Quality Assurance and Safety of Crops & Foods, 2015; 7 (1): 63-72
SPECIAL ISSUE: *Food safety in Asia*

Fumonisins in China: update on occurrence, epidemiology, exposure and regulation

J. Chen, Z. Jia, J. Song, Y. Yuan and L. Zhang*

Sichuan University, West China School of Public Health, 16# Third Section, Renmin South Road, Chengdu, Sichuan 610041, China P.R.; lishizhang_56@163.com

Received: 6 December 2012 / Accepted: 19 April 2013

REVIEW ARTICLE

Abstract

Fumonisins (FBs) are one of the most frequently occurring mycotoxins in China. FBs have attracted worldwide attention because of the significant economic losses they cause as well as their impact on human health and animal productivity. FBs have been associated with high incidences of oesophageal and liver cancer in several areas in China. However, the extrapolation of the effects of FBs on humans has always been inaccurate, which can be explained by the lack of adequate food consumption data, lack of knowledge about related health risks associated with specifically proposed limits, and the possibility of synergism with other mycotoxins present in the same food commodities. Domestic legislation is necessary to control contamination by FBs. The assessment of fumonisin exposure and development of preventative strategies in China depend not only on the determination of fumonisin levels in food contributors but also on the distinct dietary patterns of a specific population. Although limited data is available, this review aims to critically evaluate the extent of fumonisin contamination in China as well as epidemiologic studies and human exposure estimates. This review also aims to establish perspectives to aid future research and legislation.

Keywords: China, contamination, exposure, fumonisins, legislation

1. Introduction

Ensuring food safety has always been the focus in the recent decades. The contamination of food or animal feed by mycotoxins among other chemical hazards has been characterised by the World Health Organization (WHO) as a significant source of food-borne diseases (WHO, 2002). Mycotoxins currently present a major food safety issue in China. Such issue is aggravated by the large distribution of grain-producing areas across mostly subtropical and temperate zones. *Fusarium verticillioides* is one of the most prevalent fungi associated with the contamination of maize throughout the world. Fumonisins (FBs) were first isolated from cultures of *F. verticillioides* strain MRC 826 (Gelderblom *et al.*, 1988; Marasas *et al.*, 1988). FBs have been frequently detected in maize and/or maize-based food. FBs form chemically related groups, including at least 15 identified members among which FB_1 is the most frequently detected contaminant. FB_1 accounts for 70 to 80% of the total FBs content in *F. verticilioides* cultures and naturally contaminated foods. The co-contamination of FB_1-containing samples by small amounts of other mycotoxins, mainly FB_2 and FB_3, is common (WHO, 2000). FB_1 was proven to be responsible for most of the toxicological effects of *F. verticillioides* culture materials on animals (JECFA, 2001; WHO, 2001).

The kinetics and metabolism of FBs have not been clearly illustrated to date. FBs are poorly absorbed, rapidly eliminated via a two- or three-compartment model, and not metabolised in animals (JECFA, 2001; WHO, 2001). FBs cannot be metabolised by cytochrome P450 enzymes. However, FBs have been proven to alter the activity of these enzymes by interrupting sphingolipid biosynthesis through structurally related sphingoid bases. The elimination of FB_1 in mice can be done rapidly; however, given that elimination rate is a function of body weight, the elimination of FB_1 in humans could be much longer (Van der Westhuizen *et al.*, 1999).

FB_1 has been associated with animal mycotoxicoses such as equine leukoencephalomacia (Kellerman *et al.*, 1990;

Marasas *et al.*, 1988) and porcine pulmonary edema (Harrison *et al.*, 1990). Subsequent studies on laboratory animals revealed a relationship between FB_1 intake and hepatotoxicity, nephrotoxicity, as well as developmental and reproductive toxicity (Gelderblom *et al.*, 2001; Marasas *et al.*, 2004; NTP, 2001; Riley *et al.*, 1994; Voss *et al.*, 1995). Nephrotoxicity and renal carcinogenicity were identified as the most sensitive endpoints of toxicity for FB_1 in rodent bioassays conducted by the National Toxicology Program (NTP, 2001). The genotoxicity of FBs is under debate, although the lack of DNA interactive reactivity in different short-term genotoxicity assays utilizing bacteria (Gelderblom and Snyman, 1991) and *in vivo* and *in vitro* DNA repair assays in rat liver and primary hepatocytes (Gelderblom *et al.*, 1992; Norred *et al.*, 1992) is generally accepted. No direct interaction of FB_1 with oligonucleotides has been detected (Pocsfalvi *et al.*, 2000). The International Agency for Research on Cancer (IARC) evaluated FB_1 to determine if it should be classified as a carcinogenic compound. Based on animal studies and cases of human oesophageal cancer, IARC evaluated FB_1 as possibly carcinogenic to humans (group 2B) (IARC, 1993). FBs contamination has been associated with impact on human including as a risk factor for oesophageal cancer.

FBs have been frequently detected in maize in all parts of the world (WHO, 2001). The occurrence of FB_1 in maize imported from 18 countries worldwide in the Netherlands demonstrated the presence of this mycotoxin in 93% of the samples (with average level of 1,359 ng/g) (De Nijs *et al.*, 1998). Several new sources of FBs have been reported recently, including cornflakes (Castells *et al.*, 2008), the Portuguese maize bread broa (Lino *et al.*, 2006), black and herbal tea as well as medicinal plants (Martins *et al.*, 2001; Omurtag and Yazicioglu, 2004), and spices and flavours (star aniseed, chili, pepper, garlic, cinnamon, paprika, ginger, and parsley) in China (Shi *et al.*, 2003). Beer and other plant-based alcoholic drinks are also considered contributors to FBs exposure (Pietri *et al.*, 2010; Scott and Lawrence, 1995; Shale *et al.*, 2012; Tamura *et al.*, 2011, 2012). FBs contamination in grain-based beer is an important concern because to date, no investigation has been conducted on this topic in China.

Legislation on FBs is essential to protect public health and avoid trade barriers. No wide-scale limits for FBs have been established. The complexity of developing regulations and/or standards designed to protect consumer health is reflected in attempts to explore accurate exposure estimates (Lineback *et al.*, 2009). Considering that maize and maize-based food commodities such as biscuits and beer are commonly consumed in China (Sun *et al.*, 2007, 2011), research must be conducted to determine whether the contamination of these commodities by FBs results in considerable human exposure to this mycotoxin. Likewise, insights into the development of new standards must be offered.

This review aims to thoroughly examine the occurrence of FBs in maize and different food commodities for human consumption. This review also aims to evaluate human epidemiologic studies and exposure estimates conducted in China to provide perspectives to future research.

2. Occurrence

International databases (Pubmed, Embase and Web of Science) and Chinese literature databases (China Academic Journal Network Publishing Database and CBMdisc) were searched for relevant literature. The available reports on the occurrence of FBs in food are summarised in Supplementary Table S1. The table shows that information on annual variation in FBs concentrations in maize harvested in consecutive years is limited. However, the table clearly shows that considerable variation can occur with annual/seasonal climate fluctuation.

The contamination levels of FBs in food vary according to inherent *Fusarium* species contamination and environmental (climate, soil type, rainfall, and season) and processing (storage time/temperature and methods of preservation/preparation) factors. The high levels of FB_1 found in maize in Sichuan and Guangxi (Gong *et al.*, 2009) are in agreement with the meteorological data of these two provinces (moderate mean temperature and high relative humidity and rainfall) (Supplementary Table S1), which are also in agreement with the data obtained by Hennigen *et al.* (2000) who discovered an association between high levels of FBs in maize and relatively high humidity in Argentina. The most recent survey conducted in 2010 (Feng *et al.*, 2011) indicate that the mean concentration for all maize samples is 1,318 μg/kg for FB_1 and 319 μg/kg for FB_2. These values are lower than those obtained by Gong *et al.* (2009), which indicate that all maize samples except one are positive for FB_1 at levels varying from 3 μg/kg to 71,121 μg/kg with mean level of 6,662 μg/kg, but higher than those reported in Croatia (459.8 μg/kg for FB_1) (Domijan *et al.*, 2005), Spain (22 μg/kg for FB_1 and 43 μg/kg for FB_2) (Arino *et al.*, 2007), and Portugal (329 μg/kg for FB_1 and 131 μg/kg for FB_2) (Lino *et al.*, 2006).

The summarised reports in Supplementary Table S1 also reveal that data on co-contamination of FBs and other mycotoxins are quite rare albeit the possibility of synergistic or additive toxic effects on experimental animals. Therefore, further monitoring of FBs in China is essential to cover as many food contributors as possible and to illustrate seasonal and regional contamination trends.

3. Association with human diseases

Oesophageal cancer

The association between the consumption of maize infected by *F. verticillioides* and oesophageal cancer was first established in the former Transkei region in South Africa (Rheeder *et al.*, 1992; Sydenham *et al.*, 1990) and later reported in northern Italy (Franceschi *et al.*, 1990) and Iran (Shephard *et al.*, 2000). Studies exploring the association of FBs contamination with oesophageal cancer were conducted in China over the last decades; the results are shown in Supplementary Table S2. Chu and Li (1994) study established that homegrown maize in areas in China with high incidence of oesophageal cancer may be contaminated with extremely high levels of FB_1. In Chu and Li (1994) study, maize samples from Linxian and Cixian, areas with high incidence of oesophageal cancer, were analysed to determine the presence of various mycotoxins. High levels of FB_1 were found in the mouldy samples; however, no control condition was set in the study. Yoshizawa *et al.* (1994) comparative study of maize samples from Linxian and Shangqiu in the same province indicated that no significant differences exist in the FB_1 contamination level in the two areas (with high contamination incidence of 48% vs. 25%), which was further confirmed in a subsequent study (Gao and Yoshizawa, 1997) that investigated wheat and maize. The samples collected in the two studies were also frequently co-contaminated by trichothecenes whose concentration was significantly higher in Linxian than in Shangqiu. Zearalenone was detected only in the maize samples from Linxian.

Samples of maize, cornmeal, unfermented and fermented pancake batter, and cooked fermented pancakes from Linqu County, an area in China with high incidence of gastric cancer, were analysed to explore the association between consumption of FBs-contaminated maize and gastric cancer. The results did not support the hypothesis that mycotoxin contamination in Chinese fermented maize-based food increases the risk of gastric cancer (Groves *et al.*, 1999).

The association of FB_1 with oesophageal cancer in Huaian (an area with high incidence of oesophageal cancer) and Huantai (an area with low incidence of oesophageal cancer) was investigated by surveys conducted in 2001 to 2002 and 2010 (Sun *et al.*, 2007, 2011). Supplementary Table S2 shows that in 2001, the FB_1 contamination frequency was 95.7% in Huaian and 83.3% in Huantai. The level of FB_1 contamination in maize samples from Huaian was significantly higher than that of the samples from Huantai. Furthermore, 42.0% of the positive Huaian samples had an FB_1 level higher than 2,000 μg/kg, which was significantly higher than the 10.0% FB_1 level of the Huantai samples. This significant difference in FB_1 contamination in maize was further observed in a subsequent study conducted in 2010. Co-contamination of FB_1 and aflatoxin B1 (AFB_1) was observed in rice, wheat flour, and maize. A significant difference in AFB_1 contamination level was also noted in the two areas in both studies.

No clear relationship between FBs contamination and oesophageal cancer was confirmed by the abovementioned studies. The high consumption of maize in the high-incidence areas increased the risk of exposure of the population to a cocktail of mycotoxins such as FBs, AFBs, deoxynivalenol, nivalenol, and zearalenone. Considering that differences in demography, ethnicity, genetic susceptibility, culture, economy, and nutritional status affect the rates of oesophageal cancer, no definite conclusion can be drawn from these comparative studies. However, capable of adjusting confounding factors, well-designed case-control and prospective studies should be further conducted to provide confirmative and valuable evidence.

A case-control study conducted in Linxian found that the risk of oesophageal cancer tends to increase with the increase in the intake of wheat and maize (the participates in the upper three quartiles of maize consumption were at a higher risk compared with the lowest quartile of <24 meals per year) (Li *et al.*, 1989). The possible association between maize consumption, which was the primary staple food for 95% of Linxian residents back then (60% consumed it daily), and oesophageal (including gastric and cardia) cancer was also reported in a retrospective cohort study conducted in Linxian (Yu *et al.*, 1993). Other risk factors, including age, positive family history, education level, surface water use, pork consumption, and smoking, were also identified in the two studies.

Another prospective study conducted in Linxian did not report any significant association between sphingolipid levels (as a biomarker of FBs exposure) at baseline and oesophageal cancer during 5.25 years of follow-up (Abnet *et al.*, 2001a). The results of this study are consistent with those of an earlier study conducted in Africa, which also reported the absence of correlation between sphingolipids and oesophageal cancer (Van der Westhuizen *et al.*, 1999). However, considering that sphingolipids only indicate short-term differences in exposure, the seasonal and annual variation in maize consumption and level of FBs contamination limit the effectiveness of baseline sphingolipids as a long-term biomarker for FBs exposure. Dietary patterns and FBs contamination could have been different during the 5.25 years of follow up; therefore, the null association observed in these studies must be carefully analysed. To sum up, no definite conclusion can be established regarding the association between FBs and oesophageal cancer based on current scientific evidence. Further epidemiological studies in this area are required.

Liver cancer

Apart from possible involvement with oesophageal cancer, epidemiologic studies in China implicate that FBs may also affect the development of liver cancer. The results of related studies are summarised in Supplementary Table S3. The role of FBs in the development of liver cancer was first evaluated in Haimen and Penlai County. The mortality rate in Haimen (52-65 per 100,000) is approximately four times higher than that in Penlai back then (Ueno *et al.*, 1997). A three-year survey revealed that the maize harvested in Haimen was highly contaminated with FBs to the extent that the mean contamination levels in 1993 and 1995 were 10 to 50 times higher than those in Penlai; the percentages of the positive samples were also higher. The co-contamination of various mycotoxins in maize harvested in 1993 was also analysed. The results revealed the cross-contamination of maize by AFBs, FBs, and DON, suggesting that the synergistic interactions among FBs, AFBs, and trichothecenes contribute to the development of liver cancer.

A similar comparative study was conducted in Guangxi, South China (Li *et al.*, 2001). The maize samples collected in 1998 from areas with high and low incidence of liver cancer revealed no significant differences in FBs levels. However, a significantly high contamination by AFBs was observed; 60% of the samples were co-contaminated by both toxins. The surveys conducted in Fusui (high-incidence area) and Huantai (low-incidence area) from 2001 to 2002, shown in Supplementary Table S3, revealed no significant difference in the frequency of occurrence and contamination levels of FB_1 in the two areas. This result was further confirmed in a subsequent 2010 study. The co-contamination of AFB_1 and FB_1 was also observed in rice, wheat flour, and maize samples in 2010 (Sun *et al.*, 2007, 2011).

Aside from comparative studies, a recent meta-analysis also reported two case-control studies conducted in two large cohorts: the Haimen City cohort and General Population Study of Nutritional Intervention Trials cohort in Linxian (Persson *et al.*, 2012). Nail FB_1 levels were determined in 271 liver cancer cases and 280 control samples in the Haimen City cohort. Nail FB_1 levels were determined in 72 liver cancer cases and 147 control samples in the General Population Study of Nutritional Intervention Trials cohort. The results did not reveal an association between FB_1 exposure and liver cancer in Haimen City (odds ratio (OR)=1.10, 95% confidence interval (CI)=0.64 to 1.89), Linxian (OR=1.47, 95% CI=0.70 to 3.07), or the pooled meta-analysis (OR=1.22, 95% CI=0.79 to 1.89). However, the nail specimens in this preliminary study reflected only exposure throughout nail growth; thus, FB_1 exposure in the past was not represented well.

To conclude, current scientific evidence does not support the association between FBs and liver cancer. Further studies are required to provide more perspectives.

Neural tube defects

A neural tube defects (NTD) outbreak occurred in the Texas-Mexico border from 1990 to 1991. Epidemiologists hypothesised that the outbreak could have been caused by the high FB_1 levels observed in maize in the previous years (Missmer *et al.*, 2006). Several regions in China with high maize consumption also exhibit a high prevalence of NTD (Moore *et al.*, 1997). However, the association of NTD with FBs exposure is based only on theoretical hypothesis at this time. Animal experiments suggest that FB_1 is not teratogenic until general toxicity occurs and does not pass the placenta. However, some evidence suggest that the disruption of sphingolipid metabolism caused by FB_1 affect folate uptake and result in increased risk of NTD (Marasas *et al.*, 2004).

Limitations and challenges

The possible involvement of *F. verticillioides* and FBs in the development of oesophageal and liver cancer in various regions of the world, including China, is often evaluated based on the incidence rates and occurrence of FBs. Monotonous diets that consist mainly of wheat or maize might lead to nutritional deficiencies and exposure to mycotoxins. Comparative studies are limited by the lack of controlled conditions, particularly for established confounding risk factors (e.g. alcohol consumption). Only a few case-control or prospective studies on the relation of FBs to human diseases have been conducted in China partly because of the lack of well-validated biomarkers for FBs and the short half-life of FBs in serum (Delongchamp and Young, 2001; Shephard *et al.*, 2007). The mechanism by which FBs interrupt sphinganine metabolism has been elucidated recently. The elucidation revealed that sphingolipid levels are very promising biomarkers. However, several issues remain. A number of studies, including several in China (Abnet *et al.*, 2001b; Qiu and Liu, 2001), on the possible elevation of sphinganine (or its ratio with sphingosine) in human blood and urine have failed to correlate the estimates of FBs exposure. The failure to correlate was partly due to the fact that naturally existing sphingoid bases have normal ranges in human blood and urine, which can be influenced by dietary factors other than FBs ingestion. A cross-sectional study of serum sphingoid bases in Linxian residents indicated that serum sphingosine levels are correlated with a number of factors such as tocopherols, carotenoids, cholesterol, and selenium (Abnet *et al.*, 2001b). Several biomarker studies did not measure FB_1 levels in food; thus, determining how well the utilised biomarkers (nail FB_1 in the study of Persson *et al.* (2012) and sphingolipid levels in the study of Abnet *et al.*

(2001b)) correlate with dietary intake levels is impossible. It has been proposed that combining urinary FB_1 with serum sphingosine levels could improve the assessment of the impact of FB_1 exposure, which is still in validation (Van der Westhuizen *et al.*, 2013).

4. Exposure estimates

The NTP finding (2001) that the no observed effect level for nephrotoxicity is 0.2 mg/kg body weight (b.w.)/day was considered by the Joint FAO/WHO Expert Committee on Food Additives (JECFA) to induce the provisional maximum tolerable daily intake (PMTDI) for FBs in humans, which is 2 μg/kg b.w./day (JECFA, 2001). The WHO's International Programme on Chemical Safety and the European Commission's Scientific Committee on Food (SCF) have also concluded that the tolerable daily intake (TDI) for FB_1, FB_2, and FB_3 alone or in combination is 2 μg/kg b.w./day (SCF, 2000, 2003; WHO, 2001). Some concerns exist regarding the PMTDI for China where in some areas the staple component of the human diet in some parts is maize. Meeting the PMTDI is considerably more difficult in these regions than in the USA or Europe where maize consumption is much lower (Shephard *et al.*, 2002).

The exposure estimates that were conducted in China are summarised in Table 1. Several aspects should be noted. Considering that FBs exposure may vary in regions with different dietary patterns and climate, the exposure estimates (some of them are above the TDI) still require further investigation. Except for one study (Sun *et al.*, 2011), all the other studies were based solely on FBs occurrence in maize and maize consumption data. Such specific reliance might lead to the underestimation of actual overall exposure. Two of the studies targeted areas with high incidence of cancer (Li *et al.*, 2001; Sun *et al.*, 2011), which could indeed provide insights into the association of FBs exposure with certain types of cancer; however, limited its generalisation to other populations and regions.

Two other studies aimed to generalise the exposure estimate based on the average maize consumption data of the 2002 Chinese National Nutrition Survey rather than the actual specific maize consumption data of the investigated areas (Feng *et al.*, 2011; Gong *et al.*, 2009). These studies presented an unpredictable uncertainty. Moreover, FB_2 and FB_3 contamination were not included in the exposure estimates, the situation might be worse.

Table 1. Fumonisins exposure estimates in China.

Toxins	Region specification	Food investigated	Intake of food contributors (/day)	Toxins intake (μg/day) (mean)	Adults body weight (kg)	PDI[1] (μg/kg b.w./day) (mean)	Ration of PDI/TDI[2] (mean)	Reference
FB_1	Guangxi Province (high-risk area for liver cancer)	Maize	Average daily intake of maize product of 400 g[3]	150.80	50	3.02	1.50	Li *et al.*, 2001
FB_1	Six main maize-producing provinces	Maize	Mean maize consumption of 10.05 g[4]	66.95	60	1.11	0.56	Gong *et al.*, 2009
FB_1	Three main maize-producing provinces	Maize	Mean maize consumption of 10.05 g[4]	18.00	60	0.30	0.15	Feng *et al.*, 2011
FB_1	Huantai (low-risk area for oesophageal and liver cancer)	Main contributing food commodities, including rice, wheat flour, maize, peanuts, and plant oil	Specific food consumption survey of the subjects in the three areas	92.40	60	1.54	0.77	Sun *et al.*, 2011
	Huaian (high-risk area for liver cancer)			460.00	60	7.67	3.835	
	Fusui (high-risk area for oesophageal cancer)			138.60	60	2.30	1.15	

[1] PDI = provisional daily intake.
[2] Tolerable daily intake (TDI) of 2 μg/kg b.w./day is recommended by JECFA (2001).
[3] Extrapolated from the fact that the vast majority of maize consumption was 300 g to 500 g per day per person in that area.
[4] Food consumption data of Feng *et al.* (2011).

The study conducted by Sun *et al.* (2011) revealed that the staple food of the three target areas are different (the staple food in Huantai is wheat flour, whereas rice and corn are the staple food in Huaian and Fusui). The study represented the different staple food patterns in the northern and southern areas of China, which is an important factor that should be considered in assessing the risk of mycotoxin exposure.

The overall exposure estimates of FBs intake from all possible food contributors in representative areas across China and/or various sub-populations remain indefinite. Several aspects should be considered in future exposure estimation. First, the intake of food commodities rather than raw agricultural commodities should be investigated. Second, reliance on a single food category (maize in this case) as a source of exposure could be misleading; other possible food contributors, including peanuts, plant oil, several other food commodities, e.g. beer, spices, and processed grain-based products, should also be considered.

Considering atypical dietary behaviours in China, careful attention should be provided to three specific groups. The first group consists of subsistence farmers who grow and eat their own produce. The high risk associated with this group may be due to the distinct maize dietary patterns, including the use of hand grinding and 'grinding' rocks or stamping of homegrown maize to produce maize meals or soup (Burger *et al.*, 2010; Sun *et al.*, 2011). The next group consists of infants and children whose food consumption pattern includes a significant portion of maize-based food such as biscuits and cereals. Comparison of the exposure estimates in terms of PDI among child consumers in Canada and Argentina demonstrated that values in Canada (Kuiper-Goodman *et al.*, 1996) are ten times lower than those in Argentina (where maize flour is utilised for infant feeding) (Solovey *et al.*, 1999). The last group consists of regular and heavy alcohol drinkers. Heavy beer drinkers face an increased risk of FBs contamination depending on the amount of FB-contaminated grains utilised to produce the beer (Kuiper-Goodman *et al.*, 1996).

A proportion of the current population may be chronically exposed to FBs similar to the previously cited groups. The use of the probabilistic approach should be considered in refining the exposure by considering the fact that the level of contamination is not constant but varies based on climate change and differences in dietary patterns.

5. Legislation

Internationally harmonised legislation on FBs is essential to avoid trade barriers and competition distortions. No wide-scale limits for FBs have been established to date. Only a few of the countries and regions have set limits for FBs. No official upper limit has been established in China. The limitation standards in several countries and regions are listed in Table 2.

The various patterns of distribution of FBs occurrence in China restrict the establishment of regulatory criteria. China has experienced few export losses because of FBs contamination owing to the fact that very few nations have developed guidelines (Wu, 2004). With the tightened FBs standards, China would probably experience serious economic challenges considering that it is the second maize exporting nation (USDA, 2003).

6. Moving forward: challenges and current trends

China aims to reduce and eventually prevent mycotoxin occurrence in food to protect human health and improve international trends. China has exerted significant efforts to establish food safety risk assessment and management. Strengthening the nationwide food safety surveillance network and total diet study (TDS) is imperative in exposure assessment (Zhang *et al.*, 2014). Referring to the Global Environmental Monitoring System/Food Contamination Monitoring and Assessment Programme, food safety surveillance network has been established and developed in China in 2000. The overall surveillance system has

Table 2. Recommended maximum limits for fumonisins (FBs) in food products.

Food products	Limits	Country/region	References
Degermed dry-milled corn products	2 mg/kg for FBs	USA	FDA, 2001
Whole or partially degermed dry-milled corn products/dry-milled corn bran/cleaned corn intended for mass production	4 mg/kg for FBs	USA	FDA, 2001
Cleaned corn intended for popcorn	3 mg/kg for FBs	USA	FDA, 2001
Corn products	1 mg/kg for FB_1 and FB_2	Switzerland	Zoller *et al.*, 1994
Whole maize	3 mg/kg for FBs	France	Soriana and Dragacci, 2004
Whole maize	1 mg/kg for FB_1 and FB_2	EU	EC, 2007
Corn-based breakfast cereals and snack food	0.8 mg/kg for FB_1 and FB_2	EU	EC, 2007
Processed corn-based foods and baby foods for infants and young children	0.2 mg/kg for FB_1 and FB_2	EU	EC, 2007

covered 31 provinces, 1,196 sites, 17 food categories, and 109 parameters to date. Recommended by international bodies (EFSA, 2011; WHO, 2005) and aimed at providing data to monitor the dietary intakes of the population with respect to food contaminants and micronutrients, the first Chinese TDS was conducted in China in 1990. The second TDS was conducted in 1992, the third in 2000, and the fourth in 2007 (Zhou *et al.*, 2012). FBs contamination is currently included in the latest TDS and food safety surveillance network in China. The food consumption data obtained from the 2007 Chinese TDS revealed that in 12 representative regions across Chinese territory, maize consumption vary from 0.75 g in Sichuan Province to 48.6 g in Guangxi Province. Local education and assistance to ensure that food grains and animal feed are harvested correctly, dried completely, and stored properly continue to require improvement. Mycotoxin reduction and control depend on the concerted efforts of all actors along the food production chain. Multidisciplinary approaches are therefore critical.

In summary, this review provided updated information on FBs contamination at a national scale. One clear conclusion that can be drawn is that grains and food commodities in China may be subject to significant FBs contamination. The risk assessment for FBs in China is limited at present because of the lack of sufficient and reliable data. Setting realistic regulatory limits and continuously monitoring programmes for FBs are therefore crucial. The development of the Chinese TDS and surveillance system would aid in controlling FBs contamination and in clearly illustrating the association between FBs and related diseases.

Acknowledgements

This work was supported by MoniQA Association (Monitoring and Quality Assurance in the Food Supply Chain), National Natural Science Foundation of China (grant number 81030053).

Conflict of interest

The authors declare that there is no conflict of interest.

Supplementary material

Supplementary material can be found online at http://dx.doi.org/10.3920/QAS2012.0230.

Table S1. Occurrence of fumonisins in China.

Table S2. Comparison of occurrence of fumonisins (FBs) in areas in China with high and low incidence of oesophageal cancer.

Table S3. Comparison of occurrence of fumonisins (FBs) in areas in China with high and low incidence of liver cancer.

References

Abnet, C.C., Borkowf, C.B., Qiao, Y.L., Albert, P.S., Wang, E., Merrill Jr., A.H., Mark, S.D., Dong, Z.W., Taylor, P.R. and Dawsey, S.M., 2001a. Sphingolipids as biomarkers of fumonisin exposure and risk of esophageal squamous cell carcinoma in China. Cancer Causes Control 12: 821-828.

Abnet, C.C., Borkowf, C.B., Qiao, Y.-L., Albert, P.S., Wang, E., Merrill Jr., A.H., Mark, S.D., Dong, Z.W., Taylor, P.R. and Dawsey, S.M., 2001b. A cross-sectional study of human serum sphingolipids, diet and physiologic parameters. Journal of Nutrition 131: 2748-2752.

Arino, A., Juan, T., Estopanan, G. and Gonzalez-Cabo, J.F., 2007. Natural occurrence of *Fusarium* species, fumonisin production by toxigenic strains, and concentrations of fumonisins B_1 and B_2 in conventional and organic maize grown in Spain. Journal of Food Protection 70: 151-156.

Burger, H.M., Lombard, M.J., Shephard, G.S., Rheeder, J.R., Van der Westhuizen, L. and Gelderblom, W.C., 2010. Dietary fumonisin exposure in a rural population of South Africa. Food and Chemical Toxicololgy 48: 2103-2108.

Castells, M., Marin, S., Sanchis, V. and Ramos, A.J., 2008. Distribution of fumonisins and aflatoxins in corn fractions during industrial cornflake processing. International Journal of Food Microbiology 123: 81-87.

Chu, F.S. and Li, G.Y., 1994. Simultaneous occurrence of fumonisin B_1 and other mycotoxins in moldy corn collected from the people's Republic of China in regions with high incidences of esophageal cancer. Applied and Environmental Microbiology 60: 847-852.

Delongchamp, R.R. and Young, J.F., 2001. Tissue sphinganine as a biomarker of fumonisin-induced apoptosis. Food Additives and Contaminants 18: 255-261.

De Nijs, M., Van Egmond, H.P., Nauta, M., Rombouts, F.M. and Notermans, S.H., 1998. Assessment of human exposure to fumonisin B_1. Journal of Food Protection 61: 879-884.

Domijan, A.M., Peraica, M., Jurjevic, Z., Ivic, D. and Cvjetkovic, B., 2005. Fumonisin B_1, fumonisin B_2, zearalenone and ochratoxin A contamination of maize in Croatia. Food Additives and Contaminants 22: 677-680.

European Commission (EC), 2007. Commission Regulation (EC) no. 2007/1126 of 28 September 2007 amending Regulation (EC) no. 1881/2006 setting maximum levels for certain contaminants in foodstuffs as regards *Fusarium* toxins in maize and maize products. Official Journal of the European Union L255: 14-17.

European Food Safety Authority (EFSA), 2011. Joint guidance of EFSA, FAO and WHO. Towards a harmonised total diet study approach: a guidance document. EFSA, Parma, Italy. Available at: http://www.efsa.europa.eu/en/efsajournal/doc/2450.pdf.

Feng, Y.Z., Lu, X.H., Tao, B., Pang, M.H., Liu, Y.C. and Dong, J.G., 2011. Natural occurrence of fumonisins B_1 and B_2 in corn from three main production provinces in China. Journal of Food Protection 74: 1374-1378.

Food and Drug Administration (FDA), 2001. Fumonisin levels in human foods and animal feeds. Guidance for industry. Available at: http://www.fda.gov/food/guidanceregulation/guidancedocumentsregulatoryinformation/chemicalcontaminantsmetalsnaturaltoxinspesticides/ucm109231.htm.

Franceschi, S., Bidoli, E., Baron, A.E. and La Vecchia, C., 1990. Maize and risk of cancers of the oral cavity, pharynx and esophagus in Northeastern Italy. Journal of National Cancer Institute 82: 1407-1411.

Gao, H.P. and Yoshizawa, T., 1997. Further study on *Fusarium* mycotoxins in corn and wheat from a high-risk area for human esophageal cancer in China. Mycotoxins 45: 51-55.

Gelderblom, W.C., Abel, S., Smuts, C.M., Marnewick, J., Marasas, W.F., Lemmer, E.R. and Ramljak, D., 2001. Fumonisin-induced hepatocarcinogenesis: mechanisms related to cancer initiation and promotion. Environmental Health Perspectives 109: 291-300.

Gelderblom, W.C.A., Jaskiewicz, K., Marasas, W.F.O., Thiel, P.G., Horak, R.M., Vleggar, R. and Kriek, N.P., 1988. Fumonisins-novel mycotoxins with cancer-promoting activity produced by *Fusarium moniliforme*. Applied and Environmental Microbiology 54: 1806-1811.

Gelderblom, W.C.A., Kriek, N.P., Marasas, W.F. and Thiel, P.G., 1991. Toxicity and carcinogenicity of the *Fusarium moniliforme* metabolite, fumonisin B_1, in rats. Carcinogenesis 12: 1247-1251.

Gelderblom, W.C.A., Semple, E., Marasas, W.F.O. and Farber, E., 1992. The cancer initiating potential of the fumonisin mycotoxins produced by *Fusarium moniliforme*. Carcinogenesis 13: 433-437.

Gelderblom, W.C.A. and Snyman, S.D., 1991. Mutagenicity of potentially carcinogenic mycotoxins produced by *Fusarium moniliforme*. Mycotoxin Research 7: 46-52.

Gong, H.Z., Ji, R., Li, Y.X., Zhang, H.Y., Li, B., Zhao, Y., Sun, L., Yu, F. and Yang J., 2009. Occurrence of fumonisin B(1) in corn from the main corn-producing areas of China. Mycopathologia 167: 31-36.

Groves, F.D., Zhang, L., Chang, Y.S., Ross, P.F., Casper, H., Norred, W.P., You, W.C. and Fraumeni Jr., J.F., 1999. *Fusarium* mycotoxins in corn and corn products in a high-risk area for gastric cancer in Shandong Province, China. Journal of AOAC International 82: 657-662.

Guo, Y., Liu, X. and Liu, J., 1999. Studies on immunoassay for detecting fumonisin B_1. Journal of Hygienic Research 28: 238-241.

Harrison, L.R., Colvin, B.M., Green, J.T., Newman, L.E. and Cole Jr., J.R., 1990. Pulmonary edema and hyfrotorax in swine produced by fumonisin B_1, a toxic metabolite of *Fusarium moniliforme*. Journal of Veterinary Diagnostic Investigation 2: 217-221.

He, S., Xin, Y. and Liu, J., 1999. An investigation on the contamination of fungi and mycotoxins in staple crops in Sichuan province. Journal of Preventive Medicine Information 15: 199-202.

Hennigen, M.R., Valente Soares, L.M., Sanchez, S., Di Benedetto, N.M., Longhi, A., Eyherabide, G., Torroba, J. and Zanelli, M., 2000. Fumonisins in corn hybrids grown in Argentina for 2 consecutive seasons. In: De Koe, W.J., Samson, R.A., Van Egmond, H.P., Gilbert, J. and Sabino, M. (eds.) Proceedings of the Xth international IUPAC symposium on mycotoxins and phycotoxins May 21-25, 2000, Guaruja, Brazil, pp. 331-339.

International Agency for Research on Cancer (IARC), 1993. Toxins derived from *Fusarium moniliforme*: fumonisins B_1 and B_2 and fusarin C. In: Some naturally occurring substances: food items and constituents, heterocyclic aromatic amines and mycotoxins. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans, volume 56. IARC, Lyon, France, pp. 445-466.

Joint FAO/WHO Expert Committee on Food Additives (JECFA), 2001. Safety evaluation of certain mycotoxins in food. WHO Food Additives Series 47: 103-279.

Kellerman, T.S., Marasas, W.F., Thiel, P.G., Gelderblom, W.C., Cawood, M. and Coetzer J.A., 1990. Leukoencephalomalacia in two horses induced by oral dosing of fumonisin B_1. Onderstepoort Journal of Veterinary Research 57: 269-275.

Kuiper-Goodman, T., Scott, P.M., McEwen, N.P., Lombaert G.A. and Ng, W., 1996. Approaches to the risk assessment of fumonisins in corn-based foods in Canada. Advances in Experimental and Medicine Biology 392: 369-393.

Li, F.Q., Yoshizawa, T., Kawamura, O., Luo, X.Y. and Li, Y.W., 2001. Aflatoxins and fumonisins in corn from the high-incidence area for human hepatocellular carcinoma in Guangxi, China. Journal of Agricultural and Food Chemistry 49: 4122-4126.

Li, J.Y., Ershow, A.G., Chen, Z.J., Wacholder, S., Li, G.Y., Guo, W., Li, B. and Blot, W.J., 1989. A case-control study of cancer of the esophagus and gastric cardia in Linxian. International Journal of Cancer 43: 755-761.

Lineback, D.R., Pirlet, A., Van der Kamp, J.W. and Wood, R., 2009. Globalization, food safety issues and role of international standards. Quality Assurance and Safety of Crops & Foods 1: 23-27.

Lino, C.M., Silva, L.G., Pena, A.L. and Silveira, M.I., 2006. Determination of fumonisins B_1 and B_2 in Portuguese maize and maize-based samples by HPLC with fluorescence detection. Analytical and Bioanalytical Chemistry 384: 1214-1220.

Liu, Q., Liu, G. and Liu, H., 2008. Investigation into the status of contamination of strong carcinogen – fumonisin in peanut and corn and their products and rapid detection. China Tropical Medicine 8: 1906-1909.

Lv, B., Xie, M. and Yang, X., 2005. Contamination of fumonisin B(1) in staple crops in Hubei province of China. Journal of Public Health and Prevention Medince 16: 60-61.

Marasas, W.F., Kellerman, T.S., Gelderblom, W. C., Coetzer, J.A., Thiel, P. G. and Van der Lugt J.J., 1988. Leukoencephalomalacia in a horse induced by fumonisin B_1 isolated from *Fusarium moniliforme*. Onderstepoort Journal of Veterinary Research 55: 197-203.

Marasas, W.F.O., Riley, R.T., Hendricks, K.A., Stevens, V.L., Sadler, T.W., Gelineau-Van Waes, J., Missmer, S.A., Cabrera, J., Torres, O., Gelderblom, W.C., Allegood, J., Martínez, C., Maddox, J., Miller, J.D., Starr, L., Sullards, M.C., Roman, A.V., Voss, K.A., Wang, E. and Merrill Jr., A.H., 2004. Fumonisins disrupt sphingolipid metabolism, folate transport, and neural tube development in embryo culture and *in vivo*: a potential risk factor for human neural tube defects among populations consuming fumonisin contaminated maize. Journal of Nutrition 134: 711-716.

Martins, M.L., Martins, H.M. and Bernardo, F., 2001. Fumonisins B_1 and B_2 in black tea and medicinal plants. Journal of Food Protection 64: 1268-1270.

Missmer, S.A., Suarez, L., Felkner, M., Wang, E., Merrill Jr., A.H., Rothman, K.J. and Hendricks K.A., 2006. Exposure to fumonisins and the occurrence of neural tube defects along the Texas-Mexico border. Environmental Health Perspect 114: 237-241.

Moore, C.A., Li, S., Li, Z., Hong, S., Gu, H., Berry, R.J., Mulinare, J. and Erickson, J.D., 1997. Elevated rates of severe neural tube defects in a high-prevalence area in northern China. American Journal of Medical Genetics 73: 113-118.

National Toxicology Program (NTP), 2001. Technical report on the toxicology and carcinogenesis studies of fumonisin B_1 (CAS no. 116355-83-0) in F344 rats and B6C3F1 mice (feed studies). NTP Technical Report 496. NTP, Research Triangle Park, NC, USA.

Norred, W.P., Plattner, R.D., Vesonder, R.F., Bacon, C.W. and Voss, K.A., 1992. Effects of selected secondary metabolites of Fusarium moniliforme on unscheduled synthesis of DNA by rat primary hepatocytes. Food and Chemical Toxicology 30: 233-237.

Omurtag, G.Z. and Yazicioglu, D., 2004. Determination of fumonisins B1 and B2 in herbal tea and medical plants in Turkey by high-performance liquid chromatography. Journal of Food Protection 67: 1782-1786.

Persson, E.C., Sewram, V., Evans, A.A., London, W.T., Volkwyn, Y., Shen, Y.J., Van Zyl, J.A., Chen, G., Lin, W., Shephard, G.S., Taylor, P.R., Fan, J.H., Dawsey, S.M., Qiao, Y.L., McGlynn, K.A. and Abnet, C.C., 2012. Fumonisin B_1 and risk of hepatocellular carcinoma in two Chinese cohorts. Food and Chemical Toxicology 50: 679-683.

Pietri, A., Bertuzzi, T., Agosti, B. and Donadini, G., 2010. Transfer of aflatoxin B_1 and fumonisin B_1 from naturally contaminated raw materials to beer during an industrial brewing process. Food Additives and Contaminants Part A 27: 1431-1439.

Pocsfalvi, G., Ritieni, A., Randazzo, G., Dobó, A. and Malorni, A., 2000. Interaction of *Fusarium* mycotoxins, fusaproliferin and fumonisin B_1 with DNA studied by electrospray ionization mass spectrometry. Journal of Agricultural and Food Chemistry 48: 5795-5801.

Qiu, M. and Liu, X., 2001. Determination of sphinganine, sphingosine and Sa/So ratio in urine of humans exposed to dietary fumonisin B_1. Food Additives and Contaminants Part A 18: 263-269.

Rheeder, J.P., Marasas, W.F.O., Thiel, P.G., Sydenham, E.W., Shepard, G.S. and Van Schalkwyk, D.J., 1992. *Fusarium moniliforme* and fumonisins in corn in relation to human oesophageal cancer in Transkei. Phytopathology 82: 353-357.

Riley, R.T., Hinton, D.M., Chamberlain, W.J., Bacon, C.W., Wang, E., Merrill Jr., A.H. and Voss, K.A., 1994. Dietary fumonisin B_1 induces disruption of sphingolipid metabolism in Sprague–Dawley rats: a new mechanism of nephrotoxicity. Journal of Nutrition 124: 594-603.

Scientific Committee on Food (SCF), 2000. Opinion of the scientific committee on food on *Fusarium* toxins. Part 3: fumonisin B_1 (FB_1). SCF, Brussels, Belgium. Available at: http://ec.europa.eu/food/fs/sc/scf/out73_en.pdf.

Scientific Committee on Food (SCF), 2003. Updated opinion of the scientific committee on food on fumonisin B_1, B_2 and B_3. SCF, Brussels, Belgium. Available at: http://ec.europa.eu/food/fs/sc/scf/out185_en.pdf.

Scott, P.M. and Lawrence, G.A., 1995. Analysis of beer for fumonisins. Journal of Food Protection 58: 1379-1382.

Shale, K., Mukamugema, J., Lues, R.J. and Venter, P., 2012. Toxicity profile of commercially produced indigenous banana beer. Food Additives and Contaminants Part A 29: 1300-1306.

Shephard, G.S., Leggott, N.L., Stockenström, S., Somdyala, N.I.M. and Marasas, W.F.O., 2002. Preparation of South African maize porridge: effect on fumonisin levels. South African Journal of Science 98: 393-396.

Shephard, G.S., Marasas, W.F., Leggott, N.L., Yazdanpanah, H., Rahimian, H. and Safavi, N., 2000. Natural occurrence of fumonisins in corn from Iran. Journal of Agriculture and Food Chemistry 48: 1860-1864.

Shephard, G.S., Van der Westhuizen, L. and Sewram, V., 2007. Biomarkers of exposure to fumonisin mycotoxins: a review. Food Additives and Contaminants Part A 24: 1196-1201.

Shi, J., Han, W. and Gu, M., 2003. Contamination of AFB, FB, SET in spices in China. Journal of Health Laboratory Technology 13: 589-594.

Solovey, M.M., Somoza, C., Cano, G., Pacin, A. and Resnik, S., 1999. A survey of fumonisins, deoxynivalenol, zearalenone and aflatoxins contamination in corn-based food products in Argentina. Food Additives and Contaminants Part A 16: 325-329.

Soriana, J.M. and Dragacci, S., 2004. Intake, decontamination and legislation of fumonisins in foods. Food Research International 37: 367-374.

Sui, W., Liu, G., Huang, M., Kang, Y., Zhang, W., Liu, Y., Xin, L. and Liang, Y., 2003. Contamination of fumonisin and *Fusarium moniliforme* in staple crops in Jilin province of China. Journal of Health Laboratory Technology 13: 80-81.

Sun, G., Wang, S., Hu, X., Su, J., Huang, T., Yu, J., Tang, L., Gao, W. and Wang, J.S., 2007. Fumonisin B_1 contamination of home-grown corn in high-risk areas for esophageal and liver cancer in China. Food Additives and Contaminants Part A 24: 181-185.

Sun, G., Wang, S., Hu, X., Su, J., Zhang, Y., Xie, Y., Zhang, H., Tang, L. and Wang, J.S., 2011. Co-contamination of aflatoxin B_1 and fumonisin B_1 in food and human dietary exposure in three areas of China. Food Additives and Contaminants Part A 28: 461-470.

Sydenham, E.W., Thiel, P.G., Marasas, W.F.O., Shepard, G.S., Van Schalkwyk, D.J. and Koch, K.R., 1990. Natural occurrence of some *Fusarium* mycotoxins in corn from low and high esophageal cancer prevalence areas of Transkei, Southern Africa. Journal of Agriculture and Food Chemistry 38: 1900-1903.

Tamura, M., Takahashi, A., Uyama, A. and Mochizuki, N., 2012. A method for multiple mycotoxin analysis in wines by solid phase extraction and multifunctional cartridge purification, and ultra-high-performance liquid chromatography coupled to tandem mass spectrometry. Toxins 4: 476-486.

Tamura, M., Uyama, A. and Mochizuki, N., 2011. Development of a multi-mycotoxin analysis in beer-based drinks by a modified QuEChERS method and ultra-high-performance liquid chromatography coupled with tandem mass spectrometry. Analytic Sciences 27: 629-635.

Ueno, Y., Iijima, K., Wang, S.D., Sugiura, Y., Sekijima, M., Tanaka, T., Chen, C. and Yu, S.Z., 1997. Fumonisins as a possible contributory risk factor for primary liver cancer: a 3-year study of corn harvested in Haimen, China, by HPLC and ELISA. Food and Chemical Toxicology 35: 1143-1150.

United States Department of Agriculture (USDA), 2003. Countries and regions. China. Available at: http://www.fas.usda.gov/regions/east-asia-and-pacific/china.

Van der Westhuizen, L., Brown, N.L., Marasas, W.F.O., Swanevelder, S. and Shephard, G.S., 1999. Sphinganine/sphingosine ratio in plasma and urine as a possible biomarker for fumonisin exposure in humans in rural areas of Africa. Food and Chemical Toxicology 37: 1153-1158.

Van der Westhuizen, L., Shephard, G.S., Gelderblom, W.C.A., Torres, O. and Riley, R.T., 2013. Fumonisin biomarkers in maize eaters and implications for human disease. World Mycotoxin Journal 6: 223-232.

Voss, K.A., Chamberlain, W.J., Bacon, C.W., Herbert, R.A., Walters, D.B. and Norred, W.P., 1995. Subchronic feeding study of the mycotoxin fumonisin B_1 in B6C3F1 mice and Fischer 344 rats. Fundamental and Applied Toxicology 24: 102-110.

Wang, Z., Li, X., Tong, Z., Cheng, S., Gao, W., Zhu, X., Feng, J. and Cai, H., 2001. Correlation study between fumonisin contamination and generate-sources in foodstuffs. Chinese Journal of Health Laboratory Technology 11: 9-13.

Wang, J., Zhou, Y. and Wang, Q., 2008a. Analysis of mycotoxin fumonisins in corn products by high-performance liquid chromatography coupled with evaporative light scattering detection. Food Chemistry 107: 970-976.

Wang, J., Zhou, Y., Liu, W., Zhu, X., Du, L. and Wang, Q., 2008b. Fumonisin level in corn-based food and feed from Linxian County, a high-risk area for esophageal cancer in china. Food Chemistry 106: 241-246.

World Health Organization (WHO), 2000. Fumonisin B_1 (environmental health criteria 219). International programme on chemical safety. WHO, Geneva, Switzerland. Available at: http://whqlibdoc.who.int/ehc/WHO_EHC_219.pdf.

World Health Organization (WHO), 2001. Safety evaluation of certain mycotoxins in food. International programme on chemical safety. WHO food additives series 47: 103-279.

World Health Organization (WHO), 2002. WHO global strategy for food safety: safer food for better health. Food Safety Programme 2002. WHO, Geneva, Switzerland.

World Health Organization (WHO), 2005. Total diet studies: a recipe for safer food. Available at: WHO, Geneva, Switzerland. http://www.who.int/foodsafety/publications/chem/en/gems_brochure.pdf.

Wu, F., 2004. Mycotoxin risk assessment for the purpose of setting international regulatory standards. Environmental Science and Technology 38: 4049-4055.

Yan, Y., Zhang, Y. and Li, R., 1999. Contamination of fumonisin in staple crops in Shandong province of China. Chinese Journal of Health Laboratory Technology 9: 201–204.

Yoshizawa, T., Yamashita, A. and Luo, Y., 1994. Fumonisin occurrence in corn from high- and low-risk areas for human esophageal cancer in China. Applied and Environmental Microbiology 60: 1626-1629.

Yu, Y., Taylor, P.R., Li, J.Y., Dawsey, S.M., Wang, G.Q., Guo, W.D., Wang, W., Liu, B.Q., Blot, W.J. and Shen, Q., 1993. Retrospective cohort study of risk-factors for esophageal cancer in Linxian, People's Republic of China. Cancer Causes Control 4: 195-202.

Zhang, W.W., Ye, Z.M., Jin, Y., Wang, S.Y., Zhang, L.S. and Pei, X.F., 2014. Management of mycotoxin contamination in food and feed in China. World Mycotoxin Journal 7 :53-62

Zhou, P., Zhao, Y., Li, J., Wu, G., Zhang, L., Liu, Q., Fan, S., Yang, X., Li, X. and Wu Y., 2012. Dietary exposure to persistent organochlorine pesticids in 2007 Chinese total diet study. Environmental International 42: 152-159.

Zoller, O., Sager, F. and Zimmerli, B., 1994. Vorkommen von Fumonisinen in Lebensmitteln. Mitteilungen aw dem Gebiete der Lebensmitteluntersuchung und Hygiene 85: 81-99.