PJN
ISSN 1680-5194

PAKISTAN JOURNAL OF NUTRITION

ANSInet
308 Lasani Town, Sargodha Road, Faisalabad - Pakistan
Mob: +92 300 3008585, Fax: +92 41 8815544
E-mail: editorpjn@gmail.com

Pakistan Journal of Nutrition 5 (5): 398-403, 2006
ISSN 1680-5194

Nutritional and Health Implications of Mycotoxins in Animal Feeds: A Review

K.E. Akande, M.M. Abubakar, T.A. Adegbola and S.E. Bogoro
Animal Production Programme, Abubakar Tafawa Balewa University,
Bauchi, P.M.B. 0248, Bauchi State, Nigeria

Abstract: Mycotoxins are harmful substances produced by fungi in various foods and are estimated to affect as much as 25% of the world's crop each year. Most of these mytocoxins belong to the three genera of fungi: *Aspergillus*, *Penicillium* and *Fusarium*. Although over 300 mytocoxins are known, those of most concern based on their toxicity and occurrence, are aflatoxin, vomitoxin, ochratoxin, zearaleone, fumonisin and T-2 toxin. They are produced in cereal grains as well as forages before, during and after harvest in various environmental conditions. The presence of mycotoxins in feeds may decrease feed intake and affect animal performance. In addition, the possible presence of toxic residues in edible animal product such as milk, meat and eggs may have some detrimental effects on human health. Fungal contamination affects both the organoleptic characteristics and the alimentary value of feeds and entails a risk of toxicosis. The biological effects of mycotoxin depend on the ingested amounts, number of occurring toxins, duration of exposure to mycotoxin and animal sensitivity. Mycotoxins display a diversity of chemical structures, accounting for their different biological effects. Depending on their precise nature, these toxins may be carcinogenic, teratogenic, mutagenic, immunosuppressive, tremor genic, hemorrhagic, hepatotoxic, nephrotoxic and neurotoxic. Controlling mould growth and mycotoxin production is very important to the feed manufacturer and livestock producer. Control of mould growth in feeds can be accomplished by keeping moisture low, feed fresh, equipment clean and using mould inhibitors. In addition, control of mycotoxins in animal diets would reduce the likelihood that mycotoxin residues would appear in animal products destined for human consumption.

Key words: Nutritional, health, mycotoxins, moulds, animal feeds

Introduction

Mycotoxins are diverse range of molecules that are harmful to animals and humans. They are secondary metabolites secreted by moulds, mostly *Penicillium* and *Fusarium*. They are produced in cereal grains as well as forages before, during and after harvest, in various environmental conditions. Due to the diversity of their toxic effects and their synergetic properties, mycotoxins are considered as risky to the consumers of contaminated foods and feeds (Yiannikouris and Jonany, 2002).

Mycotoxins are metabolized in the liver and the kidneys and also by microorganisms in the digestive tract. Therefore, often the chemical structure and associated toxicity of mycotoxin residues excreted by animals or found in their tissues are different from the parent molecule (Ratcliff, 2002).

No region of the world escapes the problem of mycotoxins and according to Lawlor and Lynch (2005) mycotoxins are estimated to affect as much as 25 percent of the world's crops each year.

Whether grain is produced in temperate, sub-tropical or tropical climates, if rainfall and humidity are experienced in the harvest season, infection of the grain by fungi is likely. Fungi are extremely adaptable organisms being able to metabolize a large variety of substrates over a wide range of environmental conditions mycotoxins are produced only under aerobic condition (Ratcliff, 2002).

In farm animals, mycotoxins have negative effects on feed intake, animal performance, reproductive rate, growth efficiency, immunological defense as well as been carcinogenic, mutagenic, teratogenic, tremor genic-cause tremor or damage the central nervous system, haemorrhagic, as well as causing damage to the liver and kidneys. Ruminant animals are generally more tolerant of feed contaminated by mycotoxins than non-ruminant species due to the detoxifying capabilities of rumen micro organisms. Swine are generally the most sensitive with poultry intermediate (Ratcliff, 2002). Mycotoxins occur sporadically both seasonally and geographically. The formation of mycotoxins in nature is considered a global problem, however, in certain geographical areas of the world, some mycotoxins are produced more readily than others (Devegowda *et al*., 1998; Ratcliff, 2002; Lawlor and Lynch, 2005). Table 1, shows the mycotoxin that may be found in feeds that come from different global locations. They occur naturally in a wide variety of feedstuffs used in animal feeds. In most European countries aflatoxins are not considered to be a major problem. In contrast, vomitoxin, ochratoxin, zearalenone are found more frequently. Aflatoxins are common in humid climatic conditions like those existing in Asian and African countries and certain parts of Australia. Mycotoxins are regularly found in feed

Table 1: Geographic occurrence of mycotoxins

Location	Mycotoxins
Western Europe	Ochratoxin, vomitoxin, zearalenone.
Eastern Europe	Zearalenone, vomitoxin.
North America	Ochratoxin, vomitoxin, zearalenone, aflatoxins.
South America	Aflatoxins, fumonisins, ochratoxin, vomitoxin, T-2 toxin.
Africa	Aflatoxins, fumonisins, zearalenone.
Asia	Aflatoxins.
Australia	Aflatoxins, fumonisins.

Source: Devegowda *et al.* (1998)

ingredients such as maize, sorghum, barely, wheat, rice meal, cotton seed meal, groundnuts and other legumes. The problem of mycotoxins does not just end in animal feed or reduce animal performance, many become concentrated in meat, egg and milk of animal and can pose a threat to human health. Some examples of foods of animal origin which may be naturally contaminated with mycotoxins are shown in Table 2. There is increasing concern about levels of mycotoxin in human foods, both from vegetable origin and animal origin. In milk, aflatoxin appears as aflatoxin M_1, which is one of its metabolites.

Common mycotoxins in feedstuffs: The most commonly encountered mycotoxins in feedstuffs and foods are aflatoxins, zearalenone, ochratoxins, fumonisin, T2-toxin and deoxynivalenol (vomitoxin).

Aflatoxins: Aflatoxins are fluorescent compound, they are chemically classified as difurocoumarolactones and their biosynthesis by the producing fungi is via the polyketide pathway (Smith and Moss, 1985). Four major aflatoxins produced in feedstuffs and foods are aflatoxins B_1, B_2, G_1 and G_2. The most potent and the most frequently occurring of the four compounds is aflatoxin B_1. Aflatoxin is a metabolite of aflatoxin B_1 that occurs in various tissues and fluids from animals (Richard *et al*., 1993).

Two major species of *Aspergillus* are responsible for the production of aflatoxins in feedstuffs and foods, *Aspergillus flavus* and *Aspergillus parasiticus*. Infection and production of aflatoxins in field crops by these species is often associated with drought stress and insect damage (Richard *et al*., 1993).

While young animals are most susceptible to the effects of aflatoxin, all ages are affected; and clinical signs include gastrointestinal dysfunction, reduced productivity, decreased feed utilization and efficiency. Nursing animals may be affected by exposure to aflatoxin metabolites secreted in the milk.

Aflatoxin causes a variety of symptoms depending on the animal species. However, in all animals, aflatoxin can cause liver damage, decreased reproductive performance, reduced milk or egg production, embryonic death, teratogenicity (birth defects), tumors and suppressed immune system function, even when low levels are consumed (Jones *et al*., 1994). Aflatoxin contaminated feed is detrimental to the swine industry (CAST, 1989). Reduced feed intake, lowered gains and in some cases reduced feed efficiency has been observed for swine fed contaminated feed (Harvey *et al*., 1988). The physiological effects of aflatoxin consumption include liver damage characterized by enlargement, release of enzymes into the blood (for example, asparatase aminotransferase and alkaline phosphatase) and impaired protein synthesis (CAST, 1989). Aflatoxins M_1 appears in milk of sows consuming aflatoxin contaminated feeds and may affect piglets nursing those sows (Jones *et al*., 1994). Aflatoxin affects all poultry species. Although it generally takes relatively high levels to cause mortality, low levels can be detrimental if continually fed. Young poultry, especially ducks and turkeys are very susceptible. As a general rule, growing poultry should not receive more than 20ppb aflatoxin in their diet. However, feeding levels lower than 20ppb may still reduce their resistance to disease, decrease their ability to withstand stress and bruising and generally make them unthrifty (Jones *et al*., 1994).

Zearalenone: Zearalenone, a non–steroidal estrogenic mycotoxin and it major metabolic products (α-zearalenol and β-zearalenol). Zearalenone is insoluble in water and heat-stable and it persists in both animal feeds and human food prepared from contaminated grains (Shipchandler, 1975).

Fusarium graminearum (*Gibberella zeae* and *Fusarium culmorum* are the major zearalenone producing species and are distributed worldwide (Marasas *et al*., 1984). Zearalenone mimics the effect of the female hormone estrogen and at low doses, increases the size or early maturity of mammary glands and reproductive organs. At higher doses zearalenone interferes with conception, ovulation, implantation, fetal development and the viability of newborn animals. (Jones *et al*., 1994). Zearalenone causes estrogenic responses in dairy cattle, and large doses of this toxin are associated with abortions. Other responses of dairy animals to zearalenone may include reduced feed intake, decreased milk production, vaginitis, vaginal secretions,

Table 2: Some Food of Animal Origin which may be Naturally Contaminated with Mycotoxins

Mycotoxins	Potential Effects on Humans	Occurrence	Maximum Level Reported (ppb)
Aflatoxin B_1	Hepatic cancer	Eggs	0.4
Pig liver			0.5
Pig muscle			1.04
Pig kidney			1.02
Aflatoxin M_1	Carcinogenic	Cow milk	0.33
Ochratoxin A	Renal damage	Pig liver	98
		Kidney	89
		Sausages	3.4
Zearaelenone	Oestrogenic	Pig liver	10
		Pig muscle	10

Source: (FAO, 2002)

poor reproductive performance and mammary gland enlargement in heifers. It is recommended that zearalenone should not exceed 250 ppb in the total diet (Jones *et al.*, 1994).

Ocharatoxins: The ochratoxins are metabolites produced by certain species of the genera *Aspergillus* and *Penecillium* (Wood, 1992). Ochratoxin A, was discovered in 1965 by South African Scientists as a toxic secondary metabolite of *Aspergillus ochraceus* (Van der Merwe *et al.*, 1965). Other species of *Aspergillus ochraceus* group and several P*enicillium* species, including *Penicillium viridicatum*, have been shown to form ochratoxin A (Harwig *et al.*, 1974).

Ochratoxin A is the major metabolite of toxicological significance and it is mainly a contaminant of cereal grains (corn, barely, wheat and oats). It has also been found in beans (soyabeans, coffee, cocoa) and peanuts and meat in some countries (Krogh, 1987). Ochratoxin A is teratogenic in rat, hamster and chick embryo and is an inhibitor of hepatic mitochondrial transport systems. Ochratoxin A have also been reported to cause damage to the liver, gut, lymphoid tissue and renal tubular damage (Harwig *et al.*, 1974).

Fumonisins: The fumonisins are a group of compounds originally isolated from *Fusarium moniliforme* (Gelderblom *et al.*, 1988). Six different fumonisins (FA_1, FA_2, FB_1, FB_2, FB_3 and FB_4) have been reported, the A series are amides and the B series have a free amine (Gelderblom *et al.*, 1992).

In most animals fumonisin impairs immune function, causes liver and kidney damage, decreases weight gains, and increases mortality rates. It also causes respiratory difficulties in swine (Jones *et al.*, 1994).

The fumonosins (FB_1 and FB_2) were recently isolated from *Fusarium moniliforme* cultures and found to promote cancer in rats (Gelderblom *et al.*, 1988). These toxins occur naturally in corn and have been associated with equine leukoencephalomalacia (Ross *et al.*, 1990).

T-2 Toxin: The T-2 toxin, produced mainly by *Fusarium tricinctum*, was the first trichothecene to be found as a naturally occurring grain contaminant in the United States (Hsu *et al.,* 1972). It was associated with a lethal toxicosis in dairy cattle that had consumed moldy corn in Wisconsin. This mycotoxin rarely associated with disorders in animals or humans in other countries (Mirocha, 1984).

Yoshizawa *et al.*, 1981, stated that the chance of finding T-2 toxin as a residue in edible tissue is remote because it is rapidly metabolized *in vivo*.

In dairy cattle T-2 toxin has been associated with feed refusal, production losses, gastroenteristis, intestinal haemorrhages and death. T-2 has also been associated with reduced immune response in calves. In poultry, T-2 toxin has been implicated to cause mouth and intestinal lesion as well as impair the bird's immune response, causing egg production declines, decreased feed consumption, weight loss and altered feather patterns (Jones *et al.*, 1994).

Vomitoxin: Vomitoxin also called deoxynivalenol is stable, survives processing, milling and does occur in food products and feeds prepared from contaminated corn and wheat. The most common producer of vomitoxin is *Fusarium graminearum* (Marasas *et al.*, 1984). Vomitoxin is perhaps, the most commonly detected *Fusarium* mycotoxin. Vomitoxin has been associated with reduced milk production in dairy cattle, vomiting by swine consuming contaminated feed or their refusal to eat feed containing the toxin, and inhibiting reproductive performance and immune function in several animal species (Jones *et al.,* 1994).

Effects of mycotoxins on animal nutrition and health: Mycotoxins have significant economic and commercial impact, in that both the productivity and nutritive value of the infected cereal and forage is affected (Ratcliff, 2002). The effect of mould contamination on nutritional value of stored maize is presented in Table 3. The nutritive value drops after contamination by mould.

Contamination by moulds affects both the alimentary value and organoleptic characteristic of feed and entails a risk of toxicosis. The biological effects of mycotoxins

Table 3: Effect of mould contamination on the nutritional value of stored maize

	ME (Kcal/kg)	CP (%)	Fat (%)
Good corn	3,410	8.9	4.0
Mouldy corn	3,252	8.3	1.5
Loss in nutrient	158	0.6	7.5
% Loss in nutrient	4.6	6.7	62.5

Source: O'Keeffe (2003). ME = Metabolisable energy. CP = Crude protein

depend on the ingested amounts, number of occurring toxins, duration of exposure to mycotoxins and animal sensitivity. Also mycotoxins can induce health problems that are specific to each toxin as shown in Table 4, or affect the immune status of animals, favouring infections. This is the major reason for the difficulty of diagnosing mycotoxicoses (Yiannikouris and Jonany, 2002).

Mycotoxins produce a wide range of harmful effects in animals. The economic impact of reduced animal productivity, increased incidence of disease due to immunosuppression, damage to vital organs and interference with reproductive capacity is many times greater than the impact caused by death due to mycotoxin poisoning. Mycotoxins in combination appear to exert greater negative impact on the health and productivity of livestock in comparison to their individual effects (Smith and Seddon, 1998).

Control of Moulds and Mycotoixins in Animal Feeds:

Controlling mould growth and mycotoxin production is very important to the feed manufacturer and livestock producer. Control of mould growth in feeds can be accomplished by keeping moisture low, feed fresh, equipment clean and using mould binders and inhibitors. Grains and other dry feedstuffs should be stored at a moisture level of less than 14 percent to prevent mould growth. Aeration of grain bins is important to reduce moisture migration and keep the feedstuff dry (Jones *et al.*, 1994).

The use of chemical mould inhibitors is a well-established practice in the feed industry. However, mould inhibitors are only one of several tools useful in the complex process of controlling the growth of moulds, and they should not be relied upon exclusively. The main types of mould inhibitors are

1 Individual or combinations of organic acids (for example, propionic, sorbic, benzoic and acetic acids).
2 Salts of organic acids (for example, calcium propionate and potassium sorbate), and
3 Copper sulphate, solid or liquid forms work equally well if the inhibitor is evenly dispersed through the feed.

Generally, the acid form of a mould inhibitor is more active than its corresponding salt (Jones *et al.,* 1994).

Binding agents such as bentonite, aluminosilicates, spent canola oil, bleaching clays and alfalfa fibre have been used in feeds containing mycotoxins to prevent intestinal absorption of the toxins (Smith and Seddon, 1998). Mineral clay products such as bentonites, zeolites and aluminosilicates have been found to be effective in binding/adsorbing mycotoxins (Ramos *et al.,* 1996). Among these, aluminosilicates have been found to be more effective. The molecular surfaces of these additives, when saturated with water, attract the polar functional atomic structure of the mycotoxin and trap it against its surface. This isolates the mycotoxin from the digestive process and thereby prevents it from entering into circulation. Hydrated sodium calcium aluminosilicate (HSCAS) at 1.0% of the feed (10kg per tonne) can significantly diminish the adverse effects of aflatoxin in chickens, pigs and cows (Scheildler, 1993). However, these clays have a number of disadvantages, the high inclusion levels and narrow range of binding efficacy. They appear to have little or no beneficial effect against vomitoxin, T-2 toxin and Ochratoxin (Huff *et al.,* 1992). In addition, they reduce mineral utilization at a higher level of inclusion (Chestnut *et al.*, 1992).

Modified yeast cell wall mannanoligosaccharide (MOS) has been reported to effectively bind aflatoxin and to bind ochratoxins. This product has advantage over other binding agents in that it does not bind vitamins or minerals (Lawlor and Lynch, 2005).

Some foods may contain compounds that are inhibitory to fungal growth and mycotoxin production (Bullerman *et al.,* 1984). Certain herbs, spices and essential oils contain naturally occurring antifungal substances that may exert a protective effect at normal usage levels. Hitokoto *et al.* (1978) reported that mustard, green garlic, cinnamon bark and hops inhibit mould growth, whereas peppers, cloves, thyme and green tea inhibited toxin production only. Llewellyn *et al.,* (1981), also found that cinnamon, cloves and mustard had antimycotic properties and thyme and oregano were anti aflatoxigenic. Cinnamon, cinnamon oil, clove and clove oil have been demonstrated to have strong antimycotic properties (Bullerman, 1974; Bullerman *et al.,* 1977). All four substances inhibited growth and aflatoxin production. The essential oils of lemon and orange have been shown to be inhibitory to *Aspergillus niger* and *Aspergillus flavus* and to suppress aflatoxin formation (Subba, 1967; Alderman and Marth, 1976). Wellford *et al.,* 1978 reported that honey had an antifungal effect against *Aspergillus flavus* and *Aspergillus parasiticus* and an even stronger antiaflatoxigenic effect.

Other substances such as the antifungal antibiotic natamycin (pimaricin) has strong antimycotic properties but are only permitted for limited use in the United States. Azzouz and Bullerman (1982) reported that 0.0005% (5ppm) natamycin delayed the growth of seven mycotoxigenic species for 5 to 21 days.

At the farm level a number of management strategies

Table 4: Mycotoxins and their effects on different species of livestock

Mycotoxins	Species susceptibility	Effects
Aflatoxin	All domestic animals and poultry	Hepatoxic, carcinogenic, immunosuppressive
Zearalenone	Mainly pigs and dairy animals	Estrogenic and reproductive disorder
Vomitoxin	Mainly pigs and dairy animals	Dermatotoxic, feed refusal
Ochratoxin	Mainly pigs and poultry	Nephrotoxic, gout
T-2 toxin	Mainly pigs and poultry	Mouth lesions, loss of appetite
Fumonisins	Mainly pigs and horses	Neurological disorders, liver damage.

Source: Ratcliff (2002)

can be employed to minimize the development of mycotoxins in feeds. One of the key factors is the time feed is stored on the farm. More frequent delivery of feed with shorter residence time reduces the build up of mycotoxins (Good and Hamilton, 1981).

Various techniques on dietary manipulation have been reported to reduce the adverse effects of mycotoxins. Increasing the levels of selenium, methionine, carotenoids and vitamin supplementation have been found to be beneficial although not necessarily cost effective (Ratcliff, 2002). Jones *et al.,* 1994 also reported that increasing nutrient such as protein, energy (fats and carbohydrates) and vitamins in the diet may also be advisable. The addition of antioxidants to animal diets may assist in dealing with the effects of mycotoxins.

Conclusion: It is clear that mycotoxins will be of increasing importance for all those involved in feed manufacturing, farming and food production. Quality of raw materials, prevention of the occurrence of mycotoxins, control and testing systems are all essential to reducing the exposure of humans and animals to mycotoxins.

References

Alderman, G.G. and E.H. Marth, 1976. Inhibition of growth and aflatoxin production of *Aspergillus parasiticus* by Citrus Oils. Z. Lebensm-Unters-Forsch., 160: 355-358.

Azzouz, M.A. and L.B. Bullerman, 1982. Comparative antimycotic effects of selected herbs, spices, plant components and commercial antifungal agents. J. Food Protec., 45: 1298-1301.

Bullerman, L.B., 1974. Inhibition of aflatoxin production by cinnamon. J. Food Sci., 39: 1163-1165.

Bullerman, L.B., F.Y. Lieu and S.A. Seier, 1977. Inhibition of growth and aflatoxin production by cinnamon and clove oils, cinnamic aldehyde and eugenol. J. Food Sci., 42: 1107-1108.

Bullerman, L.B., L.L. Schroeder and K.Y. Park, 1984. Formation and control of mycotoxins in food. J. Food Protec., 47: 637-646.

CAST, 1989. Mycotoxins: Economic and Health Risks. Council for Agricultural Science and Technology Ammes, I. A.

Chestnut, A.B., P.D. Anderson, M.A. Cochran, H.A. Fribourg and K.D. Twinn, 1992. Effects of Hydrated Sodium Calcium Aluminosilicate (HSCAS) on Fesue Toxicosis and Mineral Absorption. J. Anim. Sci., 70: 2838 -2846.

Devegowda, G., M.V.L. Radu, A. Nazar and H.V.L.M. Swamy, 1998. Mycotoxin picture worldwide: Novel Solutions for their counteraction. In: Proceedings of Alltech's 14th Annual Symposium, Biotechnology in Feed industry. Passport of the year 2000. Nottingham University Press.

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

Gelderblom, W.C.A., W.F.O. Marasas, R. Vleggar, P.G. Thiel and M.E. Cawood, 1992. Fumonisins: Isolation, Chemical Characterization and biological effects. Mycopathologia, 117: 11.

Good, R.E. and P.B. Hamilton, 1981. Beneficial effect of reducing the feed residence time in a field problem of suspected mouldy feed. Poult. Sci., 60: 1403-1405.

Harvey, R.B., L.F. Kubena, W.E. Huff, D.E. Corrien and T.D. Phillips, 1988. Progression of aflatoxicosis in growing barrows. Am. J. Vet. Res., 49: 482.

Harwig, J., Y.K. Chen and D.L. Collins-Thompson, 1974. Stability of Ochratoxin A in beans during Canning. Can. Inst. Food Sci. J., 7: 288-289.

Hitokoto, H., S. Morozumi, T. Wauke, S. Sakai and I. Ueno, 1978. Inhibitory effects of Condiments and herbal drugs on the growth and toxin production of toxigenic fungi. Mycopathologia, 66: 161-168.

Hsu, I.H.C., E.B. Smalley, F.M. Strong and W.E. Ribelin, 1972. Identification of T-2 toxin in mouldy corn associated with a lethal toxicosis in dairy cattle. Appl. Microbiol., 24: 684.

Huff, W.E., R.D. Wyatt, T.L. Tucker and P.B. Hamillton, 1992. Efficacy of hydrated sodium calcium aluminosilicate to reduce the individual and combined toxicity of aflatoxin and ochratoxin. Poult. Sci., 71: 64-69.

Jones, F.T., M.B. Genter, W.M. Hagler, J.A. Hansen, B.A. Mowrey, M.H. Poore and L.W. Whitlow, 1994. Understanding and coping with effects of mycotoxins in livestock feed and forage. North Carolina Cooperative Extension Service, pp: 1-14.

Krogh, P., 1987. Ochratoxins in food. In: Mycotoxins in Food (ed. P. Krogh) Academic Press London, pp: 97.

Lawlor, P.G. and P.B. Lynch, 2005. Mycotoxin management. African Farming and Food Processing. 46:12-13 (January/February, issue).

Llewellyn, G.C., M.L. Burkett and T. Eadie, 1981. Potential mould growth, aflatoxin production and antimycotic activity of selected natural species and herbs. J. Assoc. Official Analytical Chem., 64: 955-960.

Marasas, W.F.O., P.E. Nelson and T.A. Toussoun, 1984. Toxigenic *Fusarium* species, Identity and mycotoxicology, pp.328. Pennsylvania State University Press, University Park.

Mirocha, C.J., 1984. Mycotoxicoses associated with *Fusarium*. In: M. O. Moss and J. E. Smith (Ed) The Applied Mycology of *Fusarium*. P 141. Cambridge University Press, U.K.

O'Keeffe, M., 2003. Mycotoxins in Foods and Feeds. In: Farm and Food – The Teagasc Research and Digest. Ashtown. http://ww.food assurance.teagac.

Ramos, A.J., Fink Gremmels and E. Hernandez, 1996. Prevention of toxic effects of mycotoxins by means of non-nutritive adsorbent compounds. J. Food Protec., 59: 631-641.

Ratcliff, J., 2002. The role of mycotoxins in Food and Feed Safety. Presented at AFMA (Animal Feed Manufacturers Association) on 16th August, 2002. http://www.facs.org.uk.

Richard, J.L., G.A. Bennett, P.F. Ross and P.E. Nelson, 1993. Analysis of Naturally occurring Mycotoxins in Feedstuffs and Food. J. Anim. Sci., 71: 2563-2574.

Ross, P.F., P.E. Nelson, J.L. Richard, G.D. Osweiler, L.G. Rice, R.D. Plattner and T.M. Wilson, 1990. Production of fumonisins by *Fusarium moniliforme* and *Fusarium proliferatum* isolates associated with equine leukoencephalomalacia and a pulmonary edema syndrome in swine. Appl. Environ. Microbiol., 56: 3225.

Scheildler, S.E., 1993. Effects of various types of Aluminosilicate and Aflatoxin B_1 on Aflatoxin toxicity, chick performance and mineral status. Poult. Sci., 72: 282-288.

Shipchandler, M.T., 1975. Chemistry of zearalenone and some of its derivatives. Heterocycles, 3: 471.

Smith, J.E. and M.O. Moss, 1985. Mycotoxins, formation, analysis and significance. John Wiley and Sons, New York.

Smith, T.K. and I.R. Seddon, 1998. Toxicological synergism between *Fusarium* mycotoxins in feeds. In: Biotechnology in the Feed Industry. (eds, Lyons, T. P. and Jacques, K. A., Nottingham University Press, Loughborough, U. K. 257-269.

Subba, M.S.,T.C. Soumithri and R.S. Rao, 1967. Antimicrobial action of citrus oils. J. Food Sci., 32: 225- 227.

Van der Merwe, K.J., P.S. Steyn and L. Fourie, 1965. Mycotoxins. Part II. The Constitution Ochratoxins A, B, and C, metabolites of *Aspergillus ochraceus*. J. Chem. Soc., 7083-7088.

Wellford, T.E., T.T. Eadie and G.C. Llewellyn, 1978. Evaluating the inhibitory action of honey on fungal growth, sporulatiion and aflatoxin production. *Z. Lebensm. Unters. Forsch*. 166: 280 – 283.

Wood, G. E. (1992). Mycotoxins in foods and feeds in the United States. J. Anim. Sci., 70: 3941-3949.

Yiannikouris, A. and J. Jonany, 2002. Mycotoxins in feeds and their fate in animals: a review. Anim. Res., 51: 81- 99.

Yoshizawa, T., C.J. Mirocha, J.C. Behrens and S.P. Swanson, 1981. Metabolic fate of T-2 toxin in a lactating cow. Food Cosmet. Toxicol., 19: 31.