DEATHS FROM CONSUMPTION OF CONTAMINATED MAIZE IN EASTERN PROVINCE, KENYA.

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Abstract.

In Kenya's Eastern province, the consumption of the dietary staple maize meal – ugali- presents a significant risk for premature deaths. The aridity of the region provides ideal conditions for the fungi, aspergillus flavus to thrive. This fungus produces the aflatoxin poison, which contaminates 25% of food crops worldwide. However, the most extreme exposures globally have been documented in Kenya's Eastern province resulting in over 600 deaths. A comparative analysis was done on peer-reviewed literature to identify risk factors linked to increased human exposure to aflatoxins, disease outcomes and possible intervention strategies to mitigate exposure. In Kenya, locally produced maize has aflatoxin levels that exceed government recommended level of 20 parts per billion [ppb] by thousands. The main target organ of aflatoxin is the liver, and thus jaundice, sudden liver failure and other liver related diseases are reportedly the main cause of deaths following aflatoxin exposure. Cost effective intervention strategies include cultivating drought resistant maize strains, proper drying and storage practices and promotion of diversified dietary options have been proposed.

Keywords: Maize, Aflatoxins, Aflatoxicosis.

Introduction

In the tropical and subtropical regions of the world, hot and humid conditions provide ideal environments for a group of fungus known as aspergillus to thrive. While there are many different species of the aspergillus fungi, the strains *aspergillus flavus* and *aspergillus parasiticus* are known to produce aflatoxins. Aflatoxins are naturally occurring and are recognized food poisons prevalent in maize, peanuts, cassava, spices and other nuts. These food products form the basis of diet staples in many low and middle-income countries. Over 4.5 billion people are exposed to aflatoxin contamination through their diets. In addition, due to increased global trading, aflatoxins have also been found in imported food products in both the United States and the European Union.

Aflatoxins are intensely fluorescent to ultraviolet light and thus categorization depends on color emitted and level of toxicity. For instance, Aflatoxin B1 [AFB1] and Aflatoxin B2 [AFB2] emit blue light while Aflatoxin G1 [AFG1] and Aflatoxin G2 [AFG2] emit yellow-green fluorescence under ultraviolet light. There are other groupings of aflatoxins but most of them are degradation products of the above four major categories. AFB1 is the most toxic form of all the aflatoxins. AFB1 is an established carcinogen, potent immune-toxicant and anti-nutritional agent (IARC, 1993, 2004; Williams et al., 2004). Human exposure to aflatoxins is prevalent and can occur to unborn fetus in utero, via breast milk, during weaning and consistently throughout an individual's lifetime (Y. Y. Gong et al., 2003; P. C. Turner et al., 2007).

Short term exposures to extremely high doses of aflatoxins in the diet results in aflatoxicosis – a disease characterized by jaundice, hemorrhage, acute liver damage, edema and sometimes death. On the other hand, consistent long-term exposure to low quantities of aflatoxins in the diet contributes to development of primary liver cancer (de Oliveira & Germano, 1997; Ledda et al., 2017; Wang et al., 2001). Since aflatoxins are naturally occurring, it is not possible to eliminate exposure completely. Subsequently, regulatory limits set in different countries range from 4 – 30ppb for humans and up to 300 ppb in animal feeds. Globally, aflatoxicosis incidences are prevalent in Kenya's Makueni County and other counties in close proximity. Over 600 deaths have been documented from recurrent aflatoxins outbreaks over the years in Kenya.

The Study

In this study, an assessment of peer-reviewed literature on Aflatoxins and associated health effects was evaluated with special focus on Kenya where aflatoxin contamination is a recognized public health problem.

Findings

Aflatoxin contamination to both farm animals and people is widely documented in Kenya. The very first documented poisoning was reported in ducklings in a white settler farm in former rift valley province. Over 16,000 ducklings died after consuming groundnut feed that was contaminated with aflatoxins (Peers & Linsell, 1973). In 1977, dogs and poultry died in large numbers in Nairobi, Mombasa and Eldoret after they were fed with aflatoxin-contaminated grains (FAO/WHO/UNEP, 1977). Reported deaths of farm animals have long been recognized as a signal for high aflatoxin levels in animal feed.

In 1981, the first occurrence of human deaths from consumption of aflatoxin-contaminated maize was reported in Machakos. Preceding human deaths, numerous deaths had been reported for dogs, poultry and other farm animals. Shortly afterwards, 20 people fell ill and visited health facilities for symptoms related to acute hepatitis. The patients were admitted at the hospital and treated conservatively. While 8 patients recovered, 12 of the patients developed hepatic failure and died between 1 to 12 days after admission at the hospitals (Ngindu et al., 1982).

In 1987, up to 3 people in Meru North suffered from acute effects related to consumption of aflatoxin-contaminated maize and eventually died (Autrup, Seremet, Wakhisi, & Wasunna, 1987). Between 1988 and 2000, there is limited documentation of aflatoxin related outbreaks in Kenya, however, intermittent exposure to aflatoxins in low quantities over a long period of time increases the risk of developing hepatitis and other liver related diseases. In addition, many cases go unreported, as many villagers may choose not go to the hospital. During the 1981 aflatoxicosis outbreak, the reported meantime from onset of aflatoxin related symptoms to hospital admissions was about 10.2days (Ngindu et al., 1982). Since published literature captures affected individuals who visit health care facility, there is a possibility that the burden of aflatoxicosis is underestimated.

Moreover, 2001, 16 deaths were reported in Maua, Meru County after consumption of moldy maize. In 2002, large numbers of dogs and poultry also succumbed to death in Coast after consumption of contaminated feed (Probst, Njapau, & Cotty, 2007). The worst and most reported outbreak of aflatoxicosis happened in 2004 in Makueni, Kitui, Machakos and Thika Counties of Kenya. About 317 patients were admitted in hospitals after display of symptoms such as abdominal discomfort, jaundice, and low-grade fever. Kenya's Ministry of Health invited the United States Center for Disease Control and the World Health Organization to probe into the aflatoxicosis outbreak [CDC 2004]. Meanwhile, 125 people died shortly after hospital admissions (Lewis et al., 2005). The results revealed that the deaths were attributed to high aflatoxin levels found in maize grains collected from the affected households. Maize samples from affected households had aflatoxin levels of up to 8,000 ppb when the recommended levels are 20ppb (Azziz-Baumgartner et al., 2005; Daniel et al., 2011).

Numerous studies have been completed after the 2004/2005-aflatoxicosis outbreak in Kenya. Makueni is the most affected region due to significant plant stress from drought conditions, which leaves crops vulnerable to aflatoxin contamination. In addition, food insecurity promotes storage of maize in hot humid households, thus providing ideal conditions for the aflatoxin producing *aspergillus* fungi to thrive. The government of Kenya has in the past provided replacement maize to affected household [CDC 2004]. Currently, the county government is tasked with similar efforts. For instance, in July 2017, the county government of Makueni delivered 10,600 bags of maize consignments to educational institutions in response to drought conditions. The county government ensured that maize was tested for safe levels of aflatoxins. Nevertheless, long term solutions that will ensure food safety and total eradication of aflatoxin contamination of maize in Makueni and other counties must be initiated.

Discussion and Conclusion

In Kenya, aflatoxin contamination is a recognized public health problem that can occur when the crops are still in the fields or after harvesting. The much-needed interventions to mitigate widespread exposure can therefore be initiated pre-harvest or post-harvest (Khlangwiset & Wu, 2010). Pre-harvest strategies that have been implemented include field inoculation with non-toxic aflatoxigenic fungi, use of pesticide and irrigation to minimize plant stress and vulnerability to infestation by the aflatoxin producing fungi. During post-harvest, satisfactory drying of maize to less than 10% moisture content is ideal followed by storage in conditions that allow adequate airflow.

Other strategies include dilution where highly contaminated grain is mixed with grain of low contamination to avoid economic losses that may ensue from destroying the maize. In Mexico, decontamination is achieved through nixtamalization, where maize is soaked and cooked in limewater before food products are made. The process is very efficient and results in over 90% reduction of aflatoxin in maize. There are other efforts targeted towards affected humans such as chemoprevention and use of drug therapy (Kensler et al., 1998). Chemoprevention or drug components used either increase detoxification processes within the body or prevent the formation of toxic biological metabolites that promote disease outcomes. These processes are expensive and could result in long-term side effects. Moreover, use of entero-sorbents such as novasil clay has been proposed as a short-term intervention during aflatoxicosis outbreaks (Afriyie-Gyawu et al., 2008; Williams et al., 2004).Novasil clay binds aflatoxin in the human body and thus facilitates elimination through fecal matter.

While there is robust evidence on the contributory role of aflatoxins to primary liver cancer (Wang et al., 2001; Wogan, Kensler, & Groopman, 2012), recent epidemiology studies have shown that dietary exposure to aflatoxins also contributes to micronutrient deficiency(Y. Gong et al., 2004; Watson, Gong, & Routledge, 2017), immune suppression (Paul C. Turner, Moore, Hall, Prentice, & Wild, 2003) and growth impairment in children (Y. Gong et al., 2004). Due to these recognized adverse health effects associated with aflatoxin contamination, further studies should be done to provide an in depth understanding of the mechanism of action of aflatoxins in the human body.

Successful eradication of aflatoxin contamination is possible when multiple key players in Agriculture, Public Health and Community members work together. Successful intervention has been reported in Qidong, China. Historically, Qidong region was characterized by high prevalence of primary liver cancer. Before 1980, locally produced maize, which was the dietary staple in Qidong, was characterized by high levels of aflatoxins. In 1980s however, agricultural and food policy reforms were extensively implemented in Qidong's region resulting in a shift from maize to rice production. Normally, rice is more resistant to aflatoxin contamination and gradually, Qidong's population adapted rice as the dietary staple. Progressively, aflatoxin contamination decreased from 100% to 23% and up to 65% reduction of mortality from liver cancers was reported in Qidong's Cancer Registry (Chen et al., 2013).

In conclusion, similar cost effective interventions can be promoted in Kenya and other countries' that are over reliant on maize as a dietary staple. Increased campaigns to shift from maize-based dietary staple to consumption of food products that are less vulnerable to aflatoxin contamination such as bananas, potatoes, rice, sorghum or millet based ugali are local solutions that can be easily implemented to mitigate long term health effects associated with consumption of aflatoxin contaminated maize.

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