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A Review on Environmental Enteropathy and Effect of Aflatoxins and Fumonisin on Stunting

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Abstract

Environmental enteropathy is a subclinical condition caused by constant fecal-oral contamination, resulting in blunting of intestinal villous and intestinal inflammation. It is common in LMICs and affects both adults and children. This review will demonstrate environmental enteropathy as well as the effects of Aflatoxin and Fumonisin on stunting. Aflatoxicosis is a relatively unknown and underreported cause of liver damage; aflatoxin is a human liver carcinogen. Aflatoxin exposure causes stunting, underweight, neurological impairment, immunosuppression, and mortality in children. Several studies have found positive associations. Fumonisin exposure, either alone or in combination with aflatoxin, may contribute to stunted growth in early childhood and the observed high prevalence of stunting among children. Appropriate intervention measures to prevent mycotoxin exposure in children should be considered one of the key initiatives for improving childhood growth and health in various regions of developing countries. the entire world It is critical to combat environmental enteropathy and stunting. Concerned government and non-government stakeholders must raise awareness and collaborate at the household and community levels.

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Introduction

Environmental enteropathy (EE) is a state of intestinal inflammation without obvious diarrhea that occurs in people who have been exposed to poor sanitation and hygiene over time (Jones, 2012). When several reports of abnormalities on jejunal biopsy of asymptomatic individuals from tropical countries surfaced in the 1960s, environmental enteropathy was dubbed "Tropical Enteropathy." Asian, African, and Latin American publications confirmed these findings in all age groups studied, from infants to adults. On biopsy, there was a decrease in villous height, an increase in crypt depth, lymphocytic infiltration of the lamina propria, and an increase in intra-epithelial. Furthermore, the majority of these people had problems with the small bowel's absorptive function (Andrew and Paul, 2012).

People in developing countries are frequently affected by a spectrum of enteropathy characterized by small intestinal inflammation, reduced absorptive capacity, and increased intestinal permeability. Microbial translocation across the compromised intestinal barrier is facilitated by this subclinical intestinal pathology, resulting in chronic systemic inflammation that can be harmful to one's health. EE is common among people living in unsanitary

conditions, and it is thought to mediate two interconnected childhood public health issues: stunting and anemia. It also contributes to poor oral vaccine efficacy in developing countries (Rosie *et al.*, 2014).

EE is also thought to play a role in the ineffectiveness of nutritional therapy. Various trials of supervised feeding interventions have failed to show significant growth improvement. Even when breastfeeding is adequate, infants as young as three months have been found to have growth stunting and associated pathologic changes in the intestinal mucosa. Chronic fecal pathogen exposure is thought to cause inflammation and structural changes in the small bowel, leading to functional changes (Steve, 2014).

Because EE increases susceptibility to other morbidities, this subclinical condition may have a greater impact on global Disability Adjusted Life Years (DALYs) than any specific pathogen. Better methods of diagnosing EE are required before treatments can be developed. Noninvasive diagnostic parameters are especially important because endoscopy is impractical at the public health intervention scale that is likely to be required for an ailment that affects up to 40% of the world's children.

WASH (2013) also found new evidence of another intestinal disease, environmental enteropathy, that affects child growth.

Environmental enteropathy is a small intestine disease caused by repeated exposure to fecal microbes and poor sanitation during childhood. It lowers a child's ability to absorb nutrients. Environmental enteropathy has been coined to describe a pathology that appears to be caused by environmental factors in developing countries (McKay *et al.*, 2010). Although the exact mechanisms underlying EE are unknown, it is thought to be caused by poor sanitation and recurrent fecal ingestion, which lead to chronic intestinal inflammation and histological changes (Humphrey, 2009). EE, on the other hand, coexists with other causes of enteropathy in developing countries, such as HIV and micronutrient deficiencies (Prendergast and Kelly 2012).Zinc deficiency, an essential micronutrient, may be linked to EE, either as a result of EE-induced decreased absorption capacity or as a potential contributor to EE through its effects on intestinal function, immunity, and inflammation (Greta *et al.*, 2014).Furthermore, environmental enteropathy is a condition of the small intestine marked by:

Villi flattening, reducing the surface area

Thickening of the surface through which nutrients must be absorbed

In otherwise healthy children, the gold standard of endoscopy and biopsy to diagnose EE is both impractical and unethical, so substitute measures are commonly used to assess small intestinal structure and function (Diamant *et al.*, 2011). The small monosaccharide mannitol passes through many small, water-filled pores in enterocyte membranes (the transcellular pathway), and a decrease in mannitol indicates a reduction in small intestine absorption capacity, which could indicate villous damage. As a result, the lactulose-mannitol (L: M) ratio in urine can be used as a replacement marker for small intestinal structure and function. It's also clear that some developing-world emerging diseases are linked to an underlying enteropathy. Increased intestinal permeability is associated with obesity, diabetes, and metabolic syndrome (Diamant *et al.*, 2010).Increased permeability to large molecules and cells (microbes).

The impact of malnutrition on child health, which affects 25% of all children and is estimated to cause more than a million deaths annually due to increased susceptibility to infection, necessitates an immediate solution to EE. Efforts to treat EE are thus underway, even as work continues to identify it using non-invasive biomarkers and delineate its pathogenesis (Poonum *et al.*, 2013).

Therefore, the objective of this review is to illustrate Environmental enteropathy as well as the effects of Aflatoxin and Fumonisin on Stunting.

Epidemiology of Environmental Enteropathy

The development of enteropathy, which is characterized by abnormal intestinal architecture and increased permeability, is aided by several biologics, environmental, and possibly genetic factors. Microbial translocation is enabled by enteropathy, resulting in a widespread activation of the innate and adaptive immune systems. This chronic inflammatory pathway may contribute to stunting and anemia in children in developing countries, exacerbate immune activation and CD4 decline in HIV-positive people, and reduce the efficacy of oral vaccines.

Environmental enteropathy is common in low-income tropical countries, where feces-contaminated food, water, and the environment are common. It's something you pick up when you're a kid. In endemic countries, stillborn children have normal intestinal cellular structures. After

2–5 years, it was resolved by migration to developed countries. Peace Corps volunteers and US soldiers in Vietnam, for example, developed environmental enteropathy within three to six months. However, after returning to a developed country, it was resolved within a year (Steve, 2014). Lindenbaum *et al.,* (1966) wanted to see if this condition of subclinical malabsorption could be acquired and, if so if it could be reversed. Many of the peace corps volunteers in Pakistan had mild diarrhea, and a few had lost weight, according to the author. On jejunal biopsy, the same abnormalities were found, as well as abnormal carbohydrate absorption, as in native Pakistanis. The volunteers were found to have regained normal carbohydrate absorptive function two to three years after returning to the United States (Andrew and Paul, 2012).

When immigrants living in New York City were studied within the first two years of migrating to the US, nearly half had mal-absorption, but absorption and changes on jejunal biopsies improved with increasing periods of residence in the US. These observed changes in histopathology in the same individuals in both developed and developing countries suggested that this condition may have an environmental etiology. A study from Vellore, India, discovered that stillborn fetuses had normal finger-like intestinal villi, but architectural abnormalities were seen in infants as early as 8 weeks of life, suggesting an environmental rather than genetic etiology. A study of 200 adults in Zambia discovered that none of the 200 subjects had 'normal' jejunal biopsies.

The subjects were found to have more severe intestinal permeability abnormalities than higher socioeconomic status controls. Because these subjects were studied serially over three years, seasonal variations in villous height and carbohydrate absorption were discovered (Poonum *et al.,* 2013). In Ethiopia, approximately 44 percent of under-five children are stunted, which can be strongly linked to the childhood incidence of diarrhea, the country's leading cause of under-five mortality, accounting for 23 percent of all under-five deaths (73,341 children per year) (UNICEF, 2014).

Malnutrition, a well-known problem in the developing world, where 26 percent of children under the age of five are underweight, maybe the most significant impact of EE. Even more concerning is the fact that screams/wraps of malnutrition are responsible for 21% of deaths in children under the age of five (Dewey and Adu-Afarwuah, 2008).

Causes and Immediate Consequences of Environmental Enteropathy

Environmental enteropathy is caused by poor sanitation and hygiene, according to Poonum *et al.,* (2013), but there is little direct evidence. The unrestrained stimulation of gut T-cells causes EE. Villous atrophy/degeneration, crypt hyperplasia, and inflammatory cell infiltration occur quickly after human gut T-cells are overstimulated. So, through a T-cell-mediated process, high concentrations of bacteria colonize or cause repeated infections in the small intestine, causing EE morphologic changes (Poonum *et al.,* 2013). Failure to address malnutrition and poverty results in 2-3% losses in national GDP; reduced physical productivity of the workforce (due to short stature); reduced cognitive development; delays in starting school (7 months); losses of schooling (0.7 grades); reductions in lifetime earnings; overall reduced economic productivity, wages, and income; and poor reproductive performance, including smaller babies, in women. These factors, when combined, result in the intergenerational transmission of malnutrition and poverty (Poonum *et al.*, 2013).

Models depicting the use of all known interventions, including vitamin A and zinc supplementation, balanced energy protein supplementation, complementary feeding, breastfeeding promotion, and micronutrient supplementation in pregnancy, revealed that using these interventions in 99 percent of children around the world would only reduce stunting by 33 percent (Bhutta, 2008). Subclinical yet pathologic changes in the absorptive and immune function of the gastrointestinal tract of infants and children in developing countries must be considered (Dewey and Adu-Afarwuah, 2008). Aside from the failure of nutritional interventions, it has been observed that oral vaccines, including those for polio and rotavirus, are less immunogenic in developing-country children, implying that EE may cause altered mucosal immunity in children (Ratnaswamy, *et al.,* 1973; Naik and Krishnan, 1996; Hasan, *et al.,* 2004 and Levine, 2010).

Aflatoxins

Aflatoxins (AF) are highly mutagenic and toxic secondary metabolites that are harmful to both human and animal health. Aflatoxins are toxic metabolites produced by fungal species during growth under favorable temperature and moisture conditions. *Aspergillus flavus, Aspergillus parasiticus*, and the much

rarer *Aspergillus nomius* are the major aflatoxin-producing species. Furthermore, they are potentially toxic, carcinogenic, mutagenic, and immunosuppressive agents produced on a variety of food products by the fungi *Aspergillus flavus* and *Aspergillus parasiticus* (Barzaville, 2005). These fungi produce metabolites known as AFB1, AFB2, AF, G1, and AFG2, all of which occur naturally. These fungi can produce toxic compounds on nearly any food that promotes growth (Klich, 2007).

Maize, sorghum, rice, and wheat, as well as other crops like groundnuts and cassava, are the main cereals affected (Cotty, 1997; Kabak *et al.,* 2006). While these toxins do not appear to have any physiological functions for the fungus, they are now known to be carcinogens, teratogens, mutagens, immune suppressants, and estrogenic in humans (Amaike and Keller, 2011). This threat has not abated in the vast majority of Sub-Saharan Africa, and it appears to be increasing. In that year, the highest level of aflatoxin was 830 ppb (parts per billion) (FAO, 2011). Aflatoxin contamination has also been found in Ethiopian staple cereals, red chili pepper, and ground peas, according to studies (Fufa and Urga, 1996; Ayalew *et al.,* 2006).

These molds can colonize a variety of crops both in the field and in storage as non-destructive plant pathogens, and they can grow and produce aflatoxins at very low moisture levels (Aw = 0.82), and over a wide temperature range (13-37oC). Although they can be found all over the world, they are more common in tropical areas with extreme variations in temperature, rainfall, and humidity. Drought stress is known to favor *A. flavus* invasion of groundnut crops in the field, and maize crops are vulnerable if insect pests damage them. Mold growth and aflatoxin production are also important during crop storage, especially if drying is insufficient or storage conditions allow insect or animal pests access (Laura *et al.,* 2015).

Aflatoxins M1 and M2 (named after milk aflatoxins and related to meat aflatoxins) are thermo-resistant hydroxylated metabolites produced by lactating animals fed aflatoxin-contaminated feed. Livestock metabolizes ingested AFB1 and AFB2 into AFM1 and AFM2, with an estimated conversion ratio of 1–3 percent between AFB1 and AFM1 (Herzallah, 2009). The presence of aflatoxin-contaminated milk has been the most concerning issue over time because cows and goats are greatly affected when eating contaminated forage all over the world (López *et al.,* 2003). According to Dawit *et al.,* (2015), there is an average of 1% pass-through of aflatoxin contamination from feed to milk. According to the authors, the high level of AFM1 in milk in the greater Addis Ababa milk shed is critical because it is one of the largest in the country and most Addis Abeba residents get their milk from these sources. Given that young children are weaned on cow's milk and are not immune-competent at this age, drinking AFM1-contaminated milk may further suppress their immunity and contribute to stunting (Bondy and Pestka, 2000; Gong *et al.,* 2004).

Effects of Aflatoxins on Human Health

The incidence of chronic aflatoxicosis in humans is unknown and is almost impossible to estimate because the symptoms are so difficult to recognize. Human liver cancer is common in parts of the world where aflatoxin contamination of food is common, and there may be a link, though this has yet to be proven. Aflatoxin exposure at high enough levels can cause acute toxicity and death in mammals, birds, and fish, as well as humans. The liver is the most commonly affected organ, but high levels of aflatoxin have also been discovered in the lungs, kidneys, brains, and hearts of people who have died from acute aflatoxicosis. The liver is the most commonly affected organ, but high levels of aflatoxin have also been discovered in the lungs, kidneys, brains, and hearts of people who have died from acute aflatoxicosis. Acute necrosis and cirrhosis of the liver, as well as hemorrhaging and edema, are common. Animals' LD (lethal dose) values range from 0.5 to 10

Chronic toxicity is likely to be more important in terms of food safety, especially in more developed parts of the world. Aflatoxin B1 is a powerful carcinogen and mutagen in many animals, including humans, and the liver is once again the primary target organ. Low-dose ingestion over time has been linked to primary liver cancer, chronic hepatitis, jaundice, cirrhosis, and impaired nutrient conversion, as well as conditions like Reye's syndrome and kwashiorkor. Aflatoxin G1 and M1 have less information about their chronic toxicity, but they are also thought to be carcinogens, albeit less potent than B1 (Kitty C., 2012).mg/kg body weight (Laura *et al.,* 2015).

Acute human aflatoxicosis is uncommon, especially in developed countries where food contamination levels are closely monitored. However, outbreaks have occurred in some developing countries, particularly in Sub-Saharan Africa, where maize and groundnuts are common and the climate is conducive to rapid mold growth on crops in

the field and during storage. In India, a notable outbreak occurred in 1974, when nearly 400 people became ill with fever and jaundice after eating maize contaminated with 0.25 to 15 mg/kg aflatoxin, with more than 100 people dying. In Kenya, there have been major outbreaks, the most recent in 2004, when 317 people were infected and 125 died, most likely as a result of eating contaminated maize (Hoffmann *et al.,* 2004).

Chronic malnutrition during childhood has far-reaching implications for national development. A child with early linear growth retardation is more likely to have poor cognitive development and academic performance (Suraiya Ismail *et al.,* 2014). Poor academic performance can limit one's ability to earn enough money. A short child is more likely to grow up to be a short adult, with lower physical productivity and a higher risk of poor pregnancy outcomes, including low birth weight babies in women. Furthermore, an adult who is short as a result of nutritional abuse during fetal or early childhood is more likely to develop obesity, coronary heart disease, diabetes, and hypertension later in life, especially when exposed to relative affluence. This has significant implications for the cost of health care in developing countries (Barker 1997).

Fumonisin

Fumonisins are naturally occurring mold toxins produced mainly by *Fusarium verticillioides*, *Fusarium proliferatum*, and *Gibberella fujikuroi* consisting of a group of seven structurally related analogs but only fumonisins B1 (FB1), B2 (FB2) and B3 (FB3) occur worldwide as natural contaminants. Infants exposed to Fumonisin intakes above the provisional maximum tolerable daily intake of 2 mg/kg body weight were significantly shorter by 1.3 cm (Kimanya *et al.,* 2012). Fumonisins hurt the kidneys, pancreas, testes, thymus, gastrointestinal tract, and blood cells, in addition to the brain, liver, and lungs. Consumption of fumonisins during early pregnancy has also been linked to an increased risk of neural tube defects in the developing fetus (Paul and Gary 2002). Exposure to fumonisin alone or in combination with aflatoxin in infants and young children could be one of the factors contributing to stunting in early childhood and the high prevalence of stunting among children (Candida, *et al.,* 2015).

The presence of fumonisin in human foods is common; contamination of maize and maize-based products is well-documented, but fumonisin has also been found in other cereals such as barley, wheat, rice, and sorghum. Human exposure ranges from micrograms to milligrams per day and is highest in areas where maize products are the main source of food. Stunting has also been linked to fumonisin exposure.

Fumonisin exposure through maize in complementary foods was found to be negatively associated with linear growth in infants in rural Tanzania. In areas of South Africa and China where fumonisin-contaminated corn is widely consumed, the mycotoxin fumonisin has been linked to esophageal cancer in humans. Damaged grain is malted and unmalted forms are consumed in Ethiopia in various ways. Mold-damaged maize is particularly popular in some areas for making traditional drinks like 'tela,' 'arek,' and 'borde.' Malted maize, on the other hand, is only used in the preparation of local beverages. In Ethiopia, Fusarium species that produce a variety of mycotoxins, such as *F. verticillioides*, *F. subgutinans*, and *F. graminearum*, are common maize contaminants. As a result, products made from toxigenic Fusarium species-infested maize grain pose a risk to human health. However, in Ethiopia, there is a low level of awareness about the health risks of mycotoxins from eating moldy grain and food (Tesfaye and Dawit, 2000).

Stunting

Childhood stunting, defined as a child's height for age falling two standard deviations or more below the World Health Organization's (WHO 2014) growth reference (HAZ -2), is a slowed rate of development in humans (NLiS, 2014). Stunting is the result of long-term malnutrition, which causes linear growth to be slowed. There are 195 million stunted children under the age of five in the world, with Sub-Saharan Africa and South Asia bearing the brunt of the burden. Childhood stunting has been linked to cognitive impairment and an increased risk of infection. Genetics, nutritional factors (energy intake, macronutrients, and micronutrients), infections (which cause injury to the gastrointestinal mucosa/epithelium), systemic effects (repeated diarrheal diseases), immune modulation, maternal factors (maternal nutrition, behavioral factors), and, most likely, dietary and environmental toxins (NLiS, 2014).In India, for example, living in an environment where many people defecate in the open due to a lack of sanitation is a major cause of stunted growth in children (Spears, 2013).

Malnutrition hurts a child's health. Under nutrition (fetal growth restriction, stunting, wasting, micronutrient deficiency, and inadequate breastfeeding) is estimated to

be the cause of 45 percent of deaths in children under the age of five around the world. Severe wasting is linked to a nearly nine-fold increase in the likelihood of death. Stunting is difficult to stop after two years of age, and it has long-term consequences for one's health and development. According to a meta-analysis published in 2007, 200 million children are stunted each year, preventing them from reaching their full developmental potential (Andrew *et al.*, 2014). Stunting and its effects, according to another study, are usually permanent once established. Stunted children may never regain the height they lost as a result of stunting, and most will never gain the weight they lost as a result of stunting (Spears, 2013). Certain adult non-communicable diseases are linked to childhood stunting, possibly due to epigenetic regulation (which refers to a long-term change in gene expression) or chronic inflammation (Rosie *et al.*, 2014).

Because the brain develops rapidly during the first 1000 days of a child's life, this "window of opportunity" is critical in laying the foundation for future cognitive and social abilities. It's also the time of year when young children are most vulnerable to infections that cause diarrhea. It is at this age that they stop breastfeeding (weaning), crawl, put things in their mouths and become exposed to feces from open defecation and enteropathies caused by the environment (Franck and Regine, 2014).

Stunting is considered "low prevalence" in terms of public health significance, according to the World Health Organization, if less than 20% of the population is affected. Values of 40% or more are considered very high prevalence, while values in the middle are considered medium to high prevalence (NLiS, 2014).

According to UNICEF, "more than a quarter (26%) of children under the age of five were stunted globally in 2011 (roughly 165 million children)," and "in Sub-Saharan Africa, 40% of children under the age of five are stunted; in South Asia, 39% are stunted" (UNICEF, 2013). Timor-Leste, Burundi, Niger, and Madagascar are the four countries with the highest prevalence, with more than half of children under the age of five being stunted (UNICEF, 2013).

The Effects of Stunting on the Public

Stunting is significant in terms of public health because it is linked to effects such as increased susceptibility to infectious diseases and cognitive impairments that last well beyond childhood (Ricci *et al.*, 2006). Apart from the obvious impact of the affected person's shorter stature, stunted growth in children has the following public health consequences:

A higher risk of illness and premature death may result in delayed mental development and thus poorer school performance, as well as lower productivity and cognitive capacity later in life.

Due to their smaller pelvis, women of shorter stature have a higher risk of complications during childbirth and are more likely to deliver a baby with low birth weight.

Growth retardation can even be passed down to future generations. This is known as the "intergenerational malnutrition cycle" (NLiS, 2014).

Effect of Aflatoxins on Stunting

Children who have been exposed to aflatoxin suffer from stunting, a condition in which the child's height for age is two standard deviations or more below the World Health Organization (WHO) growth reference. According to Khlangwiset and Ricci *et al.*, 2006, evidence from human and animal studies, as well as current knowledge of the biological mechanisms of action of aflatoxins, suggest that chronic exposure to aflatoxins may result in stunted growth.

According to Gong's studies in Togo and Benin in West Africa, higher aflatoxin exposure reduces children's height and weight in a dose-dependent manner (Gong *et al.*, 2002, 2004). Aflatoxin-albumin adduct (AF-alb) levels in maternal blood, cord blood, infant blood, and children's blood were associated with poorer growth indicators in similar studies of infants and children in Gambia (Turner *et al.*, 2003). In humans, AF-alb is a biomarker of aflatoxin exposure and biological activation. Perhaps more concerning from the standpoint of global public health is the link between aflatoxin exposure and childhood stunting, which can lead to a variety of long-term health problems. Aflatoxin exposure through diet is also strongly linked to age (Turner *et al.*, 2007).

Aflatoxins' known biological mechanisms make effects on linear growth plausible. According to human and animal studies, aflatoxins suppress the immune system, retard growth in young children, impair protein synthesis, and alter the hepatic metabolism of micronutrients (Khlangwiset *et al.*, 2011). It has also been proposed that aflatoxins, in conjunction with fumonisin, mediate intestinal damage similar to that seen

in environmental enteropathy (Smith and Prendergast, 2012). This condition causes chronic systemic immune activation and nutrient malabsorption, which can lead to growth retardation. According to the evidence, aflatoxins are a likely cause of linear growth retardation in children with enteropathy (Smith and Prendergast, 2012).

Association of Aflatoxins, Fumonisins, and Stunting

People and their livelihoods are affected by aflatoxin contamination of foods; this is especially true for poor people who have little choice in the food they eat. Previous studies reporting positive associations between aflatoxins or fumonisins biomarkers and child growth used sample sizes ranging from around 100 to several hundred (Gong *et al.,* 2002). Liquid chromatography-mass spectrometry was used to measure serum AF-alb as well as urinary FUM and DON (Scholl *et al.,* 2006; Gerding *et al.,* 2015). These parent mycotoxins and metabolites have a quantitative relationship with the toxin's dietary intake, so they can be used as quantitative exposure biomarkers (Gong *et al.,* 2008; Turner *et al.,* 2008; Turner, 2013). At enrollment, the AF-alb adduct was measured in all mothers. At enrollment (median, 14 weeks gestation) and around 32 weeks gestation, the AF-alb adduct was measured in all mothers, as well as in infants at 12 and 18 months of age. At 6, 12, and 18 months, all three mycotoxins (AF-alb adduct, urinary FB1, and urinary DON) were measured in a subgroup of 200 infants. These multiple exposure analyses were exploratory, but they were significant findings because FUM and DON biomarkers have only been evaluated in African infants in two studies to date (Srey *et al.,* 2014; Shirima *et al.,* 2015).

Acute aflatoxicosis, a Group 1 human liver carcinogen, is an under-recognized and under-reported cause of liver damage. Aflatoxin exposure is linked to stunting, underweight, neurological impairment, immunosuppression, and mortality in children. Because aflatoxins suppress the immune system, they may interact with HIV/AIDS and other infections. Mycotoxins are fungi metabolites that frequently contaminate children's staple foods in developing countries. Aflatoxin, fumonisin, and deoxynivalenol (DON) are among the chemicals that can harm a child's growth. Although these toxins have different mechanisms of action, they all cause intestinal damage by inhibiting protein synthesis, increasing proinflammatory cytokines in the blood, and inhibiting ceramide synthase. The intestinal pathology caused by mycotoxins is very similar to that caused by EE. Childhood stunting is a serious problem. Childhood stunting is a serious and inflexible public health issue that accounts for 20% of deaths in children under the age of five in developing countries (Barzaville, 2005).

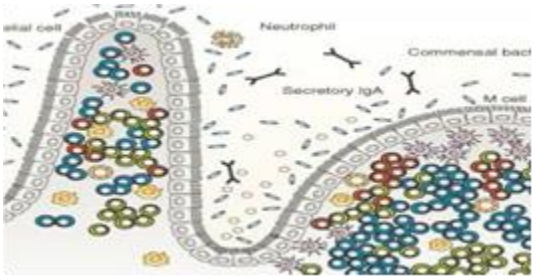
Pesticides, antibiotics, and mycotoxins (aflatoxins) in foods pose serious health risks in the Sub-Saharan African region. In countries like Gambia, Ghana, Guinea, Nigeria, Senegal, South Africa, and Uganda, high levels of aflatoxins have been discovered in groundnuts and cereal grains. Outbreaks in Somalia (1997/98) and Kenya (1982, 2001, 2004, and 2005) provide evidence of the problem's scope. A strong link has been found in epidemiological studies between aflatoxins exposure and primary liver cancer. A synergistic effect between aflatoxins and the hepatitis B virus (HBV) increases the risk of liver cancer by a factor of twelve. The presence of aflatoxins and the hepatitis B virus (HBV) has a synergistic effect that increases the risk of liver cancer by a factor of twelve times that of people infected with the virus, which already increases the relative risk five-fold (Vivian *et al.,* 2013).

Gong *et al.,* (2002) discovered that serum aflatoxin-albumin adducts were linked to stunting in rural Benin and Togo children aged 9–60 months, with a significant dose-response relationship with height-for-age and weight-for-age z scores. In a separate study from the Gambia, maternal aflatoxin exposure during pregnancy was found to be strongly inversely related to infant growth velocity during the first 12 months of life, with a predicted 0.8 kg weight gain and 2-cm height gain if maternal aflatoxin exposure (AF-alb) was reduced from 110 pg/mg to 10 pg/mg (Turner *et al.,* 2007). The first population-based longitudinal study in Tanzanian infants (aged 6–14 months at recruitment) found a urinary biomarker for urinary fumonisin B1 (FB1) (Gong *et al.,* 2008).

Gong *et al.,* (2008) discovered a urinary biomarker for urinary fumonisin B1 (FB1), and the first population-based longitudinal study in Tanzanian infants (aged 6–14 months at recruitment) found a significant link between urinary FB1 and growth faltering (Shirima *et al.,* 2015). Both aflatoxin and fumonisins were found in the study areas, exposing infants and young children.

Fig.1 Microbial translocation epithelium (Veitch, 2001)

Normal



Damaged

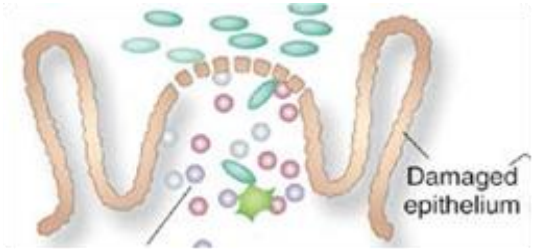


Fig.2 Aspergillus

Fig.3 Aflatoxin

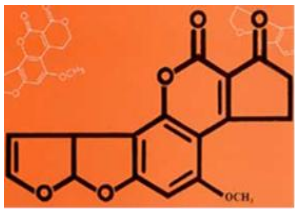


Fig.4 Image of noug cake obtained from one of Addis Ababa's dairy producers. (Dawit *et al.*, 2015.)

Fig.5 Fusarium kernel rot scattered occurrence of damaged kernels and Gibberella ear rot.

Exposure to fumonisin alone or in combination with aflatoxin could be one of the factors contributing to stunting in children's early years. In different parts of the world where fumonisin exposure is likely, appropriate intervention measures to prevent children from being exposed to mycotoxins should be considered as one of the key initiatives for improving childhood growth and health. It is recommended that studies be conducted to look into the specific epidemiological circumstances in which aflatoxin or fumonisin can cause growth defects (Shirima *et al.,* 2015).

More than 2 million children under the age of 5 years die as a result of stunting, wasting, and fetal growth retardation, accounting for 21% of disability-adjusted life-years worldwide (Black *et al.,* 2008). In 2010, it was estimated that 171 million preschool children around the world were stunted (de Onis *et al.,* 2012). Although nutritional and protein deficiencies are the most common causes of stunting in Africa and Asia, aflatoxin, common maize, and peanut contaminant, have also been linked to stunting in Sub-Saharan Africa (Gong *et al.,* 2002; Gong *et al.,* 2004; Turner *et al.,* 2008).

The fact that children in Sub-Saharan Africa are chronically exposed to AF has prompted new research into the role of AF in growth stunting and its impact on morbidity and mortality rates. Turner and colleagues (Turner *et al.,* 2007) tracked the growth of 138 Gambian infants from birth to 12 months, comparing growth to maternal AF exposure.

A higher mean maternal exposure level was found to be associated with lower weight-for-age and height-for-age z scores in this study. Over 8 months, children aged 16–37 months from Benin showed a significant negative correlation between AF exposure and height increase (Gong *et al.,* 2004). When compared to children who were not classified as stunted, children with a height-for-age z score of £ 2 had 30–40 percent higher AF-alb (Gong *et al.,* 2002; Gong *et al.,* 2003; Turner *et al.,* 2003; Obuseh *et al.,* 2010).

Environmental Enteropathy and Stunting

Stunting is more common in areas with higher levels of fecal contamination. Because of animal husbandry and biological feasibility. The failure of health interventions in developing countries, particularly those aimed at malnutrition and using oral vaccines, can be directly attributed to EE, a subclinical, pathologic process that occurs in the gastrointestinal tracts of millions of people worldwide who live in impoverished and unsanitary conditions (IFPRI, 2012).

In addition to the failure of nutritional interventions, oral vaccines, such as those for polio and rotavirus, be less immunogenic in children from developing countries, implying that EE may cause altered mucosal immunity in

these children (William, 2012). Biopsies and abnormal sugar absorption tests show that EE causes fundamental changes in both small intestinal structure and function. The small bowel's immunologic function may also be significantly impacted. In EE, nonspecific host defenses are breached, and innate and adaptive immune responses are activated as a result of constant exposure to enteric pathogens (Steve, 2014). According to Robert *et al.*, 2013, there is growing evidence of a link between a child's contaminated environment and stunted growth. Drinking water quality, sanitation, hygiene, and nutrition all improved as a result of a WASH intervention in Bangladesh and Kenya, resulting in less diarrhea, fewer parasites, less EE, improved child growth, and improved child development.

Successful Interventions against Stunting and Environmental Enteropathy

Some studies have looked into potential treatments to counteract the negative effects of EE on nutritional status. Improved complementary feeding, zinc supplementation, and improved hygiene are effective during the first 24 months of a child's life. The impact of universal coverage with all interventions is a 36% reduction in stunting prevalence at 36 months of age. It is unclear what factors contribute to the remaining 64%, or how much exposure to mycotoxins may play a role.

According to USAID's (2014-2025) strategy, if scaled up in high-burden countries, timely nutrition-specific interventions at critical points in the lifecycle can have a dramatic impact on reducing malnutrition globally. It is estimated that ten evidence-based, evidence-based interventions could reduce stunting by 20% and severe wasting by 60% if scaled to 90% coverage. As a result, the following interventions can help to some extent manage severe acute malnutrition.

Preventive zinc and Vitamin A supplementation

Breastfeeding promotion and appropriate complementary feeding

Management of moderate acute malnutrition

Folic acid supplementation or fortification before conception

Maternal multiple micronutrients and balanced energy protein supplementation

These include balanced energy-protein supplementation, multiple micronutrient supplementation, and malaria prevention treatment during pregnancy on an as-needed basis.

A new WASH paradigm emerged in the first 1000 days. Children should wash their hands with soap after playing outside of a safe play area. The latrine should be used to dispose of all feces, including that of children. Preventing infants and young children from ingesting human and animal feces can help to prevent environmental enteropathy.

EE is characterized by changes in intestinal architecture, increased intestinal inflammation and permeability, a dysregulated gut immune response, and slow growth in children and adults who live in a fecally contaminated environment. These changes have significant implications for dealing with diseases that afflict the developing world, such as malnutrition and the control of infectious diseases. To reduce stunting, there is a compelling case for developing interventions that prevent or ameliorate enteropathy. Millions of people in developing countries could benefit from such interventions, particularly children under the age of five, who are most affected by the overlapping epidemics of stunting, malnutrition, and other factors. As a result, preventing environmental enteropathy and stunting is critical because it affects welfare, human rights, equity, and survival. The global burden of stunting is enormous, with maternal and child under nutrition accounting for more than 10% of the total global burden. From an economic standpoint, it is necessary to break the vicious cycle of under nutrition and poverty; reduced physical productivity of the workforce (due to short stature); reduced cognitive development; delays in starting school; losses of schooling; reductions in lifetime earnings; overall reduced economic productivity, wages, and income; and poor reproductive performance in women, including smaller babies. These factors combine to cause under nutrition and poverty to be passed down through generations. The many underlying risk factors for developmental deficits necessitate an integrated approach to address the many causes of poor growth and development. As a result, the importance of hygiene must be considered. Along the entire infant food value chain, there is a need to raise awareness of aflatoxins and fumonisin risk mitigation practices. Policymakers and development organizations must encourage the spread of information about good agricultural and storage practices, as well as other risk-reduction strategies.

Recommendation

To fight against environmental enteropathy and stunting, concerned government and nongovernment stakeholders should create awareness and work collaboratively at household and community levels on the following:

Awareness creation on health risks of mycotoxins from contamination of moldy crops and good agricultural practices to prevent animal feeds and food crops at field level, storage, transportation, and marketing from the risks of contamination by aflatoxins and fumonisins

Mothers should consider providing safe playgrounds for their children to avoid ingesting contaminated soils and animal feces.

In general, community members and family members should be taught how to prepare and use safe and hygienic feces and water for drinking and food preparation, as well as personal hygiene and environmental sanitation.

Preventive zinc supplementation, breastfeeding promotion, appropriate complementary feeding, management of moderate acute malnutrition, pre-conceptual folic acid supplementation or fortification, maternal multiple micronutrient supplementation, Vitamin A supplementation, and maternal balanced energy prowess should all be considered in high-burden areas.

Future research should focus on the role of mycotoxins in the pathogenesis of EE, as well as interventions to reduce childhood stunting by limiting mycotoxins exposure.

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