

Epidemiology and pathological consequences of mycotoxicoses in Africa: a minireview

Adekola Hafeez Aderinsayo¹, Idris Abdullahi Nasir², Fatima Muhammad Sani³

¹. Department of Medical Microbiology, College of Health Sciences, University of Ilorin, Kwara State, Nigeria

². Department of Medical Microbiology, University of Abuja Teaching Hospital, PMB 228, Gwagwalada, FCT Abuja, Nigeria

³. Department of Medical Laboratory Science, College of Medical Sciences, University of Maiduguri, PMB 1069, Borno State, Nigeria

Correspondence address: eedris888@yahoo.com

Abstract: Mycotoxicosis causes range of disease conditions which result from intoxication of the body system by mycotoxins produced by certain micro-fungi genera especially in foods and grains. Intoxication depends on the route of mycotoxin entry and type of mycotoxins. The clinical manifestations range from mild systemic dysfunctions to chronic pathologies with high propensity to induce tissue/ organ damages, cancers and even death. The occurrence of mycotoxin in food-stuffs has been considered a serious public health threat. In recent time, there has been paucity of information in regards to the detection/ diagnosis of mycotoxicosis in African. In view of this we conducted this review of relevant published articles using extensive literature search made through PubMed and Scopus on the biology, pathology and epidemiology of mycotoxicoses in Africans. Findings from this study showed that there have been under-diagnosis of mycotoxicosis in Africa and this had led to poor understanding of its epidemiology. Due to the fact that there is no single effective antitoxins for humans who suffer from mycotoxicoses, prompt and appropriate intervention strategies provide effective way of minimising and curbing the scotch; mycotoxicoses.

[Adekola Hafeez Aderinsayo, Idris Abdullahi Nasir, Fatima Muhammad Sani. **Epidemiology and pathological consequences of mycotoxicoses in Africa: a minireview.** *N Y Sci J* 2015;8(11):14-18]. (ISSN: 1554-0200). <http://www.sciencepub.net/newyork>. 3

Keywords: Intervention strategies; Mycotoxins; Micro-fungi

1.0 Introduction

The term “mycotoxin” describes a low molecular weight toxic substances which are chemically diverse and are produced mainly by micro-fungi belonging mainly to the *Aspergillus*, *Fusarium* and *Penicillium* genera, which ranges from about 300 to 400 compounds, toxins produced by macro-fungi such as mushrooms are not included in this group (Bennett and Klich, 2003). Mycotoxins can pile up in foods and grains such as maize, cereals, soybeans, sorghum, peanuts etc., on the field, during transportation and during improper storage (Bennett and Klich, 2003)

Most micro-fungi are facultative anaerobes and are ubiquitous. In favourable environment where humidity, temperature and organic matter are sufficient, micro-fungi develop into colonies and thus mycotoxin production begins (Fox and Howlett, 2008). Mycotoxins are synthesized primarily in the hyphae and remain there to be integrated in to the conidia during conidiogenesis, or released directly into the environment (Fox and Howlett, 2008). The logical explanation for production of mycotoxins is still being sought for because they are not required for either fungal growth or its development, but since it weakens receiving host, it is assumed that they are used for ecological purposes for survival in order to increase fungal proliferation by reducing competition

for nutrients and living space by other micro-organisms (Hussein and Brasel, 2001). Mycotoxin production depends on the fungal internal and external environment, this toxins varies in their severity depending on the virulence of the organism and the susceptibility of the host (Fox and Howlett, 2008). In view of these, mycotoxicoses are major public health problems. Identifiable diseases and death without diagnosis to a particular toxin are very common all over the world (Keller et al., 2005). Thus, this study sought to summarize the epidemiology and possible disease conditions caused mycotoxicoses especially in African nations.

2.0 Review and Discussion

2.1 Mycotoxicoses

Mycotoxicoses can be defined as the disease condition that arises as a result of mycotoxin intoxication. The disease condition ranges from loss of appetite, food refusal to cancer and mortality in humans (and livestock) (Idahor, 2003). The adverse effect of these toxic compounds and their frequency in the environment plays major role in the exposure of mycotoxin and their resulting effects on humans. Mycotoxins has been implicated in one of the ten plagues of Egypt (Idahor, 2003).

There are three routes in which humans and livestock are exposed to mycotoxins. Firstly, through consumption of contaminated food and grains, this is the most common route. Secondly, through absorption into the tissue following skin contact and then lastly through inhalation of spores containing mycotoxin present in the air (Akila, 2004).

Mycotoxin can be present in foods like cereals and grains as a result of fungal infection of the mother plant which might be either eaten by human directly or by livestock as livestock feed. Mycotoxins do not decompose easily or digest easily, so they remain in food and even some of them can withstand cooking and freezing temperature (Bennett and Klich, 2003). In most underdeveloped countries who rely on single grain or cereal source, mycotoxin contamination can be a worrying issue, instances of mycotoxicoses due to aflatoxin, ochratoxin and fumonisins consumption are common in these communities (Akila, 2004).

Mycotoxins can be absorbed on contact with the skin or mucous membranes, causing necrosis and systemic effects affecting the gastrointestinal tract or the hematopoietic system, presently tricothecenes possess this property (Annaissie et al., 2009). Spores of mycotoxin producing fungi can be inhaled from contaminated environment when dust is dispersed. Buildings are another source of mycotoxins and people living or working in areas with mycotoxins producing fungi gradually becomes susceptible to adverse health conditions. Micro-fungi colonizing buildings can be grouped into three; primary, secondary, and tertiary colonizers, based on the ability to grow at a certain water activity requirement. Identification of mycotoxin production by these building colonizers is difficult due to different factors, such as being masked as derivatives, production of different metabolites on the building materials. The genera *Aspergillus*, *Penicillium* and *Stachybotrys* produce most of the mycotoxins in the indoor environment (Fog, 2003). The duration of exposure, concentration, and susceptibility of occupants play major role in triggering ill-health (Fog, 2003).

2.2 Incidences of Mycotoxicoses

Several studies have been carried out on natural occurrence of mycotoxin in maize products, maize-based foods and other foods. The results of the survey showed that 60% of the 5211 samples studied were contaminated with mycotoxins. Oceania had the highest incidence of mycotoxin contamination (82% of 82 samples), followed by Africa (77% of 383 samples), Latin America (85% of 266 samples), North America (63% of 1662 samples), Europe (53% of 1918 samples) and Asia (52% of 900 samples). Maize foods had the highest record of mycotoxin contamination (82% of 1112 samples), followed by ground maize products like flour, grits, polenta,

semolina and gluten (73% of 517 samples), maize kernels (52% of 2525 samples) and miscellaneous maize foods (40% of 892 samples) (Idahor, 2003). There were also mycotoxin contamination in other foods such as rice, asparagus, beer, and sorghum, but there were no mycotoxin contamination in foods such as wheat, rye, barley and oat (Abbas et al., 1998; Logrieco et al., 1998; Torres et al., 1998; Bhat et al., 1997; Meister et al., 1996).

There have been a documented report on both suspected and reported incidences of mycotoxicoses in both humans where maize and groundnuts serve as the major vehicle of mycotoxin transmission in South Africa (Merrill et al., 2001). This study also provided correlation between rates developing oesophageal cancer and occurrence of mycotoxins in Transkei, South Africa (Merrill et al., 2001).

In another finding, the black population had higher rate of contracting mycotoxicosis and this was higher in males than females in Butterworth and Kentani Districts when compared to Bizana and Lusikisiki Districts (IARC, 1993). These rates could be associated to the dependence of both districts on locally grown maize that constitutes about 50 and 100% yearly supply of maize for beer or porridge. The adults deliberately drink beer produced from discarded moldy maize, this particular type of maize has been shown to contain up to 118mg/kg mycotoxins at climax in a 30mg/litre of beer made from the wort of discarded moldy maize (Scott et al., 1995). It was common in males because they more frequently engage in sociocultural practices and lifestyles that predisposed them to contracting mycotoxins. Synergic mycotoxic activities have also been shown to exist in fumonisins, deoxynivalenol and zearalenone. *F. graminearum* and *F. verticillioides* were found in some cases of locally grown maize for human consumption. A case study recently conducted showed that deoxynivalenol and zearalenone levels were significantly higher in the locally grown maize (Sydenham et al., 1990). According to a cancer registry data in transkei, South Africa consistently had high rates of oesophageal cancer (Makaula et al., 1996).

In Nigeria, high rates of aflatoxin exposure has been recorded in humans. It has been estimated that increase number of cases of hepatocellular carcinoma (liver cancer) has been attributed to aflatoxins (McDonald et al., 1965) According to screening studies initiated by the Federal Government of Nigeria, it was revealed that aflatoxin was found in groundnut samples from Zaria, Kano and Mokwa (McDonald et al., 1965) also from palm wine samples at Ibadan city of Nigeria. It also revealed that various moldy food were offered for sale in Ibadan markets (Bassir and Adekunle, 1969).

2.3 Frequently encountered mycotoxins

Some mycotoxins are frequently encountered than others in cases of mycotoxicoses. Some frequently encountered mycotoxins as provided by Bennett (2003), Idahor (2003) and Merrill (2001) include the following:

a. Aflatoxins: They are metabolites of *Aspergillus flavus* and *Aspergillus parasiticus*, they are hepatocarcinogenic and genotoxic

b. Fumonisin: They are metabolites of *Fusarium verticillioides* and *Fusarium proliferatum*, they are carcinogenic, hepatotoxic and immunotoxic

c. Zearalenone: They are metabolites of *Fusarium graminearum* and *Fusarium culmorum*, they cause hyperestrogenicity and induce reproductive disorders

d. Trichothecenes: They are metabolites of several *Fusarium*, *Stachybotrys* and *Cephalosporium* species, they are dermatotoxic and immunotoxic. They also cause several gastrointestinal disorders and headaches

e. Ochratoxins: They are metabolites of *Aspergillus ochraceus* and *Penicillium verrucosum*. They are carcinogenic and nephrotoxic

f. Citrinin: They are metabolites of *Penicillium* and *Aspergillus* species, they are nephrotoxic and cause renal damage, vasodilatation and bronchial constriction

g. Gliotoxin: They are metabolites of *Alternaria*, *Penicillium* and *Aspergillus* species. They are immunotoxic

h. Patulin: Mainly metabolites of *Penicillium* and *Aspergillus* species. They cause brain and lung hemorrhage

i. Sterigmatocystin: Metabolites of *Aspergillus versicolor*, they are hepatotoxic, nephrotoxic and carcinogenic

2.4 Regulation Limits of mycotoxin in food (as prescribed by worldwide regulations for mycotoxin, 2003)

a. Aflatoxins: the minimal amount of total aflatoxins in food depends on the country regulations, however the median limit is 10µg/kg. The most often occurring limit provided by 29 European countries is 4µg/kg, but a limit of 20µg/kg is also employed by 17 other countries which included the Latin American and African countries.

b. Fumonisin: Fumonisin were subjected to regulations in six countries for maize grains which ranged from 1000µg/kg to 3000µg/kg.

c. Zearalenone: regulations of this mycotoxins are carried out in 16 countries, the limits in maize and other cereals vary between 50 to 1000µg/kg.

d. Trichothecenes: the most important of this group of mycotoxins, Deoxynivalenol, has been

regulated by 37 countries, the limit ranged between 300 and 2000µg/kg.

e. Ochratoxin: the most important of this group of mycotoxins, Ochratoxin A. It has been regulated by a number of countries in cereals, the major source. The limit ranged between 3 to 50µg/kg among 37 countries which peaks.

f. Patulin: regulation of patulin in fruits and fruit juices was carried out by 48 countries have which ranges between 5 to 100µg/kg but the vast majority including 44 countries sets its limits at 50µg/kg.

2.5 Clinical symptoms and diagnosis

Many factors determines the appearance of symptoms of mycotoxicoses as provided by Idahor (2004). These may include the following:

a. Level of contamination of mycotoxin in food

b. Length of exposure to mycotoxin

c. Type of mycotoxin causing diseases

d. Degree of combination with other mycotoxins

Other factors such as individual differences, species-specific resistance, gender, pre-existing pathological and physiological status of the victim also play role in symptoms manifestation. Mycotoxicoses can occur as either an acute or a chronic disease condition. Symptoms of acute mycotoxicoses appear within seven days prior to contamination and inappropriate intervention strategies are not instituted, victim may die as a result of complications. In chronic mycotoxicoses, the symptoms appear for a long period and victim may live longer with an illness caused by the disease (Idahor, 2004).

Symptoms of mycotoxicoses could manifest in form general body weakness, cyanosis, oedema, weight loss, heart hypertrophy, immunosuppression, abortion, lethargy, dyspnea, abdominal pains, organ/tissue (mainly brain, kidney, liver) haemorrhage, convulsion, fibrosis, hyperplasia and sudden death (Idahor, 2004).

Mycotoxicoses can be diagnosed through mycological cultural and toxin identification, hematological, biochemical, histochemical analyses, urinalysis, radiography and autops (Idahor, 2004). It depends on the body system presenting with clinical symptoms of mycotoxicosis.

One of the most challenging aspects during the diagnosis of mycotoxicoses is collecting appropriate samples and technical expertise of laboratory scientists. This should represent adequately the amount of food being contaminated by mycotoxins, because mycotoxins are not evenly distributed in foods, so the most contaminated part might have been ingested before symptoms occur thus making collection of meaningful sample very difficult (Idahor, 2004).

2.6 Treatment

Treatment of mycotoxicoses in diseased human or animal is often supportive and usually not very effective. Antitoxins for mycotoxins are generally unavailable, so the most effective treatment is by using the possible intervention strategies (Idahor, 2004).

2.7 Possible intervention Strategies of Mycotoxicoses

The possible intervention strategies can be divided in to two categories:

1. Prevention strategy: which involves stopping and preventing further exposure to mycotoxin producing fungi.

2. Detoxification strategy: which involves elimination of mycotoxins from the body system by inactivation of mycotoxin, thus reducing the level of mycotoxins in the body system and restoring the body to a healthy state.

2.7.1 Prevention strategy

The prevention strategy involves following certain procedures that will prevent further exposure to mycotoxins and mycotoxin producing fungi. The procedures include:

i. Foods or grains free of mycotoxins should be purchased.

ii. Foods or grains suspected to be contaminated with mycotoxins should be discarded.

iii. Use of mycotoxin binders should be employed to reduce level of contamination.

iv. Insect-infested and drought-stressed grains should be harvested at early maturity as soon as the moisture content allows minimum grain damage.

v. Combine header speed should be adjusted to reduce cracking of grains for example kernels, the content of trash, fines, and small broken and mold-infected grains should be reduced.

vi. The grains should be dried to at least 13- to 14 percent moisture content as quickly as possible and should not exceed 2 days after harvest to avoid aflatoxin production.

vii. The grains should be cooled after drying then dry storage conditions should be maintained.

viii. The grains should be thoroughly screened and cleaned to remove dirt, dust, and other foreign matter, crop debris, chaff, cracked or broken grains.

ix. Grains should be stored in water, insect and rodent tight structures.

x. Periodic aeration should be continued and there should be hot spots probing at intervals of 1 to 4 weeks throughout the period of storage.

xi. The grain can be prepared and stored as high moisture grains or should be treated with propionic acid, but grains treated with propionic acid can only be used as livestock and poultry feeds.

xii. Soil should be irrigated thoroughly during hot dry periods in order to avoid moisture stress during grain filling period.

2.9 Detoxification strategy

The detoxification strategy involves following certain procedures that facilitate the elimination of mycotoxins from infested body system. These procedures include:

a. Cell membrane flush or rescue should be employed to stop mycotoxin damage to cells.

b. Healthy cells and membranes can be restored by removal of mycotoxins from polar and non-polar regions of the body.

c. Consumption of vegetables and green supplements should be employed to repair damaged cells in the gut.

d. Use of air filters should be employed because it largely aid the reduction of airborne molds and mycotoxins intake.

e. A yeast free diet should be employed to stop ingestion of more molds and mycotoxins.

3.0 Conclusion and Recommendations

There have been underreporting of mycotoxicosis and as such led to poor understanding of its epidemiology. Sadly, there is no viable will for developing effective antitoxins for human who suffer from mycotoxicoses. The quality of evidence in this minireview can lead to the following recommendations:

a. The possible intervention strategies should be employed in order to reduce the level of mycotoxins present in food and grains for human and livestock consumption thus reducing the rate of mycotoxicoses among human and livestock.

b. Regulation limits of mycotoxins should be strictly adhered to reduce the spread of mycotoxicoses in human and livestock

c. Infected humans should employ detoxification strategy for fast recovery from mycotoxicoses.

Corresponding author:

Idris Abdullahi Nasir

Department of Medical Microbiology

University of Abuja Teaching Hospital

PMB 228, Gwagwalada, FCT Abuja, Nigeria

Email: eedris888@yahoo.com

Phone number: +2348030522324

References

1. Abbas HK, Cartwright RD, Shier WT, Abouzied MM, Bird CB, Rice LG et al. Natural Occurrence of fumonisins in rice with Fusarium sheat rot disease. *Plant Dis.* 1998; 82:22-25.

2. Akila W. Biological warfare. Department of Chemistry. University of California, USA. 2004
3. Annaissie EJ, McGinnis MR, Pfaller MA. *Clinical Mycology*. 2nd ed. 2009; 635
4. Bassir O, Adekunle A. Comparative toxicities of Aflatoxin B1 and palmotoxins Bo and Go. *West African Journal of Biological and Applied Chemistry*. 1969; 12(1):7-19.
5. Bennett JW, Klich M. "Mycotoxins". *Clin Microbiol*. 2003; 16 (3): 497-516.
6. Bhat RV, Shetty PH, Amruth RP, Sudershan RV. A foodborne disease outbreak due to the consumption of moldy sorghum and maize containing fumonisin mycotoxins. *J Clin Toxicol*. 1997; 35:249-255.
7. Fog NK. Mycotoxin production by indoor molds. *Fungal genetics and biology*. 2003; 39 (2): 103-17.
8. Fox EM, Howlett BJ. Secondary metabolism: regulation and role in fungal biology. *Curr Opin Microbiol*. 2008;11 (6): 481-7.
9. Hussein HS, Brasel J.M. Toxicity, metabolism, and impact of mycotoxins on humans and animals. *Toxicol*. 2001; 167 (2): 101-34.
10. IARC. Toxins derived from *Fusarium moniliforme*: Fumonisms B1 and B2 and fusarin C. In: Some naturally occurring substances: Food items and constituents, heterocyclic aromatic amines and mycotoxins. 1993; 56:445-466.
11. Idahor KO. Physiological Implications of Mycotoxicoses in Humans and Livestock: *Plant Dis*. 2003; 6-8.
12. Logrieco A, Doko MB, Moretti A, Frisullo S, Visconti A. Occurrence of fumonisin B1 and B2 in *Fusarium* proliferation infected asparagus plants. *J Agric Food Chem*. 1998; 46:5201-5204.
13. Makaula NA, Marasas WF, Venter FS, Badenhorst CJ, Bradshaw D, Swanevelder S. Oesophageal and other cancer patterns in four selected districts of Transkei, Southern Africa: 1985-1990. *Afr J Health Sci*. 1990; 3:11-15.
14. McDonald DC, Harkness A, Stonebridge WC. Growth of *Aspergillus flavus* and production of Aflatoxin in groundnuts. Part VI. Samaru research bulletin. Institute of Agricultural research, Ahmadu Bello University, Zaria, Nigeria. 1965
15. Meister U, Symmank H, Dahlke H. Investigation and evaluation of the contamination of native and imported cereals with fumonisins. 1996; 203: 528-533.
16. Melina R. "Sex-Change Chicken: Gertie the Hen Becomes Bertie the Cockerel". *Live Sci*. 2014.
17. Merrill AH, Sullards MC, Wang E, Voss KA, Riley RT. Sphingolipid metabolism: Roles in signal transduction and disruption by fumonisins. *Environ Health Perspect*. 2001;109 (2), 283-289
18. Scott PM, Kanhere SR, Lawrence G, Daley EF, Farber JM. Fermentation of work containing added ochratoxin A and fumonisins B1 and B2. *Food Addict Contam*. 1995; 12:31-40.
19. Sydenham EW, Thiel PG, Marasas WF, Shephard GS, Van Schalkwyk DJ, Koch KR. Natural Occurrence of some *Fusarium* mycotoxins in corn from low and high oesophageal cancer prevalence areas of the Transkei, Southern Africa. *J. Agric Food Chem*. 1990; 38:1900-1903.
20. Torres MR, Sanchis V, Ramos AJ. Occurrence of fumonisins in Spanish beers analyzed by an enzyme-linked immunosorbent assay method. *Int. J. Food Microbiol*. 1998;39:39-143.
21. Worldwide regulations for mycotoxins in food and feed. 2003; 17-39.

10/28/2015