MYCOTOXINS IN FOODS AND FEEDS
4-FUMONISINS

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REVIEW ARTICLE

ABSTRACT :

Mycotoxins are toxic metabolites produced by naturally occurring fungi under certain ecological conditions. Mycotoxins contaminated food and feed supplies could increase the economic and health risks to humans and animals. Fumonisins are a group of recently discovered secondary metabolites produced by Fusarium spp. especially F.*moniliforme* and occasionally F.*proliferatum* that commonly contaminate corn and corn screenings.

Human consumption of fumonisins− contaminated corn has been associated with increased incidence of esophageal cancer in South Africa, India and China. Fumonisins have been shown to be associated with some major toxicological effects in animals including equine leukoencepalomalacia (ELEM) in horses and porcine pulmonary oedema (PPE) in pigs. Experimentally high doses of fumonisins caused also adverse effects in cattle, sheep, goats, rabbits, poultry and catfish.

Although many domestic animals are susceptible, natural outbreaks of clinical toxicosis from fumonisins have only been reported in horses and swine. Clinical signs of ELEM may include depression, convulsion, ataxia, sweating, apparent blindness, head pressing, recumbency, convulsions and death. The pathognomic lesions are liquefactive necrosis of white matter of brain leaving fluid− filled cavities. Actually affected horses may have centrilobular hepatic necrosis which may be present with or without brain lesions.

Signs of fumonisin toxicosis in swine (PPE) include poor weight gain, weakness, dyspnoea, cyanosis and death. Lesions include pulmonary edema and hydeothorax, hepatic and pancreatic necrosis.

INTRODUCTION:

Natural occurrence:

Fumonisins are a group of recently discovered secondary metabolites mainly produced by *Fusarium moniliforme* (*Giberella fujikuroi*) and F.*proliferatum* (Gelderblom *et al.,* 1988; Edrington *et al.,* 1995).

Fumonisin B_1 (FB_1) was isolated in 1988 by Gelderblom *et al.,* and it was chemically characterized by Bezuidenhout *et al.* (1988) and Laurent *et al.* (1989), from cultures of F.*verticillioides* and F.*moniliforme*.

Fumonisin B_1 is produced by other species of Fusarium including, *F.anthophilum*, F.*beomiforme*, F.*diamini*, F.*globosum*,

F.*hapiforme*, F.*nygamai*, F.*oxysporum*, F. *polyphialidicum*, F.*subglutinans* and F.*thapsinum* (WHO, 2000).

The mold is usually found in maize such as screening, that has been damaged by insects or adverse weather conditions (Plumlee, 1997). F.*moniliforme* has also been isolated from pelleted feeds (Plumlee, 1997).

A part from maize and maize products, fumonisins have seldom been found in other products, such as rice (Abbas *et al.,* 1998), asparargus (Logrieco *et al.,* 1998) and sorghum (Shetty and Bhat, 1997). Surveys on other cereals, such as wheat, rys, barley and oats did not show the occurrence of the toxin (Meister *et al.,* 1996).

Maize contaminated naturally by FB_1 can be simultaneously contaminated with other F.*verticillioides* or F.*proliferatum* toxins or with other agriculturally important toxins including deoxynivalenol, zearalenone, aflatoxin and ochratoxin

Many factors including environmental conditions and host susceptibility, determine the incidence and severity of grain mold and subsequent mycotoxin contamination (Stack, 2003). High levels of fumonisins are associated with hot and dry weather, followed by periods of high humidity (Shelby *et al*., 1994).

Fumonisin levels in raw corn are also influenced by storage conditions and the optimal growth of fumonisin-producing mold that leads to increased levels of fumonisin in the raw corn can occur when the moisture content of harvested raw corn during storage is 18-23% (Bacon and Neslon, 1994). High levels of fumonisins may also occur in raw corn that has be damaged by insects (Bacon and Neslon, 1994; Miller, 1999). The best temperature for the production of FB1 on corn was 20C (Weidenborner, 2001).

Types and chemical structure:

The main fumonisins are currently recognized and are designated as fumonisin B_1 (FB_1), fumonisin B_2 (FB_2) and fumonisin B_3 (FB_3), based on the hydroxylation of the hydrocarbon chain (Edrington *et al*., 1995). FB_1 is the predominating fumonisins in naturally contaminated maize kernels with a ratio of 3:1 (FB_1: FB_2) and 12:1 (FB_1:FB_3) which corresponds to about 70% of the total fumonisins concentration detected. However, in vitro there is some isolates of F.*moniliforme* producing more FB_2 than FB_1 (Weidenborner, 2001).

At least 15 different fumonisins have so far been reported and other minometabolites have been identified, although most of them have not been shown to occur naturally (WHO, 2000). They have been grouped into four main categories FA_1, FA_2, FA_3 and FAK_1; FB_1, FB_2, FB_3 and FB_4, FC_1, FC_2, FC_3 and FC_4; FP_1, FP_2 and FP_3 (Plattner, 1995; Abbas and Shier, 1997; Musser and Plattner, 1997).

Fumonisis are water soluble, heat stable, alkaline-resistant, aliphatic hydrocarbons with a terminal amine group and two tricarboxylic acid side chains (Osweiler, 1999). The number of position of hydroxyl groups on the aliphatic hydrocarbon determines the structure as FB_1, FB_2 or FB_3 (Bezuidenhoudt *et al*., 1988; Steyn, 1995).

The FB_1 form is hydroxylated at the 3, 5 and 10 positions, whereas FB_2 or FB_3 are reduced at the 10 and 5 positions, respectively (Osweiler *et al*., 1993).

Fumonisin B_1 has the empirical formula $C_{34}H_{59}NO_{15}$ and is diester of propane-1,2,3-tricarboxylic acid and 2-amino-12,16-dimethyl-3, 5, 10, 14, 15-pentahydroxyeicosane (relative

molecular mass:721). The pure structure is a white hygroscopic powder, which is soluble in water, acetonitrile-water or methanol, is stable in acetonitrile-water (1:1), and at food processing temperature and to light (WHO, 2000).

FA_1, FA_2 or FA_3 are the N-acetyl derivatives of FB_1, FB_2 and FB_3, respectively. Within each series different hydroxyl substitution result in different fumonisins FC_1, FC_3 and FC_4 lacking the C-1 terminal methyl group which is characteristic for other fumonisins. In comparison to FC_1, the hydroxylated $FC_1(OH-FC_1)$ has one more hydroxyl group at the C-3 position (Weidenborner, 2001).

Occurrence in foods and feeds:

Maize based human foodstuffs from retail outlet in 5 countries were analysed for FB_1 and FB_2 (Syndenham *et al.*, 1991). The highest man concentration occurred in 2 Egyptian samples 2.38 mg/kg FB_1 and 0.595 mg/kg FB_2. Only of 4 Peruvian samples contained 0.66 mg/kg FB_1 and 0.135 mg/kg FB_2, while only 1 of 2 Canadian samples containe4d acceptable level of FB_1 (0.05 mg/kg). The corn meal and 10 corn grits products from the USA contained mean concentration of 1.048 mg FB_1 and 0.298 mg/kg FB_2 and 0.601 mg/kg FB_2 and 0.375 mg/kg FB_2, respectively. The mean concentration in 52 corn meal and 15 corn grits samples from South Africa were 0.138 mg/kg FB_1 and 0.083 mg/kg FB_2 and 0.125mg/kg FB_1 and 0.85 mg/kg FB_2, respectively. Only 1 of 10 corn flakes/ lime treated samples contained a low level of FB_1.

Dry milling of maize results in the distribution of fumonisin into the bran, germ and flour (WHO, 2000). In dry milled maize fractions, the fumonisin concentration was approximately 3 times higher in germ and bran than in whole maize, 13 times higher in "C"

flour and 29 times higher than in corn meal and corn grits (Broggi *et al.,* 2002). In experimental wet milling, fumonisin was detect in steep water, gluten fiber and germ, but not in the starch (WHO, 2000).

Fumonisin B_1 levels in animal feedstuffs can be exceptionally high, and reached maximum concentration of 330, 70, 38, 9 and 2 mg/kg in North America, Italy, Brazil, South Africa and Thailand, respectively (WHO, 2000). FB_1 has been recorded to occur in maize and screening servings at levels as high as 195 and 330 mg/kg, respectively (Ross *et al.,* 1991).

The center of veterinary medicine conducted as survey on fumonisin in maize and maize screenings in 1991, in response to an equine leukoencephalomalacia (ELEM) outbreak occurring in late 1990 in the united states. Fumonisin was detected in 13% of the shelled maize samples (1.2 to 3.2 mg/kg), and in 100% of corn screening samples (2.6 to 32 mg/kg) (Price *et al.,* 1993). Another survey found 85% of 158 maize samples (0.6 to 96 mg/kg) (Russel *et al.,* 1993).

In Uruguay all samples of maize- based animal feeds were positive for FB_1. However, highest FB_1 were observed in South Africa for compound feed (11 mg/kg), and Thailand and China for maize (18.8 and 25.97 mg/kg, respectively). In a study of Argentina maize, FB_1 was the major fumonisin at values of up to 11.30 mg/kg (Placinta *et al.,* 1999).

In 1998, random samples of maize and poultry feeds were collected from poultry farms, feed manufactures and markets in Haryana, India and analyzed for FB_1. 91% of maize samples and 42% of poultry feed samples were found to contain FB_1. FB_1 concentrations in the maize samples ranged from 0.1 to 87 ppm, whereas the poultry feed samples contained FB_1

in the range of 0.2 to 28 ppm (Jindal *et al.,* 1999).

Concentrations of fumonisin in foods and feeds associated with human and animal health problems:

Of several samples obtained from a high oesophageal cancer risk area in the USA 7 of 7 contained FB_1 at levels of 0.105 to 1.915 mg/kg and 6 of 7 FB_2 at levels of 0.07 to 0.46 mg/kg (Sydenham *et al.,* 1991).

The fumonisin B_1 contamination of maize samples collected from two areas of Iran during 1999 was determined (Shephard *et al.*, 2002). The FB_1 levels in Mazandaran province, an area of high esophageal cancer were 0.68-7.66 mg/kg, a mean level of 3.18 mg/kg, while the FB_1 levels found in maize samples form Isfahan, an area of low esophageal cancer were 0.01 to 0.88 mg/kg, a mean level of 0.22 mg/kg.

The fumonisin levels associated with episodes of ELEM and PPE have been found to be higher (Ross *et al.,* 1991). A total of 98 samples of feeds associated with 44 cases of ELEM and 83 samples of feed associated with 44 cases of PPE in USA were analysed for FB_1. Feeds associate with ELEM contained FB_1 ranging form < 1 to 126 mg/kg, with 75 of cases have at least 1 sample > 10 mg/kg. While, feeds associated with PPE ranged from < 1 to 330 mg/kg, with 71% of the cases having at least 1 sample > 10 mg/kg (Ross *et al.,* 1991).

Fourteen feed samples form the USA that were fed to horses prior to the development of LEM were analysed for FB_1 and FB_2 (Thiel *et al.*, 1991 b). All samples contained both FB_1 (1.3-27.0 mg/kg) and FB_2 (0.1-12.6 mg/kg). FB_1 was found to be the major fumonisin in feed samples (53-93%).

A total of 21 *F.moniliforme* contaminated feed sample associated with outbreaks of confirmed and suspected cases of mycotoxciosis

in various animal species (mainly horse and pigs) were collected from farms in the state of Parana, Brazil and analysed for FB_1 and FB_2 (Syndenham *et al.,* 1992). FB_1 and FB_2 were detected in 20 and 18 of the 21 samples, respectively at concentration of 0.2 to 38.5 mg/kg FB_1 and 0.1 to 12.0 mg/kg FB_2.

Absorption, distribution and excretion:

As described by WHO (2000), there is no reports available for fumonisin absorption through inhalation or dermal exposure. However, because fumonisins are present in F.*verticilloides* cells (mycelia, spores and conidiophores) (Tejada- Simon *et al.,* 1995), there is a potential for absorption through inhalation or buccal exposure. The risk from absorption due to animal exposure would seem slight, since fumonisins are very water soluble and, typically polar compounds do not easily penetrate the undamaged skin (Flynn, 1985).

Fumonisins have been reported to be poorly absorbed, rapidly excreted and persistent in small amounts in liver and kidney (Norrd *et al.,* 1996). Fumonisine when dosed orally to vervet monkeys, dairy cows and pigs are poorly absorbed (2 to < 6% of dose) (Prelusky *et al.,* 1994, 1995, 1996 a, b; Shephard *et al.,* 1994a, b).

In ruminants, rumen metabolism may reduce the bioavailability of FB_1 as the hydrolyzed form of FB_1, comprised 60-90% of the total amount of FB_1 found in feces. In non-ruminants the parent compound was the dominant species present (Rice and Ross, 1994). In orally dosed laying hens and dairy cows, the quantity of FB_1 detected in plasma and tissues is very low (< 1% of dose) (Scott *et al.*, 1994; Vudathala *et al.,* 1994 and Prelusky *et al.,* 1996a).

No FB_1 was detected in the milk of lactating sows fed diets containing non- lethal levels of FB_1 and there was no evidence of toxicosis in their suckling pigs (Becker *et al.,* 1995). However, in a study with lactating cows administered FB_1 intravenously, the carry over rate of FB_1 into the milk reached a maximum of 0.11% (Hammer, *et al.,* 1996). In other studies no fumonisins were detected in cows milk (Scott *et al.,* 1994; Richard *et al.,* 1996). FB_1 was found in only one of 165 samples of milk from Wisconsin, USA at a level close to 5 ng/ ml (Maragos and Richard, 1994).

There are also no data demonstrating, that fumonisin consumption results in transfer to chicken eggs (Vudathala *et al.,* 1994; Prelusky *et al.,* 1996a). Also, FB_1 was not found in milk, meat or eggs from animals fed grains containing FB_1 levels that would not affect the health of the animals (WHO, 2000).

Mode of action:

Experiments by Wang *et al*. (1991) indicated that fumonisins are inhibitors of sphingolipids biosynthesis through inhibition of ceramide synthetase and by blocking protein synthesis. Sphingolipids are found in large quantities in brain and nerve tissues. Sphingosin is synthesized in the endoplasmic reticulum. Ceramide is form by a combination of either a free fatty acid or an acyl-coenzyme A and sphingosine (Mayes, 1988).

The site of inhibition occurs where sphingosine and fatty acyl-coenzyme A combine to form dihydroceramide, which would result in accumulation of free sphingosine in tissues and serum (Norred *et al*., 1992; Merrill *et al*., 1993 b). It has been postulated that the similarities between fumonisins and long-chain (sphingoid) basis allow them to be recognized as substrate (transition state/or product analogs) of sphingosine or sphinganine-N-acetyl

transfearse. Disruption of this pathway could explain at least some of the pathological effects of fumonisins (Leeson *et al.,* 1995).

The degeneration of neuronal cells seen in ELEM may be due to inhibition of sphingolipid biosynthesis because of their high concentration in the brain. On the other hand accumulation of sphinganine in cells exposed to fumonisins may lead to cell death (since long- chain bases are highly cytotoxic), or to cell proliferation, these compounds are mitogenic for some cell types (Wang *et al.,* 1991).

The alteration in sphingolipid metabolism caused by fumonisins can be monitored by measuring serum lipids of sphingosine (So), sphinganine (Sa) and complex sphingolipids (Leeson *et al.,* 1995). Analysis of sphingosine and sphinganine levels in livers and serum revealed an increase in the level of sphinganine, with no change in the level of sphingosine, that result in an increase in the sphiganine: sphingosine ratio (Henery *et al.,* 2000).

Studies in horses (Wang *et al.,* 1991, 1992), pigs (Riley,1993), sheep (Gurung *et al.,* 1998), broiler chicks (Weibking *et al.,* 1993a), turkeys (Weibking e*t al.,* 1993b) and catfish (Goel *et al.,* 1994) have demonstrated a significant increase in the serum Sa to So ratio in animals fed a ration containing FB_1.

Toxicity:

Species difference: FB_1 is extremely toxic to horses, moderately toxic to swine, weakly toxic to cattle and has been associated with oesophageal cancer in humans. Poultry are even more resistant to adverse health effects from fumonisin (Stack, 2003). Pregnant New Zealand rabbits are found to be very sensitive to the toxic effects of FB_1 (LaBord *et al.,* 1997). The species, age and health of the animal as well as the level and duration of exposure to the

mycotoxin will determine the magnitude of the effect of exposure (Stack, 2003).

The feeding of fumonisins contaminated corn produced leukoencephalomalacia (LEM) in horses (Wilson *et al.,* 1990; Ross *et al.,* 1991) and pulmonary edema (PE) and hydrothorax in swine (Harrison *et al.,* 1990; Osweiler *et al.,* 1992). Experimental high doses of fumonisine caused adverse effects in cattle (Osweiler *et al.,* 1993) and poultry (Weibking *et al.,* 1993a). Human consumption of corn has been correlated with increased incidence of oesophageal cancer in certain parts of the world (Sydenham *et al.,* 1991; Chu and Li, 1994). In addition to their adverse effects on the brain, liver and lungs, fumonisins also affect the kidneys, pancreas, tests, thymus, gastrointestinal tract and blood cells (Vencelli and Parker, 1999).

Fumonisine have produced liver damage and changes in the levels of certain classes of lipids especially sphingolibids, in all animal studies (Merill *et al.,* 1997), kidney lesions were also found in many animals (Norred *et al.,* 1998; Merrill *et al.,* 1997).

Chronic feeding of purified FB_1 at levels of 50 ppm or more produced liver cancer and decreased life span in female mice and kidney cancer in male rats without decrease life spans (NTP, 1999).

Disturbances of thermoregulation has been observed in some animals exposed to the mycotoxin (Becker *et al.,* 1995). In horses, FB_1 causes profuse sweating, the major cooling mechanism for this species, even though body temperature remain normal (McCue, 1989). Cattle with fuminisin toxicosis has hyperthermia, with heat intolerance developing at a lower body temperature than normal for cattle (Schmidt and Osborn, 1993; Thompson and stuedermann, 1993). Although,

hyperthermia has not been reported in swine with FB_1 toxicosis, the reduced lung function associated with edema would reduce the ability to pant. In swine panting is a major mechanism for coping with high temperature, thus high environmental temperature may exacerbate FB_1 toxicosis attributable to reduced pulmonary function associated with FB_1 induced pulmonary edema (Becker *et al.,* 1995).

Human health effects:

Currently, there is no direct evidence that fumonisins cause adverse health effects in humans. Studies currently available demonstrate only inconclusive association between fumonisins and human cancer (FDA, 2001b).

A very high incidence of oesophageal cancer among the black population of the Transkei, South Africa has been reported in several surveys (Jaskiewics *et al.,* 1987; Makaula *et al.,* 1996). Investigation in South Africa suggested an association between high levels of fumonisin producing molds on corn used to make alcoholic beverages and oesophageal cancer in humans (Rheeder *et al.,* 1992). This corn has been found to contain up to 118 mg/kg fumonisins.

Based on experiments conducted on beer made from worth containing FB_1, such beers could contain fumonisin concentration of 30 mg/ liter beer (Scott *et al.,* 1995). However, these studies were limited by the lack of controlled conditions, particularly for established confounding risk factors (e.g. alcohol consumption) and therefore do not allow any definite conclusions to made about cancer causation in humans (FDA, 2001 b).

An outbreak of poisoning, characterized by abdominal pain and diarrhea caused by fumonisin-contaminated maize and sorghum in India during 1995, was reported (Bhat *et al.,*

1997). An epidemiological survey was conducted in the affected villages and a detailed house to house in selected villages. People in 27 out of 50 villages surveyed in Karnatake state were affected and disease was seen only in households and subjects consuming rain damaged moldy sorghum or maize.

All 20 sorghum and 12 maize samples collected from household had fusarium sp. as the dominant microflora and contained FB_1 in the range of 0.14 to 7.8 and 0.25 to 64.7 mg/kg, respectively. In contrast, samples collected from unaffected households has FB_1 in low levels ranging from 0.07 to 0.36 and 0.05 to 0.24 mg/kg, respectively.

However, this study lacked control of established risk factors. In addition, contaminants other than mycotoxins can not be eliminated as causative factors, and a similar dissociation was not detected in studies conducted in other countries (FDA, 2001 b).

Other studies associated with high levels of fumonisin producing molds on corn with oesophageal cancer in China reported that corn samples from areas with high incidence of esophageal cancer, contained FB_1 at levels raining from 18 to 155 mg/kg. These results established that home- grown maize in high incidence areas of oesophageal cancer in China may be contaminated with very high levels of FB_1. These studies were correlation studies where there was no clear picture on the association of either fumonisins or *F.verticilliouides* contamination with osophageal cancer (WHO, 2000).

Further in an area of China with high incidence of gastric cancer, Groves *et al.* (1999) observed a lack of association between consumption of fumonisin- contaminated corn with gastric or any other human cancer.

Animal health effects:

Ruminants:

Cattle and sheep are mildly affected by dietary concentrations of fumonisin greater than 100 ppm, but fatalities do not occur (Osweiler *et al.*, 1993). Very few studies have focused on the effects of fumonisins in ruminants. (Kriek *et al.*, 1981) demonstrated that *F.moniliforme* cultures capable of inducing ELEM and PPE were also toxic to sheep. Beasly and Buck (1982) reported feed refusal in cattle given corn contaminated with F.*moniliforme*, but existence of fumonisins was not know at that time.

Dietary fumonisins concentration greater than 148 mg/kg of diet to calves may adversely affect liver and immune functions (Osweiler *et al.*, 1993). Livers from treated calves had mild hydropic degeneration and cloudy swelling in a periacinar pattern throughout the liver. These changes were consistent with the mild to moderate elevation of liver enzymes. Cattle fed 530 mg of total fumoninis /kg diet, supplied by fumonisin- containing- culture material for 30 days, showed no signs of toxicosis (Smith and Thakur, 1996), although liver function tests revealed some hepatobiliary compromise.

Fumonisin B_1- contaminated feed (95 mg/kg), fed for 112 days caused no treatment- related effects on feed intake, nutrient digestibility, or weight gain in weanling angora goats (Gurung *et al.*, 1998). However, sphingolipid disruption and mild liver and kidney damage were observed in weanling goats.

In contrast, (Edrington *et al.*, 1995) observed clinical signs such as marked decrease in feed intake diarrhea, lethargy and ultimately death in lambs dosed intraruminally with 11.1,

22.2 and 45.5 mg of total fumonisins/ kg of b.wt. for only 4 days. The actual amounts of total fumonisin intake were determined to be 355.2, 710.4 and 1456 mg/ lamb/ day. Such as concentrations have not been reported to occur under normal feeding conditions (Gurung *et al.,* 1998).

Monogastric animals:

Equidae:

Horse are the most sensitive of the domestic animals and dietary concentration as low as 10 ppm for more than 50 days may cause leukoencephalamalacin (LEM) (Wilson *et al.,* 1990; Ross *et al.,* 1992). The ELEM has been recognized since 19th century as a sporadically occurring condition (WHO, 2000), and was experimentally produced by feeding moldy maize obtained from a field case in Kansas by Butler in 1902. The disease was known as "moldy corn poisoning" but attempts to identify the responsible fungus failed. History has referred to this disease by many names including: moldy corn poisoning, corn stalk disease, forage poisoning, leukoencephalomyelitis, blind staggers, epizootic cerebritis and non virus encephalo-myelitis (Wilson *et al.,* 1985; Asquith, 1991; Masri, 1992).

As described by WHO (2000), Wilson and Maronport (1971) succeeded in establishing the causative agent when they isolated F.*verticillioides* as the predominant contaminant of moldy maize that has caused cases of ELEM in Egypt, and reproduced the disease by feeding culture material of the fungus on maize to 2 donkeys. Subsequently investigators in South Africa confirmed the ability of F.*verticillioides* (MRC826) culture material to induce the characteristic clinical signs and pathological changes of ELEM as well as hepatosis in horses

and donkeys (Kellerman *et al.,* 1972; Marasas *et al.,* 1976, 1988 and Kriek *et al.,* 1981).

ELEM has also produced in horse by intravenous administration of pure FB_1 (Bezuidenhout *et al.,* 1988; Gelderblam *et al.,* 1988) and in horses given pure FB_1 by stomach tube (Kellerman *et al.,* 1990). Moreover the disease has been observed naturally in horses and ponies fed feeds contaminated with fumonisins (Wilson *et al.,* 1992; Ross *et al.,* 1993).

The lowest FB_1 dose that has resulted in ELEM in a controlled experiment is 22 mg/kg in diets formulated with naturally contaminated maize screenings (Wilson *et al.,* 1992). Ross *et al.* (1992) suggested that concentrations ≥ 10 mg/kg FB_1 in horse feeds were likely to be involved in LEM. In 40 to 45 confirmed cases of ELEM, FB_1 levels in feeds were ≥ 10 mg/kg, while non-problem feeds contained ≤ 10 mg/kg FB_1.

Clinical signs:

The clinical signs of ELEM appear at a variable length of time after feeding of the mycotoxin is begun (Kibluk *et al.,* 1995). In experimental cases, clinical signs have appeared as early as day 7 and as late as day 75 (Ross *et al.,* 1993). Clinical signs of the disease usually appear abruptly and the clinical course is relatively short, with most affected horses dying within 2 to 3 days (Wilson *et al.,* 1985; Marsi *et al.,* 1987; Kobuk *et al.,* 1995).

The first sign may be inappetence, followed quickly by neurologic abnormalities. Behavioral signs vary from depression and a pathy to apprehension, hyperexcitability or frenzy (Kellerman *et al.,* 1990; Masri *et al.,* 1992; Ross *et al.,* 1993; Kobluk *et al.,* 1995). Other neurologic signs may include head pressing, aimless circling, unilateral or bilateral blindness, ataxia, tremors, cranial nerve deficits

(such as lip and tongue paresis and dysphagia), and weakness, progression of the condition leads to recumbency, convulsions, coma and death (Wilson *et al.,* 1985, 1990; Masri *et al.,* 1987; Marasas *et al.,* 1988; Kellerman *et al.,* 1990; Thiel *et al.,* 1991b; Ross *et al.,* 1993 and Konluk *et al.,* 1995).in a horse injected intravenously 7 times over 10 days with 0.125 mg of FB_1/kg b.wt./day, clinical signs of neurotoxicosis included nervousness followed by a pathy, a wide- based stance, trembling, atoxin, reluctance to move, paresis of the lower limb and tongue and an inability to eat or drink were recorded (Marasas *et al.,* 1988). The principal lesions were severe oedema of the brain and early bilaterally symmetrical focal necrosis in the medulla oblengata.

Death frequently occurs within 5 days of the onset of clinical signs (Smith, 2000): sudden death, without prior clinical signs, has been reported. Animals that survive often have persistent neurologic deficit (Plumlee, 1997). The hepatic form of the disease may be accompanied by icterus, oedema, hemorrhage and hepatoencephalpaathy (Plumlee, 1997).

Gross and histopathological findings:

ELEM syndrome is characterized macroscopically by the presence of liquefactive necrotic lesions in the white matter of the cerebrum (WHO, 2000). Other lesions include encephalomalacia of both white and grey matter in the cerebrum, cerebellum, midbrain or brain stem (Masri *et al.,* 1987; Kobluk *et al.,* 1995). Acutely affected horses may have centrilobular hepatic necrosis, which may be present with or without the brain lesions (Marasas *et al.,* 1988; Wilson *et al.,* 1990).

Microscopic lesions included: liquefative necrosis of white matter, with complete loss in some areas perivascular edema, hemorrhage

and perivascular cuffing in the white matter adjacent to malacic foci. Eosinophils and plasma cells predominated in perivascular cuffs. Foci of myelin degeneration were seen in non-necrotic areas of white matter. Lesions in grey matter included satellitosis and neuronophagia. Leptomeningeal edema was present in some cases (Marasas *et al.,* 1976; Rooney and Robertson, 1996).

In conjunction with ELEM, muscular melting and cardiac failure have been reported (Vencilli and Parker, 1999). In addition to brain lesions, histopathological abnormalities in liver and kidney have been reported in horses orally dosed with pure fumonisins, maize screening, naturally contaminated with fumonisins, or culture material containing known amounts of fumonisins (Kellerman *et al.,* 1990; Wilson *et al.,* 1992; Caramalli *et al.,* 1993 and Ross *et al.,* 1993). Lesions of hepatic form include centrilobular necrosis, periportal fibrosis, periportal vacuolation and bile duct proliferation; lipofuscin deposition in macrophages and kupffer cells, and infiltration with a mixture of inflammatory cells (Marasas *et al.,* 1976; Plumlee, 1997). Perirenal edemo and congestion may be present (Marasas *et al.,* 1976).

Elevated serum enzyme level indication of liver damage (Wilson *et al.,* 1992) are produced by elevation of the serum Sa/So ratio (Wang *et al.,* 1992). The protein concentration and white cell count in the cerebrospinal fluid are elevated (Plumlee, 1997).

Natural occurrence of ELEM:

Outbreak of ELEM has been described throughout the world in Argentina, China, Egypt, New Caledonia, South Africa, Greece and other parts of Europe and the United States

(Jubb and Kennedy, 1985 and Marasas *et al.,* 1988).

The disease occurs most commonly in the United states from September to May, Certain growing and harvest conditions (dry summers and/or drought, followed by rainy Autumns) increase the amount of *Fusarium moniliforme* on feed corn (Rooney and Robertson, 1996). Epizootics have occurred in years in which corn was harvested during periods of high humidity (Badiali *et al.,* 1968; Masri *et al.,* 1987).

In 1934 to 1935, an episode of ELEM was recorded in Illinois (Graham, 1936), where 5000 horses died following a summer drought and early fall rains, when horse were allowed to forage in the stalk fields, and others were fed moldy corn after harvesting. Sporadic occurrences of the disease were reported in Egypt by Badiali *et al.* (1968), following the flooding of the Nile, resulting in mold growth on the autumn crop which was fed to donkeys.

An outbreak of ELEM was reported in a riding club in new Caledonia in 1981, where horses were being fed a ration containing high levels of locally grown corn. The development of the disease coincided with the feeding of a new batch of feed containing the corn. Out of the 45 affected horses, 5 died 10 to 28 days after the onset of clinical signs. *F.moniliforme* was recovered from all samples of corn that were examined. A second episode occurred in 1983 involving a 15- year- old stallion that had been fed locally produced corn. In this case, the major gross pathology was a focus of liquefaction of the white matter in the right cerebral hemisphere (Domench *et al.,* 1985; Asquith, 1991).

A survey conducted in 1984 to 1985, evaluated 22 farms with confirmed cases of ELEM in North Carolina. Gross lesions of necropsied animals revealed liquefaction

necrosis in the white matter of one or both cerebral hemispheres preliminary data suggested that some of the oats used in the feed preparation may have been infected. This was the first report of the disease occurring from the ingestion of commercial pelleted and non-pelleted horse rations and indicated that F.*moniliforme* may be capable of remaining viable even after the pelleting process (Wilson *et al.,* 1985; Asquith, 1991).

During the autumn of 1989, in an ELEM involving 18 of 66 purebred Arabian horses at breeding, training stable in Arizona, USA, the condition was fatal in 14 horses (Wilson *et al.,* 1990). These horses had been fed a diet containing 37 to 122 ppm of FB_1. Gross pathological findings included, liquelactive necrosis in parts of the cerebral white matter and hemorrhagic foci of various sizes in the brain stem. Histopathological findings included reafied white matter with pyknotic nuclei and oesinophilic cytoplasm. This was the first definite report on ELEM and associated FB_1 concentration.

An outbreak of ELEM affecting 6 of 10 pleasure horses in adjacent paddocks at a boarding facility in Pennsylvania, USA is reported (Wilklins *et al.,* 1994). The horses showed depression, lethargy, ataxia, blindness, seizures and recumbency after eating whole ear maize. Four horses died or were euthanized. F.*moniliforme* (*Gibberella fujikuroi*) was cultured from the ear maize and fumonisins B_1, B_2 and B_3 at concentrations of 370, 105 and 41 ppm, respectively, were detected.

The first report of ELEM in Italy was observed by Caramelli *et al.* (1993) on a farm in Piedmont, where 3 of 40 horses showed sudden anorexia, hyperexcitability, incoordination, flacid lips, blindness and recumbency followed by death in 72 hours. These horses had been fed

for some days on maize screenings containing 60 mg/kg FB_1 and 14.6 mg/kg FB_2.

ELEM was first diagnosed in Hungary in September 1995, in a 13- years- old castrated male, a 6- ears- old stallion and a 2- years- old foal (Fazekas and Bajmicy, 1996). The disease started with lack of appetite followed by difficulties in swallowing and chewing indicating the paralysis of cephalic and pharyngeal muscles. Paralysis of the cephalic and cervical muscles spread to the muscles of the extremities and trunk and ataxia developed. Sings of blindness appeared in an animal. At the final stages of the disease, the affected animals became recumbent and died. Post-mortem examination of one horse showed oedema of the lungs and white matter of the brain. The affected animals were fed green maize containing FB_1 at 18.5 mg/kg of the whole plant.

Pigs:

Swine consuming more than approximately 120 ppm dietary fumonisins for 4 to 10 days develop acute porcine pulmonary oedema (PPE) (Coloin and Harrison, 1992; Colvin et al., 1993), more than 50 ppm of fumonisins causes mild liver lesions within 7 to 10 days, but concentrations of 25 ppm or less cause no apparent clinical effects. Mild microscopic hepatic lesions can be produced from diets as low as 23 ppm (Mortelin et al., 1994; Osweiler, 1999). The sphinganine- sphingosine ratio may be altered in the serum by feed concentrations as low as 5 ppm and in the lung, liver and kidney by feed concentrations of 10 ppm, although the clinical significance of this is not established (Mortelin et al., 1994; Riley et al., 1993 and Rotter et al., 1996).

PPE represents the acute effects of FB_1 (Becker et al., 1995), while, the chronic effects of FB_1 in weaned pigs showed hyperplastic plaques in the oesophageal mucosa (Casteel et al., 1993). Swine fed a ration containing 100- 190 ppm FB_1 for up to 83 days developed nodular hyperplasia of the liver and hyperplasia of the esophageal mucosa (Casteel et al., 1993). Swine fed 150- 170 ppm FB_1 for up to 210 days developed medial hyperplasia of the pulmonary arteries and changes in gravimetric measurements of the heart (Casteel et al., 1994). In experimental trials, culture material of F.moniliforme (NRC826) was fed to horses, pigs, sheep, rats and baboons (Krick et al., 1981) lung oedema occurred only in pigs.

Acute PPE might be induced by increases in pulmonary interstitial macrophages resulting in release of vasoactive mediators (Huschek et al., 1992), pulmonary hypertension caused by vasoconstriction (Smith et al., 1996) and acute left- sided heart failure (Smith et al., 1999). There responses have been hypothesized to lead to pulmonary oedema either by increased pulmonary hydrostatic pressure or by pulmonary capillary endothelial cell damage (Cumpecht et al., 1998; Osweiler, 1999).

Clinical signs of PPE typically occur soon (2-7 days) after pigs consume diets containing large concentrations of fumonisins over a short period of time (WHO, 2000). These signs include labored or difficult breathing (dyspnoea), weakness, cyanosis and death (Osweiler et al., 1992; Mortelin et al., 1994 and Stack, 2003). Once signs appear, death may occur in less than 4 hours (Colvin and Harrison, 1992; Riley et al., 1993). Pregnant sows may abort from 1 to 4 days after the onset of signs (Osweiler, 1992), probably as a sequela to fetal anoxia from severe pulmonary oedema (Osweiler, 1999).

At necropsy, the diseased animals exhibit varying degrees of interstitial or interlobular oedema, with pulmonary oedema and

hydrothorax. Varying amounts of clear cell- free yellow fluid accumulate in the pleural cavity (Colvin and Harrison, 1992; Colvin *et al.,* 1993). Early histologic changes in the lung consisted of perivasicular oedema followed by interlobular and peribronchial oedema. Ultrastructurally, alveolar endothelial cells contained unique accumulations of membranous material in the cytocavitry network (Cumpecht *et al.,* 1998). Acidophilic fibriller material in alveoli and interlobular lymphatics, hyalinized alveolar capillary thrombi and increased numbers of pulmonary intravascular macrophages filled with osmophilic material (Colvin *et al.,* 1992; Riley *et al.,* 1993 and Osweiler, 1999).

Dietary fumonisin concentrations of 75 to 120 ppm for 1 to 4 weeks cause icterus, reduced feed intake weight loss and occasionally diarrhoea (Osweiler *et al.*, 1993; Osweiler, 1999). Toxic hepatosis occurs concurrently with PPE (Osweiler *et al.*, 1992; Colvin *et al.,* 1993) and in also observed in animals that consume high levels of fumonisins but do not develop PPE (Hasckek *et al.,* 1996). Typically the liver contains multiple foci of coagulative necrosis with disrupted hepatic architecture, increased mitotic figures in heptocytes and single hepatic necrosis (Osweiler *et al.*, 1992; Colvin and Harrison, 1992; Colvin *et al.,* 1993 and Riley *et al.*, 1993).

Rabbits and Poultry:

Rabbits:

Rabbits given multiple doses of FB (0.15, 0.3, 0.5 or 1.0 mg/kg b.wt. for 4 or 5 days) intravenously, were lethargic and anoretic and had decreased urine production (Gumprecht *et al.,* 1995). Renal lesions consisted of severe proximal tubular necrosis. Liver lesions were variable and consisted of mild hepatic vacuolation and bile stasis. A single dose of FB_1

at 1.0 mg/kg b.wt induced renal but not hepatic injury.

Bucci *et al.* (1996) studied LEM and hemorrhage in the brain of rabbits gavaged with FB_1 (1.75 mg/kg b.wt. daily) and observed that of 5 gavaged pregnant rabbits, 2 died after receiving 9 and 13 doses, respectively. Microscopic examination revealed focal small hemorrhages in cerebral white matter in both animals, with malacia and hemorrhage also present in the hippocampus of one. Both animals also had marked degeneration of renal tubules epithelium and of hepatocytes. Apoptosis was the dominant degenerative change in kidney and liver.

Pregnant New Zealand white rabbits are very sensitive to the toxic effects of Fb_1 (LaBorde *et al.,* 1997). Maternal toxicity was observed at daily gavage dosage 0.25 mg/kg b.wt from gestational day 3 to gestational day 19. The maternal kidney, serum and urine Sa/So ratios were increased, but there was no increases in these ratios in fetal liver, brain or kidney. The lowest observed effect level for maternal toxicity was 0.1 mg FB_1/kg b.wt., which is equivalent to a calculated dietary FB_1 level of 2.3 mg/kg diet (LaBorde *et al.,* 1997; WHO, 2000). This level is very close to the 2.24 mg/kg of fumonisin in the diet of young colts that could be theoretically be hazardous. Consequently, rabbits were grouped with horses (FDA, 2001a).

Poultry:

Turkey poults are more sensitive to the toxic effect of FB_1 than broiler chicks (Weibking *et al.,* 1993b) but chickens, turkeys and ducklings are relatively resistant to FB_1 in comparison to horses and swine, where significant toxicity and mortality occur at levels greater than 10 and 20 mg FB_1/ kg diet, respectively (Ross *et al.,* 1991;

Osweiler *et al.,* 1992). On the other hand, one day- old chicks are more sensitive to the adverse effects of FB_1 than 7 or 21 days- old chicken (Javed *et al.,* 1993).

Broilers:

A day- old chicks fed diets containing 200 or 300 mg FB_1/kg diet for 3 weeks had thymic cortical atrophy, multifocal hepatic necrosis, bitiary hyperplasia and widening of the proliferating cartilage zone in proximal tibiotarsal physes (Ledoux *et al.,* 1992). Acute randomly oriented hepatic necrosis with moderate biliary hyperplasia and hypertrophy of kupffer cells, mild- villus atrophy and goblet cells hyperplasia in the lower small intestines, small foci of acute myocardial and skeletal muscle necrosis and widening of both proliferating and hypertrophic zones in proximal tibiotarsal physes were found in a day- old chicks fed 300mg FB_1/kg diet for 2 weeks (Brown *et al.,* 1992).

A day- old chicks fed diets containing 89, 190, 283, 289, 481, 592 and 681 mg of total fuminisins/ kg diet for 3 weeks, had increased sphinganine: sphingosine ratios. Isolated foci of hepatic necrosis with a mild heterophil and macrophage infiltration, moderate diffuse hepatocellular hyperplasia, mild biliary hyperplasia and moderate to severe periportal granulocytic cell proliferation were noted only in broiler fed levels equal to or greater than 289 mg/kg (Weibking *et al.,*1993a; FDA, 2001a).

Dose response clinical signs, reduced body weight and mortality were observed in chicken fed diets containing 125 or 274 mg purified FB_1/kg diet (Javed *et al.,* 1993).

Immunosuppression in chickens was produced in broilers fed diets containing 10 mg pure FB_1/kg diet or diets formulated from F.verticillioides (MRC826) culture material

containing 30 to 80 mg FB_1/kg diet. Birds had reduced spleen and/ or bursa weights and altered haematological parameters (Espada *et al.,* 1994, 1997).

Decreased prothrombin time and increased fibrinogen concentration and the activity of antithrombin III were observed in blood from broiler chicks that had been fed dietary FB_1 at a concentration of 10 mg/kg for only 6 days (Henry *et al.,* 2000). Dietary FB_1 at concentration of 80 mg/kg or less did not adversely affect body weight, feed efficiency or water consumption in broiler chicks. Liver sphinganine concentration and the sphinganine: sphingosine ratio were increased significantly in all treated groups that had received 20, 40 and 80 mg FB_1/kg diet (Henry *et al.,* 2000).

Ducks:

A day- old white Pekiny ducklings fed diets containing 120.5, 240.9 and 481.8 mg of total fumonisins for 21 days had statistical increase in the mean kidney weights, liver sphinganie: sphingosine ratio and serum gamma glutamyl transferase activity as well as, mild to moderate hepatocellular hyperplasia (Bermudez *et al.,* 1995; FDA, 2001a).

Turkeys:

Turkey poults have been shown to be susceptible to the adverse effects of FB_1. Biliary hyperplasia, hypertrophy of kupffer cells, thymic cortical atrophy and moderate widening of the proliferating and degenerating hypertrophied zones of tibial physes were observed in turkey poults fed diets containing 100-200 mg FB_1/ kg diets (Weibking *et al.,* 1993b). Poor performance, increased organ weight, diarrhea, biliary hyperplasia, hepatocellular hyperplasia and rickets are

caused in poults by dietary levels of FB_1 ($\geq$75 mg/kg) (Weibking *et al.,* 1993a, 1995).

A day- old turkey poults fed diets containing graded levels of total fumonisins (33, 66, 99, 132, 231, 330, 429, 528 and 627 mg/kg) for 21 days showed liver lesions at levels equal to or grater than 99 mg/kg. hepatocellular hyperplasia was mild at 99 and 132 mg/kg, moderate to severe at 330 mg/kg and severe at 429, 528 and 627 mg/kg (Leudoux *et al.,* 1996; FDA, 2001a).

Turkey poults fed FB_1- contaminated diet for 21 days showed mild hepatic lesions at 75 mg/kg, cardiac lesions at 475 mg/kg, decreased performance at 325 mg/kg and increased liver weights at 25 mg/kg (Ledoux *et al.,* 1996). Increased liver weight with moderate diffure hepatocellular hyperplasia and elevated enzymes were observed in poults fed 200 mg FB_1/ Kg of diet (Bermudez *et al.,* 1997).

Gross lesions induced by fumonisin toxicity in poultry comprise consistent enlargement of the liver, and variable enlargement of kidney, pancrease, proventriculus and gizzard, atrophy of lymphoid organs and rickets (Calnek *et al.,* 1997).

Histologically, the liver has multifocal necrosis of hepatocytes, hyperplasia of hepatocytes and bile ductules and hypertrophy of Kupffer cells. The intestine has villus atrophy and goblet cell hyperplasia. Growth plates are widened in both the zones of proliferating and hypertrophic cartilage. Myocardium and skeletal muscles have mild lesions and lymphoid tissues are depleted (Gureshi and Hagler, 1992; Calnek *et al.,* 1997).

From the results of experimental studies conducted on fumonisins in poultry, it appears that: the turkey poult study (Ledoux *et al.,* 1996) showed toxic effects at 99 ppm, the broiler study (Weibking *et al.,* 1993a) at 190 to 280 ppm and the duckling study (Bermudez *et al.,* 1995) at

120 ppm of total fumonisins in the diet. These toxic effects center around liver lesions.although chicken may be slightly more resistant to fumonisins than turkeys and duckling, it appears that these 3 species should be considered fairly resistant to the toxic effects of fumonisins and should be grouped into one category (poultry fed for slaughter) (FDA, 2001a).

Catfish:

In channel catfish dosed with F.*moniliforme* culture, the lowest dietary concentrations of fumonisin associated with significantly elevated ratios of sphinganine to sphimgosine in kidney, serum, liver and muscle were 10, 20, 40 and 80 mg of FB_1/ kg of diet, respectively (Goel *et al.,* 1994). Catfish fed diets containing FB_1 levels equal to or higher than 40 mg/kg of diet for12 weeks showed decreased feed consumption, feed efficiency and weight gain (Li *et al.,* 1994; Li and Robinson, 1995). The minimum level of FB_1 that depressed growth was 20- 40 mg/kg (Li *et al.,* 1994).

Catfish fed diets containing 40 mg FB_1/ kg diet for 12 weeks had increased liver glycogen, increased vacuolation in nerve fibers and perivascular lymphohistiocytic investment in the brain compared with fish fed diets containing lower levels of fumonisins (0.7 to 20 mg/kg) (Li *et al.,* 1994). Small white foci (2 to 4 mm diameter) of subcapsular adipocyte hyperplasia were observed in the livers of channel catfish fed 20 mg of FB_1/ kg of diet for 10 and 14 weeks. Livers had also swollen hepatocytes with lipido containing vacuoles, lymphocyte infiltration and scattered necrotic hepatocytes (Lumlertdacha *et al.,* 1995).

Regulatory control:

Based on the wealth of available information on the adverse animal health effects associated with fumonisins, human health risks associated with exposure to fumonisins are possible. Therefore, human exposure to such natural toxins should not exceed levels achievable with the use of good agricultural and good manufacturing practices (FDA, 2001b).

The recommended maximum levels for fumonisins in human foods and animal feeds (Tables, 1 and 2) that FDA considers achievable with the use of good agricultural and good manufacturing practices are presented below (FDA, 2000; FDA, 2001 b, c).

Table (1): Recommended maximum levels of fumonisins in human food products, U.S.

Product	Total fumonisins $(FB_1 + FB_2 + FB_3)$
Degermed dry milled corn products (e.g., flaking grits, corn grits, corn meal, corn flouer with fat content of < 2.25%, dry weight basis)	2
Whole or partially degermed dry milled corn products (e.g. flaking grits, corn grits, corn meal, corn flour with content of < 2.25%, dry weight basis)	4
Dry milled corn bran	4
Cleaned corn intended for masa production	4
Cleaned corn intended for popcorn	3

Table (2) Recommended maximum levels for fumonisins in corn, corn by products and the total ration for various animals species

Animal or class	Maximum level of total fumonisins in corn and corn prosucts (ppm[1])	Feed factor [2]	Maximum levels of total fumonisins in total ration (ppm)[1]
Horses[3]	5	0.2	1
Rabbit	5	0.2	1
Catfish	20	0.5	10
Swine	20	0.5	10
Ruminants[4]	60	0.5	30
Milk[5]	60	0.5	30
Poultry[6]	100	0.5	50
Ruminant, poultry and mink breeding sock[7]	30	0.5	15
All others[8]	10	0.5	5

1. Total fumonisins $FB_1 + FB_2 + FB_3$.
2. Fraction of corn or corn by- products mixed into the total ration
3. Includes asses, zebra and onagers.
4. Cattle, sheep, goats and other ruminants that are $\geq$ 3 months old and fed for slaughter.
5. Fed for pelt production.
6. Turkeys, chickens, ducklings and other poultry fed for slaughter.
7. Includes laying hens, roosters, lactating dairy cows and bulls.
8. Includes dogs and cats.

In six instances, the U.S. FDA (2000) grouped species together because the animals seemed to have a similar sensitivity to fumonisins. This is an attempt to avoid a multitude of guidance levels and does not necessarily imply that the species are biologically similar.

1-Horses and rabbits were grouped together as the most sensitive species. Corn and corn- by

products used in rations of horses and rabbits should contain less than 5 ppm of total fumonisins and comprise no more than 20% the dry weight of the total ration (Table 2). The total ration should contain less than 1 ppm of total fumonisins.

2-Catfish and swine were grouped together as intermediate in sensitivity to fumonisins. Corn and by products used in rations of catfish and

swine should contain less than 20 ppm of total fumonisins,and comprise no moe than 50% of the dry weight of the total ration. The total ration should contain less than 10 ppm of total fumonisins.

3-Ruminants, mink and poultry were considered more resistant than horses, rabbits, catfish and swine to fumonisins. Corn and corn by products used in rations of ruminants that are at least 3 months old fed for slaughter and in rations of mink fed for pelt production should contain less than 60 ppm of total fumonisins, and comprise no more than 50% of the dry weight of the total ration. The total ration should contain less than 30 ppm of total fumonisins.

4-Corn and corn- by products used in the rations of poultry fed for slaughter should contain less than 100 ppm of total fumonisins, and comprise no more than 50% of the dry weight of the total ration. The total ration should contain less than 50 ppm of total fumonisins.

5-Corn and corn- by products used in the rations of mink, ruminants and poultry breeding stock should contain less than 30 ppm of total fumonisins, and comprise no more than 50% of the weight of the total ration.the total ration should contain less than 15 ppm of total fumonisins.

6-Corn and corn- by products used in rations of dogs and cats should contain less than 10 ppm of total fumonisins, and comprise no more than 50% of the dry weight of the total ration. The total ration should contain less than 5 ppm of total fumonisins.

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السموم الفطرية فى الأغذية والأعلاف
4- الفيومونيزينس

بدير إبراهيم عجاج

قسم بحوث الكيمياء والنقص الغذائى والسموم – معهد بحوث صحة الحيوان بالدقى – مركز البحوث الزراعية

السموم الفطرية هى مركبات كيميائية تنتجها أنواع من الفطريات التى تنمو على المواد الغذائية والأعلاف الحيوانية وتسبب أضرارا صحية لكل من الإنسان والحيوان. وتعتبر سموم الفيومونيزينس والتى تم اكتشافها حديثا ذات أهمية كبرى نظرا لإمكانية تواجدها بكميات كبيرة فى الأغذية والأعلاف (خاصة المحتوية على منتجات الذرة) الملوثة بفطريات الفيوزاريوم المفرزة لها.

ففى الإنسان لاحظ العلماء وجود علاقة بين إزدياد معدل حدوث سرطان المرىء وتواجد سموم الفيومونيزينس أو الفطريات المفرزة لها فى منتجات الذرة المستخدمة فى الغذاء فى بعض دول العالم. وفى الحيوان لوحظ كثرة حدوث حالات الأعراض العصبية والنفوق فى الخيول ، وحالات الأوديما الرئوية والنفوق فى الخنازير فى العديد من دول العالم ومنها مصر. كما أثبت العلماء تجريبيا أن سموم الفيومونيزينس لها تأثيرات سمية أخرى فى العجول والأغنام والماعز والدجاج والبط والرومى والأسماك.