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The risk management dilemma for fumonisin mycotoxins

Gordon S. Shephard [a,*], Martin E. Kimanya [b,1], Kafui A. Kpodo [c], G.J. Benoit Gnonlonfin [d], Wentzel C.A. Gelderblom [a,e]

[a] PROMEC Unit, Medical Research Council, P.O. Box 19070, Tygerberg 7505, South Africa
[b] Tanzania Food and Drugs Authority, P.O. Box 77150, Dar es Salaam, Tanzania
[c] Food Research Institute, P.O. Box M.20, Accra, Ghana
[d] International Livestock Research Institute, P.O. Box 30709, Nairobi 00100, Kenya
[e] Department of Biochemistry, University of Stellenbosch, Private Bag X1, Matieland 7602, South Africa

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ABSTRACT

Since the discovery of the fumonisins in 1988, extensive academic studies have generated much knowledge, including data on chemistry, biochemistry, toxicology, methods of analysis, natural occurrence in food supplies, fate during various processing procedures, and human and animal exposures. These mycotoxins have also been assessed by the International Agency for Research on Cancer (IARC) and have twice been the subject of risk assessments by the Joint FAO/WHO Expert Committee on Food Additives (JECFA). The outcome of these investigations has been of a nature to alert risk managers to the necessity of controlling human exposure. However, the fumonisins occur mostly in maize, a world staple crop which is consumed in various communities at levels which can be as many as 100-fold different. Compounding the problem is the fact that maize is widely used as animal feed in many developed countries, whereas in Africa and some other developing countries, it is the primary food source. This contrast produces a problem for risk managers, partly solved at national level by the regulation of maximum tolerated levels (MTLs) applicable to individual countries. MTLs at an international level are currently under discussion at the Codex Committee on Contaminants in Food. The divergence in use and consumption and the fact that maize in various regions of the world can also vary greatly in contamination levels, leads to a dilemma for setting such MTLs, which would need to be low to protect the high maize consumers, but might then cause rejection of high amounts of the world supply. Higher MTLs, acceptable to maize exporters, would only protect the low maize consumers. This dilemma may only be solved by accepting that harmonizing regulations for raw maize is problematic and a more nuanced approach may be required.

1. Introduction

The fumonisin mycotoxins, of which fumonisin B_1 (FB_1) and B_2 (FB_2) are the most important, occur ubiquitously in maize and hence in maize products worldwide (Bolger et al., 2001). Although sporadic reports exist of fumonisins in other products, maize and possibly sorghum remain the primary sources of human exposure (Bulder et al., 2012). The effects of fumonisin ingestion in animal species have been documented and are very species specific. Ingestion of FB_1 causes leukoencephalomalacia in horses (Kellerman et al., 1990) and pulmonary oedema in swine (Harrison, Colvin, Green, Newman, & Cole, 1990). In rodents, fumonisins are hepato- and nephrotoxins and carcinogens (Gelderblom, Kriek, Marasas, & Thiel, 1991; Howard et al., 2001).

The acute intake of fumonisins from mouldy maize and sorghum has been linked to an outbreak in the Deccan Plateau in southern India of food-borne disease characterized by borborygmy, abdominal pain and diarrhoea (Bhat, Shetty, Amruth, & Sudershan, 1997). Chronic ingestion of fumonisins has been linked as one possible risk factor for the occurrence of oesophageal cancer in areas such as the former Transkei region of South Africa, where fumonisin exposure from contaminated maize is high (Rheeder et al.,1992). Similar associations have been reported in maize grown in Linxian County, Henan Province and Cixian County, Hebei

* Corresponding author. Tel.: +27 21 938 0279; fax: +27 21 938 0260.
E-mail addresses: gordon.shephard@mrc.ac.za (G.S. Shephard), mekimanya@yahoo.co.uk (M.E. Kimanya), kpodofri@ghana.com (K.A. Kpodo), bgnonlonfin@yahoo.fr (G.J.B. Gnonlonfin), wentzel.gelderblom@mrc.ac.za (W.C.A. Gelderblom).
[1] Current address: School of Life Sciences and Bio-Engineering (LSBE), The Nelson Mandela African Institute of Science and Technology (NM-AIST), Tengeru, P.O. Box 447, Arusha, Tanzania.

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Province, China (Zhang, Nagashima, & Goto, 1997), maize grown in Huaian County, Jiangsu Province, China (Sun et al., 2007), maize grown in Santa Catarina state, southern Brazil (Van der Westhuizen et al., 2003) and in polenta produced in northern Italy (Pascale, Doko, & Visconti, 1995). Fumonisins have also been linked as a risk factor for primary liver cancer in China (Sun et al., 2007; Ueno et al., 1997). The International Agency for Research on Cancer (IARC) has classified FB_1 as a possible human carcinogen (group 2B) (IARC, 2002).

Evidence has emerged of the detrimental effects of fumonisin on the developing foetus and young infants. A cluster of cases of neural tube defects (NTDs) in infants in southern Texas has provided epidemiological evidence that fumonisins may have played a role in these cases in which mothers are presumed to have consumed fumonisin contaminated food (Missmer et al., 2006). It is also known that other areas of the world where fumonisin exposure is high, such as former Transkei region in South Africa and regions in northern China, have elevated incidences of neural tube defects (NTDs) (Marasas et al., 2004). Support for this association has come from evidence that fumonisins, via their depletion of sphingolipids, interfere with the folate receptor, inhibiting uptake of folate, the cellular deficiency of which is a known cause of NTDs (Stevens & Tang, 1997). Further evidence for these interactions comes from a dose-dependent rise in NTDs in fumonisin-dosed experimental mice, an effect that could be prevented by folate supplementation (Gelineau-van Waes et al., 2005). Further studies in infants in Tanzania have shown that ingestion of fumonisin was associated with growth retardation as measured in infants at 12 months of age (Kimanya, De Meulenaer, Roberfroid, Lachat, & Kolsteren, 2010).

2. Risk assessment

In 2001, the 56th meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA) evaluated fumonisins and established a provisional maximum tolerable daily intake (PMTDI) of 2 μg/kg body weight/day for FB_1, FB_2 and FB_3, either alone or in combination, based on a no observed effect level (NOEL) of 0.2 mg/kg body weight/day for renal toxicity and a safety factor of 100 (Bolger et al., 2001). This PMTDI was re-assessed and confirmed in 2010 at the 74th meeting of JECFA in which benchmark dose modelling for a 10% extra risk was applied to the observation of megalocytic hepatocytes in male mice dosed with pure FB_1 (Bulder et al., 2012).

A large number of fumonisin exposure assessments have been performed. These include international assessments by JECFA using either the WHO GEMS/Food regional diets (Bolger et al., 2001) or the WHO GEMS/Food clusters (Bulder et al., 2012). Other national, regional or group specific estimates have been summarized by JECFA (Bulder et al., 2012). Exposure assessments for fumonisins in various countries and populations are heavily influenced by the fact that exposure is governed by maize consumption, which can vary by as much as 100-fold between developed countries and developing countries, particularly in Africa. Thus maize consumption can be less than 10 g/person/day in various European countries, but can rise to an average of 400–500 g/day in rural Africa (Shephard, Leggott, Stockenström, Somdyala, & Marasas, 2002), with a 90 percentile value of over 700 g/person/day (Shephard et al., 2007). The effect of this variation is that to attain the PMTDI of 2 μg/kg body weight/day, a European consumer at an assumed body weight of 60 kg would need to consume 10 g maize at a fumonisin contamination level of 12,000 μg/kg, whereas an African consumer of 500 g per day, would exceed the PMTDI if the contamination level was above 240 μg/kg. Urbanized African communities can still consume maize as a dietary staple at levels intermediate between these extremes. One such study in South Africa recorded mean daily maize consumption of 267 g in an urban area (Gelderblom et al., 1996).

These international evaluations have highlighted the need for risk management for fumonisins. However, the setting of maximum tolerated levels (MTLs) represents a dilemma for managers due to the variations in human maize consumption highlighted above.

3. Effects of processing

The primary source of fumonisin exposure is via consumption of maize or its processed products. Thus, the processing of maize and the exact form of the product used as food has an important bearing on the exposure assessment and is an important consideration in the setting of regulations. The fate of fumonisins during various processing stages has been the subject of various research papers and published results indicate that large reductions in contamination levels can be achieved. The cleaning of maize by screens and gravity separation during discharge from a silo into trucks has been shown to produce a reduction from 7.90 mg/kg to 3.24 mg/kg, with a mass loss of 7% (Malone, Richard, Romer, Johansson, & Whitaker, 1998). There are two commercial milling processes relevant for maize. Of these, wet milling results in the loss of some fumonisins in steep water and the subsequent industrial milling process partitions the fumonisin into the fractions for animal feed (gluten, fibre and germ) and a starch fraction intended for human consumption and which has negligible fumonisin content (Bennett, Richard, & Eckhoff, 1996; Saunders, Meredith, & Voss, 2001). Dry milling is the other milling process and is the most important for production of maize flour/meal and grits. Contrary to wet milling, no fumonisin is lost during dry milling, but the mycotoxin is distributed in the products based on its occurrence in the maize kernel (Brera, Debegnach, Grossi, & Miraglia, 2004; Katta, Cagampang, Jackson, & Bullerman, 1997; Saunders et al., 2001; Scudamore & Patel, 2009). Although complex industrial milling process streams and sometimes non-uniform terminology can cause confusion, most data suggests that the processing from maize kernels to grits and flour will greatly reduce fumonisin levels in these products which are destined for human food preparation. Specifically, Brera et al. (2004) in an Italian mill reported that flour for human consumption derived from two different maize lots contained, in both cases, 9% of the FB_1 level determined in the kernels and 8–20% in various grit fractions. Another product, designated as animal feed flour, contained 135% and 206% of the starting FB_1 levels. The increase in contamination was presumably due to the fact that this product is derived via the recycling of waste streams from various milling stages. Similarly, germ (188% and 196%) and bran (156% and 159%) contained higher levels than the starting maize lots. However, a note of caution for risk managers has been provided by Scudamore and Patel (2009) in studies in three large UK maize mills. Whereas their data indicated a mean 93% reduction in the FB_1 levels in flaking grits, mean FB_1 levels in maize flour varied between 75% and 131% of the relative levels in maize kernels. The exact reasons for the difference in results between the UK and Italian study are difficult to determine, but undoubtedly relate to the complex recycling processes within a commercial operation. Further industrial processes such as frying and extrusion cooking have been shown to reduce fumonisins based on temperature and the processing time involved (Bullerman, Ryu, & Jackson, 2002).

All the above maize handling and industrial processes are relevant to consumption and exposure in the developed world. However, maize in the developing world and particularly in Africa, is frequently consumed as local dishes, which may include the kernels themselves or locally ground maize meal, produced without the beneficial effects of commercial dry milling and its