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The Risk of Mycotoxins Contamination of Dairy Feed and Milk on Smallholder Dairy Farms in Kenya

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Abstract: Mycotoxins are toxic secondary metabolites produced by fungi that thrive in warm humid environments. Because Kenyan climate is favourable for growth of mycotoxins causing moulds, the threat of mycotoxin related livestock and human poisoning is real and of major concern. This threat is made even more palpable by the fact that, staple diets in many Kenyan households are based on crops such as maize, which are highly susceptible to mycotoxins contamination. The objective of the current study was to highlight the existing but grossly ignored danger of mycotoxin contamination of dairy feeds possibly leading to animal and human poisoning. During the study, qualitative and quantitave information were obtained through extensive review of scientific articles, magazines and books touching on this subject. Consultations were also held with resource persons (Toxicologists) to help validate some of the assertions made by various authors. A very clear illustrated facts, as revealed by the current study are that, aflatoxin is one of the most widely occurring and dangerous of all mycotoxins known. The term aflatoxin refers to a closely related group of metabolites produced by toxigenic strains of *Aspergillus flavus* and *A. parasiticus*. Aflatoxins are potent carcinogenic, mutagenic, teratogenic, and immunosuppressive agents. Four different aflatoxins, B1, B2, G1 and G2, have been identified with B1 being the most toxic. Their contamination of agricultural feed grains poses a serious threat worldwide. Although occurrence and magnitude of mycotoxin contamination varies with geographical and seasonal factors and also with the conditions under which a food or feed crop is grown, harvested, and stored, those grown under tropical and subtropical conditions are more prone to contamination than those in temperate regions due to favourable humidity and temperature levels for mould growth (10 - 40°C, pH range of 4 - 8 and above 70% equilibrium relative humidity). Aflatoxin B1 is potent when it contaminates food grains. This potency was illustrated by an outbreak of aflatoxin poisoning in Kenya (January - July 2004). This outbreak resulted in 125 recognized deaths and hospitalization of over 300 others across various districts (Makueni: N = 148; Kitui: 101; Machakos: 19; Thika: 12 and Kenyatta National Hospital: 37). Of 342 samples tested, a total of 182 (53.2%) had >20 ppb of aflatoxin. In addition, a substantial percentage of samples from each district had aflatoxin levels >1,000 ppb: Makueni (12.1%), Kitui (9.6%), Thika (3.9%), and Machakos (2.9%). Livestock get poisoned when they consume contaminated feeds. Virtually all feeds are susceptible so long as conditions permit mould colonization. Mouldy protein supplements, poultry manure, cereal grains and their by-products are the primary sources of mycotoxins found in homemade dairy concentrates on smallholder farms. Aflatoxin M1 is metabolic breakdown product of aflatoxin B1 and can appear in the milk of lactating cows consuming significant quantities of aflatoxin B1 emanating from mouldy feedstuffs. When the level of M1 appearing in milk and other dairy products is more than 20 ppb (concentration accepted by Kenya authorities), then it becomes a food safety hazard. Control of mycotoxins in dairy diets on smallholder farms would reduce the likelihood of livestock poisoning and concentration of mycotoxin residues in milk and other animal products destined for human consumption.

Key words: Aflatoxins poisoning, ruminant livestock, mould colonization

Introduction

In most developing countries, livestock production is an important part of the national economy and more importantly, of the subsistence and semi-commercial smallholder farming systems, dominated by resource-poor farm households. In Kenya, dairy industry is increasingly becoming a smallholder farmers' domain. At preset, they own over 80% of the 3 million heads of dairy cattle, producing about 56% of the total milk production and contributing 80% of the marketed milk (Peeler and Omore, 1997; Staal *et al*., 1999; Conelly, 1998; Thorpe *et al*., 2000). This therefore means, focusing dairy development efforts to this category of farmers will not only provide a good potential to improve national milk self-sufficiency and family incomes, but also a greater potential in improving public health

through improvement of milk quality and hygiene. Currently, a major obstacle to increased milk production on smallholder farms is the chronic shortage of affordable feeds of adequate quality and quantity, particularly during the dry season. A closer look a the use of available feed resources on these farms, indicates that, the problem of feed shortage is further complicated by farmers inability to use them before they spoil, especially during the wet season when there is a production peak. During the peak production, one of the major agents of feed spoilage, are moulds. Though their prevalence and rate of colonization are highly dependent on the climate and farm practices, the growth of moulds in raw materials and finished feeds for livestock is a universal problem. Mould spores, of the so-called field fungi, can contaminate cereal grains and stover in the standing crop and in severe conditions (too wet or too dry season). Their spores can germinate, especially under high moisture conditions. During storage a different range of moulds (the storage fungi) become more important. These moulds can grow at moisture contents above 11%. Both the field and storage fungi can lead to a build up of mycotoxins. Mycotoxins are metabolites produced by these fungi (Moulds), which serve as a protective mechanism (Merck Veterinary Manual, 1986). Mycotoxins are among the most common contaminants in animal feeds, causing great economic loss in both the livestock industry and aquaculture (Sharlin *et al.*, 1981; Hafez *et al.*, 1982; Jantrarotai and Lovell, 1990). Problems associated with mycotoxins tend to be worse in the tropics where high humidity and temperature create optimal conditions for fungal growth. Mycotoxins include metabolic by-products produced by a number of different fungi that may or may not be toxic. Aflatoxin is one of the most widely occurring and dangerous mycotoxins (C.A.S.T., 1989). Aflatoxin, is produced by the fungi *Aspergillus flavus* and *Aspergillus parasiticus*. Four different aflatoxins, B1, B2, G1 and G2, have been identified with B1 being the most toxic, carcinogenic, hepatotoxic and potentially mutagenic, while also being the most prevalent (Stoloff, 1980). Mycotoxins are not only toxic to animals but also to exposed humans. Usually, exposure is through consumption of contaminated foods (Nelson *et al.*, 1993). This paper examines some of the potential factors and pathways that could expose both livestock, particularly dairy cattle and human beings to mycotoxins poisoning. Special reference is hereby made to the smallholder resource-poor farmers in Kenya where lack of awareness and limited feeds are believed to aggravate chronic mycotoxins (i.e aflatoxin) poisoning.

Materials and Methods

The current study was conducted over 2-month period (May - June, 2005) in Kenya. During the study, relevant scientific publications touching on mycotoxicosis and aflatoxicosis in both livestock and humans formed the primary source of qualitative and quantitative information discussed herein. Secondary sources included books, newsletter, newspapers and magazines. Consultations were also held with technical persons specialized in toxicology and institutions that have registered cases of aflatoxin poisoning (hospitals). The purpose of consultations was to help validate some of the obtained baseline data and assertions made by various authors of documents reviewed. The objective of the study was to highlight the present risks of feed and therefore milk contamination on smallholder dairy farms in Kenya. By highlighting this highly ignored subject, the authors of this paper hope to create awareness on the need to take appropriate health precautionary measures during feed handling and feeding of dairy animals on these farms so as to minimize chances of feed and food contamination. Thus, minimizing the risk of mycotoxin related poisoning on both livestock and humans. Relevant data and graphic presentations from different sources were adapted to illustrate various aspects of the discussion and to provide the basis for drawing conclusive inferences and recommendations.

Results and Discussion

Mycotoxin causing moulds: Mycotoxins are secondary metabolites of fungal origin that are toxic. The term Mycotoxin literally means poison from a fungi. Among the thousands of species of fungi, only about 100 are known to produce mycotoxins. There are three major genera of fungi that produce mycotoxins: *Aspergilius*, *Fusarium* and *Penicilium*. And, although between 300 and 400 mycotoxins are known, those mycotoxins of most concern, based on their toxicity and occurrence, are aflatoxin, deoxynivalenol (DON or vomitoxin), zearalenone, fumonisin, T-2 toxin, and T-2-like toxins (trichothecenes). Deoxynivalenol (DON), zearalenone, T-2 toxin and fumonisin are all produced by mould of the genus *Fusarium*. Moulds in this genus are found in virtually every cereal crop, especially maize (in the field or in store) and collectively are capable of producing 70 different mycotoxins. Some strains of *Fusarium* may produce as many as 17 mycotoxins simultaneously. Thus *Fusarium* mycotoxins are the most frequently identified group of mycotoxins in grains and feeds. Aflatoxins are one of the most potent toxic substances that occur naturally. These are a group of closely related mycotoxins produced by fungi *Aspergillus flavus, A. parasiticus* and *A. nomis*. The genus *Aspergillus* includes over 185 species. Around 20 species have so far been reported as causative agents of opportunistic infections in man. Among these, *Aspergillus fumigatus* is the most commonly isolated species, followed by *Aspergillus flavus* and *Aspergillus niger*. *Aspergillus clavatus*, *A. nomis, Aspergillus parasiticus*, *Aspergillus glaucus* group, *Aspergillus nidulans, Aspergillus oryzae, Aspergillus terreus, Aspergillus ustus*, and *Aspergillus versicolor* are among the other species less commonly

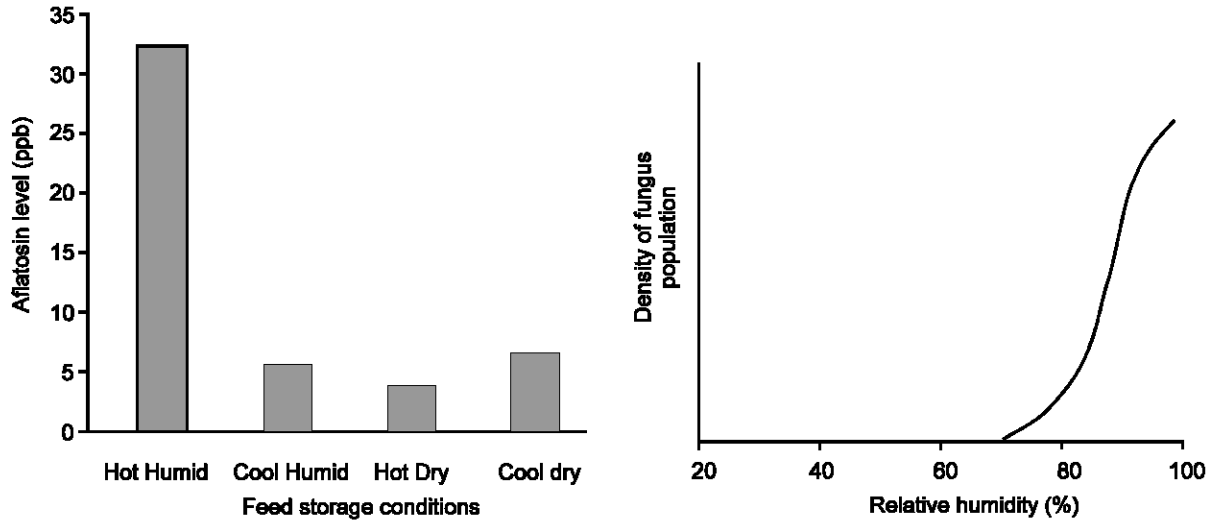
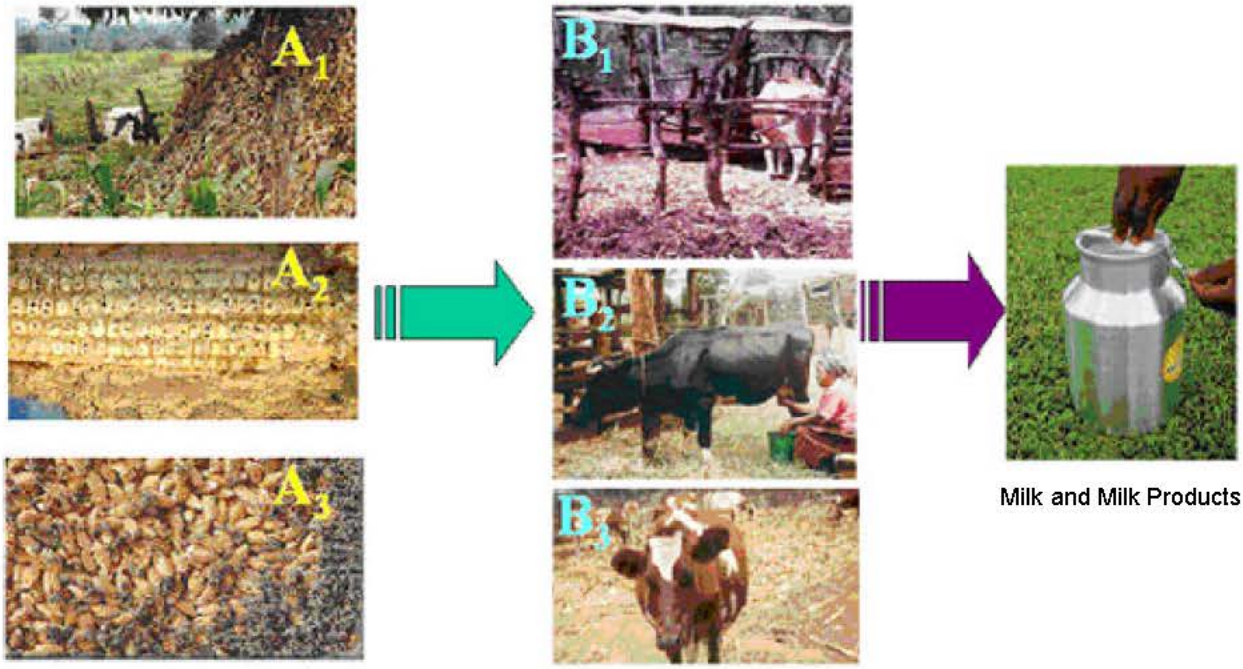


Fig. 1: Influence of temperature and relative humidity on fungal colonization and aflatoxin production in animal feeds (Thomson and Henke, 2000)

isolated as opportunistic pathogens. *Aspergillus* is a group of moulds, which is found worldwide. These fungi are ubiquitous and their potential for contamination of foodstuffs and animal feeds is widespread. Under favourable environmental conditions, some of these toxigenic moulds can produce mycotoxins on a wide range of agricultural commodities. The occurrence and magnitude of any mycotoxin contamination varies with geographical and seasonal factors, and also with the conditions under which a food or feed crop is grown, harvested, and stored. Crops in tropical and subtropical areas are more susceptible to contamination than those in temperate regions, since the high humidity and temperature (Fig. 1) in these areas provide optimal conditions for toxin formation.

Feed and food contamination: Many different mycotoxins have been found to occur on forages and grains either in the field or in storage (Lacey, 1991). Main factors contributing to mould colonization and therefore mycotoxins production include: the feedstuff (quality), moisture level of feed, temperature, relative humidity, pH, plant stresses such as drought or insect infestation, and damaged or broken grain kernels (Jacques, 1988; Ashworth *et al.*, 1969). Weather condition is the primary factor influencing mould colonization (Coulumbe, 1993) (Fig. 1). Moulds grow over a temperature range of 10 - 40°C, pH range of 4-8 and above 70% equilibrium relative humidity. Unlike yeast, which requires free water, moulds are also capable of growing on a dry surface (Lacey, 1991) and on feeds containing 12-13% moisture (Christensen *et al.*, 1977). In wet feeds such as silage, higher moisture levels allow rapid mould growth if oxygen is available. Because most moulds are aerobic, high moisture concentrations that exclude adequate oxygen can prevent mould growth. *Aspergillus flavus* colonization and aflatoxin production in maize and oil seeds are encouraged by high humidity (86-87%) and heat (Davis and Deiner, 1983; Smith, 1997). Drought stress has also been found to increase the number of *Aspergillus* spores in the air (Sorenson *et al.*, 1984). Thus, when drought stress occurs during pollination, the increased population of *Aspergillus* spores in the air greatly increases the chance of infection. Also, nitrogen stress (low soil fertility) and other stresses that affect plant growth during pollination can increase the level of aflatoxin produced by the *Aspergillus* fungi. Mature maize that remains in the field (as dry heaps - a common practice in Kenya) or maize that is stored without properly drying can be susceptible to *Aspergillus* growth and aflatoxin production. Though poorly stored feeds and grains can and indeed does become contaminated with aflatoxin (Lillehoj and Fennell, 1975; Shotwell *et al.*, 1975), past studies have clearly demonstrated that most aflatoxins contamination originates from the field (Smith, 1997). Time of harvest has also been shown to have an influence on aflatoxin levels because *Aspergillus* does not compete well with other molds when maize is above the 20 percent moisture. Thus, harvesting maize with a moisture content of above 20 percent and then drying it down to at least a moisture content of 15 percent, within 24 to 48 hours of harvest, will keep *Aspergillus* growth and toxin production at a minimum. This requirement is hard to achieve on smallholder resource-poor farms in Kenya. Insect (i.e Weevil) and physical damage (running trucks on maize grain as often seen in maize growing belts such as Kitale) can greatly increase *Aspergillus* infection and the levels of aflatoxin.

Protein rich supplements (cotton seed cake, sun flower



A1 - Poorly conserved maize stover (susceptible to moulding); A2 - Heavily pest infested un-shelled maize cob; A3 - Mould colonized wheat grain; B1 - Dump (dairy cow) feeding environment; B2 - Smallholder farmer milking and grass hay on dirty ground; B3 - Dairy cows scavenging on maize stover on dirty yard

Fig. 2: Potential sources of aflatoxin poisoning of dairy cows and pathway for contamination of milk on smallholder farms in Kenya

cake, fish meals and other oil seed by-products), cereal grains and their by-products (maize bran, maize germ, wheat bran and other grain milling by-products) which are often poorly stored, are the primary source of the moulds found in homemade dairy concentrates on smallholder farms. Majority of smallholder farmers in Kenya are not keen on controlling moisture through appropriate storage, which is the single most important factor in determining if and how rapidly moulds will grow in feeds. This is further compounded by the farmers' wide spread practice of using spoilt (physically, pest or mould damaged) grains and other feed resources (mouldy crop residues) to formulate dairy rations (Fig. 2). Today, crop residues constitute the major part of ruminant livestock rations on smallholder farms. However, their efficient utilization is highly militated against by rapid mould colonization, exacerbated by poor handling and storage. Majority of farmers do not protect maize stover against scorching sunshine, termite and other pest damage and more importantly mould colonization. This leads to both chronic aflatoxin poisoning of dairy animals and heavy wastage of this very important feed resource. To control mould growth, obvious sources of moisture in the feed handling and storage must therefore be eliminated. In sound feed management systems the first important step in controlling mould and therefore feed contamination, is to reduce moisture level in hay, cereal and protein supplements used for ration formulation. It is important to note that, although drying of feed has been shown to reduce mould counts many mould spores remain in the feed or feed material after it has been dried. After drying, the remaining spores can grow if conditions are right. In Kenya majority of smallholder dairy herds are kept in either total (zero-grazing) or semi-confinement (semi-zero-grazing) in highly moist environments. Their respiration, defecation, water spillages and rainfall make the environments where these animals are kept highly laden with moisture. This therefore means that, feed that was initially very low in moisture content will rapidly gain moisture when placed into this kind of environment, hence spurring the growth of mould and production of mycotoxin. Under such environment provision of feed troughs will greatly reduce growth of mould and production of mycotoxin on the feed being offered to the animals.

Livestock poisoning: Mycotoxin related poisoning (i.e. Aflatoxicosis) is reported from all parts of world in almost all domestic animals (poultry, cattle, horses, rabbits, and dogs). Aflatoxicosis is poisoning that result from ingestion of aflatoxins in contaminated feed. The

aflatoxin group consists of B1, B2, G1, and G2 (Cotty *et al.*, 1994; Smela and Curier, 2001). Aflatoxin B1 is the most potent of the group and has been shown to be a potent carcinogen (Allcroft and Carnagham, 1963). Like other mycotoxins, aflatoxins occur in a variety of feedstuffs, which are routinely used by farmers to feed dairy animals. In agreement with this assertion, past research studies have confirmed that, most cases of animal poisoning by aflatoxins can be traced to the growth of fungi in poorly handled feeds (Jacques, 1988; Smith, 1997). When ingested through contaminated feed mycotoxins produce a wide range of harmful effects in animals. Though it depends on the affected animal species, the general effects of aflatoxicosis include decreased feed utilization and efficiency (reduced appetite) leading to low weight gain, liver and kidney damage, gastrointestinal dysfunction (hemorrhage, and necrosis throughout the digestive tract), embryonic and early death of the newborn (interferes with conception, ovulation, implantation, fetal development, and the viability of newborn animals), teratogenicity (birth defects), tumors and suppressed immune system function (even when low levels are consumed), reduced productivity (reduced milk production and reproductive efficiency), anaemia, jaundice, carcinogenesis, and death, (Pier, 1992). Depending on interaction with other factors, aflatoxin concentration as low as 100 ppb may be toxic to cattle (Garrett *et al.*, 1968). However, the toxic level is generally considered to be between 300 and 700 ppb. Garrett *et al.* (1968) reported a significant negative effect on weight gain and dry matter intake of cattle offered diets containing 700 ppb aflatoxins. A significant increase in liver weight was also observed during the same study and this was used as an indicator for toxicity. Guthrie (1979) reported a decline in reproductive efficiency when lactation dairy cattle fed on diets containing 120 ppb aflatoxin. When the same cows were offered aflatoxin-free diets, milk production increased by more than 25%. The general indication, as pointed out by many past research reports is that, milk production loss appears to occur when diets contain more than 300 ppb (DON). However, Patterson and Anderson (1982) and Masri *et al.* (1969) suggested that depending on many other factors, aflatoxin concentration of as low as 100 ppb may be detrimental to the dairy stock. Young animals in all species are more sensitive to the effects of aflatoxin than mature animals. Calves may be affected by exposure to aflatoxin metabolites secreted in the milk. Pregnant and growing animals are less susceptible than young animals, but are more sensitive to aflatoxin than mature non-pregnant animals. Even when fed at non-lethal levels, aflatoxin will reduce productive efficiency and may increase the disease susceptibility of the animals. Because of partial degradation in rumen, mycotoxins are less toxic to cattle than most other farm animals. However, mycotoxins are not completely degraded and some of the degradation products remain toxic (Kiessling *et al.*, 1984). The extent of ruminal degradation appears to be variable. It is speculated that feeds and/or feeding situations resulting in a faster rate of ruminal feed passage or a low population of protozoa in the rumen may reduce mycotoxin degradation in the rumen (Kiessling *et al.*, 1984). Ruminal degradation of mycotoxins appears to be more dependent on protozoan than bacterial activity (Kiessling *et al.*, 1984). Acute toxicity is caused when large doses of aflatoxins are ingested through heavily contaminated feed. Though it has been reported that mouldy feed is unpalatable, totally confined dairy animals on smallholder farms in Kenya have little chance of selecting feed. Farmers offer what is available and the question of whether the feed is mouldy or not, is less considered. This is particularly critical during dry season when feeds are scarce. Rather than acute type aflatoxin toxicity, chronic type posses more serious challenge on these farms. Chronic toxicity occurs through long-term exposure of dairy (and other farm) animals to low - moderate aflatoxin concentrations which impacts negatively on herd productivity and posses serious risks to humans that depend on it for milk (Robens and Richard, 1992; Reeds and Kasali, 1987). The economic impact of chronically reduced animal productivity, increased incidences of morbidity due to immunosuppression, damage to vital organs, interferences with reproductive capacity and the ever present source of milk contaminant, is many times greater than the economic impact caused by an animal death due to acute poisoning. For this reason therefore, preventive detection in the feed is essential.

Human poisoning: Outbreaks of mycotoxin related human poisoning is a worldwide phenomenon. Though the problem of mycotoxin contamination of animal products destined for humans is not as frequent as observed with grains, the risk is still real and of major concern. While remaining hopeful that many of our healthy ruminant livestock are capable of "filtering out" or detoxifying many of the mycotoxins to which they are exposed during feeding, the issue of mycotoxin residues in milk, milk products and meat should not be ignored. Of the many reported mycotoxins, aflatoxins are the most potent toxic fungal substabstance that occurs naturally. These toxins have continued to wreck havoc across the tropical world, especially where cereal grains form the basal human diet. Human exposure to levels of aflatoxins from nanograms to micrograms per day occurs through consumption of maize, peanuts and other contaminated agricultural foodstuffs. Maize is the staple food in Kenya. It is milled into flour to make delicacies referred to locally as “Ugali” and “Uji” (a porridge). During periods of food scarcity the government of Kenya imports maize grains to bridge the shortfall. It is the contamination of these foodstuffs that

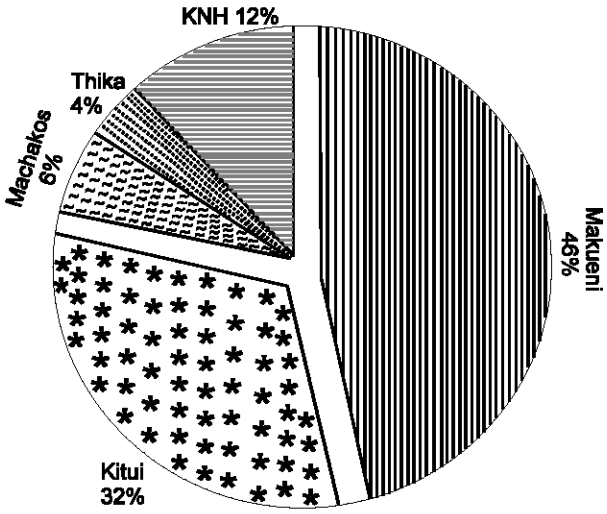


Fig. 3: Distribution of aflatoxcosis cases in five recording site and according to date of reporting in Eastern and Central provinces in Kenya (January - July, KMOH, 2004)

has been incriminated for both acute (Fig. 3) and chronic aflatoxin poisoning in Kenya. The chronic incidence of aflatoxin in diets is evident from the presence of aflatoxin M1 in human breast milk in Ghana, Kenya, Nigeria, Sierra Leone, Sudan, Thailand, and the United Arab Emirates, and in umbilical cord blood samples in Ghana, Kenya, Nigeria, and Sierra Leone (Bhat and Vasanthi, 2003; Maxwell *et al.*, 1998). Frequent consumption of low levels of aflatoxin has also been associated with chronic diseases like cancer. If animals consume contaminated feeds (sometimes containing high levels of aflatoxin up to 8,000 ppb) and then these toxins find their way from feed into milk or meat, they become a food safety hazard in these products too (Bhat and Vasanthi, 2003). This often happens, especially where animals exposed to aflatoxin contaminated feeds such as observed on many smallholder resource-poor farms in Kenya (Fig. 2). One structural type of aflatoxins - aflatoxin B1 is considered the most toxic and the type commonly found type in Kenya.

Aflatoxin M1 is metabolic breakdown product of aflatoxin B1 and can appear in the milk of lactating cows consuming significant quantities of aflatoxin B1 (Fig. 2). Aflatoxin M1 is not as carcinogenic as B1 but can be as toxic. The conversion of aflatoxin B1 in feed to aflatoxin M1 in milk is about 1% to 2%. Aflatoxin M1 residues in milk is reportedly approximately equal to 1 to 2 percent (1.7 percent average) of the dietary level. This ratio is not influenced greatly by milk production level since higher producing cows consume more feed and have a slightly higher transmission rate. Due to risks of milk residues, dietary aflatoxin should be kept below 25 ppb.

In May 2004, the Kenya Ministry of Health (KMOH, 2004) and key stakeholders undertook an investigation to establish the cause of an outbreak of jaundice with a high case-fatality rate (CFR) in the districts of Makueni and Kitui, Eastern Province. Laboratory testing of food collected from the affected area revealed high levels of aflatoxin, suggesting that, the outbreak was due to aflatoxin poisoning. Evidence that this outbreak resulted from aflatoxin poisoning included 1) high levels of aflatoxin (up to 8,000 ppb) in maize samples collected from patient households, 2) a clinical illness consistent with acute aflatoxin poisoning, 3) clustering of cases among residents of the same household, and 4) reports of deaths among animals known to have eaten the same maize as the patients during the same period. Food samples collected from household visits during May 10-19 included maize flour, maize grains, dry maize cobs, muthokoi (i.e., maize in which the outer hulls have been removed), millet, sorghum, and beans. During this investigation, a total of 342 samples were submitted for testing at the Kenya National Public Health Laboratory Services. The results revealed varied aflatoxin B1 levels (range: 20 to 8,000 ppb). Out of the 342 samples tested, a total of 182 (53.2%) had >20 ppb of aflatoxin. In addition, a substantial percentage of samples from each district had aflatoxin levels >1,000 ppb: Makueni (12.1%), Kitui (9.6%), Thika (3.9%), and Machakos (2.9%).

An outbreak of acute aflatoxicosis (20 cases; CFR = 60%) was reported previously in Makueni district, Eastern Province, Kenya, in 1981 (Ngindu, 1982). During the Kenya ministry of health study in Eastern province in 2004, patients were clustered in family groups that shared meals consisting of aflatoxin-contaminated maize (1,600-12,000 ppb). Acute hepatitis associated with consumption of moldy grains also has been reported in other areas in Africa, Western India, and Malaysia (Krishnamachari *et al.*, 1975a, b; Lye *et al.*, 1995), where most affected persons came from areas prone to drought and malnutrition. Unpredictable change in rainfall pattern sometimes causes forced harvest of grains before adequate drying, leading to heavy mould colonization and mycotoxin production. Typically, increased reports of jaundice and hepatitis followed within weeks of such harvests (Krishnamachari *et al.*, 1975a; b; Lye *et al.*, 1995). Ingestion of 2-6 mg/day of aflatoxin for a month can cause acute hepatitis and death (Patten, 1981; Krishnamachari *et al.*, 1975a, b). Locally produced maize associated with this outbreak was harvested in February during peak rains, and the first illnesses were reported in Makueni district in late March and early April. As of 20th July, a total of 317 cases had been reported, with 125 deaths (CFR = 39%). An increase in case reports began in the third week of April, with new cases continuing to occur through mid-July. It is worth pointing out that, for every symptomatic case of aflatoxicosis identified in affected areas in Kenya, several other additional persons were likely to

have been exposed to unsafe levels of aflatoxin through consumption of contaminated grains or animal products and thus, might face future adverse health consequences. It is not only by consumption of contaminated food that humans get poisoned. It has also been reported that, handling mouldy feeds (hay, stover, silage and others) may be harmful due to the presence of mycotoxins and actinomycetes which are responsible not only for poisoning but also for the allergic disease affecting man known as 'farmer's lung'. This is particularly serious in asthmatics and patients suffering from cystic fibrosis. There is no cure for aflatoxin B1 poisoning and it damages vital organs in the body including the liver, kidneys and lungs. Symptoms include yellow eyes, swollen legs, vomiting and bleeding.

Conclusion: It is uncontestable fact that mycotoxins pose a serious health risk to both livestock and human beings world-over, particularly in the tropics where climatic conditions (high moisture and temperature) spur the growth of moulds. Other than the direct health risk, economic losses arising from mycotoxicoses are equally enormous. Controlling mould growth and mycotoxin production is therefore very important. Solutions to mycotoxin menace can be based only on application of recommended preventive measures and research focusing on detoxification/decontamination of mycotoxin-contaminated foods and feeds. The corner stone to a successful mycotoxin reduction in feeds is control of moisture and temperature, which in turn has a direct bearing on the extent of mould colonization. Thus, control of mould growth in feeds can be accomplished by keeping moisture low, temperature moderately low, keeping feed fresh, keeping equipments used on-farm clean, and where possible using mould inhibitors. First step in mould control is to ensuring that the food (grains) or feed (crop residues, hay and agro-industrial processing by-products) is dried adequately. The dried grains and feeds (including poultry manure) should then be stored at a well-aerated barn and at low moisture level (14 percent or less) to discourage mould growth. In silages (forage, brewers waste) mycotoxins can be prevented by following accepted ensiling practices aimed at inhibiting quality deterioration primarily through elimination of oxygen. Some silage additives (such as ammonia, propionic acid, microbial cultures, or enzymatic silage) may be beneficial in preventing mycotoxins because they are effective at reducing mould growth. Silage silo size should be matched to herd size to ensure daily removal of silage at a rate faster than deterioration. Feed troughs and barns should be cleaned regularly to prevent contamination of fresh feed. At research level, deliberate efforts should be made to prevent mycotoxin contamination before harvest through crop and forage breeding research. Longer-term solutions would include strengthening nationwide surveillance, increased food and feed inspections to ensure food safety, and local education and assistance to ensure that food grains and animal feeds are harvested correctly, dried completely, and stored properly. Agricultural and public health frontline extension staffs need to demonstrate improved methods of feed utilization to resource-poor farm households and educate them against poor practices that encourage mycotoxin contamination of food and feeds on-farm.

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