Ass. Univ. Bull. Environ. Res. Vol. 6 No. 2, October 2003

AUCES

PREVENTION AND CONTROL OF MYCOTOXINS IN FEEDS

B.I. Agag

Biochemistry and Nutritional Deficiency Diseases Department,
Animal Health Research Institute, Agricultural Research Center

REVIEW ARTICLE

ABSTRACT :

Mycotoxins are compounds produced by toxigenic strains of fungi in grains and feedstuffs. Mycotoxins can contaminate agricultural products before harvest, after harvest, during processing, transport and storage. The contamination levels are influenced by environmental factors and insect damage.

Most of the mycotoxins are of greatest potential risk to humans and animals. The toxicity of mycotoxins to animals range from acute death to chronic disease and interference with reproductive efficiency. Contamination of meat, milk and eggs can result from animal consumption of mycotoxin contaminated feed or feed-ingredient. These contaminants of mycotoxins and their metabolites are hazardous to humans.

Management practices to avoid mycotoxin accumulation in stored grains or feed are conducted by several authors. Optimization of environmental conditions, good sanitation practices, addition of antifungal agent of low toxicity to animals and good insect/rodent control are valuable. Moreover, several procedures for the decontamination/detoxification of mycotoxin containing feeds have been reported. These include physical separation, thermal inactivation, irradiation, microbial degradation and chemical treatment This article summerises the different methods used for prevention and control of mycotoxins in animal feeds.

METHODS OF PREVENTION AND CONTROL :

Prevention of the contamination of fungi and their mycotoxins in agricultural commodities or feed ingredients can be divided into the following three levels:

I-PRIMARY PREVENTION:

Several hundreds (probably thousands) of fungus species have the capacity to produce toxins. All these fungi have three critical environmental requirements (temperatures above freezing, moisture above 14% and oxygen). Limiting any one of these requirements will reduce or prevent the production of toxins (Gotlieb, 1999).

Primary prevention should be initially carried out before the fungal infection and mycotoxin production. Several practices have been recommended to keep the conditions unfavourable for any fungal growth (Suttajit, 2002). These include:

1-Control of the environmental and other factors that influence fungi invading:

A-Pre-harvest control :

Pre-harvest control is the first step in ensuring a safe product. Once the crop becomes infected under field conditions growth will continue during post- harvest stages and storage (Lupez-Garcia *et al*., 1999). The pre-harvest manage-ment is focused on controlling critical factors that have been shown to enhance mycotoxin production, such as:

Irrigation and soil condition: High moisture and high relative humidity are essential for spore germination and fungal proliferation. Therefore, adequate efforts should be made to avoid extreme conditions of either drought or excessive moisture (Lupez-Garcia *et al.,* 1999).

Weed control: A sound weed control is necessary for optimum yield and crop quality but also help in reducing level of inoculum (Janet and Canale, 1997).

Control of insect infestation: Control of insect infestation in the field may help to prevent proliferation and subsequent mycotoxin production (Suttajit, 2002).

Management of crop residues: when the crop is harvested, some residues remain in the field, these provide an environment that is conductive to the survival of fungal spores and the subsequent infection of the next crop. Proper management of crop residues would help to avoid this problem (Lupez-Garcia *et al.,* 1999).

B-Post-harvest control:

Post-harvest control and decontamination procedures represent an important tool in avoiding consumer exposure to mycotoxin.

Dry the harvest grain: Grain and oil seed crops should be harvested at their optimum maturity and dried as soon as possible to moisture content levels that not allow fungal growth (less than 14%) (Jones *et al*., 1994; CAST, 1989). Sun drying is probably the oldest and most common way of reducing the moisture content of any crop. It has been practiced while the crops are still in the field (Noomhorm and Cardoma, 2002). Other methods of grain drying include mechanical drying, in-bin drying, infrared, microwave or sonic and solar energy drying (Goyal, 2002).

Control the physical condition of grain: Damaged portions of crops should be removed during the harvesting process whether the damage is caused by physical or biological causes. Care should be taken to remove foreign materials and high moisture plant parts during harvesting (CAST, 1989).

C-Good management of feed bins (stores):

Good management of stores will minimize mold related problems. A good management programme should include the following points:

-Chick bins for leaks and poor ventilation (Vest, 1997). Bins Should have good ventilation to minimize heat buildup and sweating of side walls (Waldroup, 1997).

-Minimize feed storage time and do not leave feed in bins between flocks (Vest, 1997). Ground feed is an ideal source of food for fungal growth, therefore it should be utilized rapidly (Royes and Yanong, 2002).

-Clean storage bins regularly, particularly roof areas and upper inside walls (Vest, 1997),and remove old grains or feeds, damaged kernels

and any foreign matter from bottom of the bin (Boyles and Eastridge, 2002), to prevent bridging of the feedstuffs and creation of hotspots (Royes and Yanong, 2002).

-Bagged feed: Storage of finished feed or raw material in bags presents problems especially, in humid climates. Condensation and mold growth can occur easily in solid walled and lined bags as temperature change. Jute and plastic mesh bags are particularity troublesome if recycled and used more than once for feed. Furthermore, the open nature of these bags allows humidity and mold spores to penetrate to the feed within (Waldroup, 1997).

-Grains with elevated moisture should be aerated and/or dried with heat (Bolyles and Eastridge, 2002).

-Chick grains periodically for temperature, moisture and insect damage (Boyles and Eastridge, 2002).

Grain temperature, moisture and duration interact in regulating the growth of storage fungi. Table (1) lists safe storage duration for corn, depending on grain temperature and moisture. Note that from a starting point of 14% moisture and 50°F, every 10°F increase in temperature or 1.5% increase in storage moisture reduces the safe storage period by half (Raney *et al.,* 1987).

Table (1): Safe storage duration for corn held at specific moistures and temperatures.

Temperature (F)	Moisture percent				
	14	15.5	17	18.5	20
	Days of safe storage				
50	256	128	64	32	16
60	128	64	32	16	8
70	64	32	16	8	4
80	32	16	8	4	2
90	16	8	4	2	1
100	8	4	2	1	0

N.B. When considering silage, it is neither practical nor desirable to limit temperature or moisture. Limiting oxygen is the key to successfully limiting toxin production during ensilement. Oxygen is like a light switch. It can turn toxin production on and off during storage (Gotlieb, 1999).

-Store grains at controlled atmosphere by cooling and depleting the oxygen content of the storage atmosphere to the desired level where the microbes and insects can not grow, e.g. underground storage (Goyal, 2002).

-Silage: Discard mouldy areas when silo is first opened or when moldy pockets are found during feeding, and feed sufficient rate per day to minimize spoilage (Boyles and Eastridge, 2002).

d-Control infestation in stored bulk grains:

Insects damage grains by removing the germ, eating the endosperm and cotyledons, exposing external layers of the seed to the dust, depositing fecal pellets, dying and leaving their mortal remains in the grain, and creating favorable conditions for the development of fungi (Williams, 1991) and can also serve as carriers of fungal spores (Harris and Staples, 2002). Once fungal growth starts, the water of

metabolism from the fungus will provide sufficient water for further growth and mycotoxin development (Maracas and Nelson, 1987).

2-Development of fungal resistant varieties of growing plants:

It has been clear that the fungal-resistance of each variety is genotypic. For example the resistance to invasion of *A. flavus* has been attributed to several biochemical, environmental and physical factors. Some strains would require physical damage for their infestation and others would not. During long grain storage, the biochemical activity of grain is much reduced, while invasion of storage fungi and mycotoxin contamination would increase (Suttajit, 2002).

Genotypes of peanuts and biochemical properties of its seed such as tannin content, thin pericarp (Rao and Tupule, 1967), small amount of circular wax (La Prade *et al*., 1973), and chemical composition of the pericarps and embryos (Lindsey and Turner, 1975) have been shown to inhibit fungal invasion by *A. flavus* and aflatoxin formation.

Recently, antifungal enzymes, chitinase (Roberts and Selitrennikoff, 1986) and B-(1-3) D-glucanase (Nelson *et al*., 1969), found in a number of plant seeds, may acts as defense against pathogenic fungi, since chitin and glucan are major polymeric components of many fungal cell walls. Such polysaccharides in fungal cell wall could be enzymatically hydrolysed into smaller products resulting the damage or killing of fungal mycelia or spores. It is foreseen that seeds rich in such antifungal enzymes likely resist the infestation of fungi. Even there are many technical problems in searching for the "super" plant against pathogencitiy, the development of fungal-resistant plant verities utilizing genetic resistance to mycotoxin contamination is still possible and encouraged (Suttajit, 2002).

On the other hand, studies have identified the chromosome regions associated with aflatoxin resistance. This line of research is therefore a good option for future pre-harvest control and prevention of mycotoxin formation. Genetic engineering has also been useful in the development of host resistance through the addition or enhancement of antifungal genes. Many endogenous compounds with low molecular weight and bio-macromolecules in maize kernel tissues have been identified as antifungal compounds. Enhancing the production of the compounds may also enhance resistance to mycotoxin contamination (Lupez-Garcia *et al*., 1999).

3-Use antifungal agents (mold inhibitors):

Antifungal agents used to prevent fungal growth have no effect on the toxin already formed. The main types of mold inhibitors are:

-Organic acids e.g. acetic, propionic, butyric, benzoic, formic, lactic and sorbic acids (Sherwood and Puberdy, 1974; Suttajit, 2002).

-Salts of organic acids e.g. sodium or calcium proprionate and potassium sorbate. Generally the acid form as a mold inhibitor is more effective than its corresponding salt (Jones *et al*., 1994). The critical concentrations of all these acids, whether used singly or in combination was found to be between 0.1 and 1% W/W. Below 0.1% the fungus was able to develop (Sherwood and Peberdy, 1974). According to Herting and Drury (1974) propionic and formic acids are effective antifungal agents or corn, grain sorghum, wheat, oats, barley and soybeans. Propionic acid was found by Sauer and Burroughs (1974) to be effective as a mold inhibitor for corn and

grain sorghum at moisture contents of 18 to 24%, isobutyric, acetic and formic acids followed, in order.The particular value of propionic acid as a preservative stems from its low order of toxicity towards human and other animals. In fact, it is a normal component of the digestive tract of ruminants (Kiessling and Pettersson, 1991). A positive effect of this was observed by Jones *et al.* (1970) when they reported that propionic acid, besides being an effective preservative of high-moisture corn, also brought about an improvement in animal performance. Grains treated with propionic acid can be used only for livestock and poultry feeding (Jacobsen *et al*., 1993). Mold inhibitors (sodium or calcium propionate or organic acids) are added to stored grains to prevent further development of molds at levels of 0.2 to 0.25% to feeds with 14-17% moisture and 0.5 to 0.6% to feeds with 18-24% moisture (Tarr, 1996).

-Benzoic acid derivatives e.g. O-nitrobenzoate, O-aminobenzoate, P-aminobenzoate, benzo-caine (ethylaminobenzoate) ethyl benzoate, methyl benzoate and o-acetoxy benzoic acid (Davis and Diener, 1967).

-Potassium sulfite and potassium fluoride (Suttajit, 2002).

-Fumigants: Ammonia and phosphine (Vandergraft *et al.,* 1975).

-Gentian violet (0.5-1.5 g/kg) and thiabendazol (100 mg/kg) (Luxsanakoses, 2002).

-Copper sulphate: Copper sulphate is a poor mold inhibitor for poultry feeds. Moreover, excessive levels of copper are toxic to young animals and sheep and will accumulate in the environment (Jones *et al*., 1994).

There are many factors influencing the effectiveness of mold inhibitors (Jones *et al*., 1994), these include:

-Particle size: Mold inhibitors can not be effective unless they are completely and thoroughly distributed throughout the feed. The smaller the inhibitor particles, the greater the effectiveness.

-Feed ingredients: Protein or mineral supplements e.g. fish meal, poultry by- product meal, soybean meal and limestone tend to reduce the effectiveness of propionic acid. These materials can neutralize free acids and convert them to their corresponding salts, which are less active as inhibitors. Dietary fat tends to enhance the activity of organic acids probably by increasing their penetration into feed particles.

-Pelleting of feed: Pelleting feed has among its numerous benifits, destruction of fungal spores and a decrease in the fungal burden. Also, the heat produced during pelleting of feeds enhances the effectiveness of organic acids. Generally, the higher the pelleting temperature, the more effective the inhibitor.

-Organic acids are corrosive to metals and irritating to skin, but some have been modified to counteract this characteristic. Concrete or steel surfaces that will contact acid-treated feed should be covered with plastic or coated with acid resistant paint.

4-Animal management:

-Moldy or toxic feeds should be removed and replaced with unadulterated one.

-Acidic diets should be avoided as they intensify the effects of mycotoxin (Jones *et al*., 1994).

-Addition of nutrients such as proteins, amino acids (choline, methionine), energy (fats and carbohydrates), vitamins and trace elements to animal diet may also be advisable (Jones *et al*., 1994). Some of these have effective, while others appear to have little effect Crude protein: Because aflatoxin metabolites appear to have an effect on protein and amino acid

metabolism, researchers have examined the possibility of increasing dietary protein levels as a means of alleviating aflatoxicosis (Waldroup, 1997). Smith *et al.* (1971) demonstrated that increasing the crude protein levels alleviated the growth depressing effects of aflatoxin. For ochratoxin A, Gibson *et al.* (1989) observed that increasing dietary crude protein helped to alleviate but did not eliminate the adverse effects of ochratoxin A (OA) on body weight and feed conversion, but mortality rates did not appear to be affected. However, increasing protein levels is a costly approach to mycotoxins control (Waldroup, 1997).

Methionine: Increasing the dietary total sulfur amino acids to levels in excess of NRC protected chicks from the growth depressing effect of aflatoxin, possibly through an increasing rate of detoxification by glutathione, a sulfer amino acid metabolite (Veltmann *et al.*, 1981 & 1983).

Phenylalanine: In virtue studies have indicated that the toxicity of OA may be alleviated by phenylalanine supplementation (Creppy *et al.*, 1980; Klinkert *et al.*, 1981). Further studies by Bailey *et al.* (1980) suggested that supplemented phenylalanine improved the health status of birds fed diets containing ochratoxin A.

Lipids: Dietary high levels of oils (like olive, coconut, safflower) or animal fat, reduced mortality and in some instances improved body weight of chickens (Waldroup, 1997). The lipids extended their effects in part by interfering with absorption of the aflatoxins, i.e. the aflatoxin was not being absorbed from the high-fat diet (Smith *et al.*, 1971).

Vitamins: Adequate vitamin supplementation is particularly important when feeds contain mycotoxins, because fungi can destroy vitamins in feeds. Interactions of aflatoxins with riboflavin, vitamin D, vitamin A and thiamin have been reported (Diekman *et al.*, 1992). Recent work with fusarium toxicity (Waldroup, 1997) suggests that an antithiamin factor is a major toxic factor of *F. proliferatum*, and supplementation of diets with thiamin aided in ameliorating the toxicity of the mycotoxins in chick diets. The acute lethality of aflatoxin in rats was increased by a deficiency of vitamin A, while the carcinogenicity of aflatoxin was lessened by the deficiency (Rogers and Newberne, 1971). A diet deficient in vitamin D rendered chickens sensitive to growth inhibition by doses of aflatoxin too small to inhibit growth in a sufficient diet (Hamilton *et al.*, 1974). Riboflavin and pantothenol had a sparing effect against aflatoxin when incorporated into the diets of ducklings (Lynd and Lynd, 1971). Dietary supplementation with biotin, which is involved in certain fatty liver syndromes, reduced in chickens the accumulation of lipid and retarded any change in the fatty acid profile of liver fat caused by aflatoxin (Bryden *et al.*, 1979).

Antibiotics: Addition of 55 ppm of chlortetracycline to broiler diets helped to alleviate, but did not overcome the adverse effects of 10 ppm aflatoxin on body weight, feed conversion and mortality (Waldroup, 1997).

Antioxidants: Antioxidants aid in the overall detoxification process in the liver and in cells and thus may aid in alleviation of mycotoxicosis. Both vitamin E and selenium are involved in the formation of glutathione peroxidase, a compound vital in the cellular detoxification mechanism (Waldroup, 1997).

II- SECONDARY PREVENTION:

If the invasion of some fungi begins in commodities at early phase, several measures are suggested as follows:

-Stop growth of infested fungi by re-drying the product (Suttajit, 2002).

-Remove the contaminated seeds or grains. Removal of contaminated seeds, physical separation of infected grains is an efficient and feasible method of minimizing mycotoxins contamination. This is offered by either manual operation or with the help of an electronic sorter. Fungal infection of seeds or grains usually imparts characteristic colour or other physical properties (Goyal, 2002).

-Protect stored products from any conditions which favour continuing fungal growth (Suttajit, 2002).

-Decontamination or detoxification of mycotoxins contaminated products (Suttajit, 2002).

Inactivation of mycotoxins and decontamination or detoxification of commodities containing them can be classified as physical, chemical or microbiological:

I-Physical inactivation:

a-Thermal inactivation: Aflatoxins and most Fusarium mycotoxins are resistant to thermal inactivation for example, Fumonisinine B_1 requires a high temperature (150- 200°C) to achieve 87- 100% loss in corn (Chrevatidis *et al.,* 2003b).

Boiling and autoclaving: Aflatoxins are heat-stable and are not totally destroyed by boiling, autoclaving and a variety of food and feed processing procedures (Christensen *et al*., 1977).

Heating and cooking under pressure can destroy nearly 70% of aflatoxin in rice compared to atmospheric pressure only 50% destroyed. Since aflatoxin resists to higher temperature up to 260ºC, long-time cooking and overheating would destruct essential vitamins and amino acids in treated foods (Suttajit, 2002).

Roasting reduce the aflatoxin content of nuts, oil seed meals and corn (Marth and Doyle, 1979 and Conway *et al.,* 1978) and its content in foods from 40-60% (FDA, 1979). Roasting temperature more than 250°C are necessary for effective aflatoxin degradation; increasing the moisture content of the substrate will enhance degradation (Weidenborner, 2001). Dry roasting of naturally contaminated peanuts produced a 40 to 50% loss of AFB1 and AFG1 and a 20 to 40% of AFB2 and AFG2, whereas microwave roasting of contaminated peanuts completely destroyed aflatoxins (Luter *et al.*, 1982; Waltking, 1971). Roasting corn at 145 to 165°C reduced the aflatoxin B1 by 4080% (Conway *et al.*, 1978).

b-Storage of feed ingredients: Ochratoxin A can slowly breakdown merely during storage of grains and grain products as is documented by a decrease of more than 60% in naturally contaminated barley over a storage period of 2 years (Weidenborner, 2001).

c-Storage and physical treatments of milk may lead to some degradation of aflatoxin M1, as follows:-

Storage: It is observed that AFM_1 in milk decreases with time. Approximately 40% of M_1 was lost after 4 days and about 80% after 6 days of storage at 0°C of AFM_1 contaminated milk (Applebaum *et al*., 1982). Storage at 5°C for 1-3 days caused the amount of M_1 to decrease by 11-25% (Kiermeier and Mashaley, 1977).

Freezing at -18°C for 30 and 120 days caused degradation of 14% and from 43 to 68% of M_1, respectively (Mckinney *et al*., 1973). Repeated freezing at -20°C and defreezing is also efficient in liquid medium to reduce the OA production (Deberghes *et al*., 1993).

Pasteurization reduces AFM_1 content of milk: Bulk pasteurization at 62°C for 30 min.

caused a reduction of 32% of M_1. High temperature short time processes of 72 and 80°C for 45 seconds, reduced the amount of M_1 content by 45 and 64%, respectively (Purchase *et al*., 1972).

Sterilization of milk will cause a loss of some AFM_1. Processing of milk using steam pressure of 1 kg/cm^2 for 20 min. with preheating and cooling for an additional 10 min. each, reduced 80% of M_1 (Purchase *et al*., 1972).

Concentration of milk to half its volume at reduced pressure and 40°C lowered the content of M_1 64%. Drying with reduced pressure and 40°C resulted in 61% loss of the M_1 (Applebaum *et al*., 1982).

d-Sunlight: Sunlight degrades aflatoxin somewhat (FDA, 1979). Sunlight destroys 83% of the aflatoxin added to casein and 50% of that added to groundnut cake flour (Shantha and Murthy, 1981). Exposure of aflatoxin contaminated groundnut oil to sun light has given very promising results as it destroyed about 99% of aflatoxins (Goyal, 2002).

e-Gamma irradiation: Exposure of milk to gamma irradiation (UV) for 60 minutes, resulted in 100% degradation of aflatoxin. Gamma irradiation is also efficient to prevent the production of OA or destroy it. Two to 3 kg and 4 to 5 kg are need for solid and liquid medium, respectively (Deberghes *et al*., 1993).

f-Wet and dry milling: Wet and dry milling processes, which are widely used for maize and cereal grains, have been shown to result in reduced mycotoxin levels (zearalenone, fumonisins, aflatoxins, trichothecenes and ochratoxin A) in several fractions such as milling solubles, gluten, fiber, starch and germ (Lupez-Garcia and Park, 1998). Wet milling of maize, produces starch free, or almost free of zearalenone, fuminisins and aflatoxins, but T- 2 toxin is increased in maize germ (Riley and Norred, 1999). During dry milling of maize, high levels of zearalenone were found in the maize germ, degermer fines, bran meal, hull and high fat fractions and low levels (10-22%) occurred in the prime products (grits, low-fat meal and flour). Dry milling caused an accumulation of fumonisins in the bran, germ and fines fractions that are widely used in the production of animal feeds. Increasing refinement of maize meal caused lowered fumonisin levels by as much 95% in the fine maize meal compared to maize screenings. During experimental wet-milling of maize the maize bits (starch, fiber and gluten) contained most of the OA (51%) of all the maize farctions (Weidenborner, 2001).

g-Simple washing procedures, using water or sodium carbonate solution, result in some reduction in concentrations of deoxynivalenol. Zearalenone, zearalenol, and fumonisins in grains or corn cultures (Chrevatidis *et al.,* 2003b).

h-Adsorbents: Addition in the diet of nutritionally inert adsorbents such as hydrated sodium calcium aluminosilicate, activated carbon, bentonite, clays and special polymers reduces the absorption of mycotoxins from the gastrointestinal tract thus avoiding or/ reducing the toxic effects for livestock and their carry over of mycotoxins into animal products (Chrevatidis *et al.,* 2003d).

Hydrated sodium calcium aluminosilicates (HSCAS) are a phyllosilicate clays which are very effective with regard to preventing aflatoxicosis in a great variety of animals including chickens, turkey poults, goats, cows, pigs and lambs (Ramos *et al.,* 1996). HSCAS has been shown to adsorb AFB_1 with high affinity and high capacity in the aqueous solutions including milk, in contaminated oils, diminish

the effects of aflatoxins in young animals and decrease the level of AFM_1 in milk from lactating dairy cattle and goats (Lupez-Garcia *et al.*, 1999). The efficacy of HSCAS was quite limited against zearalenone and OA and totally ineffective for trichothecences such as T-2 toxin, diacetoxyscripenol and dexynivalenol (Chrevatidis *et al.,* 2003d).

Activated carbon (AC) has also been studied for its ability to bind aflatoxins in vivo and in vitro (Lupez-Garcia *et al.,* 1999). AC reduces dietary conversion of AB_1 to AM_1 in cows (Marquez *et al.,* 1993). AC is also quite effective in absorbing OA from aquesus solutions but has no beneficial effect when tested in vivo (Rotter *et al.,* 1989).

Sodium bentonite has been used as a binding and lubricating agent in the production of pelletted feeds. Dietary addition of bentonite or zeolite have shown to alter the effects of T-2 toxin and zearalenone (ZEA) in rats (Carson, 1982 and Smith, 1980).

Cholestyramine (CH), a resin used for pharmaceutical purposes in decreasing total and LDL cholesterol, adsorbed almost 100% of ZEA from gastric and intestinal fluids.

In particular 1 gm of CH was able to adsorb up 2mg of ZEA (Ramos *et al.,* 1996). In studies on rats CH was tested as a protective agent against OA and was found to be able to decrease the concentration of OA in plasma, the excretion of OA and its metabolites in urine and bile and to increase OA excretion in feces (Kerkadi *et al.,* 1998).

II-Biological detoxification:

Microbial strains including yeast, molds and bacteria, have been seened for their ability to modify and/or inactivate aflatoxins, deoxynivalenol, zearalenone, T-2 toxin and other trichothecenes, but their practical use has not been shown (Chrevatidis, 2003b; Ciegler *et al.*, 1966). *Flavobacterium aurantiacum* was reported to significantly remove aflatoxin from a liquid medium without producing toxic by-products (Ciegler *et al.*, 1966). It removes AFB1 form milk, maize, peanuts and soybeans, while AFG1 and AFM1 are also metabolized (Weidenborner, 2001). Black yeast fungus and a bacterium strain have been found to hydrolyze fumonisin B1 to aminopentol and tricarboxylic acid. The same microorganisms seem to be able to further detoxify aminopentol with release of CO_2 (Chrevatidis, 2003 b).

Lactic acid bavteria and yeasts expressing mycotoxin degrading enzymes may offer a natural way of providing these activities in fermentation process (Chrevatidis, 2003b). Fermentation with yeasts has also effective in destroying patulin and rubratoxin B (Lopez-Garcia and Park. 1998). Probiotic mixtures of Lactobacillus and Propionibacterium may reduce bioavailability of dietary AF (Ahokas *et al.*, 1998). Fermentation of contaminated grains, resulted in degradation of aflatoxins (Dam and Satterlee, 1977).

Some mycotoxins are detoxified during ensiling and other fermentation processes (aflatoxins, mycophenolic acid and patulin) while others are reable to degrade or transform their own products under suitable conditions (Karlovsky, 1999). Ensiling contaminated high-moisture corn did not adequately degrade aflatoxins. It was postulated that insufficient acid was produced by this procedure to catalyze the transformation of aflatoxin B_1 to aflatoxin B_{2a}. (Lindenfelser and Ciegler, 1970).

III-Chemical detoxification:

Aflatoxins are crystalline substances, freely soluble in polar solvents such as chloroform, methanol and dimethyl sulfoxide and dissolve in

water to the extent of 10-20 mg/L. Crystallin aflatoxins are extremely stable in the absence of light and practically UV radiation, even at temperatures in excess of 100ºC. A solutions prepared in chloroform, or benzene is stable for years if kept cold and in the dark. The lactone ring makes them susceptible to alkaline hydrolysis, and processes involving ammonia or hypochlorite have been investigated as means for their removal from food commodities, although questions concerning the toxicity of the breakdown products have restricted the use of this means of eradicating AFs from food and animal feeds. If alkaline treatment is mild, acidification will reverse the reaction to reform the original aflatoxin. In acid, AFB_1 and G_1 are converted to AFB_{2a} and G_{2a} by acid catalytic addition of water across the double bond of the furan ring. Oxidizing reagents react with and the molecules lose their fluorescence properties (Chrevatidis *et al*., 2003a).

Many chemicals have been tested for their ability to degrade or inactivate aflatoxins and some mycotoxins including acids, bases, aldehydes, bisulfite, oxidizing agents and various gases.

A-Bases: Many bases are tested for decontamination of mycotoxins such as ammonia, calcium or sodium hydroxide, calcium hydroxide plus monomethylamine and sodium bicarbonate.

Ammoniation: Either anhydrous ammonia, NH_3 (gas) or ammonium hydroxide, NH_4OH (aqua-ammonia=liquid), can be used at levels of 0.5 to 1.5 percent of the dry weight of feed like corn. The lower rate (0.5 percent) would be appreciated when the corn is 16 percent moisture or more, the temperature is 80 degrees F or more and the aflatoxin levels are less than approximately 200 ppb. The 1.5 percent rate should be used for high levels of aflatoxin and/or when the temperature and moisture conditions are less lowered (PER, 2003; USDA, 1998). The method consisted of injection or spraying ammonia onto contaminated feed, storing in air-tight containers e.g. plastic bags for about 3 weeks. The feed is heated after storing in oven at 50ºC for 24 hours to eliminate the residual ammonia and finally aerated to remove some of the ammonia odor and makes the feed more acceptable for animals (Marquez *et al*., 1993).

Ammoniation yielded from 79-90 percent reduction in aflatoxin B_1 in contaminated feed. Ammonia reacts with the aflatoxin molecule by breaking an oxygen bond making the resulting products far less toxic (PER, 2003). Ammonia causes lactone ring opening of AFB1, ultimate splitting off of the cyclopentone part. Several breakdown products of AFB1 have been identified e.g. AFD and the 206 molecular weight compound. Both substances showed a 450-fold decrease in mutagenicity compared to AFB1 (Weidenborner, 2001). Ammoniation, a procedure used for decontamination of aflatoxins, yielded a 79% reduction in fumonisin B_1 levels in naturally contaminated corn. Ammoniation of ochratoxin A-containing grain is effective in eliminating its toxicity (Marquardt and Frohlich, 1992).

Since ammonia vapor is only 60 percent as heavy as air, it should be applied under the corn. Corn with moisture content below approximately 15 percent, will need rewetting so that the ammonia will be absorbed. Up to 3 percent moisture can be added to the surface of the corn by spraying with water. Let the corn absorb the water for 6-8 hours before treating with ammonia. The warmer the corn, the faster the ammonia reaction. Corn with a moisture content of at least 15 percent and at a temperature of 90 degrees F can be treated in approximately 7 days, 75 degrees F corn

requires 14 days. Corn with a lower moisture content and/or temperature will require a treatment time of 21 days. When ammonia vapor is absorbed by the corn the temperature will increase 10 to 20 degrees (USDA, 1998).

Ammonium hydroxide (NH_4OH) is usually about 29 percent ammonia (by weight) and can be sprayed directly on corn. The application of ammonium hydroxide will also add about 2 percent moisture to the corn for every percent of actual ammonia used. This may eliminate the need for rewetting dry corn (PER, 2003).

Precautions for handling and application of ammonia :

Procedures involving anhydrous ammonia are less costly, but may by hazardous because of toxic fumes and the danger of explosions (Duncan and Hagler, 1986).

Ammonia in concentration greater than 1 percent is corrosive to metal surfaces. Ammonia also discolors and darkens treated corn kernels (as the sugars are carmelized) and increases its temperature about 10-20 degrees at the time of treatment. Applicators should use the following precautions when using ammonia (USDA, 1998; PER, 2003):

-When handling corn with high levels of aflatoxin, always wear a dust mask. The contamination dust is hazardous.

-Applicators should wear protective goggles, neoprene gloves and long sleeves (ammonia is extremely caustatic and cause skin burns and respiratory and eye injuries).

-A large supply of water should be close in hand for flushing of eyes or skin if an accident occurs.

- Workers should not enter bins in which it is being applied to avoid breathing ammonia. Prolonged inhalation can cause suffocation and death.

-Do not allow any spark of flame in areas where ammonia is being used.

-Detoxified corn must be thoroughly aerated in order to prevent feed refusal or reduced feed intake due to residual ammonia (Duncan and Hagler, 1986).

Calcium hydroxide [$Ca(OH)_2$]: Detoxification of AFB_1 with calcium hydroxide [$Ca(OH)_2$] or calcium hydroxide and monomethylamine (CH_3-NH_2), simultaneously resulted in 94-100% destruction of AFB_2. The mechanism of destruction is the opening of the lactone ring and decarboxylation. The detoxification process consisted of adding Ca $(OH)_2$ to the dry diet at a concentration of 2% (W/W), mixing for 5 minutes and adding monomethylamine at a concentration of 0.5% and water to adjust moisture content to 24%. Mixing in a closed environment (in a mixer equipped with a steam jacket) for 1 hour at 100°C and finally dry in a convection oven at 40°C to remove volatile compounds (Park *et al.*, 1983).

Treatment of FB1-contaminated corn (100 mg/kg) simulating modified nixtamalization (heat treatment with $NaHCO_3$ + H_2O_2 alone or with $Ca(OH)_2$ gave 100% reduction of FB1, whereas the traditional nixatamalization (treatment with $Ca(OH)_2$ only) was not effective because it produces hydrolyzed FB1 which is still toxic (Park *et al.*, 1996).

Calcium hydroxide monomethylamine: Calcium hydroxide monomethylamine has been used to decontaminate feeds containing T-2 toxin and diacetoxyscripenol at 10 to 20 mg/kg, the success of the producer is dependent on the moisture content of the feed and the processing temperature. In particular about 50% of mycotoxin reduction was observed when the treatment was performed at about 25°C and 10% moisture in 4 hours; when the moisture content was increased to 25%, T-2 toxin level

was reduced by 95 to 99% (Chrevatidis *et al*., 2003 c).

Sodium hydroxide (NaOH) and sodium bicarbonate ($NaHCO_3$): The mixing of diet with NaOH or $NaHCO_3$ at a role of 100 ml of 5N base per Kg diet and heating the mixture in an oven for 1 hour at100°C, then storing at room temperature for 7 days, causes 67 and 71% destruction of AFB_1 (Mashaly *et al*., 1983).

Sodium bisulfite: Sodium bisulfite treatment of grain results in the greatest reduction in deoxynivalenol (Young *et al*., 1986). Sodium bisulfite (a common food additive) has been shown to react with aflatoxin (B_1, G_1, M_1 and aflatoxicol) to form sulfonate derivatives (Chervatidis *et al*., 2003C). Sodium bisulfite solutions are capable to reduce DON level (85%) in contaminated corn (4.4 mg/kg DON) and form a DON-sulfonate conjugate when the treatment was performed at 80°C for 18 hours (Young *et al*., 1987).

B-Acids: Acids effectively convert AFB1 and AFG1 to their corresponding hemiacetal forms AFB2 and AFG2 (Weidenborner, 2001). Acetic (C_2H_5OH), citric, lactic and phosphoric acid (H_3PO_4) are recorded as a mycotoxin detoxificating agents.

Acids are used at a concentration of 100ml of 5N acid per Kg diet. The detoxification process consisted of adding acid to the diet, mixing and heating in an oven for 1 hour at 100°C. After storing at room temperature for 1 week the process causes reduction in aflatoxin B_1 in diet 88, 57, 80 and 68% for acetic, citric, lactic and phosphoric acid, respectively (Mashaly *et al*., 1983). Formic, propionic and scorbic acids are found to degrade OA at concentrations from 0.25% to 1% after exposure of 3-4 hours (Chrevatidis *et al*., 2003 c).

c-Oxidizing agents: Oxidizing agents such as sodium hyprochlorite (NaOCl), hydrogen peroxide (H_2O_2) and (HCHO) are tested for detoxification of mycotoxins. Treatment of diet with NaOCl, H_2O_2 or HCHO at level of 100ml (0.02%) of antioxidant per Kg diet and storing at room temperature for 7 days produced 82, 78 and 62% destruction of AFB_1, respectively (Mashaly *et al*., 1983).

Incubation of contaminated feed with 0.05% H_2O_2 for 30 minutes at room temperature causes 100% destruction of citrinin (Fouler *et al*., 1994). The toxicity of ochratoxin A in contaminated diet can be reduced by heating of diet with H_2O_2 at 100°C in an alkaline condition.

D-Gasses: It is recorded that the treatment of contaminated corn with ozone gas provided almost complete protection against the toxicity of aflatoxins in young turkey poults. Ozonation degrades and detoxifies aflatoxins in naturally contaminated maize promising (Riley and Norred, 1999). Dwarakanath *et al.* (1968) reported reduction of aflatxoin levels in cotton seed and peanut meal by ozone treatment under various conditions of moisture, temperature and time.

III-TERTIARY PREVENTION:

Once the products are heavily infested by toxic fungi, the primary and secondary prevention would not be then feasible. Any action would not be effective as the practices mentioned above, and reduce toxin formation (Suttajit, 2002). Only a few practices are recommended:-

-Complete destruction of the contaminated products.

-Detoxification or destruction of mycotoxins to the minimal level.

REFERENCES :

1-Ahokas, J.; El- Nezami, H.; Kankaanp, P.; Mykknen, H. and Salminen, S. (1998):"A pilot clinical examining the ability of a mixture of lactobacillus and propion bacterium to remove aflatoxins from the gastrointestinal tract of healthy" Egypt.. Volunteers. Revue Med. Vet., 149: 568 (Abstr.).

2-Applebaum, R.; Brackett, R.; Wiseman, D. and Marth, E. (1982): "Aflatoxin: toxicity to dairy cattle and occurrence in milk and milk products. "A Review. J. of Food Protect., 45 (8): 752- 777.

3-Bailey, C.A.; Gibson, R.M.; Kubena, L.F.; Huff, W.E. and Harvey, R.B. (1980):"Impact of L- phenylalanine supplementation on the performance of three- week- old broilers feed diet containing ochratoxin A. 2. Effects on hematology and clinical chemistry." Poultry Sci., 69: 420- 445.

4-Boyles, S. and Eastride, M. (2002): "What do I do if mycotoxins are present." Dept. of Anim. Sci., Ohio State University. Paracelsus (1493- 1541).

5-Bryden, W.L.; Cumming, R.B. and Balnave, D. (1979):"Influence of vitamin A status on the response of chickens to aflatoxin B_1 and changes in liver lipid metabolism associated with aflatoxicosis." Br. J. Nutr., 41: 529.

6-Carson, M.S. (1982):"The effect of dietary fiber and nonnutritive mineral additives on T-2 toxin in rats" MSc. Thesis Dept. Nutr. Univ. of Guelph, Guelph, Ontaria, Canada.

7-CAST (Council for Agric. Sci. and Technol) (1989):"Mycotoxins economic and health risks" Task force report No. 116, Nov. 1989.

8-Chrevatidis, A.; Eggington, A.; Piacentini, M.; Wilson, P. and Patal. P. (2003a):"Fact sheet 2 on the aflatoxins". EMAN, Leatherhead food RA, Leatherhead, Surrey KT22 7RY, UK.

9-Chrevatidis, A.; Eggington, A.; Piacentini, M.; Wilson, P. and Patal. P. (2003b):"Fact sheet on evaluation and risk analysis. 1. Decontamination, processing effect and risk analysis aspect" EMAN, Leatherhead food RA, Leatherhead, Surrey KT22 7RY, UK.

10-Chrevatidis, A.; Eggington, A.; Piacentini, M.; Wilson, P. and Patal. P. (2003c):"Fact sheet on evaluation and risk analysis. 2.Chemical treatments for mycotoxin decontamination of raw materials." EMAN, Leatherhead food RA, Leatherhead, Surrey KT22 7RY, UK.

11-Chrevatidis, A.; Eggington, A.; Piacentini, M.; Wilson, P. and Patal. P. (2003d):"Fact sheet on evaluation and risk analysis. 3. Adsorbent compounds as feed additives to reduce mycotoxin bioavailability." EMAN, Leatherhead food RA, Leatherhead, Surrey KT22 7RY, UK.

12-Chritensen, C.H.; Mirocha, C.T. and Meronuck, R.A. (1977):"Mold mycotoxins and mycotoxicosis" Agric. Experiment station Miscellaneous Report 142. Univ. of Minnesota, St. Paul, Minnesote.

13-Ciegler, A.; Lillehoj, E.; Peterson, R. and Hall, H. (1966): "Microbial detoxification of aflatoxin." Appl. Microbiol., 14: 939- 939.

14-Conway, H.F.; Anderson, R.A. and Bagley, E.B. (1978): "Detoxification of aflatoxin – contaminated corn by roasting." Cereal Chem., 55: 115- 117.

15-Creppy, E.E.; Schlegel, M.; Roschenthaler, R. and Kirheimee, G. (1980):"Phenylalanine prevents acute poisoning by ochratoxia A in mice" Toxicol. Lett., 60: 77-80.

16-Dam, R.; Tam, S.W. and Satterlee, L.D. (1977): "Destruction of aflatoxine during fermentation and by product isolation from artificially contaminated grain." Cereal. Chem., 54: 705- 714.

17-Davis, N.D. and Diener, U.L. (1967): "Inhibition aflatoxin synthesis by P-aminobenzoic acid, potassium sulfite and potassium fluoride "Appl: Microbiol., 15: 1517.

18-Deberghes, P.; Deffieus, G.; Gharbi, A.; Betbeder, A.M.; Boisard, F.; Blanc, R.; Delaby, J.F. and Creppy, E.E. (1993): "Detoxification de l'ochratoxine A par des moyens physiques, chimiques et enzymatiques. Proceedings of the international symposium: Human ochratoxicosis and associated pathologies in Africa and developing countries, held in Brodeaux (France) on July 4- 6, 1993" Pp. 75- 82.

19-Diekman, M.A.; Coffey, M.T.; Parkhiser, E.D.; Reeves, D.E. and Youg, L.G. (1992): "Mycotoxins and swine performance." Purdue University, Cooperative extension service, west Lafayette, Indiana.

20-Duncan, H.E. and Hagler, W.M. (1986): "Aflatoxicas and other mycotoxins" NCH- 52, PEST management, purdue univ., Cooperative Extension Service West Labayatte, IN47907.

21-Dwarakanath, C.T.; Bayner, F.T.; Mann, G.E. and Dollear, F.C. (1968): J. Am. Oil chem.. Soc., 45: 93- 95. Cited by Mashaly *et al.,* (1983).

22-FDA, Food and Drug Administration (1979): "Conference on mycotoxins in animal feeds and grains related to animal health." June 1979. FDA, Rockville, Maryland.

23-Fouler, S.G.; Trived, A.B. and Kitabatake, N. (1994): "Detoxification of citrinin and ochratoxin A by hydrogen peroxide" JAOAC Int., 77 (3): 631- 637.]

24-Gibson, R.M.; Bailey, C.A.; Kubena, L.F.; Huff, W.E. and Harvey, R.B. (1989): "Ochratoxin A and dietary protein. 1- Effect on body weight, feed conversion, relative organ weight and mortality in three week- old broilers." Poultry Sci., 68: 1658- 1663.

25-Gotlieb, A. (1999): "Mycotoxins in silage: A silent loss in profits." Plant and Soil Science Dept., Univ. of Vermint. Alan. Gotlieb @ uvm. Edu.

26-Goyal, R.K. (2002):"Prevention and control of mycotoxins in foodgrains in India" In mycotoxin prevention and control in food grains. A collaborative publication of the UNDP/ FAO Regional Network Inter- country cooperation on preharvest technology and quality control of foodgrains and ASEAN grain postharvest prgrame, Bankok, Thailand.

27-Hamilton, P.B.; Tung, H.T.; Wyatt, R.D. and Donaldson, W.E. (1974):"Interaction of dietary aflatoxin with some vitamin deficiencies" Poult. Sci., 53: 871.

28-Harris, B.Jr. and Staples, C.R. (2002):"The problems of mycotoxins in dairy cattle rations" Institute of Food and Agricultural Sciences UF/ IFAS, University of Florida.

29-Herting, D.C. and Drury, E.E. (1974): "Antifungal activity of volatile fatty acids on grains." Cereal Chem., 51: 74.

30-Jacobsen, B.J.; Bowen, K.J.; Shelby, R.A.; Diener, U.L.; Kemppainen, B.W. and Floyed, J. (1993): "Mycotoxins and mycotoxicosis" Auburn University, Auburn, Alabama. Circular ANR- 767.

31-Janet, Fallon and Canale, Ch. (1997):"Crop production and feeding, strategies can reduce mycotoxin problems." Copyright © 1997, Agway Inc.

32-Jones, F.; Genter, M.; Hagler, W.; Hansen, J.; Mowrey, B.; Poore, M. and Whitlow, L. (1994): "Understanding and coping with effects of mycotoxins in livestock feed and forage" Electronic publication No. DRO- 29. NCCES, North Carolina State Univ., Raleigh.

33-Jones, G.M.; Donefer, E. and Elliot, J.J. (1970): "Feeding value for dairy cattle and pigs of high- moisture corn preserved with propionic acid." Can. J. Anim. Sci, 50: 483.

34-Karlovsky, P. (1999): "Biological detoxification of fungal toxins and its use in plant breeding, feed and food production" Natural toxins, 7(1): 1- 23.

35-Kerkadi, A.; Barrioult, C.; Tuchweber, B.; Frohlich, A.A.; Marquard, R.R.; Bouchardand, G. and Yousel, I.M. (1998): J. Toxicol. Environ. Health, 3: 231- 250. Cited by Chrevatidis *et al.* (2003d).

36-Kiermeier, F. and Mashaley, R. (1977): "Einfluss der mokereitechnischen Behanlung der produkte, Z." Lebebsm. Unters. Forsch., 164: 183- 187.

37-Kiessling, K.H. and Pettersson, H. (1991): "Chemical preservatives. In mycotoxins and animal foods." Smith, J.E. and Henderson, R.S. (1991) chapter 32, Pp. 765- 775, CRC press, Ine, USA.

38-Klinkert, W.; Lorkowsky, G.; Creppy, E.E.; Dirheimer, G. and Roschenthaler, R. (1981): "Inhibition of macrophage migration by ochratoxin A and citrinin and prevention by phenylalanine of the ochratoxin A induced inhibition" Toxical. Eur. Res., 3: 185- 189.

39-La Parde, J.C.; Bartz, J.A.; Norden, A.J. and Demunyk, T.J. (1973):"Correlation of peanut seed coat wax accumulation with lolerance to colonization by *Aspergillus flavus*" Proc. Am. Peanut Res. Educ. Assoc., 5: 89- 94.

40-Lindenfelser, L.A. and Ciegler, A. (1970): "Studies on aflatoxin detoxification in shelled corn by ensiling." J. Agric. Fd. Chem., 18: 640- 643.

41-Lindsey, D.L. and Turner, R.B. (1975): "Inhibition of growth of *Aspergillus flavus* and Trichoderma viride by peanut embryos." Mycopathologia, 55: 149- 152.

42-Lupez- Garcia, R. and Park, D.L. (1998): "Effectiveness of post- harvest procedures in management of mycotoxin hozards" In: Bhatnagar, D. and Sinha, S. eds. Mycotoxins in agriculture and food safety. PP. 407- 433, New York, Marcel Dekker.

43-Lupez-Garcia, R.L.; Park, D.L. and Phillips, T.D. (1999): "Integrated mycotoxin management system" In: food, nutrition and agriculture 23, 1999. Editorial group FAO Information Division, Rome, Italy.

44-Luter, L.; Wyslouzil, W. and Kashyap, S.C. (1982):"The destruction of aflatoxins in peanuts by microwave roasting" Can. Inst. Food Sci. Technol. J., 15: 236.

45-Luxsanakoses, S. (2002): "Mycotoxicosis in animals" In: mycotoxin prevention and control in foodgrains. A collaborative publication of the UNDP/FAO Regional Network Intercountry cooperation on preharvest technology and quality control of foodgrains and ASEAN grain postharvest programe, Bangkok, Thailand.

46-Lynd, J.Q. and Lynd, F.F. (1971):"Mucor divergence in aflatoxin effects with duckling bile and plasma" Environ., Res., 4: 316.

47-Maracas, W.F. and Nelson, P.E. (1987): "Mycotoxicology" Pennsylvania state Univ. Press, Univ. Park, Pennsylvania.

48-Marquardt, R.R. and Frohlich, A.A. (1992):"A review of recent advances in understanding ochratoxicosis" J. Anim. Sci., 70: 3968- 3988.

49-Marquez, R.M.; Bujaidar, E.M. and Hernandez, T.I. (1993): "Genotoxic evaluation of ammonium inactivated aflatoxin B_1 in mice fed with contaminated corn." Mutation Research, 299: 1- 8.

50-Marth, E.H. and Doyle, M.P. (1979): "Update on molds" Degradation aflatoxin. Fd. Technol., 33: 81- 87.

51-Mashaly, R.I.; El- Deeb, S.A.; Ismail, A.A. and Youssef, A. (1983): "Effect of some chemical treatments on detoxification of aflatoxins in cottonseed meal" Proc. Int. Symp. Mycotoxins, PP. 515- 522.

52-McKinney, J.; Cavanagh, G.; Bell, J.; Hoverslund, A.; Nelson, D.; Pearson, J. and Selbirk, R. (1973): "Effects of ammonlation of aflatoxins in rations fed lactating cows" J. Am. Oil Chem. Soc., 50: 79- 84.

53-Mirocha, C.J. (1983): "Historical aspects of mycotoxicology and developments in aflatocicosis." Proc. Int. Symp. Mycotoxins, pp. 23- 31.

54-Nelson, T.E.; Johnson, J.; Jantzen, E. and Kirkwood, S. (1969):"Action pattern and specificity of on exo-B-(1-3) D-glucanase from basidiomycetes species QM 806" J. Biol. Chem., 244: 5972- 5980.

55-Noomhorm, A. and Cardoma, T.D. (2002): "Drying and chemical treatment of grains to prevent mycotoxin contamination during storage" Pp. 1-7. In mycotoxin prevention and control in food grains. A collaborative publication of the UNDP/FAO Regional Network Inter- country cooperation on preharvest technology and quality control of foodgrains and the ASEAN grain postharvest programe, Bankok, Thailand.

56-Park, D.L.; Garcia, L.R. and Pveciado, T.S. (1996): "Reduction of risks associated with fumonisin contamination in corn" Adv. Exp. Med. Biol. 392: 335- 344.

57-Park. L.D.; Jemmali, M.; Frayssinet, C.; Lafarge, F.C. and Yvon, M. (1983): "Decontamination of aflatoxin- contaminated peenot meal, using the mono methylamine: Ca $(OH)_2$ method" Proc. Int. Symp. Mycotoxins, Pp. 257- 299.

58-PER, Pesticide Education Resources (2003): Fumigation, Chapter, Univ. of Nebrosko, Lincoln.

59-Purchase, I.F.; Steyn, M.; Rinoma, R. and Tustin, R.C. (1972): "Reduction of the aflatoxin M content milk by processing" Food Cosmet. Toxicol., 10: 383- 387.

60-Ramos, A.J.; Gremmels, J.F. and Hernandez, Z.E. (1996): J. Food. Prot., 59: 631- 641- Cited by Chrevatidis *et al.* (2003d).

61-Raney *et al.* (1987):"Management of on farm stored grain." Cooperative Extersion service, University of Kentucky, Lexington, K.Y

62-Rao, K.S. and Tupule, P.G. (1967):"Varietal difference of groundnut in the production of aflatoxins" Nature, 214: 738- 739.

63-Riley, R.T. and Norred, W.P. (1999): "Mycotoxin prevention and decontamination- a case study on maize" In: food, nutrition and agriculture 23, 1999. Editorial group FAO Information Division, Rome, Italy.

64-Roberts, W.K. and Selitrennikoff, P. M. (1986): Isolation and partial characterization to two antifungal proteins from barley" Biochemica et Biophysica Acta, 88: 161- 170.

65-Rogers, A.E. and Newberne, P.M. (1971): "Nutrition and aflatoxin carcinogenesis" Nature: 229: 62.

66-Rotter, R.G.; Frolich, A.A. and Marquadt, R.R. (1989):"Can. 8. Vet. Res., 53: 449- 453. Cited by " Chrevatidis *et al.* (2003d).

67-Royes, J.B. and Yanong, R.P. (2002):"Molds in fish feeds and aflatoxicosis. Fact sheet FA-95." Dept. of Fisheries and Aquatic Sciences, Institute of Food and Agric. Sciences. Univ. of Florida.

68-Sauer, D.B. and Burroughs, R. (1974): "Efficacy of various chemicals as grain mold inhibitors." Trans. ASAE, 17: 557.

69-Shantha, T. and Murthy, V.S. (1981): J. Assoc. Off. Anal. Chem., 64: 291- 293. Cited by Mirocha, C.J. (1983).

70-Sherwood, R.F. and Puberdy, J.F. (1974): "Production of the mycotoxin, zearolenone by fuzarium graminearum growing on stored grath." II. Treatment of wheat grain with organic acids. J. Sci. Food Agric., 25: 1089.

71-Smith, J.W.; Hill, C.H. and Hamilton, P.B. (1974):"The effect of dietary modifications on

aflatocicosis in the broiler chicken" poultry Sci., 50: 768- 774.

72-Smith, T.K. (1980): "Influence of dietary fiber, protein and zeolite on zearalenone toxicosis in rats and swine" J. Anim. Sci., 50: 278- 285.

73-Suttajit, M. (2002): "Prevention and control of mycotoxins" In: mycotoxin prevention and control in foodgrains. A collaborative publication of the UNDP/FAO Regional Network Intercountry cooperation on preharvest technology and quality control of foodgrains and ASEAN grain postharvest programe, Bangkok, Thailand.

74-Tarr, B. (1996): "Molds and Mycotoxins" Livestock@Omafra. Gov. On. Ca, Last updated: July 22, 2002.

75-USDA, Farm Service Agency (1998): "Ammoniation of aflatoxin-contaminated corn." 1400 Independence Ave., S.W. Stop 0506, Washington.

76-Vandergraft, E.E.; Hesseltine, C.W. and Shotwell, O.L. (1975): "Grain preservatives-Effect on aflatoxin and ochratoxin production" cereal chem., 52: 79- 84.

77-Veltmann, J.R.; Wyatt, R.D. and Voight, M.N. (1981): "The effect of varying the total sulfur amine acid content in the diets of chickens with aflatoxicosis" Poultry Sci., 60: 1748 (Abstr.).

78-Veltmann, J.R.; Wyatt, R.D. and Voight, M.N. and Shamsuddin, Z. (1983):"Influence of dietary sulfur amino acid levels on performance, free amino acids and biochemical parameters in plasma and hepatic glutathione of broiler chickes fed aflatoxin" Poultry Sci., 62: 1518- 1519.

79-Vest, L.R. (1997):"Preventing mycotoxins and mycotoxicosis. Poultry tips from UGA" Univ. of Georgia Dept. of poultry Sci.

80-Waldroup, P.W. (1997):"Managing molds and mycotoxins in poultry feeds" Am. Soybean Assoc., 541 Orchard Road 11- 03, Singapore 238881.

81-Waltking, A.E. (1971):"Fate of aflatoxin during roasting and storage of contaminated peanut products" J. Assoc. Off. Ana. Chem., 54: 533.

82-Weidenborner, M. (2001):"Encyclopedia of food mycotoxins." Springer Publisher, Berlin, New York and London.

83-Williams, P.C. (1991): "Storage of grains and seeds. In mycotoxins and animal foods." Smith, J.E. and Henerson, R.S. (1991) Chapter 30, Pp. 721- 746, CRC Press, Inc, USA.

84-Young, J.C.; Subryan, L.; Potts, D.; Mackare, M. and Gobran, F. (1986): "Reduction in levels of deoxynivalenol in contaminated wheat by chemical and physical treatment." J. Agric. Food Chem., 34:461-465.

85-Young, J.C. Tvbholm, H.L., Friend, D.W. and Prelusky, D.B.(1987): J.Agric. Food Chem., 38:259-261. Cited by Chrevatidis et al,. (2003c).

Ass. Univ. Bull. Environ. Res. Vol. 6 No. 2, October 2003

الوقاية والتخلص من السموم الفطرية

بدير إبراهيم عجاج

قسم بحوث الكيمياء الحيوية والنقص الغذائى والسموم

معهد بحوث صحة الحيوان الدقى – مركز البحوث الزراعية بالجيزة

السموم الفطرية هى مركبات كيميائية سامة تفرزها أنواع من الفطريات التى تنمو على المنتجات العلفية، وتختلف السموم الفطرية باختلاف تركيبها الكيميائى وتأثيرها السمى ودرجة ضراوتها.

ويؤدى تغذية الحيوانات والطيور على علائق تحتوى على مستويات مرتفعة من هذه المركبات إلى أعراض مرضية مثل ضعف النمو، انخفاض معدل إنتاج اللحم واللبن والبيض، ضعف الجهاز المناعى وارتفاع الإصابة بالأمراض وارتفاع نسبة النفوق، كما تؤدى متبقيات هذه المركبات فى المنتجات الحيوانية إلى أضرار بالغة مثل السرطانات فى الإنسان.

لذا يجب مراقبة المواد العلفية لأى نموات فطرية واختبارها دوريا لمعرفة مدى تواجد أى من السموم الفطرية وتركيزها بالإضافى إلى المعدلات المسموح بها لكل نوع من السموم وكل حيوان.

وتتلخص الإجراءات الوقائية للسموم الفطرية فى تجفيف الحبوب جيداً بعد حصادها، وخفض نسبة الرطوبة فى المنتجات العلفية وتخزينها فى ظروف ملائمة وإضافة مضادات الفطريات لها، وللتخلص من السموم الفطرية فى حالة تواجدها بنسب مرتفعة فى المنتجات العلفية يمكن استخدام أى من الطرق الآتية مثل فصل الحبوب الموجود عليها نموات فطرية واضحة، تحميص الحبوب المصابة، التخلص من السموم الفطرية بيولوجيا باستخدام بعض أنواع الميكروبات، أو استخدام العلاجات الكيماوية مثل القلويات والأحماض. وقد استهدفت هذه الورقة إلقاء الضوء على العديد من الطرق المستخدمة فى الوقاية والتخلص من السموم الفطرية فى المنتجات العلفية بالأعلاف