Effects of Mycotoxins on Cereals Grain Feed and Fodder Quality

F Waliyar, SV Reddy and RP Thakur[1]

Abstract

The mycotoxigenic mold fungi Aspergillus, Penicillium *and* Fusarium *were shown to be associated with grain molds in sorghum and pearl millet. These fungi produce several mycotoxins, some of which can affect human and animal health. The most important mycotoxins that occur at biologically significant concentrations in cereals are aflatoxins, ochratoxin A, fumonisins, zearalenone, deoxynivalenol and T_2 toxin. Reports indicate that aflatoxin B_1 was associated with liver cancer, synergistic with hepatitis B virus and childhood cirrhosis in humans. Several outbreaks of aflatoxicosis in poultry and livestock were reported. The fumonisins were associated with oesophageal cancer and T_2 toxin with alimentary toxic aleukia in humans. Fusarial mycotoxins have been implicated as causative agents in various animal diseases such as leucoencephalomalacia, pulmonary edema, infertility, diarrhea, vomiting, reduced growth rate, drop in egg production and immunosuppression.*

Mycotoxin contamination of cereals can cause economic losses at levels of food and feed production including crop distribution, processing and animal production. Health risks associated with consumption of contaminated cereals were recognized and several countries have recommended permissible levels of mycotoxins. At ICRISAT, Patancheru, India, we have developed an enzyme-linked immunosorbent assay (ELISA)-based technology to detect aflatoxin B_1, aflatoxin M_1, ochratoxin A and fumonisins in food and feeds.

A large number of agricultural commodities are vulnerable to infestation by a group of fungi that produce toxic secondary metabolites called mycotoxins. Mycotoxins are a group of chemically diverse secondary metabolites that exhibit a wide array of biological effects. Some of the mycotoxins can be mutagenic, carcinogenic, embryo-toxic, nephro-toxic, teratogenic, oestrogenic or immunosuppressive. Among the various mycotoxins, aflatoxins, ochratoxins and fusarial toxins (fumonisins) assume significance due to their deleterious effects on human beings, poultry and livestock. The toxins are produced on cereal grains both in field and storage.

In sorghum (*Sorghum bicolor*) and pearl millet (*Pennisetum glaucum*), grain molds are important in relation to mycotoxin contamination. Grain mold fungi grow on or in seed (Williams and McDonald 1983). They affect sorghum

1. ICRISAT, Patancheru 502 324, Andhra Pradesh, India.

and pearl millet grown in warm and wet conditions between flowering and harvest (Williams and Rao 1981, Williams and McDonald 1983). They are much more widespread in sorghum than in pearl millet because of the nature of the growing environments of the two crops. The problem of grain mold is encountered throughout the humid tropical and subtropical regions. It is particularly serious in areas where improved, short- and medium-duration cultivars that mature before the end of the rains have been cultivated. Under these conditions, the fungi such as *Aspergillus* spp, *Penicillium* spp and *Fusarium* spp can infect these crops and produce mycotoxins (Bandyopadhyay et al. 1988, Stenhouse et al. 1997).

According to Charmley et al. (1994), 25% of the world's food crops are affected by mycotoxins each year. The American Phytopathological Society (APS) reports that the loss in USA is estimated to be US$100 million per year (Source: APS Net). The most important mycotoxins that can frequently occur at biologically significant concentrations in cereals are aflatoxins, ochratoxins, fumonisins, zearalenone, moniliformin and trichothecenes (deoxynivalenol, nivalenol and T_2 toxin). These compounds can occur naturally in cereals, either individually or as specific clusters of two or more depending on the fungal species (or strain) implicated. These mycotoxins are associated with causative agents in various human and animal diseases. Mycotoxin contamination of crops cause economic losses at all levels of food and feed chain, including crop and animal production. Under certain environmental conditions the contamination of various cereal grains with mold fungi and mycotoxin is unavoidable. Therefore, the prevention of mycotoxin contamination of grain is the main goal of food and agricultural industries throughout the world. This report reviews some general information on the occurrence of toxigenic fungi and type of mycotoxins they produce in sorghum and pearl millet and their affect on human, animal health and economic losses. Most of the information has been accessed from the book 'Mycotoxins in agriculture and food safety' (Sinha and Bhatnagar 1998).

Toxigenic fungi on sorghum and pearl millet

Fungi that belongs to more than 40 genera are associated with molded grain including *Fusarium* spp, *Aspergillus* spp and *Penicillium* spp. These fungi produce mycotoxins in cereal grains and oil seeds (Fig. 1). The literature on toxigenic abilities of *Fusarium* species contain significant number of confusions, caused by usage of several taxonomic systems, wrong identification of toxigenic isolates or incorrect identification of mycotoxins (Chelkowsky 1989).

A. flavus *that produce aflatoxin*

Infected groundnut pod at the time of harvest

Contaminated maize seed

Figure 1. Groundnut and maize seeds affected by fungi that produce mycotoxins.

Effect of mycotoxins on human and animal health

Aflatoxins

Toxicologically, the aflatoxins, particularly aflatoxin B_1 (AFB_1) should be regarded as a quadruple threat, ie, as a potent toxin, carcinogen, teratogen and mutagen. AFB_1 induces liver cancer in several animal species, and has also been linked to liver cancer in human beings (Wang et al. 1996). Statistical

correlations between contaminated food supplies and high frequencies of human hepatocellular carcinomas in Africa and Asia have implicated aflatoxins as risk factors in human liver cancer. All epidemiological studies of aflatoxins and liver cancer conducted in Africa and Asia involving populations subjected to hepatitis B virus (HBV) infection indicates possible synergistic effect of aflatoxins and HBV infection in the etiology of liver cancer (Van Rensberg 1986, Groopman and Kensler 1996, Montesano et al. 1997). Amla et al. (1974) presented circumstantial evidence to indicate that children exposed to aflatoxins through breast milk and dietary items may develop cirrhosis. They detected AFB_1 in 7% of urine samples from cirrhotic children.

No animal species are resistant to the acute toxic effects of aflatoxins. A wide variation in LD_{50} values has been obtained in animal species tested with single doses of aflatoxins. For most species, the LD_{50} value ranges from 0.5 to 10 mg kg^{-1} body weight. Animal species respond differently in their susceptibility to the chronic and acute toxicity of aflatoxins. Environmental factors, exposure level and duration of exposure besides age, health and nutritional status of diet can influence the toxicity.

Several outbreaks of aflatoxicosis in poultry have been reported from India. The disease symptoms included severe and sudden anorexia, loss of weight, staggering gait, convulsive movements, feed refusal and drop in egg production. Post-mortem examination of dead birds revealed liver lesions of varying severity (Char et al. 1982, Choudary 1986). An increase in blood clotting time increases the susceptibility of the carcass to bruising even at doses below that to have an effect on growth. In poultry, aflatoxins impair the availability of bile salts, which decreases vitamin D_3 production. This causes a decrease in the absorption of fat-soluble vitamins.

Several outbreaks of aflatoxicosis in cattle have been reported. The lesions were confined mainly to the liver, showing degenerative changes with biliary proliferation and finally leading to diffuse cirrhosis (Allcroft and Lewis 1963). Aflatoxin M_1 (AFM_1) is a major metabolite of aflatoxin B_1 found in milk of animals that have consumed feeds contaminated with aflatoxin B_1. The toxic and carcinogenic effects of AFM_1 have been convincingly demonstrated in laboratory animals and therefore AFM_1 is classified as class 2B human carcinogen. AFM_1 is relatively stable during pasteurization, storage and preparation of various dairy products. Therefore, AFM_1 contamination poses a significant threat to human health especially to children, who are major consumers of milk.

Ochratoxin A

Ochratoxin A has been implicated with a fatal human kidney disease referred to as 'Balkan Endemic Nephropathy' (BEN) characterized by contracted kidneys. Ochratoxin A has been found more frequently in food samples and in the serum taken from people in villages with BEN than in areas where the disease is unknown (Petkova-Boeharova et al. 1988). The toxin is nephrotoxic to most of the animal species and induces liver and kidney tumors. The renal lesions associated with diseases include degeneration of the proximal tubules and interstitial fibrosis in the renal cortex. Pigs fed with ochratoxin A, showed reduced feed intake, weight loss, increased water consumption, followed by polyurea diarrhea.

Fusarium toxins

Species of *Fusarium* are widespread in nature as saprobes in decaying vegetation and as parasites on all parts of plants. Many cause diseases of economically important plants. There are a number of species that produce mycotoxins, mostly fumonisins, zearalenone and trichothecenes (T_2 toxin, deoxynivalenol and nivalenol). A few common examples are discussed below.

Fumonisins

An outbreak of poisoning, characterized by abdominal pain and diarrhea, caused by the ingestion of fumonisin-contaminated maize (*Zea mays*) and sorghum in India was reported. People in 27 villages in Karnataka, India were affected and the disease was seen only in households which consumed sorghum or maize contaminated with fumonisin (Bhat et al. 1997). Consumption of fumonisin-contaminated maize has been linked to oesophageal cancer in humans in Transkei region of South Africa, China and other countries (Rheeder et al. 1992). Ingestion of fumonisin-contaminated maize has been associated with spontaneous outbreak of leucoencephalomalacia in horses, a neurological syndrome characterized by focal, often extensive, liquefactive necrosis of the white matter of the cerebrum, and white acute pulmonary edema in pigs. Although hepatic injury has been observed in all vertebrate species studied, a number of species-specific effects have been induced experimentally by the fumonisins on other target organs including renal injury and liver cancer in rats,

immunosuppression in chicken, toxicity to broiler chicken and chicken embryos, nephrotoxicity and brain hemorrhage in rabbits (Marasas 1995).

T_2 toxin

Of the *Fusarium* trichothecenes, T_2 is the most toxic though less widely distributed than deoxynivalenol. The effects of the first trichothecene toxin, T_2, documented was in the 1940s where it was associated with an outbreak of alimentary toxic aleukia (ATA). At its peak, in 1944, the population in the Orenbury district and other districts of the then USSR suffered enormous casualties; more than 10% of the population was affected and many fatalities occurred (Joffe 1986). The term 'alimentary toxic' refers to the fact that the toxin is consumed in food and 'aleukia' refers to the reduced number of leucocytes or white blood cells in the affected person. Other symptoms included bleeding from nose and throat, and multiple, subcutaneous hemorrhages. The infected food in this case was millet, which made up a great part of the diet of the people in the region, and at times, during World War II, it was not uncommon to allow the millet to be left standing in the fields over winter because bad weather in the fall prevented its harvest at the proper time. During late winter and early spring the millet would become infected with various fungi, including *F. tricinctum*, and when the people gathered and ate this fungus, many were affected with what was diagnosed as ATA. Thousands were affected and many died (Joffe 1986).

In pigs, the clinical signs of T_2 toxicosis include emesis, posterior paresis, lethargy and frequent defecation. At low levels of contamination in the diet T_2 toxin causes reduced feed intake and animal performance. At high concentrations (>2 mg kg^{-1}) in the diet it produces diarrhea, emesis and feed refusal. T_2 toxicosis in poultry causes oral lesions, reduced feed consumption, reduced growth rate and egg production in laying hens. In ruminants the T_2 toxicosis results in a wide range of responses, such as feed refusal, leukopenia, depression, diarrhea, coagulopathy, enteritis and posterior ataxia. Reduction of humoral immunity is a common effect for pigs, poultry and ruminants and when exposed to low concentrations of T_2 toxin in the diet showed increased susceptibility to other diseases.

Zearalenone

Zearalenone and related metabolites pose strong estrogenic activity and can result in severe reproductive and infertility problem when fed to domestic

animals in sufficient amounts. Pigs appear to be most sensitive; therefore, they are most frequently reported with problems caused by zearalenone, which include enlargement or swelling and reddening of the vulva in gilts and sows, swelling of mammary gland and atrophy, and prolapse of the ovaries, vagina and rectum. In young male pigs zearalenone can cause swelling of the prepuce, testicular atrophy and enlargement of the mammary glands, while in boars it causes reduced libido and marginal reduction in sperm quality.

Deoxynivalenol

Deoxynivalenol is also called vomitoxin and is the most important trichothecene because of its high incidence in cereals, but it is not one of the most acutely toxic of this group of mycotoxins (Rotter et al. 1996). At cellular level the main toxic effect is inhibition of protein synthesis via binding to ribosomes. In animals, the overt effect at low dietary concentrations (>2 mg g^{-1}) appears to be a reduction in food consumption and weight gain, while higher doses (>20 mg g^{-1}) induce feed refusal, diarrhea and vomiting. Deoxynivalenol is known to alter brain neurochemicals, and serotoninergic system appears to play a role in mediation of the feeding behavior and emetic response. Animals fed low doses of toxin are able to recover from initial weight loss, while higher doses induce more long-term changes in feeding behavior. Pigs are more sensitive than other livestock to the presence of deoxynivalenol in their feed. Most of the clinical signs caused by the ingestion of deoxynivalenol are also observed with nivalenol, the latter being generally more toxic.

Economic losses caused by mycotoxins in sorghum and pearl millet

Some factors that influence the degree of fungal infestation and mycotoxin contamination in cereals are the prevailing weather conditions and the susceptibility of the crop to fungal invasion and mycotoxin contamination (Visconti 1996). During the seasons of extensive mycotoxin contamination, grain shortages may occur leading to elevated prices for grain and costs for livestock and poultry producers and consumers of grain products. Mycotoxigenic fungal infestation may reduce crop yields, seed germination rates, seedling vigor and grain quality. Mycotoxin contaminated grains are downgraded from food to feed, and additional cleaning and milling procedures may be required to reduce contamination, and export markets are affected.

Among all the mycotoxins, aflatoxin B_1 is the most toxic, carcinogenic and immunosuppressive agent to human beings and livestock. Mycotoxin exposure in humans increases medical and welfare costs, and reduces income potential of the individual. Consumer problems are related to less nutritious food, increased health risks in years of severe mycotoxin contamination, higher product prices and long-term chronic effects from low contamination.

Most economic losses due to consumption of mycotoxin contaminated diet by farm animals result from reduced animal production and increased disease incidence. Livestock producers are affected by increased production cost due to higher mortality rates, reproductive failures (abortions), reduced feed efficiency and overall quality loss. Presence of mycotoxins in poultry feed causes adverse effects on laying hen and broiler chicks. Moreover, consumption of feed containing a combination of toxins has a greater adverse effect on poultry than when feeds containing a single toxin are fed. Pigs are very sensitive to *Fusarium* mycotoxins in their diet. Deoxynivalenol can cause reduced feed intake, vomiting and reduced body weight gain. Delay in the time taken for pigs to reach the ideal marketing body weight or marketing pigs below normal weight can have serious economic consequences for pig producers. Economic implications of animal feed contaminated with fumonisins are significant, especially if contamination results in death of livestock.

Mycotoxin contamination in cereal and legume byproducts is hampered by strict regulation of many countries. Groundnut export was significantly reduced both in Asia and Africa because of very low permissible levels of aflatoxin B_1. Thus the reduction in the level of mycotoxins in agricultural products for food and feed is of high importance. Table 1 shows the permissible level of some of the mycotoxins in cereals.

Detection technologies

Several physio-chemical methods are available for estimation of mycotoxins. These methods are expensive and time consuming. At the International Crops Research Institute for the Semi-Arid Tropics (ICRISAT), Patancheru, Andhra Pradesh, India, we have developed an ELISA (enzyme-linked immunosorbent assay)-based detection technique, which is cost effective and less time consuming. We have produced monoclonal and polyclonal antibodies for aflatoxin B_1 and determined the AFB_1 quantities in various foods and feed

Table 1. Worldwide recommended regulatory limits on *Fusarium* mycotoxins in cereals and cereal products.

Country	Mycotoxin	Commodity	Limit (ng g^{-1})
Austria	Deoxynivalenol	Wheat, rye/durum wheat	500–750
	Zearalenone	Wheat, rye, durum wheat	60
Brazil	Zearalenone	Maize	200
Canada	Deoxynivalenol	Uncleaned soft wheat (food)	2,000
	Deoxynivalenol	Diets for swine, young calves, lactating dairy animals, cattle, poultry	1,000 5,000
	All mycotoxins	Feedstuff for reproducing animals	0
France	Zearalenone	Cereals	200
Netherlands	All mycotoxins	Cereals (products)	0
Romania	Zearalenone	All foods	30
Russia	T_2 toxin	Cereals (wheat, hard and strong type), flour, wheat bran	100
	Zearalenone	Cereals (wheat, hard and strong type), flour, wheat bran	1,000
Switzerland	Fumonisins	Maize (products)	1,000
Uruguay	Zearalenone	Maize, barley	200
USA	Deoxynivalenol	Finished wheat products (food)	1,000
	Deoxynivalenol	Grains and grain byproducts for feed	5,000–10,000[1]
	Fumonisins	Maize (products) for feed	5,000–50,000[2]

1. 5,000 ng g^{-1}, feed for pigs (not exceeding 40% of the diet); 10,000 ng g^{-1}, feed for ruminating beef and feed lot cattle older than 4 months and for chicken (not exceeding 50% of the cattle or chicken total diet).
2. Feed for horses (5,000 ng g^{-1}), pigs (10,000 ng g^{-1}), and beef cattle and poultry (50,000 ng g^{-1}).

Source: FAO (1997).

materials. We have also produced polyclonal antibodies against aflatoxin M_1 and ochratoxin A, and developed technology to detect these toxins in milk, foods and feeds. We are in the process of refining the ELISA test for fumonisin detection.

Recently we produced polyclonal antibodies to fumonisin B_1. Like most of the mycotoxins, fumonisins are also low molecular weight compounds. Since the low molecular weight compounds do not stimulate the immune system in warm-blooded animals, it is essential to tag these compounds to a bigger protein molecule, such as bovine serum albumin (BSA). To produce polyclonal antibodies for fumonisin B_1, we prepared fumonisin-BSA conjugate (Feng-Yih Yu and Chu 1996). The rabbit was immunized 5 times (3 subcutaneous and 2 intra-muscular) each with 250 mg FB1-BSA conjugate and 8–9 bleedings were made for serum collection. High titered antibodies were obtained and an ELISA method was standardized.

Several animal feed samples were tested for fumonisin content using the antibodies produced. Different extraction methods were used. We faced some difficulties in extraction of fumonisin from crop residues. One hundred sorghum straw samples meant for cattle feed were collected from markets of Andhra Pradesh and analyzed for aflatoxins and fumonisin B_1. All the samples were free from aflatoxins; however, 45% of the samples contained >100 μg kg^{-1} fumonisin B_1 (range 100–1600 μg kg^{-1}). As we have recently developed the test for fumonisins, we can now test many more samples to understand better the importance of fumonisin contamination and its implication on human and livestock health.

Conclusions

Mycotoxins are distributed widely in cereal crops, to the extent ubiquity in certain crops grown in specific regions and seasons. In cereals, grain quality as well as straw quality are important in relation to animal feed purpose. Mycotoxin contamination in sorghum and pearl millet grain was reported in different parts of the world. However, literature availability on mycotoxins in cereal straw is scanty. Since cereal straw is used for animal feed very extensively, it is essential to monitor the cereal straw for mycotoxin contamination. Moreover, after crop harvest, cereal straw in storage gets exposed to high temperature and high humidity that are conducive for the growth of mold fungi and subsequent mycotoxin production. To some extent, the presence of small amounts of mycotoxins in cereals and related food products is unavoidable. This necessitates monitoring of the food and feed samples at regular intervals. Some of the technologies available at ICRISAT can help different groups of people for better monitoring and analysis of their products for mycotoxins.

References

Allcroft R and **Lewis G.** 1963. Groundnut toxicity in cattle: Experimental poisoining of calves and a report on clinical effects in older cattle. Veterinary Records 75:487–494.

Amla I, Murthy VS, Jayaraj AP and **Parpia HAB.** 1974. Aflatoxins and Indian childhood cirrhosis – a review. Journal of Tropical Pediatrics and Environmental Child Health 20:28–33.

Ansari AA and **Srivastava AK.** 1994. Susceptibility of pearl millet varieties to toxigenic *Aspergillus ochraceus* for ochratoxin production. Indian Journal of Agricultural Sciences 64:421–422.

Bandyopadhyay R, Mughogho LK and **Prasada Rao KE.** 1988. Sources of resistance to sorghum grain molds. Plant Disease 72:504–508.

Bhat RV, Shetty PH, Amruth RP and **Sudershan RV.** 1997. A food borne disease outbreak due to the consumption of moldy sorghum and maize containing fumonisin mycotoxin. Journal of Toxicology, Clinical Toxicology 35:249–255.

Chandran N, Mall OP and **Chauhan SK** 1993. Moniliformin production by fusaria infecting bajra. National Academy of Science Letters 16:285–286.

Char NL, Rao P, Khan I and **Sarma DR.** 1982. An outbreak of aflatoxicosis in poultry. Poultry Adviser 15:57–58.

Charmley LL, Rosenberg A and **Trenholm HL.** 1994. Factors responsible for economic losses due to *Fusarium* mycotoxin contamination of grain, foods and feedstuffs. Pages 471–493 *in* Mycotoxins in grains (Miller JD and Trenholm HL, eds.). St. Paul, Minnesota,USA: Eagan Press.

Chelkowsky J. (eds.) 1989. *Fusarium* – mycotoxins, taxonomy and pathogenicity. Amsterdam, The Netherlands: Elsevier. 63 pp.

Choudary C. 1986. An outbreak of fatty liver syndrome in commercial layer farms. Poultry Adviser 19:59–60.

FAO. 1997. Worldwide regulation for mycotoxins 1995. A compendium. FAO Food and Nutrition Paper No. 64. Rome, Italy: FAO. 46 pp.

Feng-Yih Yu and **Chu FS.** 1996. Production and characterization of antibodies against fumonisin B1. Journal of Food Protection 59:992–997.

Gamanya R and **Sibanda L.** 2001. Survey of *Fusarium moniliforme* (*F. verticillioides*) and production of fumonisin in cereal grains and oil seeds in Zimbabwe. International Journal of Food Microbiology 71:145–149.

Groopman JD and **Kensler W.** 1996. Temporal patterns of aflatoxin-albumin adducts in hepatitis B surface antigen-positive and antigen-negative residents of Daxin, Qidong

County, People's Republic of China. Cancer Epidemiology Biomarkers and Preventions 5:253–256.

Gupta Rajani and **Gupta R.** 1998. Incidence of zearalenone producing *Fusarium* species on sorghum grains. Indian Phytopathology 51:251–253.

Joffe AZ. 1986. Effects of fusariotoxins in humans. Pages 225–292 *in Fusarium* species: their biology and toxicology. New York, USA: Willey-Interscience Publication.

Marasas WFO. 1995. Fumonisins: their implications for human and animal health. Natural Toxins 3:193–198.

Misra NK and **Daradhiyar SK.** 1991. Mold flora and aflatoxin contamination of stored and cooked samples of pearl millet in the Paharia belt of Santhal Paragana, Bihar, India. Applied and Environmental Microbiology 57:1223–1226.

Montesano R, Hainaut P and **Wild CP.** 1997. Hepatocellular carcinoma: from gene to public health. Journal of National Cancer Institute 89:1844–1851.

Petkova-Boeharova T, Chernozemsky IN and **Castegnaro M.** 1988. Ochratoxin A in human blood in relation to Balkan endemic nephropathy and urinary system tumors in Bulgaria. Food Additives and Contaminants 5:299–301.

Ramakrishna Y, Bhat RV and **Vasanthi S.** 1990. Natural occurrence of mycotoxins in staple foods in India. Journal of Agricultural and Food Chemistry 38:1857–1859.

Rheeder JP, Marasas WFO, Theil PG, Sydenham EW, Shephard GS and **van Schalkwyk DJ.** 1992. *Fusarium moniliforme* and fumonisins in corn in relation to human oesophageal cancer in Transkei. Phytopathology 82:353–357.

Rotter B, Prelusky DB and **Pestaka JJ.** 1996. Toxicology of deoxynivalenol (vomitoxin). Journal of Toxicology and Environmental Health 48:1–34.

Sashidhar RB, Ramakrishna Y and **Bhat RV.** 1992. Moulds and mycotoxins in sorghum stored in traditional containers in India. Journal of Stored Products Research 28:257–260.

Shetty HS, Usha CM, Patkar KL and **Lacey J.** 1994. Mycotoxin contamination in developing seeds of rice sorghum and groundnut in Mysore. Seed Research 22:31–38.

Shetty PH and **Bhat RV** 1997. Natural occurrence of fumonisin B1 and its co-occurrence with aflatoxins B1 in Indian sorghum, maize and poultry feeds. Journal of Agricultural and Food Chemistry 45:2170–2173.

Silva JB da, Pozzi CR, Mallozzi MAB, Ortega EM and **Correa B.** 2000. Mycoflora and occurrence of aflatoxin B1 and fumonisin B1 during storage of Brazilian sorghum. Journal of Agricultural and Food Chemistry 48:4352–4356.

Sinha KK and **Bhatnagar D.** (eds.) 1998. Mycotoxins in agriculture and food safety. New York, USA: Marcel Dekker, Inc. 511 pp.

Stenhouse JW, Bandyopadhyay R, Singh SD and **Subramanian V.** 1997. Breeding for grain mold resistance in sorghum. Pages 326–336 *in* Proceedings of the International Conference on Genetic Improvement of Sorghum and Pearl Millet, 22–27 Sep 1996, Lubbock, Texas, USA. USA: INTSORMIL.

Thirumala-Devi K, Mayo MA, Gopal Reddy and **Reddy DVR.** 2002. Occurrence of aflatoxins and ochratoxin A in Indian poultry feeds. Journal of Food Protection 65:1338–1340.

Van Rensberg SJ. 1986. Role of mycotoxins in endemic liver cancer and oesophageal cancer. Pages 483–494 *in* Mycotoxins and phycotoxins (Steyn PS and Vleggar R, eds.). Amsterdam, The Netherlands: Elsevier.

Visconti A. 1996. Fumonisins maize genotypes grown in various geographical areas. Advances in Experimental Medicine and Biology 392:193–204.

Wang LY, Hatch M, Chen CJ, Levin B, You SL, Lu SN, Wu MH, Wu WP, Wang LW, Wang Q, Huang GY, Yang PM, Lee HS and **Santella RM.** 1996. Aflatoxins exposure and risk of hepatocellular carcinoma in Taiwan. International Journal of Cancer 67:620–625.

Williams RJ and **McDonald D.** 1983. Grain mold in the tropics: problems and importance. Annual Review of Phytopathology 21:153–178.

Williams RJ and **Rao KN.** 1981. A review of sorghum grain moulds. Tropical Pest Management 27:200–211.

Wilson JP, Casper HS and **Wilson DM.** 1995. Effect of delayed harvest on contamination of pearl millet grain with mycotoxin producing fungi and mycotoxins. Mycopathologia 132:27–30.