical effects (immunotoxicity and haematotoxicity) in several short-term studies on pigs. The lowest LOEL of 0.029 mg/kg body weight/day for changes in white

and red blood cell counts was considered to be close to a NOEL, due to the subtlety and reversible of effects seen. A safety factor of 500 was applied to derive a PMTDI of 0.06 μg/kg body weight/day for T2 and HT2 (JECFA, 2001b).

The EU Scientific Committee on Food came to the same conclusions in establishing a temporary TDI (t-TDI) for T2 and HT2 combined of 0.06 μg/kg body weight/day (Scientific Committee on Food, 2001). Using the same toxicological data and a benchmark dose approach, EFSA have derived a group TDI for the sum of T2 and HT2 of 100 ng/kg body weight/day (0.1 μg/kg body weight/day) (EFSA, 2011).

Other trichothecene mycotoxins

RIVM (the Netherlands National Institute for Public Health and the Environment) assessed toxicological information for FX, DAS, NEO, 3ADON and 15ADON, to determine if *t*-TDIs could be derived for these toxins, but concluded that the information was too limited (Pronk *et al.*, 2002).

2.6.2.2 Benchmark doses

DON

JECFA reported a BMD_{05} and associated $BMDL_{05}$ (lower 95th percentile confidence limit for the BMD_{05}) for chronic DON exposure of 8.6 and 0.6 μg/kg body weight/day, respectively (JECFA, 2011). It should be noted that this benchmark dose includes an 'extrapolation' from the animal benchmark dose to a human equivalent benchmark dose. The animal BMD_{05} for reduced body weight gain has been estimated as 0.236 mg/kg body weight/day, with an associated $BMDL_{05}$ of 0.219 mg/kg body weight/day (Muri *et al.*, 2009a; Pieters *et al.*, 2001). Data to derive benchmark doses came from the same study used to determine the PMTDI for DON (Iverson *et al.*, 1995). For acute exposure, a lowest $BMDL_{10}$ of 0.21 mg/kg body weight was derived from studies of emesis in pigs.

NIV

Decreased WBC count data from a 90 day rat study was also used to derive benchmark doses for NIV (EFSA, 2013b). BMD_{05} and $BMDL_{05}$ estimates were 0.46 and 0.35 mg/kg body weight/day, respectively.

T2/HT2

EFSA derived a BMD_{05} and associated $BMDL_{05}$ for T2, using anti-horse globulin titres (a measure of immunotoxicity) in pigs (Rafai *et al.*, 1995a; Rafai *et al.*, 1995b). The best fitting model gave a BMD_{05} value of 15 μg/kg body weight/day and a $BMDL_{05}$ of 10.3 μg/kg body weight/day (EFSA, 2011). Using this model the corresponding BMD_{10} and $BMDL_{10}$ values would be 31.6 and 21.7 μg/kg body weight/day, respectively.

3 RESULTS AND DISCUSSION

3.1 OTA

3.1.1 Estimated dietary exposure

Table 5 summarises estimates of dietary exposure to OTA.

Table 5: Estimated ochratoxin A (OTA) dietary exposure for various New Zealand population subgroups

Age-gender group	Estimated dietary OTA exposure, lower bound – upper bound, ng/kg body weight/day		
	Mean	95th percentile	95th percentile usual
PTDI (JECFA) = 14.3 ng/kg body weight/day[1] TDI (EFSA) = 17.1 ng/kg body weight/day			
Child (5-6 years)	0.8-3.2	2.0-6.7	1.2-4.7
Female (11-14 years)	0.5-1.7	1.1-4.0	NC-3.1
Male (11-14 years)	0.5-2.1	1.4-4.7	1.0-3.8
Male (19-24 years)	0.3-1.5	0.8-2.9	NC
Female (25+ years)	0.3-1.0	0.7-2.1	NC-1.8
Male (25+ years)	0.3-1.3	0.8-2.7	0.6-2.2

NC: usual intakes could not be calculated

PTDI = provisional tolerable daily intake, TDI = tolerable daily intake, JECFA = Joint FAO/WHO Expert Committee on Food Additives, EFSA = European Food Safety Authority

[1] Both JECFA and EFSA set **weekly** exposure limits (100 and 120 ng/kg body weight/week). These have been converted to their daily equivalents by dividing by 7

No previous New Zealand estimates of dietary exposure to OTA are available for comparison.

Table 6 summarises estimates of dietary OTA exposure from other countries for comparison with the estimates in this report. Only more recent estimates (since 2006) have been summarised here. Older estimates of OTA exposure have been summarised and mean estimates were in the range 0.7-4.6 ng/kg body weight/day for adults (Cressey and Thomson, 2006).

Table 6: Overseas estimates of dietary ochratoxin A (OTA) exposure

Country	Year	Population group	Estimated dietary exposure (ng OTA/kg bw/day)	Main contributing food(s)	Reference
Belgium Czech Republic Denmark Finland France Germany Hungary Ireland Italy Netherland Sweden United Kingdom	2002-2006	Adults (16-64 years)	Mean (95%) 58 (131) 54 (117) 64 (130) 54 (119) 64 (130) 87 (171) 74 (139) 80 (177) 100 (184) 65 (135) 53 (103) 49 (108)	Vegetables, nuts and pulses food group	(Boon *et al.*, 2011)
Canada	NS	Infant (1 year) Male (31-50 years) Female (31-50 years)	Mean (95%) 4.38 (12.08) 1.62 (4.04) 1.33 (3.42)	Wheat products	(Kuiper-Goodman *et al.*, 2010)
China, Shanghai	2011-2012	Male adults (16-35 years) Female adults	Mean (95%) 1.15 (4.74) 1.05 (4.37)	Cereals and derived products	(Han *et al.*, 2013)
China, Yangtze Delta	2010	Children Adults	Mean (95%) 13.9 (27.7) 4.62 (9.23)	Only cereal consumption was considered	(Li *et al.*, 2014)
France	1998-2002	Adults (15+ years)	Mean (95%) 1.62-1.70 (3.28-3.88)[1]	NS	(Counil *et al.*, 2006)
France	2007-2009	Children Adults	Mean (95%) 0.23-2.82 (0.58-5.25) 0.28-1.92 (0.61-3.23)	Bread, alcoholic beverages (adult)	(Sirot *et al.*, 2013)
Lebanon	2005	Children (8-13 years) Teenagers (14-18)	Mean (95%)[4] 17.6-38.6 (31.0-57.5) 14.8-28.8 (24.0-43.6)	Cereals and cereal-based products	(Soubra *et al.*, 2009)
Portugal	2002	Adults (19-92 years, *n* = 104)	0.71 (range 0.19-3.35)[2]	NS	(Lino *et al.*, 2008)
Portugal	NS	Adult (65 kg body weight)	3.98	Cereals	(Duarte *et al.*, 2010)
Spain (Lleida)	NS	Blood donors (*n* = 279) (18+ years)	Mean (95%) From plasma OTA 1.69 (4.94) From dietary exposure 1.96 (3.97)	Cereals and derived products, wine	(Coronel *et al.*, 2009)
Spain (Lleida)	2008-2009	Blood donors (*n* = 325) (18+ years)	Mean (95%) From plasma OTA 2.66/1.57 (7.73/4.15)[3] From dietary exposure 1.60 (3.21)	NS	(Coronel *et al.*, 2011)

Country	Year	Population group	Estimated dietary exposure (ng OTA/kg bw/day)	Main contributing food(s)	Reference
Spain (Catalonia)	2008	Infants (0-3 years) Children (4-9) Adolescents (10-17) Adults (18-65)	Mean (95%)[4] 0.28-2.42 (1.46-7.23) 0.09-0.39 (0.30-0.98) 0.14-0.28 (0.44-0.68) 0.37-0.53 (1.14-1.31)	NS	(Coronel *et al.*, 2012)
Spain (Valencia)	2008	Blood donors (*n* = 168) (18+ years)	1.47/2.16[5]	NS	(Medina *et al.*, 2010)
Tunisia	2007-2009	Controls[6] I II III IV	Mean (range)[2] 4.4 (5.0-24.9) 26.0 (5.4-90.0) 7.7 (6.6-29.0) 8.1 (6.6-21.8) 7.7 (5.6-21.3)	NS	(Zaied *et al.*, 2011)

[1] The range of values reflects use of 1, 3 or 7 day dietary records to estimate food consumption
[2] Dietary exposure was calculated from serum OTA using the equation of Breitholtz *et al.* (1991)
[3] Differing exposure estimate depend on whether the conversion from plasma OTA to dietary OTA is based on the coefficient of Studer-Rohr *et al.* (2000) or Miraglia *et al.* (1996)
[4] The range limits were derived by assigning analytical results below the limit of detection a concentration either equal to zero or equal to the analytical limit of detection
[5] Differing exposure estimate depend on whether the conversion from plasma OTA to dietary OTA is based on the coefficient of Breitholtz *et al.* (1991) or the Klassen equation
[6] I = cases with chronic interstitial nephropathy of unknown aetiology, II = cases with chronic interstitial nephropathy of known aetiology, III = cases of chronic glomerular nephropathy, IV = cases with chronic vascular nephropathy

The European exposure estimates from the study of Boon *et al.* (2011) are anomalously high. These estimates used the European Concise Food Consumption Database[3] and the authors of the OTA exposure assessment ascribed their high estimate to a poor match between foods analysed for OTA and foods present in the database.

Mean and 95th percentile exposure estimates from the current study are generally consistent with or less than estimates for other countries. As expected, the application of techniques to correct estimates of percentile exposures for the effects of intra-person variability result in decreased estimates of dietary exposure at high percentiles (95th). The resultant 'usual' 95th percentile exposure estimates are less than other documented estimates, even at the upper bound.

The New Zealand estimates of dietary OTA exposure are quite similar to those from the second French Total Diet Study (Sirot *et al.*, 2013). This is probably not surprising, as both studies have adopted a similar methodological approach.

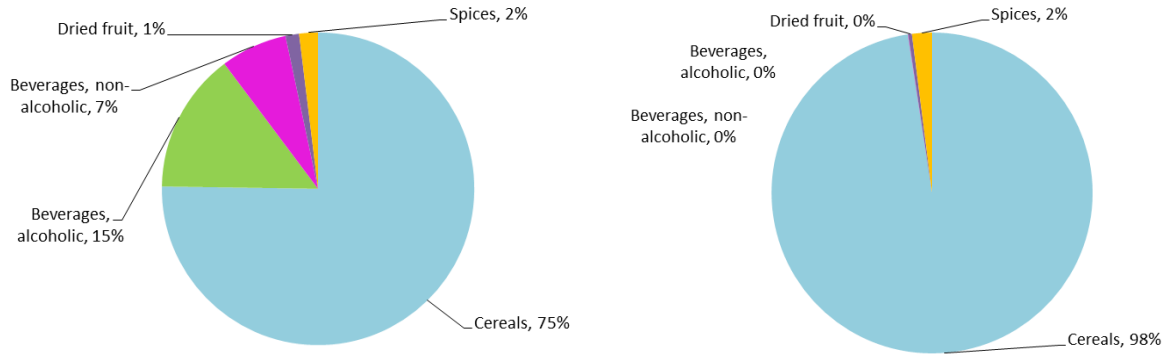
3.1.2 Contributing foods

Figure 3 shows the proportional contribution of different food groups to estimated dietary OTA exposure for an adult male and a 5-6 years old child (the group with the highest exposure on a per kilogram body weight basis). Figures are based on upper bound estimates

[3] http://www.efsa.europa.eu/en/datexfoodcdb/datexfooddb.htm Accessed 8 November 2013

of exposure. The contribution of food groups to OTA exposure for all age-gender groups are included in Appendix 4.

Figure 3: Contribution of food groups to mean upper bound estimates of ochratoxin A dietary exposure for adult males (25+ years) and children (5-6 years)



Males (25+ years) **Children (5-6 years)**

Exposure estimates for both age-gender groups are dominated by the contribution from cereals. This is particularly marked for children, with virtually all of their dietary OTA exposure coming from cereal consumption. Bread is the major contributor to exposure amongst cereals, making up about one-third of total exposure, for upper bound estimates, and more than 40% of total exposure for lower bound estimates. Noodles and pasta make up 12-24% of total OTA exposure, depending on the age group and treatment of left censored data. Coffee accounts for 6-10% of adult OTA exposure, depending on whether lower or upper bound estimates are considered, while beer is also a significant contributor to OTA exposure for adult males.

Due to the high proportion of quantified results, spices contribute 7-8% of lower bound OTA exposure estimates, but a much lower proportion of upper bound estimates.

3.1.3 Uncertainty assessment

3.1.3.1 Measurement uncertainty

Appendix 5 lists 95th percentile credible intervals for all concentration values used in the current study, considering measurement and sampling uncertainty associated with the selection and analysis of food samples. These credible intervals were derived by application of the two component uncertainty model for chemical analyses (Rocke and Lorenzato, 1995) combined with the sampling uncertainty approach of Vannoort *et al*. (2013). Credible intervals were determined by simulation. Table 7 shows the uncertainty intervals for mean and 95th percentile estimates of dietary OTA exposure resulting from measurement uncertainty. As these statistics are already represented by an uncertainty interval (upper and lower bounded), the credible interval represents the interval between the 2.5th percentile

credible limit for the lower bound estimate and the upper 97.5th percentile credible limit for the upper bound estimate.

Table 7: Uncertainty in summary statistics of dietary ochratoxin A (OTA) exposure estimates due to measurement and food sampling uncertainty

Age-gender group	Estimated dietary OTA exposure, lower bound – upper bound (95th percentile credible interval), ng/kg body weight/day	
	Mean	95th percentile
Child (5-6 years)	0.8-3.2 (0.3-3.5)	2.0-6.7 (1.3-7.8)
Female (11-14 years)	0.5-1.7 (0.2-1.9)	1.1-4.0 (0.6-4.5)
Male (11-14 years)	0.5-2.1 (0.2-2.3)	1.4-4.7 (0.8-5.4)
Male (19-24 years)	0.3-1.5 (0.1-1.7)	0.8-2.9 (0.6-3.5)
Female (25+ years)	0.3-1.0 (0.2-1.1)	0.7-2.1 (0.6-2.3)
Male (25+ years)	0.3-1.3 (0.2-1.4)	0.8-2.7 (0.6-3.2)

The credible intervals suggest that food sampling and measurement uncertainty adds further uncertainty to dietary OTA exposure estimates, over and above the uncertainty generated by the high proportion of 'not detected' results in the analytical data. However, all upper 95th percentile credible limits are still below the relevant health-based exposure limits.

Due to the manner in which they are determined, it is not possible to use the same approach to quantify uncertainty associated with 'usual' 95th percentile exposure estimates.

3.1.4 Population sampling uncertainty

The bootstrap (resampling) method was used to quantify the uncertainty in summary statistics of dietary OTA exposure due to sampling of the population through the national nutrition survey cohort. Results are summarised in Table 8.

Table 8: Uncertainty in summary statistics of dietary ochratoxin A (OTA) exposure estimates due to population sampling uncertainty

Age-gender group (*n* = number of respondents in national nutrition survey cohort)	Estimated dietary OTA exposure, lower bound – upper bound (95th percentile credible interval), ng/kg body weight/day	
	Mean	95th percentile
Child (5-6 years, *n* = 639)	0.8-3.2 (0.8-3.3)	2.0-6.7 (1.9-7.3)
Female (11-14 years , *n* = 551)	0.5-1.7 (0.4-1.8)	1.1-4.0 (1.0-4.4)
Male (11-14 years, *n* = 531)	0.5-2.1 (0.5-2.3)	1.4-4.7 (1.2-5.2)
Male (19-24 years, *n* = 124)	0.3-1.5 (0.3-1.7)	0.8-2.9 (0.6-3.4)
Female (25+ years, *n* = 1961)	0.3-1.0 (0.3-1.0)	0.7-2.1 (0.7-2.2)
Male (25+ years, *n* = 1558)	0.3-1.3 (0.3-1.3)	0.8-2.7 (0.7-2.7)

The outputs in Tables 7 and 8 suggest that the uncertainty due to the population sample selected is less than the uncertainty due to food sampling and analysis. Population sampling uncertainty is also inversely related to the size of the sample taken. For adult females and males, with cohort sizes greater than 1500 people, the difference between exposure estimates and 95th percentile credible limits is about 3-4%. For the smallest cohort (19-24 years males) this difference is about 13-18%.

Due to the manner in which they are determined, it is not possible to use the same approach to quantify uncertainty associated with 'usual' 95th percentile exposure estimates.

No similar analyses of uncertainty associated with dietary OTA exposure were found in the scientific literature or in regulatory assessment reports to benchmark these findings against.

3.1.5 Risk assessment

3.1.5.1 Comparison of dietary exposures to health-based exposure limits

Table 9 summarises comparisons of dietary exposure estimates to either the JECFA PMTWI (100 ng/kg body weight/week, equivalent to 14.3 ng/kg body weight/day) or the EFSA TWI (120 ng/kg body weight/week, equivalent to 17.1 ng/kg body weight/day). Comparisons are presented in terms of the dietary exposure estimates as percentages of the daily equivalent of the PTWI/TWI.

Table 9: Comparison of dietary ochratoxin A (OTA) exposures to health-based exposure limits

Age-gender group	Estimated dietary OTA exposure as a percentage of PMTDI/TDI		
	Mean	95th percentile	95th percentile usual
PTDI (JECFA) = 14.3 ng/kg body weight/day[1]			
Child (5-6 years)	6-22	14-47	8-33
Female (11-14 years)	3-12	8-28	NC-22
Male (11-14 years)	3-15	10-33	7-27
Male (19-24 years)	2-10	6-20	NC
Female (25+ years)	2-7	5-15	NC-12
Male (25+ years)	2-9	5-19	4-15
TDI (EFSA) = 17.1 ng/kg body weight/day[1]			
Child (5-6 years)	5-19	12-39	7-27
Female (11-14 years)	3-10	6-24	NC-18
Male (11-14 years)	3-13	8-28	6-22
Male (19-24 years)	2-9	5-17	NC
Female (25+ years)	2-6	4-12	NC-10
Male (25+ years)	2-7	4-15	4-13

NC: usual intakes could not be calculated

PTDI = provisional tolerable daily intake, TDI = tolerable daily intake, JECFA = Joint FAO/WHO Expert Committee on Food Additives, EFSA = European Food Safety Authority

[1] Both JECFA and EFSA set **weekly** exposure limits (100 and 120 ng/kg body weight/week). These have been converted to their daily equivalents by dividing by 7

In the worst case situation (child, 95th percentile upper bound exposure estimate, lower health-based exposure limit), dietary exposure estimates approach 50% of the lowest health-based exposure limit. Mean estimates of dietary exposure are all less than 25% of the health-based exposure limits.

Examination of individual daily estimates of dietary OTA exposure identified one record where health-based exposure limits were exceeded for upper bound, but not lower bound, estimates. The record related to a 5-year-old boy, who was reported to have consumed more than 1 kg of white bread at one meal.

3.1.5.2 Margin of exposure

A $BMDL_{10}$ of 15 µg/kg body weight/day (15,000 ng/kg body weight/day) for renal tumour formation in rats was used as the benchmark dose to determine MoEs for the exposure estimates in Table 5. Resulting MoEs are shown in Table 10.

Table 10: Margins of exposure for ochratoxin A (OTA) dietary exposure for various New Zealand population subgroups

Age-gender group	Margin of Exposure for OTA exposure, lower bound – upper bound		
	Mean	95th percentile	95th percentile usual
$BMDL_{10}$ = 15000 ng/kg body weight/day			
Child (5-6 years)	4730-18,750	2235-7350	3220-12,930
Female (11-14 years)	8720-33,330	3720-13,890	4870-NC
Male (11-14 years)	7010-30,000	3165-10,950	3930-15,310
Male (19-24 years)	10,200-50,000	5170-18,990	NC
Female (25+ years)	15,310-53,570	7140-22,060	8,520-NC
Male (25+ years)	12,000-46,880	5660-20,000	6980-24,190

NC: usual intakes could not be calculated

$BMDL_{10}$ = lower 95th percentile confidence limit for the benchmark dose equivalent to a 10% increase in response

Except for the 95th percentile exposure for children, the MoE ranges for dietary OTA exposure all either contain or are greater than a MoE of 10,000. MoEs of this magnitude are considered to represent situations of low public health risk (EFSA, 2005).

3.2 Trichothecene Mycotoxins

3.2.1 Estimated chronic dietary exposure

Table 11 summarises estimates of dietary exposure to trichothecene mycotoxins. Exposure estimates are presented here for DON and NIV. In the survey that these exposure estimates are based on T2 and 15ADON were detected in 1 food sample, while DAS was detected at very low concentrations in 3 food samples. These concentration data were considered to be insufficient to derive dietary exposure estimates for T2, 15ADON and DAS.

Table 11: Estimated trichothecene mycotoxin dietary exposure for various New Zealand population subgroups

Age-gender group	Estimated dietary trichothecene mycotoxin exposure, lower bound – upper bound, ng/kg body weight/day		
	Mean	95th percentile	95th percentile usual
Deoxynivalenol (DON) PMTDI/TDI (JECFA/EFSA) = 1000 ng/kg body weight/day			
Child (5-6 years)	76-77	206-208	NC
Female (11-14 years)	38-39	102-103	75-76
Male (11-14 years)	44-45	129-131	86-88
Male (19-24 years)	30-33	103-103	NC
Female (25+ years)	16.6-17.1	51-54	NC
Male (25+ years)	23.6-25.1	68-72	NC-51
Nivalenol (NIV) TDI (EFSA) = 1200 ng/kg body weight/day			
Child (5-6 years)	21.9-23.7	51-54	36-38
Female (11-14 years)	10.3-11.3	26.8-28.4	15.5-15.9
Male (11-14 years)	12.8-14.1	29.4-31.2	21.4-23.9
Male (19-24 years)	3.7-5.3	11.0-13.7	NC
Female (25+ years)	3.6-4.2	9.2-10.2	NC-7.9
Male (25+ years)	4.0-5.3	10.5-12.5	NC-9.1

NC: usual intakes could not be calculated

PMTDI = provisional maximum tolerable daily intake, TDI = tolerable daily intake, JECFA = Joint FAO/WHO Expert Committee on Food Additives, EFSA = European Food Safety Authority

No previous New Zealand estimates of dietary exposure to trichothecene mycotoxins are available for comparison.

While the analytical method used to generate the concentration data used in the current exposure assessment was extremely sensitive for the detection of DON, it was less optimal for detection of the conjugated forms of DON (3ADON, 15ADON and DON-3-G). These conjugated forms would have been present, at some level, in foods analysed. Based on information presented in EFSA's exposure assessment of DON, the total exposure of DON from these three conjugated is likely to be less than 50% of the exposure to parent DON (EFSA, 2013a). A 50% increase in the dietary exposure estimates in Table 11 would still result in estimates that were well below the health-based exposure limits.

Table 12 summarises estimates of dietary trichothecene mycotoxin exposure from other countries for comparison with the estimates in this report. Only more recent estimates (since 2006) have been summarised here. Older estimates of trichothecene mycotoxin exposure have previously been summarised (Cressey and Thomson, 2006).

Table 12: Overseas estimates of dietary exposure to trichothecene mycotoxins

Country	Population group	Mean (95th percentile) exposure, ng/kg body weight/day	Reference
DON TDI (EFSA) or PMTDI (JECFA) = 1000 ng/kg body weight/day			
Argentina	Males - 18-24 years - 25-50 years Females - 18-24 years - 25-50 years	 135 95 75 92	(Pacin *et al.*, 2011)
Belgium	Adults - DON - 3ADON - 15ADON - DON-3-G - Total DON	 35-40 (80-91) 23-24 (53-55) 9-16 (21-35) 21-25 (48-56) 89-104 (202-237)	(De Boevre *et al.*, 2013)
Brazil (Paraná state)	Individuals, 8-76 years (*n* = 260)	1130 (range 0-5090)[1]	(Sifuentes dos Santos *et al.*, 2013)
Denmark	Total population Children	170 320	(Rasmussen *et al.*, 2007)
European Union	Infants Toddlers Other children Adolescents Adults Elderly Very elderly	160-730 (920-1610)[2,4] 480-1020 (880-1810) 430-970 (760-1650) 280-580 (590-1080) 170-460 (310-1020) 160-310 (310-620) 210-330 (400-590)	(EFSA, 2013a)
France	Adult females - Before pregnancy - Third trimester	 253-282 (554-619) 198-221 (428-475)	(Chan-Hon-Tong *et al.*, 2013)
France	Children - DON - 3ADON - 15ADON Adults - DON - 3ADON - 15ADON	 540-560 (1020-1030) 0.1-29 (0. 8-54) 0.7-30 (2.8-57) 370-380 (720) 0.3-16 (1.6-29) 0.2-16 (0.9-27)	(Sirot *et al.*, 2013)
GEMS/Food cluster diets	General population	190-14500	(JECFA, 2011)
Hungary	Adults (from white bread)	260-490 (560-1050)	(Ambrus *et al.*, 2011)
India	General population	3200 (7720; 90th percentile)	(Mishra *et al.*, 2013)
Japan	From consumption of wheat products only - 1-6 years - 7-14 years - 15-19 years - Over 19 years	95th percentile 718 511 404 249	(Nakatani *et al.*, 2011)

Country	Population group	Mean (95th percentile) exposure, ng/kg body weight/day	Reference
Korea	Male, 3-6 years Female, 3-6 years Male, 7-12 years Female, 7-12 years Male, 13-19 years Female, 13-19 years Male, 20-29 years Female, 20-29 years Male, 30-49 years Female, 30-49 years Male, 50-64 years Female, 50-64 years Male, 65+ years Female, 65+ years	142 (292) 144 (302) 95 (200) 96 (200) 77 (162) 65 (138) 66 (139) 66 (132) 68 (143) 68 (141) 73 (156) 72 (155) 70 (154) 70 (154)	(Ok *et al.*, 2009b)
Korea	Population, 3-85 years (mean body weight = 57.6 kg)	9.8	(Ok *et al.*, 2009a)
Korea	Population, 3-85 years (mean body weight = 57.6 kg) - DON - 3ADON - 15ADON	 47.6 1.8 6.2	(Ok *et al.*, 2011)
Lebanon	Children, 8-13 years Teenagers, 14-18 years	545 (975) 409 (664)	(Soubra *et al.*, 2009)
Morocco	General population	0.2	(Serrano *et al.*, 2012)
Netherlands	Children (*n* = 123, duplicate diet)	291	(Bakker *et al.*, 2009)
Serbia	Children Adults	1700 1500	(Škrbić *et al.*, 2012)
South Africa[6]	Infants, 1-5 years, rural Infants, 1-5 years, urban Young children, 6-9 years, rural Young children, 6-9 years, urban Adults, 10+ years, rural Adults, 10+ years, urban	3800 2780 2730 1960 1770 1450	(Shephard *et al.*, 2010)
Spain	Infants, 0-3 years Children, 4-9 years Adolescents, 10-19 years Adult males, 20-65 years Adult females, 20-65 years Seniors, 65+ years Coeliac sufferers Ethnics	740 360 150 100 90 40 130 570	(Cano-Sancho *et al.*, 2011)
Spain	DON + 3ADON - Infants (0-3 years) - Children (5-12 years) - Adults (18-65 years)	 77.3 63.3 9.1	(Rodríguez-Carrasco *et al.*, 2013)
Tunisia	General population	189	(Serrano *et al.*, 2012)
United Kingdom	Pregnant women (*n* = 55)	168 (range 23-982)[5]	(Hepworth *et al.*, 2011)

Country	Population group	Mean (95th percentile) exposure, ng/kg body weight/day	Reference
NIV TDI (EFSA) = 1200 ng/kg body weight/day			
France	Children Adults	31-59 (72-119) 20-34 (45-67)	(Sirot *et al.*, 2013)
European Union	Infants Toddlers Other children Adolescents Adults Elderly Very elderly	2.4-140 (16-389)[2,4] 4.3-202 (12-484) 1.3-132 (3.0-259) 1.0-80 (3.0-147) 0.4-75 (1.1-224) 0.8-55 (2.3-127) 0.8-58 (1.9-111)	(EFSA, 2013b)
Korea	Population, 3-85 years (mean body weight = 57.6 kg)	76.6	(Ok *et al.*, 2011)
Morocco	General population	6.1	(Serrano *et al.*, 2012)
Spain	General population	433	(Serrano *et al.*, 2012)
Spain	Infants (0-3 years) Children (5-12 years) Adults (18-65 years)	67.5 62.7 7.5	(Rodríguez-Carrasco *et al.*, 2013)
Tunisia	General population	3411	(Serrano *et al.*, 2012)
T2 toxin TDI (EFSA) = 100 ng/kg body weight/day (combined with HT2)			
Belgium	Adult	6-12 (15-28)	(De Boevre *et al.*, 2013)
European Union	Infants Toddlers Other children Adolescents Adults Elderly Very elderly	5.9-16 (19-51)[2,4] 12-43 (23-91) 10-39 (21-71) 4.4-24 (12-47) 3.4-18 (7.2-39) 3.3-14 (6.7-26) 2.8-15 (5.3-25)	(EFSA, 2011)
France	Adult females - Before pregnancy - Third trimester	 1.95-16.62 (4.56-35.40) 1.55-12.97 (3.61-27.57)	(Chan-Hon-Tong *et al.*, 2013)
France	Children Adults	4.0-38 (9.0-73) 1.8-20 (4.8-37)	(Sirot *et al.*, 2013)
Morocco	General population	0.4[2]	(Serrano *et al.*, 2012)
Serbia	Children Adults	20 20	(Škrbić et al., 2012)
Spain	Children, 4-9 years Adolescents, 10-19 years Adult male, 20-65 years Adult female, 20-65 years	56-79[2,3] 27-40 12-19 11-16	(Cano-Sancho *et al.*, 2012)
Spain	Infants (0-3 years) Children (5-12 years) Adults (18-65 years)	85.9[2] 79.2 9.5	(Rodríguez-Carrasco *et al.*, 2013)
Tunisia	General population	43.6[2]	(Serrano *et al.*, 2012)
HT2 toxin	**TDI (EFSA) = 100 ng/kg body weight/day (combined with T2)**		
Belgium	Adults	10-18 (23-41)	(De Boevre *et al.*, 2013)

Country	Population group	Mean (95th percentile) exposure, ng/kg body weight/day	Reference
France	Adult females - Before pregnancy - Third trimester	 4.33-22.18 (9.43-46.38) 3.70-17.98 (7.94-37.35)	(Chan-Hon-Tong *et al.*, 2013)
France	Children Adults	11-53 (22-104) 7.2-32 (15-59)	(Sirot *et al.*, 2013)
Other			
Korea	Population, 3-85 years (mean body weight = 57.6 kg) - FX	 4.2	(Ok *et al.*, 2011)
Morocco	General population - DAS	 0.1	(Serrano *et al.*, 2012)
Spain	Infants (0-3 years) - FX - DAS - NEO Children (5-12 years) - FX - DAS - NEO Adults (18-65 years) - FX - DAS - NEO	 40.0 8.6 53.5 36.1 5.8 23.7 4.5 1.1 7.5	(Rodríguez-Carrasco *et al.*, 2013)
Tunisia	General population - DAS	 24.7	(Serrano *et al.*, 2012)

PMTDI = provisional maximum tolerable daily intake, TDI = tolerable daily intake, JECFA = Joint FAO/WHO Expert Committee on Food Additives, EFSA = European Food Safety Authority, DON = deoxynivalenol, 3ADON = 3-acetylDON, 15ADON = 15-acetylDON, DON-3-G = DON-3-glucoside, NIV = nivalenol, FX = fusarenon X, DAS = diacetoxyscirpenol, NEO = neosolaniol

[1] Exposures were calculated for consumption of bread and pasta. However, the DON concentration used was for harvest wheat

[2] Sum of T2 and HT2

[3] The range covers different approaches for dealing with left censored analytical data

[4] The ranges presented are from the lower bound exposure estimate for the minimum EU estimate to the upper bound estimate for the maximum EU estimate

[5] The published exposures were in units of µg/day. These were converted to ng/kg body weight/day assuming a body weight of 60 kg

[6] Exposures were calculated separately for consumption of maize meal and wheat flour. These separate estimates have been summed, but more than 95% of exposure is due to consumption of maize meal

In most cases, mean and 95th percentile exposure estimates for DON and NIV from the current study are consistent with or less than estimates for other countries. As expected, the application of techniques to correct estimates of percentile exposures for the effects of intra-person variability result in decreased estimates of dietary exposure at high percentiles (95th). The resultant 'usual' 95th percentile exposure estimates are less than other documented estimates, even at the upper bound.

For DON, a lower estimate of dietary exposure has been derived for Morocco (0.2 ng/kg body weight/day) (Serrano *et al.*, 2012). However, it should be noted that this estimate only considered dietary exposure to DON from consumption of rice.

The conclusion of low DON and NIV dietary exposure compared to most other countries is expected, as the concentration data that these exposure estimates are based on suggested lower levels of trichothecene mycotoxins contamination in New Zealand foods (Cressey *et al.*, 2014).

3.2.2 Estimated acute dietary exposure to DON

The acute exposure model used in the current study used simulation analysis to determine the probability of an adult or child consumer exceeding the ARfD during a 24-hour consumption period. Summary statistics from the simulation analysis are shown in Table 13.

Table 13: Summary of simulation results for acute dietary exposure to deoxynivalenol (DON)

	Children (5-14 years)	Adults (15+ years)
ARfD = 8000 ng/kg body weight		
Number of iterations	100,000	100,000
Mean exposure (ng/kg bw)	53.4	18.1
Exposure percentiles (ng/kg bw)		
- 95	187	67
- 99	461	173
- 99.9	1380	645
- 99.99	5055	1446
Probability of exceeding ARfD	0.00003	ARfD was not exceeded in any iteration

bw = body weight, ARfD = acute reference dose

The current study found that the ARfD was exceeded on 0.003% of child exposure days and was not exceeded on any adult exposure days.

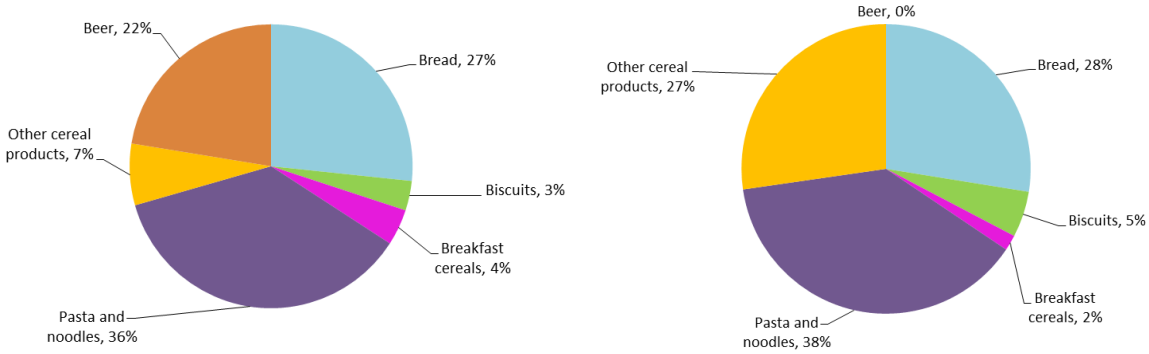
Acute estimates of DON exposure determined for European countries were generally an order of magnitude greater than those determined for New Zealand (EFSA, 2013a). This is consistent with differences in the concentrations of DON in key foods, rather than indicating major differences in dietary habits. The European study found that, for children, between 0.04 and 0.51% of exposure days exceeded the ARfD, while for adults the range was 0 to 0.09% of exposure days.

3.2.3 Contributing foods

3.2.3.1 DON

Figure 4 shows the proportional contribution of different food groups to estimated DON exposure for an adult male and a 5-6 years old child (the group with the highest exposure on a per kilogram body weight basis). Figures are based on upper bound estimates of exposure. The contribution of food groups to DON exposure for all age-gender groups are included in Appendix 4.

Figure 4: Contribution of food groups to mean upper bound estimates of dietary deoxynivalenol (DON) exposure for adult males (25+ years) and children (5-6 years)



Males (25+ years) **Children (5-6 years)**

For all age groups consumption of bread and pasta/noodles accounted for more than half of dietary DON exposure. For adult males, beer consumption also contributes appreciably to DON exposure. For young children (5-6 years), 'other cereal products' account for approximately a quarter of dietary DON exposure. This is mainly due to their greater consumption of snack foods and the high concentrations of DON encountered in some of these snack foods.

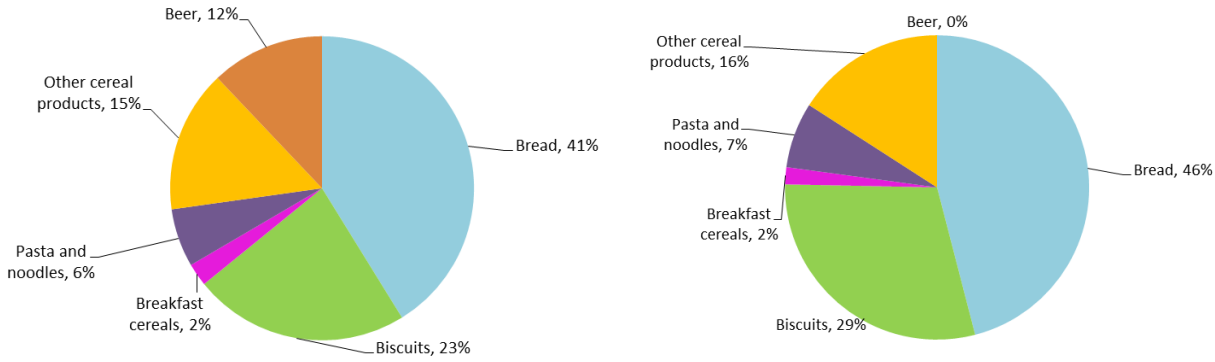
The substantial contribution of pasta/noodles to dietary DON exposure across all age-gender groups (36-54%) is noteworthy, given that this food category contains a number of imported products.

The French Total Diet Study also concluded that bread and pasta were two of the main food types contributing to DON exposure, although they found that bread was very much the main contributor to dietary DON exposure (Sirot *et al.*, 2013). Bread accounted for approximately 60% of adult dietary DON exposure. An Argentinean study concluded that 60-70% of dietary DON exposure was due to bread consumption (Pacin *et al.*, 2011). Other studies of dietary DON exposure (see Table 12) either considered only a narrow range of foods or did not present information on the contributions of individual food types to overall dietary exposure.

3.2.3.2 NIV

Figure 5 shows the proportional contribution of different food groups to estimated NIV exposure for an adult male and a 5-6 years old child (the group with the highest exposure on a per kilogram body weight basis). Figures are based on upper bound estimates of exposure. The contribution of food groups to NIV exposure for all age-gender groups are included in Appendix 4.

Figure 5: Contribution of food groups to mean upper bound estimates of dietary nivalenol (NIV) exposure for adult males (25+ years) and children (5-6 years)



Males (25+ years) **Children (5-6 years)**

Bread is the major contributor to the dietary NIV exposure for all age-gender groups. Biscuits are also a major contributor to NIV exposure, with sweet plain biscuits accounting for more than 70% of the biscuit contribution to dietary NIV exposure. Within the 'other cereal products' group, rice and flavoured snacks are consistent contributors to dietary NIV exposure, although their individual contributions do not exceed 10% of total exposure for any age-gender group.

The French Total Diet Study produced similar findings with bread accounting for 30-50% of dietary NIV exposure, depending on age groups and treatment of left-censored data (Sirot *et al.*, 2013). Pasta, rice and wheat products and mixed dishes were also major contributors to dietary NIV exposure in the French study.

Bread and rolls were also major contributors to dietary NIV exposure in an EFSA study (EFSA, 2013b). This study included a number of country-level exposure estimates, with bread and rolls accounting for more than 75% of dietary NIV exposure in some studies. In most country-level estimates, the contribution from bread and rolls was in the range 10-75% of total dietary NIV exposure. Pasta was also a consistent contributor to dietary NIV exposure. This is not surprising, as Europe contains several countries with high levels of pasta consumption.

3.2.4 Uncertainty assessment

3.2.4.1 Measurement uncertainty

Appendix 5 lists 95th percentile credible intervals for all concentration values used in the current study, considering measurement and sampling uncertainty associated with the selection and analysis of food samples. These credible intervals were derived by application of the two component uncertainty model for chemical analyses (Rocke and Lorenzato, 1995) combined with the sampling uncertainty approach of Vannoort *et al.* (2013). Credible intervals were determined by simulation. Table 14 shows the uncertainty intervals for mean

and 95th percentile estimates of dietary exposure for DON and NIV. As these statistics are already represented by an uncertainty interval (upper and lower bounded), the credible interval represents the interval between the 2.5th percentile credible limit for the lower bound estimate and the upper 97.5th percentile credible limit for the upper bound estimate.

Table 14: Uncertainty in summary statistics of dietary trichothecene mycotoxin exposure estimates due to measurement and food sampling uncertainty

Age-gender group	Estimated dietary trichothecene mycotoxin exposure, lower bound – upper bound (95th percentile credible interval), ng/kg body weight/day	
	Mean	95th percentile
Deoxynivalenol (DON)		
Child (5-6 years)	76-77 (48-104)	206-208 (159-305)
Female (11-14 years)	38-39 (28-51)	102-103 (72-147)
Male (11-14 years)	44-45 (26-57)	129-131 (81-175)
Male (19-24 years)	30-33 (17-44)	103-103 (64-149)
Female (25+ years)	16.6-17.1 (12.7-21.2)	51-54 (40-71)
Male (25+ years)	23.6-25.1 (15.0-34.4)	68-72 (48-111)
Nivalenol (NIV)		
Child (5-6 years)	21.9-23.7 (15.5-29.6)	51-54 (37-69)
Female (11-14 years)	10.3-11.3 (7.5-14.0)	26.8-28.4 (20.5-35.7)
Male (11-14 years)	12.8-14.1 (9.9-16.9)	29.4-31.2 (24.8-38.6)
Male (19-24 years)	3.7-5.3 (2.3-6.5)	11.0-13.7 (8.6-17.1)
Female (25+ years)	3.6-4.2 (2.8-5.1)	9.2-10.2 (7.7-12.9)
Male (25+ years)	4.0-5.3 (2.8-6.2)	10.5-12.5 (9.0-14.7)

The credible intervals suggest that food sampling and measurement uncertainty adds further uncertainty to dietary DON and NIV exposure estimates, over and above the uncertainty generated by the high proportion on 'not detected' results in the analytical data. However, all upper 95th percentile credible limits are still below the relevant health-based exposure limits.

Due to the manner in which they are determined, it is not possible to use the same approach to quantify uncertainty associated with 'usual' 95th percentile exposure estimates.

3.2.5 Population sampling uncertainty

The bootstrap (resampling) method was used to quantify the uncertainty in summary statistics of dietary DON and NIV exposure due to sampling of the population through the national nutrition survey cohort. Results are summarised in Table 15.

Table 15: Uncertainty in summary statistics of dietary trichothecene mycotoxin exposure estimates due to population sampling uncertainty

Age-gender group (*n* = number of respondents in national nutrition survey cohort)	Estimated dietary trichothecene mycotoxin exposure, lower bound – upper bound (95th percentile credible interval), ng/kg body weight/day	
	Mean	95th percentile
Deoxynivalenol (DON)		
Child (5-6 years, *n* = 639)	76-77 (70-83)	206-208 (188-233)
Female (11-14 years , *n* = 551)	38-39 (35-43)	102-103 (89-113)
Male (11-14 years, *n* = 531)	44-45 (40-49)	129-131 (116-146)
Male (19-24 years, *n* = 124)	30-33 (23-41)	103-103 (75-129)
Female (25+ years, *n* = 1961)	16.6-17.1 (15.5-18.2)	51-54 (48-58)
Male (25+ years, *n* = 1558)	23.6-25.1 (22.2-26.7)	68-72 (63-80)
Nivalenol (NIV)		
Child (5-6 years, *n* = 639)	21.9-23.7 (20.4-25.4)	51-54 (48-58)
Female (11-14 years , *n* = 551)	10.3-11.3 (9.5-12.3)	26.8-28.4 (25.0-30.4)
Male (11-14 years, *n* = 531)	12.8-14.1 (11.8-15.2)	29.4-31.2 (27.1-36.2)
Male (19-24 years, *n* = 124)	3.7-5.3 (3.1-6.2)	11.0-13.7 (8.2-15.7)
Female (25+ years, *n* = 1961)	3.6-4.2 (3.4-4.4)	9.2-10.2 (8.8-10.8)
Male (25+ years, *n* = 1558)	4.0-5.3 (3.8-5.5)	10.5-12.5 (9.8-13.1)

The outputs in Tables 14 and 15 suggest that the uncertainty due to the population sample selected is less than the uncertainty due to food sampling and analysis, although both sources of uncertainty will contribute to overall uncertainty in exposure estimates. As noted for OTA, population sampling uncertainty is inversely related to the size of the cohort. For adult females and males, with cohort sizes greater than 1500 people, the difference between exposure estimates and 95th percentile uncertainty limits is about 4-11%. For the smallest cohort (19-24 years males) this difference is about 15-27%.

Due to the manner in which they are determined, it is not possible to use the same approach to quantify uncertainty associated with 'usual' 95th percentile exposure estimates.

Unfortunately, no similar analyses of uncertainty associated with trichothecene mycotoxins were found to benchmark these findings against. Other studies have addressed uncertainties qualitatively (EFSA, 2013b) or not at all.

3.2.6 Risk assessment

3.2.6.1 Comparison of dietary exposures to health-based exposure limits

Table 16 summarises comparisons of dietary exposure estimates to either the JECFA PMTDI (1000 ng/kg body weight/day for DON) or the EFSA TDI (1000 ng/kg body weight/day for DON and 1200 ng/kg body weight/day for NIV). Comparisons are presented in terms of the dietary exposure estimates as percentages of the PMTDI/TDI.

Table 16: Comparison of dietary trichothecene mycotoxin exposures to health-based exposure limits

Age-gender group	Estimated dietary trichothecene mycotoxin exposure as a percentage of PMTDI/TDI[1]		
	Mean	95th percentile	95th percentile usual
Deoxynivalenol (DON) PMTDI/TDI (JECFA/EFSA) = 1000 ng/kg body weight/day			
Child (5-6 years)	8	21	NC
Female (11-14 years)	4	10	8
Male (11-14 years)	4-5	13	9
Male (19-24 years)	3	10	NC
Female (25+ years)	2	5	NC
Male (25+ years)	2	7	NC-5
Nivalenol (NIV) TDI (EFSA) = 1200 ng/kg body weight/day			
Child (5-6 years)	2	4	3
Female (11-14 years)	1	2	1
Male (11-14 years)	1	2-3	2
Male (19-24 years)	0.3-0.4	0.9-1.1	NC
Female (25+ years)	0.3-0.4	0.8-0.9	NC-0.7
Male (25+ years)	0.3-0.4	0.9-1.0	NC-9.1

NC: usual intakes could not be calculated

PMTDI = provisional maximum tolerable daily intake, TDI = tolerable daily intake, JECFA = Joint FAO/WHO Expert Committee on Food Additives, EFSA = European Food Safety Authority

[1] Although health-based exposure limits were compared to upper and lower bound estimates of dietary exposure, the percentages calculated were the same at the upper and lower bound in most cases, after rounding

Estimates of dietary DON and NIV exposure are well within the health-based exposure limits even at the upper bound 95th percentile level. The highest 95th percentile single day exposure estimate (for 5-6-year-old children) was still less than 25% of the health-based exposure limit.

Examination of individual estimates of dietary trichothecene mycotoxin exposure identified one record where the health-based exposure limit for DON was exceeded. The record related to a 9-year-old boy, who was reported to have consumed 2 very large servings of popcorn during the day. No individual estimates of dietary NIV exposure exceeded the health-based exposure limit.

3.2.6.2 Margin of exposure

An animal $BMDL_{05}$ of 0.219 mg/kg body weight/day (219,000 ng/kg body weight/day) has been determined for DON, based on decreased body weight gain in mice (Muri *et al.*, 2009b). A $BMDL_{05}$ of 0.35 mg/kg body weight/day has been determined for NIV, based on decreased WBC counts in rats (EFSA, 2013b).These were used to determine MoEs for the exposure estimates in Table 10. Resulting MoEs are shown in Table 17.

Table 17: Margins of exposure for trichothecene mycotoxin dietary exposure for various New Zealand population subgroups

Age-gender group	Margin of Exposure for trichothecene mycotoxins exposure, lower bound – upper bound		
	Mean	95th percentile	95th percentile usual
Deoxynivalenol $BMDL_{05}$ = 219000 ng/kg body weight/day			
Child (5-6 years)	2830-2880	1050-1060	NC
Female (11-14 years)	5580-5700	2130-2160	2880-2920
Male (11-14 years)	3720-4980	1270-1700	2490-2550
Male (19-24 years)	6750-7190	2140-2140	NC
Female (25+ years)	12,830-13,190	4090-4270	NC
Male (25+ years)	8720-9280	3030-3240	NC-4290
Nivalenol $BMDL_{05}$ = 350000 ng/kg body weight/day			
Child (5-6 years)	14,740-15,980	6450-6870	9210-9720
Female (11-14 years)	30,930-33,880	12,320-13,050	22,010-22,580
Male (11-14 years)	24,820-27,350	11,230-11,910	14,640-15,360
Male (19-24 years)	66,210-95,700	25,580-31,700	NC
Female (25+ years)	83,310-97,570	34,470-38,170	NC-44,300
Male (25+ years)	66,490-87,860	27,950-33,460	NC-38,460

NC: usual intakes could not be calculated

$BMDL_{05}$ = lower 95th percentile confidence limit for the benchmark dose equivalent to a 5% increase in response

The MoEs in Table 17 highlight a current issue in using MoE approaches. In 2005, EFSA stated that, "The Scientific Committee is of the view that in general an MoE of 10,000 or higher, if it is based on the $BMDL_{10}$ from an animal study, would be of low concern from a public health point of view and might be considered as a low priority for risk management actions" (EFSA, 2005). The MoEs in Table 17 are derived from $BMDL_{05}$ values, rather than $BMDL_{10}$, and will consequently produce lower MoEs. At this point in time, there is no guidance on what constitutes an acceptable MoE, with the exception of the statement by EFSA.

For NIV, almost all MoEs are in excess of 10,000 suggesting that exposure to this mycotoxin in New Zealand would currently be of low public health concern. Utilisation of a $BMDL_{10}$ would have resulted in even greater MoEs.

For DON, many of the MoE estimates are considerably less than 10,000 and it is not currently possible to comment on the public health significance of these MoEs. However, given that the PMTDI/TDI was derived from the same toxicological study as the $BMDL_{05}$, and mean and 95th percentile dietary DON exposures are all less than the PMTDI/TDI, it is likely that these MoEs do not represent a public health concern.

4 CONCLUSIONS

Foods available for consumption in New Zealand are frequently contaminated with OTA and/or trichothecene mycotoxins. However, dietary exposure to these contaminants appears to be at the low end of the range seen internationally and exposures are well within health-based exposure limits.

Mean OTA exposures range from 0.8-3.2 ng/kg body weight/day for 5-6-year-old children to 0.3-1.0 ng/kg body weight/day for adult females. The corresponding 95th percentile dietary exposure estimates are 2.0-6.7 and 0.7-2.1 ng/kg body weight/day, respectively. The lowest tolerable intake is 100 ng/kg body weight/week or 14.3 ng/kg body weight/day, derived by JECFA. Use of statistical techniques to determine the distribution of long-term usual exposures results in even lower estimates of 95th percentile dietary exposure. These estimates suggest that current levels of exposure to OTA by New Zealanders are of low public health concern.

Exposure to OTA is mainly through consumption of cereal products, particularly bread and pasta/noodles. Coffee is also a significant contributor to dietary OTA exposure for adult consumers.

Of the trichothecene mycotoxins, only DON and NIV were detected frequently in foods available in New Zealand. Occasional detections of T2, 15ADON and DAS did not provide sufficient data for exposure assessment.

Mean exposures to DON and NIV were highest for the 5-6-year-old child group, with exposures of 76-77 and 21.9-23.7 ng/kg body weight/day, respectively. The lowest mean exposures to DON and NIV were in the adult female group (16.6-17.1 and 3.6-4.2 ng/kg body weight/day, respectively). All exposure estimates (mean and 95th percentile) were less than 25% of the respective tolerable intakes and are of low public health concern.

Assessments of acute dietary exposure were also carried out for DON. For children, there was a very low probability (0.003%) of daily exposure exceeding the ARfD, while the probability of an adult exceeding the ARfD was so low that it was not able to be determined.

Bread and pasta/noodles were the major contributors to dietary DON exposure, with snack foods as a major contributor for children and beer a significant contributor for adult males. Dietary exposure to NIV was even more strongly dominated by the contribution from bread, with biscuits also contributing.

Quantification of uncertainty due to measurement and sampling produced credible intervals for exposure estimates that were still well within health-based exposure limits.

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APPENDIX 1 SURVEYS OF MYCOTOXINS IN FOODS AVAILABLE IN NEW ZEALAND

OCHRATOXIN A (OTA)

Food	Year of survey	Analytical limit of detection, µg/kg	Number of samples positive/ total samples (%)	Mean of positive results (range), µg/kg
Cereals and primary cereal products				
Rice	2000	0.1	0/3 (0)	
Rice, white, cooked	2011	0.2	0/8 (0)	
Flour, white	2000	0.1	0/5 (0)	
Flour, wholemeal	2000	0.1	5/7 (71)	0.26 (0.10-0.40)
Rye meal, rye flakes	2000	0.1	2/3 (67)	0.20 (0.20,0.20)
Barley; pearl, flakes	2000	0.2	1/2 (50)	0.63
Wheat; whole, puffed, kibbled	2000	0.1	2/3 (67)	0.58 (0.30,0.85)
Other cereals	2000	0.1	0/3 (0)	
Cereal products				
Bread, mixed grain	2007	0.2	0/5 (0)	
Bread, mixed grain	2011	0.2	3/8 (38)	0.34 (0.30-0.41)
Bread, wheatmeal	2007	0.2	1/3 (33)	0.20
Bread, wheatmeal	2011	0.2	2/8 (25)	0.49 (0.45-0.52)
Bread, white	2007	0.2	0/3 (0)	
Bread, white	2011	0.2	2/8 (25)	0.48 (0.40-0.55)
Biscuits, chocolate	2011	0.2	0/8 (0)	
Biscuits, cracker	2000	0.1	0/1 (0)	
Biscuits, cracker	2011	0.2	0/8 (0)	
Biscuits, sweet plain	2000	0.1	0/2 (0)	
Biscuits, sweet plain	2011	0.2	2/8 (25)	0.31 (0.20,0.42)
Breakfast cereals, cornflakes	2000	0.1-0.2	0/3 (0)	
Breakfast cereals, cornflakes	2007	0.2	2/5 (40)	0.37 (0.20-0.53)

Food	Year of survey	Analytical limit of detection, µg/kg	Number of samples positive/ total samples (%)	Mean of positive results (range), µg/kg
Breakfast cereals, cornflakes	2011	0.2	0/8 (0)	
Break cereals, rolled oats	2000	0.1	1/4 (25)	0.10
Break cereals, rolled oats	2011	0.2	0/8 (0)	
Breakfast cereals, wheat biscuits	2000	0.1	1/4 (25)	0.50
Breakfast cereals, wheat biscuits	2007	0.2	0/1 (0)	
Breakfast cereals, wheat biscuits	2011	0.2	0/8 (0)	
Breakfast cereals, muesli	2000	0.1-0.2	2/4 (50)	0.49 (0.20,0.77)
Breakfast cereals, muesli	2007	0.2	2/9 (22)	1.89 (0.92-2.85)
Breakfast cereals, muesli	2011	0.2	3/8 (38)	0.65 (0.24-1.42)
Breakfast cereals, bran-based	2000	0.1	0/2 (0)	
Breakfast cereals, bran flakes	2011	0.2	4/8 (50)	1.19 (0.20-2.99)
Breakfast cereals, other	2000	0.1-0.2	2/4 (50)	0.35 (0.20,0.50)
Cake, plain	2011	0.2	0/8 (0)	
Muffin	2011	0.2	1/8 (13)	0.33
Pasta, dry	2000	0.1	0/5 (0)	
Pasta, dry	2011	0.2	1/8 (13)	0.52
Spaghetti in sauce, canned	2011	0.2	0/8 (0)	
Noodles	2000	0.1	0/2 (0)	
Noodles, instant	2011	0.2	1/8 (13)	0.79
Snack bars	2011	0.2	2/8 (25)	0.26 (0.21,0.30)
Snacks, flavoured	2011	0.2	0/8 (0)	
Corn products, various	2000	0.1-0.2	0/9 (0)	
Pizza	2011	0.2	0/8 (0)	
Infant weaning food, cereal-based	2011	0.2	0/8 (0)	
Pulses				
Lentils	2000	0.1	1/3 (33)	0.10
Peas, chick or split	2000	0.1-0.2	0/3 (0)	

Food	Year of survey	Analytical limit of detection, µg/kg	Number of samples positive/ total samples (%)	Mean of positive results (range), µg/kg
Beans, mung, kidney, haricot, lima	2000	0.1-0.2	1/5 (20)	0.60
Baked beans, canned	2000	0.2	0/2 (0)	
Soy flour	2000	0.2	0/1 (0)	
Non-alcoholic beverages				
Coffee, instant, dry	2000	0.2	15/15 (100)	1.36 (0.3-3.5)
Coffee, instant, prepared	2011	0.05	0/8 (0)	
Coffee, roasted, ground, dry	2000	0.2-0.5	2/6 (33)	1.82 (0.94,2.70)
Coffee, roasted, ground, dry	2007	0.2	5/8 (63)	0.61 (0.28-1.48)
Coffee, brewed	2011	0.05	1/8 (13)	0.09
Other beverages (Milo, Cocoa)	2000	0.2	0/2 (0)	
Sparkling grape juice	2000	0.02	0/2 (0)	
Alcoholic beverages				
Wine, white	2000	0.02	0/6 (0)	
Wine, white	2011	0.05	0/8 (0)	
Wine, red	2000	0.02	2/9 (22)	0.36 (0.03,0.68)
Wine, red	2011	0.05	0/8 (0)	
Beer	2000	0.02	0/1 (0)	
Beer	2011	0.05	0/8 (0)	
Spices				
Cayenne pepper	2007	0.2	2/2 (100)	1.79 (0.83,2.74)
Cayenne pepper	2009	0.2	5/5 (100)	4.58 (1.79-7.27)
Chilli powder	2007	0.2	3/3 (100)	4.27 (0.23-10.7)
Chilli powder	2009	0.2	4/5 (80)	3.71 (1.30-6.34)
Curry powder	2009	0.2	5/5 (100)	1.50 (0.24-3.49)
Ginger, ground	2007	0.2	2/2 (100)	2.54 (0.60,4.48)
Ginger, ground	2009	0.2	4/5 (80)	0.97 (0.53-2.09)
Paprika	2007	0.2	4/4 (100)	32.2 (13.3-50.6)

Food	Year of survey	Analytical limit of detection, µg/kg	Number of samples positive/ total samples (%)	Mean of positive results (range), µg/kg
Paprika	2009	0.2	5/5 (100)	42.9 (14.7-102.9)
Pepper (black, white)	2009	0.2	4/5 (80)	3.02 (0.70-8.26)
Nutmeg	2007	0.2	2/2 (100)	13.9 (4.3,23.5)
Turmeric	2007	0.2	1/1 (100)	0.67
Dried fruit				
Dates	2000	0.1	0/4 (0)	
Dates	2007	0.2	1/2 (50)	1.02
Dates	2009	0.2	0/5 (0)	
Dried apricots	2000	0.1	0/3 (0)	
Dried apricots	2007	0.2	0/2 (0)	
Dried apricots	2009	0.2	0/5 (0)	
Dried vine fruit	2000	0.1-0.2	15/23 (65)	3.57 (0.30-22.0)
Dried vine fruit	2007	0.2	2/3 (67)	0.51 (0.28,0.74)
Dried vine fruit	2009	0.2	5/10 (50)	0.38 (0.21-0.63)
Figs	2000	0.1	1/2 (50)	0.20
Figs	2007	0.2	0/2 (0)	
Figs	2009	0.2	1/10 (10)	73.1
Prunes	2000	0.1	0/3 (0)	
Prunes	2007	0.2	0/1 (0)	
Prunes	2009	0.2	0/5 (0)	
Meat products				
Pâté	2000	0.1	0/3 (0)	

2000 = (Stanton, 2000) 2007 = Darren Saunders, ESR, personal communication 2009 = (Cressey and Jones, 2009) 2011 = (Cressey and Jones, 2011)

TRICHOTHECENE MYCOTOXINS

Food	Year of survey	Toxin	Analytical limit of detection, µg/kg	Number of samples positive/ total samples (%)	Mean of positive results (range), µg/kg	Reference
Breakfast cereals						
Cornflakes	1993	DON NIV	Not stated	6/7 (86) 7/7 (100)	260 (160-350) 490 (110-760)	(Lauren and Veitch, 1996)
Cornflakes	1996	DON[2] NIV	50 50	2/14 (14) 2/14 (14)	105 (100-110) 105 (80-130)	(Lauren and Veitch, 1996)
Cornflakes	2009	DON NIV	40 50	1/3 (33) 1/3 (33)	44 31[1]	(Kosanic, 2009)
Cornflakes	2009	DON[3] DAS	0.2 0.1	8/8 (100) 1/8 (13)	4.7 (2.4-9.5) 0.2	(Cressey *et al.*, 2014)
Breakfast cereals, various	1996	DON[2] NIV	50 50	0/6 (0) 1/6 (17)	- 150	(Lauren and Veitch, 1996)
Bran flake cereal	2009	DON[3]	0.3	8/8 (100)	13.7 (8.9-22)	(Cressey *et al.*, 2014)
Bran flake cereal	2014	DON[3]	0.3	4/4 (100)	8.1 (5.2-10.6)	(Cressey *et al.*, 2014)
Wheat biscuit cereal	2009	DON NIV	12 70	0/3 (0) 0/3 (0)	- -	(Kosanic, 2009)
Wheat biscuit cereal	2009	DON[3]	0.2	8/8 (100)	3.4 (2.4-5.0)	(Cressey *et al.*, 2014)
Muesli	2009	DON NIV	33 32	0/3 (0) 0/3 (0)	- -	(Kosanic, 2009)
Muesli	2009	DON[3]	0.2	8/8 (100)	3.7 (1.3-9.5)	(Cressey *et al.*, 2014)

Food	Year of survey	Toxin	Analytical limit of detection, µg/kg	Number of samples positive/ total samples (%)	Mean of positive results (range), µg/kg	Reference
Muesli	2014	DON[3]	0.2	4/4 (100)	1.4 (1.0-1.8)	(Cressey *et al.*, 2014)
Oats, rolled (uncooked)	2009	DON NIV	19 21	0/3 (0) 0/3 (0)	- -	(Kosanic, 2009)
Oats, rolled (cooked)	2009	No toxins detected[3]	-	-	-	(Cressey *et al.*, 2014)
Breads, biscuits and other baked cereal products						
Bread, various	1996	DON[2] NIV	50 50	2/16 (13) 2/16 (13)	90 (80-100) 225 (60-390)	(Lauren and Veitch, 1996)
Bread, various	2009	DON NIV	27 32	0/3 (0) 1/3 (33)	- 100	(Kosanic, 2009)
Bread, mixed grain	2009	DON[3] NIV	0.3 0.2	8/8 (100) 5/8 (63)	5.3 (2.0-10.4) 3.3 (2.7-3.8)	(Cressey *et al.*, 2014)
Bread, mixed grain	2014	DON[3]	0.3	4/4 (100)	10.1 (7.2-14.5)	(Cressey *et al.*, 2014)
Bread, wheatmeal	2009	DON[3] NIV	0.3 0.2	7/8 (88) 4/8 (50)	4.6 (2.6-7.4) 4.8 (3.5-6.6)	(Cressey *et al.*, 2014)
Bread, wheatmeal	2014	DON[3] NIV	0.3 0.2	4/4 (100) 3/4 (75)	8.3 (3.6-10.7) 3.8 (2.5-4.8)	(Cressey *et al.*, 2014)
Bread, white	2009	DON[3] NIV	0.3 0.2	6/8 (75) 3/8 (38)	3.2 (1.3-5.4) 2.8 (2.5-3.8)	(Cressey *et al.*, 2014)
Bread, white	2014	DON[3]	0.3	4/4 (100)	1.8 (1.4-2.2)	(Cressey *et al.*, 2014)
Biscuits, chocolate	2009	No toxins detected[3]	-	-	-	(Cressey *et al.*, 2014)

Food	Year of survey	Toxin	Analytical limit of detection, µg/kg	Number of samples positive/ total samples (%)	Mean of positive results (range), µg/kg	Reference
Biscuits, cracker	2009	DON[3] NIV	0.2 0.2	8/8 (100) 8/8 (100)	8.1 (4.8-22) 8.7 (5.8-13.5)	(Cressey *et al.*, 2014)
Biscuits, sweet plain	2009	DON[3] NIV	0.2 0.4	7/8 (88) 6/8 (75)	5.3 (2.4-13.0) 10.6 (6.9-16.4)	(Cressey *et al.*, 2014)
Cake	2009	DON[3] NIV	0.3 0.3	2/8 (25) 3/8 (38)	4.9 (2.0-7.8) 4.2 (2.3-7.0)	(Cressey *et al.*, 2014)
Muffin	2009	No toxins detected[3]	-	-	-	(Cressey *et al.*, 2014)
Noodles and pasta						
Noodles, instant (cooked)	2009	DON[3] NIV	0.2 0.1	7/8 (88) 4/8 (50)	9.2 (4.5-25) 1.5 (1.2-1.8)	(Cressey *et al.*, 2014)
Pasta, dried (cooked)	2009	DON[3] NIV	0.2 0.1	8/8 (100) 1/8 (13)	16.8 (3.0-38) 1.7	(Cressey *et al.*, 2014)
Spaghetti in sauce, canned	2009	DON[3] NIV	0.3 0.1	6/8 (75) 3/8 (38)	10.4 (5.6-22) 1.6 (1.1-2.6)	(Cressey *et al.*, 2014)
Rice						
Rice (uncooked)	2009	DON NIV	18 19	0/3 (0) 1/3 (33)	- 28	(Kosanic, 2009)
Rice, white (cooked)	2009	NIV[3] DAS	0.2 0.3	1/8 (13) 1/8 (13)	3.9 1.2	(Cressey *et al.*, 2014)
Snack foods						
Snack bars	1996	DON[2] NIV	50 50	0/13 (0) 3/13 (23)	- 87 (60-120)	(Lauren and Veitch, 1996)
Snack bars	2009	DON[3] NIV	0.2 0.3	1/8 (13) 1/8 (13)	2.5 2.5	(Cressey *et al.*, 2014)

Food	Year of survey	Toxin	Analytical limit of detection, µg/kg	Number of samples positive/ total samples (%)	Mean of positive results (range), µg/kg	Reference
Extruded snack foods	1996	DON[2] NIV	50 50	0/20 (0) 1/20 (5)	- 50	(Lauren and Veitch, 1996)
Extruded snack foods	2009	DON[3] NIV	0.2 0.3	4/5 (80) 3/5 (60)	6.7 (3.4-9.0) 5.5 (5.0-6.1)	(Cressey *et al.*, 2014)
Extruded snack foods	2014	DON[3] NIV	0.2 0.3	2/2 (100) 1/2 (50)	1.4 (1.1-1.7) 7.4	(Cressey *et al.*, 2014)
Corn chips	1996	DON[2] NIV	50 50	1/24 (4) 1/24 (4)	90 60	(Lauren and Veitch, 1996)
Corn chips	2009	DON[3] NIV 15ADON T2	0.2 0.3 6 0.04	3/3 (100) 2/3 (67) 1/3 (33) 1/3 (33)	166 (26-410) 6.8 (5.0-8.6) 41 0.4	(Cressey *et al.*, 2014)
Corn chips	2014	DON[3] NIV	0.2 0.3	2/2 (100) 1/2 (50)	2.5 (2.4-2.6) 10.1	(Cressey *et al.*, 2014)
Pizza	2009	DAS[3]	0.2	1/8 (13)	0.3	(Cressey *et al.*, 2014)
Other foods						
Corn oil	1996	DON[2] NIV	50 50	0/8 (0) 0/8 (0)	- -	(Lauren and Veitch, 1996)
Corn meal/grits/flour	1996	DON[2] NIV	50 50	7/14 (50) 6/14 (43)	207 (50-410) 324 (60-650)	(Lauren and Veitch, 1996)
Cornflour	2009	DON NIV	14 25	1/3 (33) 1/3 (33)	108 112	(Kosanic, 2009)
Cereal-based infant weaning food	2009	NIV[3]	0.3	1/8 (13)	4.6	(Cressey *et al.*, 2014)

Food	Year of survey	Toxin	Analytical limit of detection, µg/kg	Number of samples positive/ total samples (%)	Mean of positive results (range), µg/kg	Reference
Beer	2009	DON[3]	0.4	1/8 (13)	10.9	(Cressey *et al.*, 2014)

DON = deoxynivalenol, NIV = nivalenol, T2 = T-2 toxin, HT2 = HT-2 toxin, NEO = neosolaniol, DAS = diacetoxyscirpenol, FX = Fusarenon X, 3ADON = 3-acetyldeoxynivalenol, 15ADON = 15-acetyldeoxynivalenol

[1] Result was reported even though it was below the LOD given
[2] Individual analytical results were reported in this study. For some samples, duplicate results were reported and for some of these one duplicate was quantified, while one was 'not detected'. In the current summary, such samples are represented as the quantified result
[3] The survey included analyses for DON, 3ADON, 15ADON, NIV, FX, T2, HT2, DAS and NEO. If any of these toxins are not listed in the table in relation to a food then it was not detected in that food.

APPENDIX 2 PROCEDURE FOR DETERMINING THE PROPORTION OF MYCOTOXIN-CONTAINING FOODS IN RECIPES

Sources of recipes

No single standard source for recipes exists. In the absence of such a resource, the recipes used in a database must be selected based on a pre-determined strategy. While such a strategy may be discussed and even criticised, its existence provides a methodology than can be followed for subsequent additions and can be utilised by other parties. The following sources of recipes have been identified:

- New Zealand Food Composition Database. Contains recipes for 272 foods (in the version of Food Files currently held by ESR). Not all of these are true recipes, as some describe how food descriptors have been combined to produce food composition information for other descriptors. Recipes are expressed as the percentage of the ingredient in the food.
- McCance and Widdowson's The Composition of Foods (this is essentially the British equivalent of the food composition database) contains recipes for 103 foods (Holland *et al.*, 1991). Recipes are expressed in terms of the weight of the ingredients plus an estimate of the weight loss upon cooking, where relevant.
- The National Nutrition Survey (Russell *et al.*, 1999) and National Children's Nutrition Survey (Ministry of Health, 2003) 24-hour dietary recall studies include recipes, where these were provided by respondents. These have already been integrated into our working version of the database, but could be used as a resource to define recipes for situations where recipes were not provided by respondents. Recipes are in the form of the weight of the ingredients.
- Recipes used in conjunction with the USDA Nutrient database for nationwide food surveys 2007 is available on-line[4]. Recipes are expressed as percentages of ingredients in final foods.
- Various cookbooks and internet resources. Express ingredients in terms of weights or standard measures.

Yield Factors

For many recipes, particularly cooked recipes, the final weight of the prepared recipe will be different from the sum of the weights of the (uncooked) ingredients. The ratio of these two weights is often referred to as a yield factor. Weight changes during cooking mainly relate to gains or losses in moisture (Bergstrom, 1999).

Unfortunately, the form of the calculations carried out for food composition purposes is opposite to that required for management of recipes in a food consumption database. Our interest is generally in deconvoluting from a cooked composite food to uncooked ingredients. In this case the sum of the weights of the individual ingredients would be expected to be equal to or greater than the weight of the composite food. However, different ingredients will differ in their moisture content and would be expected to lose differing amounts of their initial weights during the cooking process.

[4] http://www.nal.usda.gov/fnic/foodcomp/Data/SurveyNDB7/

Utility of yield factor information will depend on the form of other information available. The following scenarios are envisaged:

- Consumed weight of recipe available. Yield factor available. Ingredient composition available in terms of standard measures. Calculate total weight of ingredients from recipe. Calculate total weight of ingredients from consumed weight and yield factor. Scale weight of ingredients to uncooked weight of prepared food.
- Consumed weight of recipe available. Yield factor available. Percentage figures available for recipe ingredients. If percentage refers to uncooked weight, use consumed weight and yield factor to determine uncooked weight then apply percentages. If percentage refers to cooked weight, then composition of uncooked recipe cannot be determined. However, this scenario is unlikely.

Procedure for Application of Recipes in Food Consumption Datasets

The following procedure is largely based on that of Reinivuo *et al.* (Reinivuo *et al*., 2009), although it has been modified to accommodate different formats of information. The two formats encountered are:

- Classical recipes, where the ingredients are listed in terms of weights or measures; and
- Database recipes, where the ingredients are listed in terms of percentages of the finished recipe.

The procedure is:

- Identify recipe from a source listed in the section 'Source of recipes'. Wherever possible, priority should be given to New Zealand sources. However, priority should be given to systematic sources of recipes over *ad hoc* sources (e.g. choose NZ Food Composition Database recipe before internet recipe).
- If recipe is in the form of percentages, apply directly.
- If recipe is in the form of weights and measures, convert all measures (cups, tablespoons, etc.) to weights using standard weights per measure (see 'CSM' file in the most recent version of Foodfiles held at ESR, currently Foodfiles 2006) or standard volumes of measures and density values for the ingredient. Standard volumes are listed in Attachment 1 and densities of food items can be found in the CSM file in Foodfiles.
- Convert weights to percentages.

So, to convert a weight of a final recipe to the weight of its ingredients:

- Take weight of final recipe.
- If recipe is cooked or processed otherwise in a manner that will cause a weight change, apply the inverse of the appropriate yield factor to give the total weight of ingredients. For example, if the final weight is 500 g and yield tables indicate that the recipe loses 9% of its weight through cooking, the weight of the ingredients is 500 x (100/100-9) or 500/0.91. This gives a weight of 549 g.
- Apply percentages determined above to give the weight of ingredients.

Examples

The NNS contains entries for Macaroni cheese (with or without added meat). A common serving size is 506 g. The Food files (New Zealand Food Composition Database) give a percentage recipe for Macaroni cheese:

- Milk, fluid, standard 45
- Macaroni, boiled 36
- Cheese processed 13
- Butter, salted 3
- Flour, wheat, white, standard 3
- Salt 0

European yield tables give a 9% weight loss for macaroni cheese on cooking (Bergstrom, 1999). For a serving of 506 g, the uncooked weight would be 506/0.91 = 556 g. The weight (g) of the uncooked ingredients would be:

- Milk, fluid, standard 250
- Macaroni, boiled 200
- Cheese processed 72
- Butter, salted 17
- Flour, wheat, white, standard 17
- Salt 0

McCance and Widdowson (Holland *et al.*, 1991) gives a recipe for macaroni cheese of:

- 350 ml milk
- 280 g cooked macaroni
- 100 g grated cheese
- 25 g margarine
- 25 g flour
- 0.5 tsp salt

Weight loss is 9.4%. Excluding salt and assuming a density of 1 g/ml for milk, the total weight of ingredients is 780 g, with a cooked weight equivalent of 707 g. For a 506 g serving the scale factor is 506/707 = 0.716. Applying this to the original recipe gives:

- Milk 251
- Cooked macaroni 200
- Grated cheese 72
- Margarine 18
- Flour 18

It appears probably that these two expressions of the recipe for macaroni cheese are from the same primary source.

Using a more challenging source for the recipe (an internet source) of macaroni cheese (ingredients list was truncated for simplicity):

- 2 cups milk
- 2 cups macaroni, cooked
- 2 cups grated cheese

- 2 TB butter
- 2 TB flour

The 'csm' file in Foodfiles contains weights of standard measures for foods in the database. Another useful resource is the USDA measurement conversion tables: http://www.ars.usda.gov/Aboutus/docs.htm?docid=9617

For this exercise the following are relevant:

- Milk. CSM gives a weight of 15.5 g/tablespoon for standard, fluid milk. USDA gives a conversion of 16 tablespoons per cup. 2 cups = 500 g
- Macaroni. CSM doesn't give the weight of a cup of cooked macaroni, but does give a density 0.596 g/ml. Therefore, 2 cups (500 ml) would be expected to weigh 300 g.
- Cheese. CSM gives the weight of a cup of shredded Gruyere cheese as 119 g. This is similar to using the density of cheddar cheese (0.47 g/ml) and the volume of a standard cup (250 ml). Therefore, 2 cups of grated cheese will weigh approximately 240 g.
- Butter. CSM gives the weight of a tablespoon of salted butter as 15 g. Therefore, 2 tablespoons will weigh 30 g.
- Flour. CSM gives the density of standard white flour as 0.489 g/ml. A tablespoon is approximately 15.5 ml giving a weight for 2 tablespoons of flour of 15 g.

Total weight of this recipe is 1085 g, corresponding to a cooked weight (-9%) of 987 g and a conversion factor for a 506 g serving of 506/987 = 0.513. The recipe weights equating to a 506 g serving, based on this recipe are:

- Milk 257 g
- Macaroni 154 g
- Cheese 123 g
- Butter 15 g
- Flour 8 g

These figures differ from those above, but are generally still recognisable.

APPENDIX 3 MAPPING OF FOODS FOR WHICH MYCOTOXIN CONCENTRATION INFORMATION WAS AVAILABLE TO NATIONAL NUTRITION SURVEY FOODS

Food group for which mycotoxin data are available[1]	NNS foods mapped
Cereal and cereal products	
Bread, mixed grain	All mixed or multi-grain breads and bread rolls, including breads containing whole or chopped seeds (e.g. linseed), sandwiches made from mixed or multi-grain bread
Bread, wheatmeal	All wheatmeal or wholemeal breads and bread rolls, including fruit breads and rye breads, all filled rolls made from wheatmeal or wholemeal rolls, hot cross buns, sandwiches made from wheatmeal or wholemeal bread
Bread, white	All white breads and bread rolls, including bagels, croissants, panini, pita bread and 'other' bread (unless specifically identified as not white bread), sweet buns and rolls, breadcrumbs, hamburger buns, bread or breadcrumb-containing recipes (e.g. asparagus rolls, meatloaf), all filled rolls made from white rolls, sandwiches made from white bread, wraps
Biscuits, chocolate	All chocolate or chocolate-coated biscuits
Biscuits, cracker	All cracker biscuits, crispbreads, cabin breads and wafers
Biscuits, sweet plain	All non-chocolate sweet biscuits, including filled biscuits, cheesecake (base), biscuit-based slices
Cornflakes	All corn flake-based breakfast cereals, all other puffed or extruded breakfast cereals
Bran flake cereal	All bran-based breakfast cereals, bran as a separate descriptor
Muesli	All toasted and untoasted mueslis, including 'lite' style mueslis
Oats, rolled	All porridge or cooked oat breakfast cereals, all other cooked cereal breakfast cereals
Cake, plain	All cakes, loaves and cake-like desserts (e.g. sponge desserts)
Muffin	All baked cereal products, not included under other descriptors, including muffins, crumpets, doughnuts, lamingtons, pancakes, pikelets, scones and slices
Noodles, instant	All noodles and noodle-containing recipes (e.g. chow mein)
Pasta	All types of pasta and pasta-containing recipes, all pastry and pastry-containing products, including dumpling, pies (pastry case), quiche (pastry base types only), samosa, savouries with a pastry base, spring rolls and wontons
Rice, white	All rice and rice-containing recipes
Spaghetti in sauce, canned	All canned pasta products
Snack bars	All snack bars, but not chocolate bars
Snacks, flavoured	All extruded or formed cereal-based snack foods (e.g. burger rings, pretzels), popcorn and masa flour products (e.g. corn chips, taco shells)

Food group for which mycotoxin data are available[1]	NNS foods mapped
Pizza	All pizzas and pizza bases
Alcoholic beverages	
Beer	All beer
Wine, red	All red wines, including fortified red wines (e.g. port)
Wine, white	All white and rosé wines, including sparkling and saki (rice wine)
Non-alcoholic beverages	
Coffee, brewed	All coffee brewed from ground beans
Coffee, instant	All prepared coffee from dry powder or paste
Coffee, instant, dry powder	All dry coffee powders or granules, coffee pastes and other concentrates
Spices	
Cayenne pepper	Various recipes
Chilli powder	Various recipes
Paprika	Various recipes
Curry powder	Various recipes
Ginger, ground	Various recipes
Pepper (black, white)	Various recipes, excluding discretionary pepper added at table
Dried fruits	
Dates	Dates, date scones, date pudding
Dried apricots	Dried apricots, apricots cooked from dry
Dried vine fruits	Raisins; sultanas; currants; mixed fruit; fruit mince; vine fruit-containing bakery products, vine fruit-containing recipes
Dried figs	Dried figs
Prunes	Prunes, dried, cooked prunes , plums, dried, prune-containing recipes

[1] All mappings are relevant for OTA, while only mapping related to cereal-based foods, including beer, are relvant for the trichothecene mycotoxins

APPENDIX 4 CONTRIBUTION OF FOOD GROUPS TO MEAN DIETARY MYCOTOXIN EXPOSURE

Ochratoxin A (OTA)

Age-gender group	Contribution of food group to estimated dietary exposure (%), based on lower bound – upper bound concentration estimates				
	Cereals	Beverages, alcoholic	Beverages, non-alcoholic	Dried fruit	Spices
Child (5-6 years)	91.9 – 97.7	0.0 – 0.0	0.0 – 0.0	0.4 – 0.9	1.8 – 7.1
Female (11-14 years)	88.4 – 96.8	0.0 – 0.0	1.2 – 4.3	0.3 – 0.7	1.7 – 6.5
Male (11-14 years)	92.7 – 98.0	0.0 – 0.0	0.2 – 0.9	0.4 – 1.1	1.3 – 5.3
Male (19-24 years)	78.8 – 83.1	0.0 – 9.6	3.9 – 7.1	0.2 – 0.5	3.2 – 13.6
Female (25+ years)	70.1 – 80.9	3.1 – 6.0	9.0 – 15.6	2.2 – 5.0	1.9 – 6.2
Male (25+ years)	71.4 – 75.2	3.5 – 14.5	6.9 – 13.9	1.4 – 3.7	1.9 – 7.4

Deoxynivalenol (DON)

Age-gender group	Contribution of food group to estimated dietary exposure (%), based on lower bound – upper bound concentration estimates					
	Bread	Biscuits	Breakfast cereals	Pasta and noodles	Other cereal products	Beer
Child (5-6 years)	27.5-27.8	5.1-5.1	1.1-1.7	38.3-38.9	27.0-27.3	0.0-0.0
Female (11-14 years)	24.3-24.5	5.6-5.6	0.5-1.0	40.3-40.9	28.4-28.8	0.0-0.0
Male (11-14 years)	28.9-29.2	5.3-5.4	1.2-1.9	44.0-44.8	19.3-19.9	0.0-0.0
Male (19-24 years)	17.7-18.2	2.0-2.0	2.9-3.0	51.3-53.5	11.3-11.7	11.9-14.4
Female (25+ years)	30.8-31.5	5.3-5.5	5.3-5.4	41.6-42.8	10.2-11.0	4.8-5.8
Male (25+ years)	26.7-28.1	3.4-3.6	4.1-4.1	36.4-38.7	6.5-7.0	19.0-22.4

Nivalenol (NIV)

Age-gender group	Contribution of food group to estimated dietary exposure (%), based on lower bound – upper bound concentration estimates					
	Bread	Biscuits	Breakfast cereals	Pasta and noodles	Other cereal products	Beer
Child (5-6 years)	46.0-47.5	29.3-31.2	0.0-1.8	6.7-7.0	14.7-15.9	0.0-0.0
Female (11-14 years)	41.4-42.3	32.0-34.2	0.0-1.2	6.2-6.5	17.4-18.8	0.0-0.0
Male (11-14 years)	46.1-47.7	28.9-31.1	0.0-1.9	6.2-6.5	15.0-16.6	0.0-0.0
Male (19-24 years)	40.3-47.0	14.7-19.1	0.0-2.0	10.9-12.1	21.5-21.8	0.0-10.5
Female (25+ years)	41.8-44.5	29.5-34.0	0.0-2.7	5.8-5.9	15.7-17.4	0.0-2.7
Male (25+ years)	41.2-48.5	23.0-29.4	0.0-2.4	6.2-6.9	15.1-15.1	0.0-12.1

APPENDIX 5 MYCOTOXIN CONCENTRATION VALUES USED IN THE CURRENT STUDY AND THEIR ASSOCIATED CREDIBLE INTERVALS, CONSIDERING MEASUREMENT AND SAMPLING UNCERTAINTY

Ochratoxin A (OTA)

Food group	Mean ochratoxin A concentration, µg/kg (95th percentile credible interval)	
	Lower bound	Upper bound
Bread, mixed grain	0.069 (0.000-0.142)	0.229 (0.174-0.284)
Bread, wheatmeal	0.105 (0.007-0.203)	0.244 (0.164-0.324)
Bread, white	0.073 (0.000-0.170)	0.242 (0.162-0.322)
Biscuits, chocolate	0.000 (0.000-0.049)	0.200 (0.137-0.264)
Biscuits, cracker	0.000 (0.000-0.049)	0.200 (0.137-0.264)
Biscuits, sweet plain	0.062 (0.000-0.154)	0.202 (0.119-0.285)
Cornflakes	0.070 (0.000-0.148)	0.226 (0.169-0.282)
Bran flake cereal	0.583 (0.109-1.06)	0.667 (0.220-1.11)
Wheat biscuit cereal	0.000 (0.000-0.035)	0.171 (0.130-0.213)
Muesli	0.330 (0.086-0.575)	0.457 (0.234-0.680)
Oats, rolled	0.000 (0.000-0.049)	0.200 (0.151-0.249)
Cake, plain	0.000 (0.000-0.049)	0.200 (0.151-0.250)
Muffin	0.041 (0.000-0.133)	0.216 (0.158-0.275)
Noodles, instant	0.099 (0.000-0.292)	0.274 (0.125-0.422)
Pasta, dried	0.065 (0.000-0.198)	0.240 (0.149-0.331)
Rice, white	0.000 (0.000-0.049)	0.200 (0.151-0.250)
Spaghetti in sauce, canned	0.000 (0.000-0.049)	0.200 (0.151-0.249)
Snack bars	0.064 (0.000-0.158)	0.214 (0.159-0.268)
Snacks, flavoured	0.000 (0.000-0.049)	0.200 (0.150-0.249)
Pizza	0.000 (0.000-0.049)	0.200 (0.150-0.249)
Infant weaning food, cereal based	0.000 (0.000-0.049)	0.200 (0.150-0.250)
Wine, still red	0.042 (0.000-0.118)	0.074 (0.000-0.147)
Wine, still white	0.000 (0.000-0.035)	0.037 (0.002-0.072)
Beer	0.000 (0.000-0.045)	0.047 (0.001-0.092)
Coffee, brewed	0.011 (0.000-0.065)	0.055 (0.005-0.105)
Coffee, instant	0.000 (0.000-0.049)	0.050 (0.001-0.099)
Coffee, instant, dry powder	1.36 (0.906-1.81)	1.36 (0.906-1.81)
Dried Figs	6.09 (0.000-17.0)	6.27 (0.000-17.2)
Dried Vine Fruits	0.229 (0.097-0.361)	0.322 (0.227-0.415)
Dried Apricots	0.000 (0.000-0.038)	0.200 (0.161-0.238)
Dried Dates	0.227 (0.000-0.510)	0.382 (0.153-0.612)
Dried Prunes	0.000 (0.000-0.054)	0.200 (0.146-0.254)
Pepper (black, white)	2.42 (0.000-5.61)	2.46 (0.000-5.62)
Chilli Powder	4.02 (1.70-6.34)	4.04 (1.74-6.35)
Cayenne pepper	3.34 (1.87-4.80)	3.34 (1.87-4.80)

Food group	Mean ochratoxin A concentration, µg/kg (95th percentile credible interval)	
	Lower bound	Upper bound
Paprika	37.2 (22.6-51.6)	37.2 (22.6-51.6)
Ginger	1.56 (0.467-2.65)	1.58 (0.506-2.66)
Curry Powder	1.50 (0.000-3.04)	1.50 (0.000-3.03)

Deoxynivalenol (DON)

Food group	Mean DON concentration, µg/kg (95th percentile credible interval)	
	Lower bound	Upper bound
Bread, mixed grain	6.81 (4.95-8.92)	6.81 (4.95-8.92)
Bread, wheatmeal	5.45 (3.76-7.18)	5.48 (3.80-7.19)
Bread, white	2.21 (1.42-3.02)	2.26 (1.51-3.02)
Biscuits, chocolate	0.00 (0.00-0.05)	0.20 (0.15-0.26)
Biscuits, cracker	8.11 (4.10-12.13)	8.11 (4.10-12.13)
Biscuits, sweet plain	4.63 (2.12-7.16)	4.66 (2.16-7.17)
Cornflakes	4.70 (3.21-6.22)	4.70 (3.21-6.22)
Bran flake cereal	11.84 (9.45-14.28)	11.84 (9.45-14.28)
Wheat biscuit cereal	3.49 (2.89-4.10)	3.49 (2.89-4.10)
Muesli	2.91 (1.68-4.17)	2.91 (1.68-4.17)
Oats, rolled	0.00 (0.00-0.08)	0.30 (0.22-0.38)
Cake, plain	1.22 (0.00-3.08)	1.45 (0.00-3.21)
Muffin	0.00 (0.00-0.05)	0.20 (0.15-0.25)
Noodles, instant	8.05 (2.26-13.87)	8.08 (2.32-13.87)
Pasta, dried	16.75 (8.45-25.12)	16.75 (8.45-25.12)
Spaghetti in sauce, canned	7.83 (2.84-12.89)	7.91 (2.97-12.94)
Rice, white	0.00 (0.00-0.05)	0.20 (0.15-0.25)
Snack bars	0.31 (0.00-0.91)	0.49 (0.00-1.03)
Snacks, flavoured	44.31 (0.00-105.7)	44.32 (0.00-105.7)
Pizza	0.00 (0.00-0.08)	0.30 (0.22-0.38)
Infant weaning food, cereal based	0.00 (0.00-0.16)	0.60 (0.44-0.76)
Beer	1.36 (0.00-3.95)	1.71 (0.00-4.21)

Nivalenol (NIV)

Food group	Mean DON concentration, µg/kg (95th percentile credible interval)	
	Lower bound	Upper bound
Bread, mixed grain	1.37 (0.48-2.27)	1.48 (0.65-2.34)
Bread, wheatmeal	2.57 (1.28-3.89)	2.66 (1.42-3.93)
Bread, white	0.70 (0.03-1.38)	0.85 (0.23-1.49)
Biscuits, chocolate	0.00 (0.00-0.11)	0.40 (0.29-0.51)
Biscuits, cracker	8.69 (6.75-10.81)	8.69 (6.75-10.81)
Biscuits, sweet plain	7.94 (4.15-11.85)	8.04 (4.32-11.84)
Cornflakes	0.00 (0.00-0.08)	0.30 (0.22-0.39)
Bran flake cereal	0.00 (0.00-0.10)	0.50 (0.39-0.61)
Wheat biscuit cereal	0.00 (0.00-0.10)	0.40 (0.29-0.51)
Muesli	0.00 (0.00-0.08)	0.40 (0.32-0.49)
Oats, rolled	0.00 (0.00-0.05)	0.20 (0.15-0.26)
Cake, plain	1.58 (0.00-3.32)	1.77 (0.15-3.41)
Muffin	0.00 (0.00-0.05)	0.20 (0.15-0.26)
Noodles, instant	0.74 (0.19-1.30)	0.79 (0.28-1.32)
Pasta, dried	0.21 (0.00-0.62)	0.30 (0.00-0.68)
Spaghetti in sauce, canned	0.78 (0.15-1.43)	0.83 (0.23-1.45)
Rice, white	0.49 (0.00-1.43)	0.67 (0.00-1.57)
Snack bars	0.31 (0.00-0.92)	0.57 (0.05-1.11)
Snacks, flavoured	3.97 (2.00-6.02)	4.10 (2.19-6.05)
Pizza	0.00 (0.00-0.03)	0.10 (0.07-0.13)
Infant weaning food, cereal based	0.58 (0.00-1.71)	0.84 (0.00-1.90)
Beer	0.00 (0.00-0.05)	0.20 (0.15-0.26)