An Overview on Major Mycotoxin in Animal: Its Public Health Implication, Economic Impact and Control Strategies

Eyob Eshetu*      Habtamu Adugna      Azeb Gebretensay
School of Veterinary Medicine, Wolaita Sodo University, Ethiopia

Summary
There has been a significant concern regarding the potential health risks for humans and animals via foods and feeds that are contaminated with different agents. Particularly, mycotoxin contamination is of great importance as it is widespread and unpreventable. In both foods and feeds, molds produce secondary metabolites called mycotoxins; these are produced generally after the fungi reach their maturity. Depending on the definition used, hundreds of fungal compounds are recognized as mycotoxins. However, the attention is mainly focused on aflatoxins, ochratoxins, fumonisins, ergot, patulin and trichothecenes, which are considered the most important threats for human and animal health. Mycotoxin contamination causes a fundamental problem all over the world including developed countries. Additionally, the economic impact of mycotoxins is another global concern on the agricultural markets. These concerns are based on toxicological data, which show that naturally occurring levels of mycotoxins have adverse effects in farm and laboratory animals as well as humans. Owing to the significant health risks and economic impacts, considerable investigations are being performed to diminish their harmful effects and to prevent their formation. In order to limit their levels, much research has been focused on detecting the mycotoxins in contaminated food and feedstuffs. This review will focus on information about primary mycotoxins, their occurrence, its public health implication, economic impact, and control strategies.

Keywords: Mycotoxins; Aflatoxin; Ergot; Public health

INTRODUCTION
The term mycotoxin was used for the first time in 1961, after the math of a veterinary crisis in England, during which thousands of animals died. It was derived from “Myco” means fungus “Toxin” means poisons (Checke and Shull, 1985). Mycotoxins are toxic secondary metabolite of low molecular weight produced by naturally occurring fungus and the disease was linked to a peanut meal, incorporated in the diet, contaminated with a toxin produced by the filamentous fungus Aspergillus flavus (Richard, 2007).

Plants can be contaminated in two ways: fungi growing either pathogen on plants in the field or on stored plants (Glenn, 2007). Biosynthesis of mycotoxins which are secondary metabolites is related to some internal factors e.g. genetic potential of fungi, substrate or to factors under which a crop is grown, harvested and stored e.g. oxygen (usually fungi need at least 1-2% O₂), humidity (usually fungi grow at 13-18% moisture), temperature (usually fungi grow at 20°C-30°C), physical damages by insects and other stress factors (Simona and mihaela, 2008). Basically three major genera of fungi are identified to produce mycotoxins: they include Aspergillus, Fusarium and Penicillium. Although other genera are also produces these toxigenic compounds. Mycotoxin producing fungi grow on a wide spectrum of feed that include cereal grains, groundnuts, beans and peas. The Food and Agriculture Organization (FAO) estimates that 25% of the world’s agricultural commodities are contaminated with mycotoxins, leading to significant economic losses (Wu, 2007). Mould species that produce mycotoxins are extremely common, and they can grow on a wide range of substrates under a wide range of environmental conditions; they occur in agricultural products all around the world (Bosco and Mollea, 2012). The presence of mycotoxins in food and feeds not only affects economy but also represents a health hazard to humans and animals constitute a risk for international trade (Cole and Cox, 2004). They are stable chemical compounds and can neither be completely removed from the food supply nor destroyed during processing and heat treatment. Always exposure is through consumption of contaminated feeds or food (marin et al., 2013).

Mycotoxicosis is diseases caused by exposure to foods or feeds contaminated with mycotoxins (Nelson et al., 1993). Direct consequences of consumption of mycotoxins-contaminated animal feed include: reduced feed intake, feed refusal, poor feed conversion, diminished body weight gain, increased disease incidence (due to immune-suppression), and reduced reproductive capacities (Fink-Gremmels and Male, 2007) which leads to economic losses (Wu, 2004, 2006a).

Mycotoxins also have been reported to be carcinogenic, tremorgenic, haemorrhagic, teratogenic, and dermatitis to a wide range of organisms and to cause hepatic carcinoma in humans (Nidhi and Manisha, 2013). A supplemented list, aflatoxins were listed as a natural mixture which has a proven carcinogenic potential for humans (IARC monographs on the evaluation of carcinogenic risk to humans 2002; http://monographs.iarc.fr/ENG/Classification/ClassificationsGroupOrder.pdf2010). It varies widely in their toxicity and the toxic effects may be both acute (after a single exposure) and chronic (after repeated exposure). The severity of which depends on the toxicity of the mycotoxins, the extent of exposure, age and nutritional status of the individual and possible synergistic effects of other chemicals to which the individual is exposed.
major OTA producing species are belonging to several Aspergillus species, mainly A.melleus (Romagnoli et al., 2007; O’Riordan and Wilkinson, 2009). Immunosuppressive, hepatotoxic, carcinogenic, mutagenic, and teratogenic effects can be observed according to animal species, sex, and aflatoxin type, exposure dose and period (Marin et al., 2013).

It has been detected that median lethal dose (LD50) of AFB1 is estimated to be between 0.3 and 18 mg/kg according to the administration route, animal species, age and health condition. Poultries are more sensitive to aflatoxin than mammals. Within poultry, ducks are the most susceptible species then turkey poultries and then chicken. Within domestic animals, the order is canine, swine, calves, cattle and sheep. Young animals are more susceptible to AFs than matures. Nutritional deficiencies, especially protein and vitamin E increase the susceptibility to AFs (Bryden, 2011).

Ochratoxin A

Ochratoxin A is the most commonly encountered and toxic member of ochratoxins group. Two genera of fungi produce OTA, Aspergillus and Penicillium. The main OTA producing species are A.ochraceus, A.carbonarius, A.melleus A.sclerotiorum, P.verrucosum and P.nordicum. Ochratoxin A nomenclature is derived from Aspergillus ochraceus, the first fungus that was discovered to produce the toxin (Zain, 2011).

Ochratoxin A has been suggested as a causative agent of Balkan Endemic Nephropathy. It disrupts several cellular functions, including ATP production; however, its toxicity is generally attributed to its inhibitory effects on the enzyme involved in the synthesis of the aminoacyl-tRNAs containing phenylalanine (Bennett and Klicha, 2003). Ochratoxin absorbed into the body is distributed at a high concentration in the kidney. It inhibits the synthesis of proteins, DNA and RNA in the cell. It shows renal toxicity by inhibiting various enzyme activities in the kidney. It is often detected at the same time with citrinin and mycotoxins similarly with renal toxicity, but there is also a report that additive renal toxicity is not observed by their simultaneous intake (Braun berg, 1994). The rumen of ruminants is highly capable of degrading ochratoxin A into phenylalanine and ochratoxin-α with low toxicity, therefore poisoning by ochratoxin is not very much likely to occur; however, calves with immature rumen functions are sensitive to ochratoxin. As for effects on human health, there is a possibility to cause renal dysfunction and the relationship with Balkan nephropathy observed in Balkan has been indicated. Ochratoxin A is observed in human blood in many countries, however at a low concentration. There also is a recent report that ochratoxin A at a higher concentration (mean 18±7 ng/mL) compared to normal persons is detected in the blood from patients with symptoms similar to Balkan nephropathy (Zaied, 2010). Moreover, ochratoxin A is found in breast milk and the positive correlation to the intake of foods including pork is indicated (Galvano et al., 2001).

Trichotheccenes

Different Fusarium species such as F.culmorum, F.sporotrichioides, F.tricinctum, F.roseum F.graminearum,
F. nivale, F. sambucinum and some members of Myrothecium are able to produce trichothecenes. Corn, barley, wheat, oats, rye, soybeans and fruits as well as animal feeds are mostly attacked by fusarium. Fungal infection appears as a red-pink colored area, mostly at the tip of the crop (Kim et al., 2014).

During the last 40 years, more than 180 trichothecene mycotoxins have been discovered. Structurally, trichothecenes have been classified according to the difference in the functional group, hydroxyl and acetoxy side, into four groups. Type A involves HT-2, T-diacetoxycircenol (DAS) and neosolanil. The trichothecenes cause the greatest problems to animal health. General signs of TCs toxicity in animals include weight loss, decreased feed conversion, feed refusal, vomiting, bloody diarrhea, severe dermatitis, hemorrhage, decreased egg production, abortion and death (Didwania and manisha, 2013).

Fumonisins

Fumonisins are a group of non-fluorescent mycotoxins, mainly produced by Fusarium moniliforme, F. proliferatum, F. napiforme, F. dlamini and F. nygamai (Marin et al., 2013). Corn is mostly infected with fumonisin producing moulds, particularly when corn is imported from humid climates (Scussel et al., 2014). As study conducted by (Diekman and Green, 1992), only the fumonisins FB1 and FB2 appear to be toxicologically significant and the occurrence of FB1 in cereals, primarily maize, has been associated with serious outbreaks of leukoencephalomalacia (LEM) in horses and pulmonary oedema in pigs. It is characterized by liquefactive necrotic lesions of the white matter of the cerebral hemispheres and has been reported in many countries, including the USA, Argentina, Brazil, Egypt, South Africa and China. FB1 is also toxic to the central nervous system, liver, pancreas; kidney and lung in a number of animal species as were FB2 is hepatotoxic in rats.

Patulin

Patulin is mycotoxin produced by some filamentous fungi of the genus Aspergillus, Penicillium and Byssochlamys. These fungi grow in fruits and vegetables, but rotten apples and apple-based products are considered the main source of this fungal toxin, and penicillicum expanses the principal responsible. Patulin was firstly proposed for therapeutic purpose because of its antibiotic properties. However, in 1960s it was reclassified as mycotoxin due to its toxicity (Puel et al., 2010). These toxins which can grow in different food products reach the humans by contaminating foods (Russel et al., 2010). It has electrophilic properties and high reactivity to cellular nucleophiles. At cellular level it can cause enzyme inhibition and chromosomal damage. Patulin causes cytotoxic and chromosome-damaging effects mainly by forming covalent adducts with essential cellular thios and Patulin has also become important to apple processors as a method for monitoring the quality of apple juices and concentrates.

The presence of high amounts of patulin indicates that moldy apples were used in the production of the juices. Patulin is being considered as a “possible toxin” in Europe and New Zealand and is regarded as the most dangerous mycotoxin in fruits, particularly apples, pears, and their products. Patulin is mainly associated with surface-injured fruits, which renders them vulnerable to fungal infection, mainly by Penicillium spp (Abramson et al., 2009).

Ergot

Ergot alkaloids are made as secondary metabolites of fungi. Their production occurs in the sclerotia of several species of the genus Claviceps, the most common is Claviceps purpurea. These compounds belong to the family of indole alkaloids, one of the ten classes of alkaloids that pharmacologists have defined from plant secondary metabolites. Lysergicacid, a tetracycline ergoline ring system is a common structure to all of the ergot alkaloids (Krska and Crews, 2008).

The disease is of greater significance because of the toxic alkaloids produced by the fungus. Grain is classified as "ergoty" if it exceeds the level and is of lower value. When infected rye (a staple for humans in European countries with cold wet climates) was ground and used to produce bread, non-lethal levels of ergot poisoning caused severe hallucinations or intense burning pain (St Anthony's fire) and gangrene of feet, hands, and whole limbs, due to the vaso-constrictive action of the ergot alkaloids (Edaie, 2004). The pharmacological activities of the fungus are due to components that include lysergic acid diethylamide (LSD) (Cunhae et al., 2009).

The occurrence and factors that affect the incidence of mycotoxins

Occurrence of Mycotoxins in Feed

The contamination of foods and feeds is a significant problem in worldwide. Food contamination with mycotoxins is a very complex process and sometimes more than one type of mycotoxins occur as reported contamination with aflatoxinB1/fumonisinB1 or ochratoxin A/aflatoxin B1 (Murphy et al., 2006). The contamination process can lead to extremely toxic interactions. It have been detected in various food commodities from many parts of the world and are presently considered as one of the most dangerous contaminants of food and livestock feed (Okoli, 2007a). Contamination of feeds with mycotoxins is considered several harmful effects on human and animal health, resulting economic losses as well as in undesirable trade barriers for raw materials and consumable products (Wu, 2006b).
Table-1: Some major mycotoxins, their main effects and species fungus which produce them in feeds

<table>
<thead>
<tr>
<th>Mycotoxins</th>
<th>Producing fungi</th>
<th>Feed affected</th>
<th>Toxicities</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aflatoxin</td>
<td><em>Aspergillus flavus,</em> A.Parasiticus</td>
<td>Cereals (maize), nuts</td>
<td>Hepatocarcinogen, teratogenic effects</td>
</tr>
<tr>
<td>Ochratoxins</td>
<td><em>Aspergillus ochraceus</em></td>
<td>Cereals (maize, wheat and barley) fruit, nuts</td>
<td>Nephrotoxin, Hepatotoxic</td>
</tr>
<tr>
<td>T-2 Toxin</td>
<td><em>Fusarium graminearum</em></td>
<td>Cereals (maize, wheat)</td>
<td>Cytotoxicity</td>
</tr>
<tr>
<td>Vomitoxin</td>
<td><em>Fusarium graminearum</em></td>
<td>Cereals (maize, wheat)</td>
<td>Vomiting</td>
</tr>
<tr>
<td>Zearalenone</td>
<td><em>Fusarium graminearum</em></td>
<td>Cereals, rice, silage</td>
<td>Estrogenic effect</td>
</tr>
<tr>
<td>Fumonisine</td>
<td><em>Fusarium moniliforme F.verticilloides</em></td>
<td>Cereals (maize, wheat)</td>
<td>Pulmonary edema, leukoencephalomalacia, hepatotoxicity</td>
</tr>
</tbody>
</table>

Source: (Murphy et al., 2006)

Condition for Occurrence of Mycotoxins

Various classifications are used in categorizing the factors that affect the incidence of mycotoxigenic fungi and mycotoxins in the food chain. Some classifications categorize these factors as extrinsic and intrinsic, some as physical, chemical and biological factors while others classify them as ecological, environmental and storage factors (Zain, 2011).

Moreover, insects play an important role through physical damage of the grains and mechanical transmission of the microorganisms (Richard, 2007; Marroquin et al., 2014). Most of cereal grains, oil seeds, tree nuts, and fruits (especially dried ones) are susceptible to fungal attack and mycotoxin formation. Agricultural products like cereal grains and forages can be contaminated during pre-harvest (field period), harvest, and post-harvest (storage and transportation period). Corn and other grains used in animal feed could also be contaminated by pathogenic moulds, thereby mycotoxins, even they may be destroyed at different rates during industrial processing (Griessler et al., 2010; Reddy et al., 2010; Bryden, 2011).

Climate represents the key agrosystem that drives fungal colonisation and mycotoxin production (Magan et al. 2003). Probably the two most important environmental components favouring mold growth and mycotoxin production are hot and humid conditions. Drought conditions actually constitute stress factors to plants rendering them vulnerable to mould infection with ensuing increase in toxin production. An indelible sign that droughts prop up toxic contamination is the fact that these conditions preceded the fatal outbreak of acute human aflatoxicosis that occurred in Kenya in 2004 recorded higher mycotoxigenic fungal contamination during the rainy season than in the dry harmattan season among produce in Nigeria. Plant, animal, and human epidemics are influenced by the climate (Thomson et al., 2006).

PUBLIC HEALTH IMPLICATIONS AND ECONOMIC IMPORTANCE

Public Health Implications

Animal feeds are an essential part of the farm animal to human food chain; therefore, infectious and non-infectious hazards present in animal feeds pose a threat to human health. Public concern on health matters related to food has increased following the recent epidemic of bovine spongiform encephalopathy (BSE) in the UK and foot and mouth disease in UK, Ireland, Belgium, France, Germany and South Africa (Hinton, 2000). Animal feeds provide a market for slaughterhouse offal; grains considered “unfit for human consumption” and similar waste products to be turned into a profit. Cereal grains are the primary ingredients of most animal feeds and these are often of sub-standard grade, which predisposes these grains to mycotoxins contamination. Mycotoxins have adverse effects on human and animal health. Many of them may be toxic to vertebrates and other animal groups and, in low concentrations, some of them can cause autoimmune illnesses, exhibit hormonal activity and have allergenic properties, while others are teratogenic, carcinogenic and mutagenic (Bezerra et al., 2014).

Their main effects on DNA, RNA, protein synthesis and their pro-apoptotic action may cause changes in physiological functions including reproduction, growth and development. Development defects, including birth ones, are possible adverse effect following exposure to mycotoxins and it may also affect the gastrointestinal system, cause skin irradiation, have hematological effects and reduce growth. Human exposure to mycotoxins may result from consumption of plant derived foods that are contaminated with toxins, the carryover of mycotoxins and their metabolites into animal products such as milk, meat and eggs or exposure to air and dust containing toxins. The disease resulting from mycotoxin exposure is a mycotoxicosis. It occurs when mycotoxins enter the bodies usually by consumption of contaminated food (Miller et al., 2001).

Chronic effects of mycotoxins in human populations

In many regions of the world, dietary staples, especially cereal grains contain low levels of mycotoxins. The impact of regular low level intake of mycotoxins on human health is likely to be significant with a number of possible consequences including impaired growth and development, immune dysfunction and the disease
consequences of alterations in DNA metabolism (Smith et al., 2007).

Growth and development
Numerous animal studies have shown that one of the first effects of mycotoxin ingestion is reduced feed intake and growth. In 2002 Gong et al. conducted a cross-sectional epidemiological survey in West Africa in which they determined the aflatoxin exposure of children between 9 months and 5 years of age and examined their growth, development and height against a WHO (2008) reference population. His study revealed a very strong association between exposure to aflatoxin in the children and both stunting and being underweight. Accordingly, significant malnutrition and exposure of the children to aflatoxin in utero is subsequently after birth. The children were also co-exposed to a number of infectious diseases and it is likely that the exposure to disease and aflatoxin would significantly compromise growth and development through reduced food intake and also the repartitioning of nutrients to maintain an upregulated immune system and away from growth and development. There are also reports linking kwashiorkor, a disease of malnutrition, to aflatoxin exposure (Hendrickse and Kwashiokor, 1991).

Immuno-suppression
Aflatoxin, trichothecenes, ochratoxin A, sterigmatocystin, rubratoxin, fumonisins, zearalenone, patulin, citrinin, wortmannin, fusarochromanone, gliotoxin and ergot alkaloids have been shown to cause immunosuppression and increase the susceptibility of animals to infectious disease (CAST, 2003). Substantial evidence exists that mycotoxins can be immune-toxic and exert effects on cellular responses, humoral factors and cytokine mediators of the immune system (Oswald et al., 2005). The effects on immunity and resistance are often difficult to recognize in the field because signs of disease are associated with the infection rather than the toxin that predisposed the individual to infection through decreased resistance and/or reduced vaccine or drug efficacy. Moreover, in animal models, immunosuppressant effects of toxins occurs at lower levels of intake than do the toxin’s effects on other parameters of toxicity such as feed intake and growth rate. Recent studies in Gambian children (Turner et al., 2003) and in Ghanaian adults show a strong association between aflatoxin exposure and reduced immune-competence suggesting that aflatoxin ingestion decreases resistance to infection in human populations. In 2004 the studies by Pestka and his colleagues have shown that DON can both stimulate and suppress the immune system. This has been demonstrated with the effect of DON on dysregulation of IgA and immunosuppression.Toxins, in particular aflatoxin, can impair the immune system to the point that it contributes to the development of kidney disease in animal models that closely resembles human glomerulo-nephritis IgA nephropathy.

Carcinogenicity, mutagenicity and teratogenicity
There has been extensive evaluation of the capacity of mycotoxins to interact with DNA and modify its action. Mycotoxins may be carcinogenic (eg. fumonizins), carcinogetic and teratogenic (eg. ochratoxin) or carcinogetic, mutagenic and teratogenic (eg. aflatoxin). When it was first appreciated some 40 years ago that aflatoxin was a potential carcinogen, it was this finding that gave significant impetus for the research that has subsequently been conducted to define the role of mycotoxins in human and animal disease. Wild and Turner (2002) have extensively reviewed the mechanism of toxicity and carciogenesis of aflatoxins. They also explains as now a day, there is a significant body of evidence demonstrating human exposure in utero to a number of mycotoxins but the relevance of this exposure to birth defects or impaired embryonic developed has received relatively little attention.

Cawdell et al. (2007) were able to identify some 40 mycotoxins that had been shown to be teratogenic and/or embryotoxic in animal models. However, most of these mycotoxins have only been evaluated in rapid screening assays that did not seek to delineate their potential teratoenicity during early pregnancy. Aflatoxin B1, ochratoxin A, rubratoxin B, T-2 toxin, sterigmatocystin and zearalenone have been shown experimentally to be teratogenic in at least one mammalian species. Recent epidemiological investigations of human populations in Texas, China, Guatemala and southern Africa that rely on foods prepared from maize, which is often contaminated with fumonisins, found a significantly higher incidence of neural tube defects in babies (Voss et al, 2007).

Bioterrorism
Mycotoxins can be used as chemical warfare agents Ciegler (1986). There is considerable evidence that Iraqi scientists developed aflatoxins as part of their bioweapons program during the 1980s. Toxigenic strains of Aspergillus flavus and A. parasiticus were cultured, and aflatoxins were extracted to produce over 2,300 liters of concentrated toxin. The majority of this aflatoxin was used to fill warheads; the remainder was stockpiled (Stone, 2001).

Economic Importance
Worldwide, approximately 25% of crops are affected by mycotoxins. Annual economic costs of mycotoxins to the U.S. agricultural economy is estimated to average $1.4 billion (CAST, 2003). A widely cited journal article by Otsuki et al. (2001) found that cereal (and cereal preparations) exports by 9 African countries to the EU during 1998 would have declined by 59 percent, or $177 million, if the EU had harmonized their aflatoxin
regulations at the proposed limit and enforced this limit on all shipments. Economic losses are due to effects on livestock productivity, crop losses and the costs of regulatory programs directed toward mycotoxins. Small proportions of crop aflatoxin content are transferred from feed to milk causing even feed crops to have stringent regulations for use at dairies (Wu and Van egmond, 2004).

Aflatoxins impact the U.S. cotton industry because dairies pay a premium for cottonseed that must contain less than 20 ppb to enter the dairy feed market. Rotation crops (particularly maize and peanuts) are frequently also affected. A production of these is often limited by frequencies and severity of aflatoxin contamination (Cotty, 2001). Under epidemic conditions, agronomic practices have modest impact on the disease (Miller, 2008).

The consumption of multiple mycotoxins contaminated diet may induce hematological, biochemical and liver physiological changes and growth depression in livestock (Gowda et al., 2008), and thus the presence of mycotoxins in poultry feeds causes significant economic losses to animal industries (Awad et al., 2006a). Sometimes mycotoxins occur at concentrations high enough to cause major losses in health and performance of animals. However, mycotoxins are more usually at lower levels that result in interactions with other stressors to cause subclinical losses in performance, increases in incidence of disease and reduced reproductive performance. To the animal producer, these subclinical losses are of greater economic importance than losses from acute effects. In addition to reduced animal products, industries costs are derived from the efforts made by producers and distributors to counteract their initial loss, the cost of improved technologies for production, storage and transport, the cost of analytical testing, especially as detection or regulations become more stringent and the development of sampling plans (Whitaker et al., 1995).

Rodrigues (2008) reviewed mycotoxin contamination of diverse feedstuff samples from throughout the world for six mycotoxins ( aflatoxin B1, ZEA, DON, fumonisin, T-2 toxin and OTA). Fumonisin was the most frequent contaminant (58% of samples) occurring commonly in corn, distillers grains and finished feeds. It was estimated that in the U.S the total annual loss due to aflatoxin in corn is about $163 million ($73 million to $332 million). The annual market loss through corn rejected for feed is about $31 million ($10M to $54M), while the loss through corn rejected for feed and through livestock losses is estimated at $132 million ($14M to $298M). BT corn would reduce aflatoxin in cases where Bt-sensitive insects was the main determinant of aflatoxin development. The level of Bt corn planting in such regions at about 17% (USDA 2004), and the assumption that Bt corn is partly effective in reducing aflatoxin only in Texas/Southeastern U.S. where 80% of the aflatoxin contamination problems occur, the upper limit of the current benefit is $14 million ($5.0M to $22M).

Table-2: Estimated losses due to mycotoxin in us corn and benefit from BT corn (average and 95% confidence interval, SUS Million).

<table>
<thead>
<tr>
<th>Loss in $ US Million</th>
<th>Fumonosin</th>
<th>Aflatoxin</th>
</tr>
</thead>
<tbody>
<tr>
<td>Market loss</td>
<td>39 (14 to86)</td>
<td>163 (73 to 332)</td>
</tr>
<tr>
<td>Animal Health</td>
<td>0.27 (0.051 to 2)</td>
<td>N/A</td>
</tr>
<tr>
<td>Total us loss</td>
<td>40 (14 to 88)</td>
<td>163 (73 to332)</td>
</tr>
<tr>
<td>Benefit from Bt corn</td>
<td>8.8 (2.3 to 31)</td>
<td>14(6.2 to28)</td>
</tr>
</tbody>
</table>

Source: (Wu, 2006a)

The economic impact of aflatoxins is derived directly from crop and livestock losses due to aflatoxins and in directly from the cost of regulatory programs designed to reduce risks to human and animal health. The Food and Agricultural Organization (FAO) estimates that 25% of the world’s crops are affected by mycotoxins, of which the most notorious are aflatoxin. Aflatoxins losses to livestock and poultry producers from aflatoxin-contaminated feeds include death and more subtle effects of immune system suppression, reduced growth rates, vaccine failure and losses in feed efficiency. By reducing the immune system, it increasing the chances of infection and targets the liver causing reduced liver function and death. Other adverse economic effects of aflatoxin include lower yields for food and fibre crop (Setamu et al., 1997).

CONTROL STRATEGIES

In recognition of the global public health importance of food borne diseases and in order to promote economic growth and development, the World Health Organization (WHO) commissioned the Food borne Disease Burden Epidemiology Reference Group (FERG) to undertake the systematic reviews of some chemicals and toxins like cyanide in cassava, aflatoxin, dioxins and peanut allergens (Hird et al, 2009). A primary strategy should aim to eliminate mycotoxins by reducing mould proliferation during Cultivation and storage. (Magan and Aldred, 2007)

Planting

Crop varieties selection Seeds must be free of pests and disease before planting, to ensure healthy, vigorous plants capable of withstanding attack during the growing season. A possible strategy is also to select crop varieties on their ability to resist mould attack (Clements and White, 2004).
Sowing date
If the sowing date flowering coincides with spore release, then more frequent and severer attacks are likely (Champeil et al., 2004). As far as is practical, crop planting should be timed to avoid high temperature and drought stress during the period of seed development and maturation (Codex Alimentarius, 2003).

Pre-harvest
In absence suitable cultivation techniques, the primary reservoir of fungal inoculum is the residues from previous crops. Also, ploughing limits mould contamination (Champeil et al., 2004). The effects of crop rotation are also significant. For example, wheat following maize in the rotation has been found to have DON concentrations six times higher than those in wheat following another cereal (wheat, barley) or soybean (Krebs et al., 2000).

Predictive models
Software applications are available to help farmers predict mycotoxin risk during the year as a function of climatic parameters (Prandini et al., 2009). Fungicides should be useful whenever a predictable risk exists (Paul et al., 2008). However, risk management must take into account potential risks to consumer health linked to the presence of fungicides in food. Thus, a recent study compared the toxicity of two fungicides and two mycotoxins, ZEA and DON (Muri et al., 2009).

Sorting
Contaminated grain does not have the same color or density as safe grain. Thus, grain can be sorted according to appearance or density. Not only sorting but also, washing food or grain can reduce mycotoxin levels. For example, the first step in spaghetti production with wheat is washing, which removes 23% of DON (Visconti et al., 2004).

Biocontrol techniques
Study conducted by (Dorner and Cole, 2002) showed that treatment of soil with non-toxigenic strains of *A. Pavus* and *A. parasiticus* significantly reduced pre-harvest aflatoxin contamination. In addition, soil treatment with nontoxicogenic strains had the beneficial carry-over effect of reducing aflatoxin contamination that occurred during storage.

Post harvest
During storage important factors should be managed to prevent crops from fungal contamination (Schro, 2004). For example, grain should be stored with less than 15% moisture content to eliminate pockets of higher moisture, at low temperature and a low oxygen concentration (51%) (Kabak et al., 2006).

Eliminating mycotoxins from food and feed
Different methods are used to decontaminate food and feed before ingestion. Presently, regulations do not permit the decontamination of food that exceeds the concentration threshold limits. Mycotoxin reduction in food could be carried out during industrial processing or by using additives which eliminate or deactivate the mycotoxin. In all cases, decontamination processes should destroy or inactivate mycotoxins, generate no toxic products, guaranty the nutritional value of the food and induce no modification to the technological properties of the product (Bullerman and Bianchini, 2007). Whitelow and Hagler (1998) were generalized that it is possible to prevent and manage mycotoxins through reduction it in preparation of silage, wet feed, dry feed, using mould inhibitor and animal management.

CONCLUSION AND RECOMMENDATIONS
Mycotoxins are ubiquitous and have been detected in many parts of the world and are presently considered as one of the most dangerous contaminants of food and animal feeds. As it is possible to understand from this paper, mycotoxins contamination of food and feed represents a serious risk for human and animal health, which is well known among the scientific community but almost unknown among the consumers. More information concerning about why mycotoxins occur, when to expect them, how to prevent their occurrence and how to deal with their presence will help to maintain, human health, economic sustainability and animal welfare of country. Therefore, based on above conclusion the following recommendations were forwarded

- Awareness of Extension workers and owner’s of livestock on impact of mycotoxins in feeds: implications to livestock, human health, and economy of a country should be conducted.
- Not only human but also animal exposure should be considered by authorities.
- Countries should have their own national policies and limits to save public health from toxic outcomes.

REFERENCES


deoxyxynivalenol, fumonisins, Toxins, 6: 20-32.
deoxyxynivalenol content of wheat. The world grows organic.Proceedings of the 13th
dairy Cattle.
Organization.
exposure assessment, Food and Chemical Toxicology. 60: 218-237.
global environment-a review, Food and Chemical Toxicology. 69: 220-230.
Miller, J.D. (2008): Mycotoxins in small grains and maize: old problems, new challenges, Food Additive and
Contaminants. 2: 219-230.

exposure assessment, Food and Chemical Toxicology. 60: 218-237.
global environment-a review, Food and Chemical Toxicology. 69: 220-230.
Miller, J.D. (2008): Mycotoxins in small grains and maize: old problems, new challenges, Food Additive and
Contaminants. 2: 219-230.
Muri, S.D., Van der, Voet H., Boon P.E., van Klaveren, J.D. and Bru¨ schweiler, B.J. (2009): Comparison of
human health risks resulting from exposure to fumigicides and fungicides via food, Food Chem
Toxicology. 12:2963–2974.
5:51-65.
International J. of Pharmacy and Pharmaceutical Science. 5:1014-1019.
commercial chilli spice preparations and subsequent development of an improved method. Food Cont.,
20:700–705.
commercial poultry feeds and feed raw material in the humid tropical environment of Imo state, Nigeria.
Pp. 115-119
European Food Safety Standards on African Exports,” Food Policy. 26: 495-514.
triazole- based fungicides for Fusarium head blight and deoxynivalenol control in wheat: a multivariate
meta-analysis. Phytopathology. 98:999–1011.
Food Microbiology.119: 3-10.
343-356.
and medicinal plants marketed in Italy, Food Cont., 18:697–700


