Building an Aflatoxin Safe East African Community

Technical Policy Paper 2

Aflatoxin and the 1,000 Days

Knowledge Platform 2015

Situational Analysis East Africa Region

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EAC JUMUIYA YA AFRIKA MASHARIKI

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Cover: A child in transition from breastfeeding to complementary foods. *IITA*

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Foreword

Adequate nutrition from conception to two years of age is the cornerstone for the establishment of good health, intellectual development, and economic productivity throughout a child's future life. The consequences of malnutrition during the first 1,000 days of life are profoundly detrimental and irreversible on all of these critical factors.

Launched in 2010, the "Scaling Up Nutrition" (SUN) movement underscored the need for an intensified focus on this unique window of opportunity to ensure an optimal quality of life for the next generation of children in the developing world. Today, 26 percent of the world's stunted children, those suffering from chronic undernutrition, live in Africa.

There is a growing body of evidence that mycotoxin contamination, especially from aflatoxins, in the diets of pregnant and lactating women, infants, and young children, interferes with the absorption of nutrients in the body, contributing to low birthweight and stunting during the most critical period of development. Aflatoxin also compromises immune response, making infants and young children more susceptible to disease. Aflatoxin damages the liver and significantly worsens liver disease in tandem with hepatitis A, B, and C. An estimated 40 percent of hepatocellular carcinoma (HCC) throughout East Africa is believed to be correlated with high levels of dietary aflatoxin.

Climatic conditions of heat and humidity, combined with poor agricultural practices, create an ideal environment for the development in food crops of the fungus that produces the toxin. Aflatoxin exposure during the 1,000 days occurs through several pathways, often beginning in utero through dietary exposure of the mother. Aflatoxin can cross the placental barrier. After a child is born, the exposure continues during infancy and early childhood through contaminated breast milk, and increases significantly when complementary foods are introduced. In the East African Community (EAC) region, complementary foods are frequently introduced prematurely, increasing infant exposure to unsafe levels of aflatoxin. Further, aflatoxin contamination levels in many of the staple foods currently recommended as complementary foods such as maize, groundnut, cassava and dairy products are in excess of safe dietary and serum limits even for adults.

In many developed countries, more stringent standards exist for foods commonly consumed by infants and young children, but no such standards exist in East Africa. Published studies of aflatoxin exposure in children for Kenya, Tanzania, and Uganda show high exposure levels during pregnancy and lactation, with levels peaking in young children progressively through weaning. While exposure data are more limited for Burundi and Rwanda, given the similarity in dietary consumption patterns, climate, and other contributing factors across the region, it is likely that throughout the 1,000 days period exposure patterns in Rwanda and Burundi are similar to that elsewhere in the region. The fact that over 90 percent of samples from young children within both the East and West Africa regions have had detectable AF-alb levels, in contrast to less than 1 percent in the developed world, clearly demonstrates an alarming public health burden in sub-Saharan Africa.

This paper summarizes the exciting research on the relationship between aflatoxin and the first 1,000 days of life, and establishes a scientifically sound knowledge platform for the timely design of policies and programs to address the issue.

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Executive Summary

This paper reviews the current state of research on aflatoxin ingestion during the 1,000 days and makes policy recommendations for actions to minimize aflatoxin contamination during this vulnerable period of childhood.The term "the 1,000 days" refers to the first 1,000 days of human life, from conception to a child's second birthday. Exposure to aflatoxin during the first 1,000 days of life exacerbates child malnutrition and causes negative health impacts because the toxin interferes with complex developmental processes taking place in the body during this time. As intellectual capacity is formed during this period, aflatoxin ingestion has an impact on a population's economic productivity far into the future. Aflatoxin ingestion during the 1,000 days also contributes to negative health events in later life, including cancer.

Aflatoxin contamination of staple foods is prevalent throughout East Africa, which has ideal climatic conditions for the development of the fungus that produces the toxin. There are several pathways for aflatoxin exposure during the first 1,000 days of life. Exposure can start in utero if the mother is exposed to the toxin, as aflatoxins can cross the placental barrier. After a child is born, the exposure continues during infancy and early childhood through contaminated breast milk, and increases significantly when complementary foods are introduced. As complementary foods are frequently introduced prematurely, infant dietary exposure to aflatoxin presents a high risk.

While published studies of aflatoxin exposure in children within the region are few, there are reports of high exposure levels during pregnancy in Kenya, and also among children in Tanzania and Uganda. There are no available exposure data for Burundi or Rwanda. However, given the similarity in dietary consumption patterns, climate, and other contributing factors across the region, it is likely that throughout the 1,000 day period exposure patterns in Rwanda and Burundi are similar to that elsewhere in the region. The fact that over 90 percent of samples from young children within both the East and West Africa regions have had detectable AF-alb levels, in contrast to less than 1 percent in the developed world, clearly demonstrates an alarming public health burden in sub-Saharan Africa.

As a result, the pervasive presence of aflatoxin in the diets of East African infants and young children is likely to compromise efforts to improve child health, such as those included as milestones for the Millennium Development Goals (MDG) and the Scaling Up Nutrition (SUN) initiative.

Reduction of aflatoxin exposure, especially for infants, children, pregnant and lactating women, is a challenge that must be expeditiously addressed throughout East Africa.

This calls for a concerted effort among policy makers, donors, national governments, and regional economic organizations to support a comprehensive program to build an aflatoxin safe community across the health, agriculture, livestock, trade, and environment sectors. Within this, priority attention should be given to the1,000 days population and other vulnerable groups.

In tandem with a framework to foster an aflatoxin safe action plan to promote good agricultural practices, a rigorous regulatory environment that ensures food safety standards are met, adequate alternative use and disposal systems for commodities deemed contaiminated, and broader initiatives to address the risk of aflatoxin to the public health, a specialized vertical intervention for the 1,000 days is important. This will entail the design and implementation of behavior change communications (BCC) programs, training of health professionals, inclusion of aflatoxin in food safety, medical, agricultural, and nutrition education curricula, and family-focused agricultural extension services to address aflatoxin in the on-farm consumption context. Due to the urgency of ameliorating the negative impacts of aflatoxin within the1,000 days, first priority should be given to actions that will affect modifications to dietary patterns, rigorous enforcement of standards for foods specifically for infants and young children, and other interventions to promote aflatoxin abatement measures at the community and household level.

Aflatoxin exposure is prevalent in Africa. Exposure is often most severe during the first 1,000 days of life—the period from conception to two years of age. It begins in utero and rapidly accelerates during complementary feeding due to contaminated complementary food and family food. In developing countries, the complementary feeding period is also often a period in which children are most susceptible to malnutrition, enteropathy, and infectious disease (Lunn 2000). These, co-occurring with increased aflatoxin exposure, inevitably have serious implications for child growth and development.

Introduction

Aflatoxins are toxic byproducts of the fungus *Aspergillus flavus*. They occur naturally in the soils throughout tropical latitudes, including all five partner states of the East African Community (EAC). For reasons still not well understood, certain crops are prone to aflatoxin contamination, including many of the staple foods comprising the core dietary intake of the region. Maize and groundnuts consumed on-farm as well as purchased in the marketplace often do not meet food safety standards, and are often consumed by infants and young children. Aflatoxins are also transferred to animal products through contaminated feeds and are prevalent in milk, dried and farmed fish, and poultry. Aflatoxin can cause fatal liver toxicity at high doses during acute outbreaks of exposure, while lower levels of chronic exposure typical throughout the EAC are associated with a range of significant health effects including liver cancer, child stunting, low birthweight, and immune suppression. Immune suppression may increase susceptibility to infections, particularly in children, affecting general health, nutrient uptake, and growth. Aflatoxin can also cause damage to intestinal epithelial cells, and this effect may contribute to environmental enteropathy by causing leaky gut and reducing the uptake of essential nutrients. Stunting associated with aflatoxin exposure in utero and during the first two years of life may be due to these mechanisms or other effects such as disruption of growth-factor pathways.

Aflatoxin exposure in humans can be monitored by measuring levels of contamination in food or by measuring biomarkers in blood or urine. Biomarkers of aflatoxin exposure are widely used for studies aimed at understanding the health effects for aflatoxin exposure. Such biomarkers have shown widespread exposure in adults and children across a range of countries, including high levels in populations in East and West Africa. The solutions to the many problems associated with aflatoxin contamination are complex and require concerted efforts and cooperation among national governments and regional organizations, the public and private sectors, as well as the dedication of substantial human and financial resources for programs to control aflatoxin contamination in a multisectoral context spanning health, agriculture, trade, and the environment.

Growing awareness throughout the global community on issues related to aflatoxin have led to the formation of a new partnership for aflatoxin control in Africa. Announced at the tenth Agriculture Growth and Opportunity Act (AGOA) forum in Lusaka, Zambia in March 2011, the Partnership for Aflatoxin Control in Africa (PACA) seeks to raise awareness, create an effective regulatory environment, scale up mitigation activities, engage high level leadership, mobilize resources, and establish an effective information sharing platform. PACA was formally launched by the Africa Union October 31, 2012, in Addis Ababa, Ethiopia following the commemoration of the Africa Food and Nutrition Security Day and in conjunction with the 2012 Conference of African Ministers of Agriculture and Trade.

The EAC responded quickly to this initiative and began building a comprehensive program to address aflatoxin issues in collaboration with partner states and regional experts. On October 12, 2013 in Arusha, Tanzania, the EAC launched its first effort to address aflatoxin contamination, announcing the "Regional Project on Aflatoxin Control and Improved Nutrition." This was followed by a coordinated activity, the Multisectoral Regional Aflatoxin Abatement Program (MRAAP) in 2013.

Knowledge Platform

Child Malnutrition Worldwide

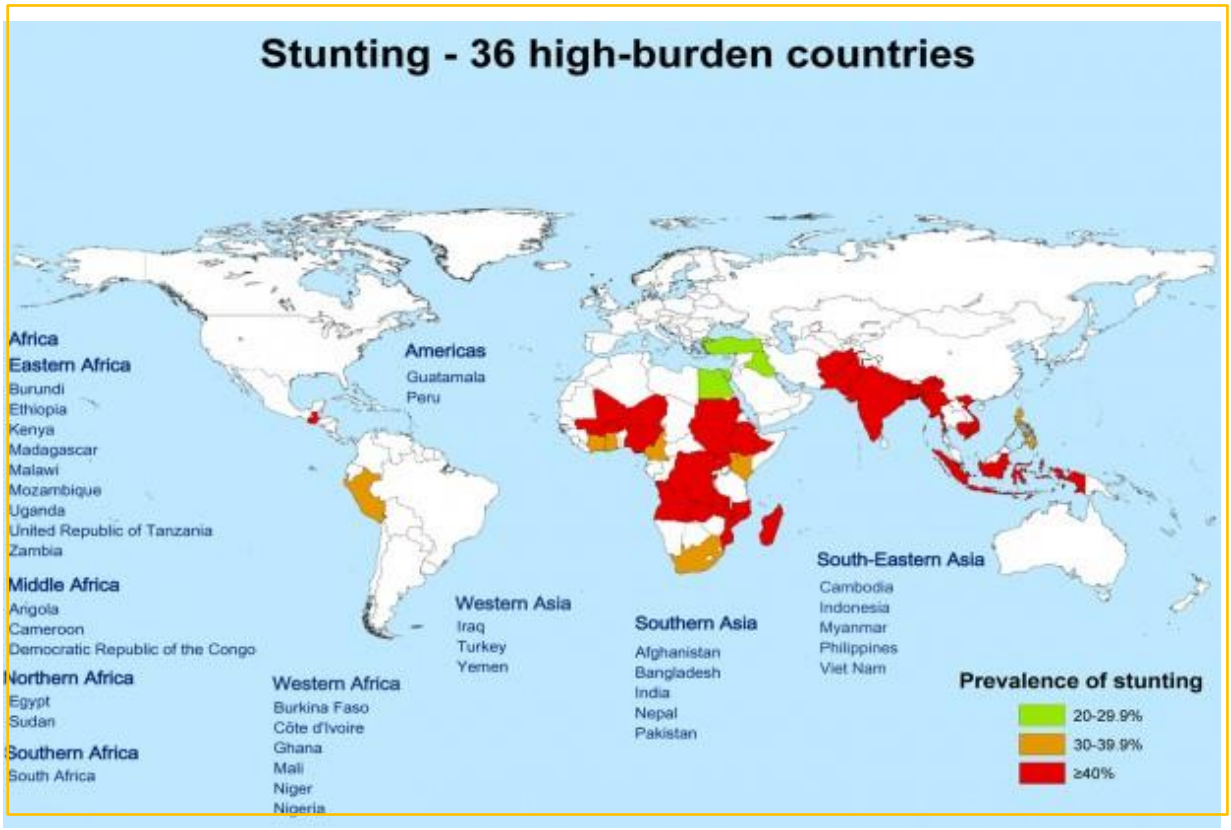
Child malnutrition in this document refers to undernutrition, which typically includes stunting, wasting, underweight, and intrauterine growth retardation (IUGR). According to the World Health Organization (WHO), stunting is defined by a height for age Z (HAZ) score of less than -2, wasting as weight for height Z (WHZ) score less than -2, and underweight as weight for age Z (WAZ) score less than -2. Growth conditions with HAZ, WHZ, or WAZ below -3 are defined as severe stunting, wasting or underweight, respectively. A baby with a term birthweight less than 2,500 grams is very likely to have been retarded during intrauterine growth (Black et al. 2008).

Complementary feeding is defined as the process starting when breast milk or infant formula alone is no longer sufficient to meet the nutritional requirements of infants, and therefore other foods and liquids are needed, along with breast milk or a breast-milk substitute. The start of complementary feeding is generally expected to occur beginning at six months (WHO 2013).

Black et al. (2013) estimated that malnutrition, including fetal growth retardation, stunting and wasting, deficiencies of vitamin A and zinc, and suboptimum breastfeeding practices, causes 3.1 million child deaths annually, with 45 percent of all child deaths in 2011 linked to these same causes. Malnutrition in the first 1,000 days of life in South Asia and sub-Saharan Africa has not declined proportionately in response to increases in economic growth, where an inverse relationship would be expected, and is often observed elsewhere. Additionally, nutritional supplementation programs have often not realized the anticipated improvements in nutritional status (Bhutta et al. 2008). This indicates that it is not simply a problem of food intake or income, but rather a multifactoral problem, involving the complex interaction of a number of variables related to diet, behavioral factors, health, and social and environmental conditions.

As of 2013, UNICEF estimated that 165 million children are stunted by the age of five. An additional 52 million worldwide are thought to be wasted (World Bank/UNICEF/WHO 2012). The *Lancet* nutrition review identified the 36 highest burden countries, most in Africa and South Asia (Figure 1). They account for 90 percent of children under five who suffer from malnutrition, and include Burundi, Kenya, Tanzania and Uganda.

Although worldwide prevalence of malnutrition in the first 1,000 days of life has greatly reduced in recent years, a decrease has not been observed in some of these high-burden countries (Figure 2).



Source: World Health Organization

Figure 1: 36 high burden countries for child stunting.

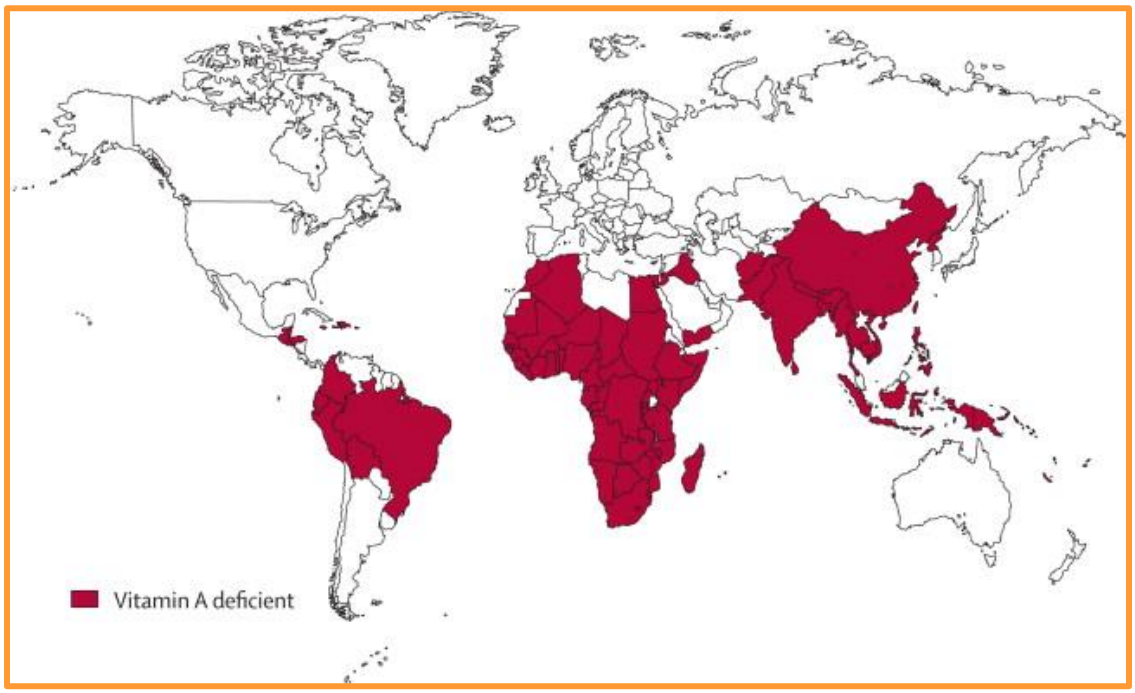
Pooled data from longitudinal studies suggests that child stunting, wasting, and underweight are associated with high mortality from common infectious diseases including malaria, and the risk increases as growth indicator Z score decreases (Black et al. 2013). Stunting (or linear growth retardation) is a reflection of long-term nutritional stresses owing to poor quality and quantity of diet and a high burden of infections, and as a result catch-up growth is insufficient (Richard et al. 2012). Stunted children have shown poorer cognitive and educational outcomes and behavior changes (Black et al. 2013). Because stunting is highly prevalent and has important consequences for health and development, it has been recommended that stunting should replace underweight as the main anthropometric indicator for childhood under-nutrition (Black et al. 2013).

Micronutrient deficiencies. It is estimated that 17 percent of the world population suffers from zinc deficiency based on food intake assessment (Wessells and Brown 2012), with Africa and Asia having the highest prevalence. This is likely due to low intake of animal products of which some low resource countries have inadequate supply. Furthermore,

persistent diarrhea and environmental enteropathy, which can affect absorption of micronutrients, are also risk factors for zinc deficiency (Lindenmayer et al. 2014).

Zinc deficiency has been shown to create a negative impact on the immune system and enhanced susceptibility to infectious diseases, especially in children (Ahmed et al. 2012). Encouragingly, evidence gathered from three meta-analyses (Brown et al. 2002 and 2009; Imdad and Bhutta 2011) shows that zinc supplementation can positively promote linear growth in childhood.

Vitamin A deficiency (VAD) is highly prevalent in developing countries. According to WHO (2009), 5 million children under five years old are estimated to have night blindness, a clinical condition of severe VAD, and 90 million to have subclinical VAD. Africa has the highest prevalence of VAD (41.6 percent), defined as having serum retinol concentrations below 0.70 umol/L. Although clinical symptoms have improved, largely due to supplementation, subclinical VAD has not decreased significantly in Africa (Black et al. 2013). VAD during pregnancy has been associated with lower birthweight (Gazala et al. 2003; Tielsch et al. 2008). However, randomized controlled trials failed to show any positive effect of vitamin A supplementation during pregnancy on birthweight (Thorne-Lyman and Fawi 2012); nor did supplementation during childhood improve growth performance. (Rivera et al. 2003).



Source: Black et al. 2008.

Figure 2: Prevalence of vitamin A deficiency in children under five.

Prevalence of anemia. Anemia is a condition characterized by a low level of hemoglobin in the blood. The common cause of anemia is nutritional deficiency due to inadequate

intake of nutrients necessary for synthesis of hemoglobin, such as iron, folate, vitamin B12, and riboflavin, especially during periods of increased requirements such as pregnancy and infancy. Iron deficiency is the most significant contributor to the onset of anemia. Anemia also results from sickle cell disease and increased blood loss due to parasitic infestation and infections such as malaria. Anemia in children is associated with impaired cognitive and physical development and increased morbidity and mortality. Anemia during pregnancy is a particularly serious problem and can lead to spontaneous abortion, premature delivery, low birthweight and even maternal mortality.

Anemia prevalences of ≤ 4.9 percent, 5.0-19.9 percent, 20.0-39.9 percent and ≥ 40 percent in a population are classified by WHO as normal, mild, moderate, and severe public health problems respectively (WHO 2001). Available data shows that anemia is a public health problem for children, pregnant women, and women of reproductive age in all East African countries, sufficient to impede initiatives to improve health and nutrition development during the 1,000 days period.

Table 1 presents prevalence of anemia among children aged 6-59 months and women of reproductive age (15-49 years). Prevalence of anemia in children aged 6-59 months is 38 percent in Rwanda and is higher in children from the other EAC partner countries, ranging from 44 percent in Burundi to 69 percent in Kenya. A similar pattern is noted for women aged 15-49 years; the prevalence ranges from 17 percent in Rwanda to 46 percent in Kenya. Anemia prevalence is the highest among children less than 12 months and declines with increasing age and is also higher in pregnant and lactating women than non-pregnant and non-breastfeeding women (ISTEEBU 2012; WHO 2008; NISR 2012; NBS and IFC Macro 2011; UBOS 2012). Given the consequences of anemia in health, social, and economic development, the prevalence of this condition should spur authorities to re-evaluate current strategies for control of anemia and ensure that all factors contributing to this problem are identified and addressed in an integrated approach.

Table 1: Prevalence of anemia in children, women of reproductive age, and pregnant women.

Country	Population	Anemia prevalence (percent)				Source
		Any anemia	Mild anemia	Moderate anemia	Severe anemia	
Burundi	Children 6-59 months	44.5	23.1	20.5	1.0	ISTEEBU, 2012
	Women 15-49 years[a]	18.5	15.0	3.2	0.3	
Kenya	Children 0-4.9 years	69.0	NA	NA	NA	WHO, 2008
	Women 15-49.9 years[b]	46.4	NA	NA	NA	
Rwanda	Children 6-59 months	38.1	24.2	13.5	0.5	NISR, 2012
	Women 15-49 years[c]	17.3	14.2	2.9	0.2	
Tanzania	Children 6-59 months	58.6	27.3	29.4	1.9	NBS and IFC Macro, 2011
	Women 15-49 years[d]	40.1	29.0	10.1	1.0	
Uganda	Children 6-59 months	49.3	22.3	25.5	1.5	UBOS, 2012
	Women 15-49 years	23.0	17.7	4.8	0.6	

[a] **Prevalence is higher in pregnant (26 percent) and breastfeeding women (20 percent) than in non-pregnant and non-breastfeeding women (16 percent)**

[b] **Prevalence is higher in pregnant women (55.1 percent) than non-pregnant women of reproductive age (46.4 percent)**

[c] **Prevalence is higher in pregnant women (20 percent) than non-pregnant women (17 percent)**

[d] **Prevalence is higher in pregnant women (53 percent) than breastfeeding, non-pregnant or non-breastfeeding women (39 percent)**

NA=data not available

Global Nutrition Programs

The Millennium Development Goals (MDG) defined eight international development goals to be achieved by 2015. Of these, eradicate extreme poverty and hunger (MDG1); reduce child mortality (MDG4); and improve maternal health (MDG5) are closely linked to child malnutrition issues. After the world food price crisis of 2007-08, international organizations and global communities realized that these MDGs cannot be achieved without urgent action to reduce global child malnutrition. The pledge for urgent action was further highlighted in the UN 2010 Summit. Reduction of childhood malnutrition will benefit communities in many ways, including boosting overall health for both children and women, raising national productivity, reducing child death and poverty, and empowering women to contribute to the society. The Scaling Up Nutrition (SUN) movement seeks to form global, regional, and national alliances, establish policies, and implement laws to align nutritional programs and mobilize resources.

To date, 56 countries—including all EAC partner states—have signed SUN agreements and developed action plans to implement specific nutrition-related strategies and programs. The specific actions for nutrition include exclusive breastfeeding up to six months, appropriate complementary feeding practices, food fortification, micronutrient supplementation, and treatment of acute malnutrition. Nutrition strategies under the SUN movement are:

- Make nutritious food more acceptable
- Provide clean water and sanitation
- Strengthen nutritional knowledge education
- Strengthen health care and social care
- Build a healthier population and a more sustainable society.

It is estimated that $25 billion will have been committed to the SUN movement by 2020. Objectives include a 40 percent reduction of stunting for children under five; 50 percent reduction in anemia among women of reproductive age; 20 percent reduction of low birthweight, an increase of at least 50 percent in exclusive breastfeeding in the first six months, and a reduction in childhood wasting levels to less than 5 percent (SUN 2014).

Aflatoxin Exposure in the First 1,000 Days

Exposure during pregnancy. Chronic aflatoxin exposure can occur in utero since aflatoxin crosses the trans-placental barrier (Autrup 1993). In The Gambia, aflatoxin exposure in pregnancy was associated with reduced body weight in babies (Turner et al. 2007). Aflatoxin exposure was reported to be higher in later pregnancy than early pregnancy (but only in the dry season) in a mother child cohort in The Gambia (Castelino et al. 2013).

Exposure in the first six months. breastfeeding is a period when aflatoxin exposure is less prevalent, as indicated by AF-alb biomarker data (Gong et al. 2003; Turner et al. 2007; Shirima et al. 2013). However, there is increasing evidence that breast milk contains AFM1 (Polychronaki et al. 2006; Adejumo et al. 2013). Nevertheless, AFM1, a hydroxylated metabolite found in milk, is less toxic than AFB1 found in food (IARC 2002), with estimated toxicity approximately 2–10 percent of the AFB1 (Zinedine et al. 2007). Therefore, exclusive breastfeeding during the first six months of life may help to reduce the negative health impacts associated with aflatoxin exposure.

Exposure at complementary feeding period. The complementary feeding period can be defined as the transition from breast milk to solid food, and in the EAC typically commences between three and six months (although WHO recommends the introduction of solid foods at six months). In the EAC, complementary foods are primarily cereal-based, and often include groundnut sauce and root crops. They are therefore prone to aflatoxin contamination. Consequently, the introduction of complementary food and family food marks a significant increase in aflatoxin exposure, as demonstrated by studies in The Gambia, Benin, and Tanzania (Gong et al. 2003; Turner et al. 2007: Shirima et al. 2013).

Figure 3 summarizes research suggesting that exposure to aflatoxin through diet shows an increasing trend up to three years of age. A child may have a different exposure profile than an adult who consumes the same food, i.e., a child could have higher exposure per kilogram body weight. Children may also be more susceptible to the toxic effects compared to adults, given the smaller body size of children and their dynamic physiological development. Children are not little adults (WHO 2008).

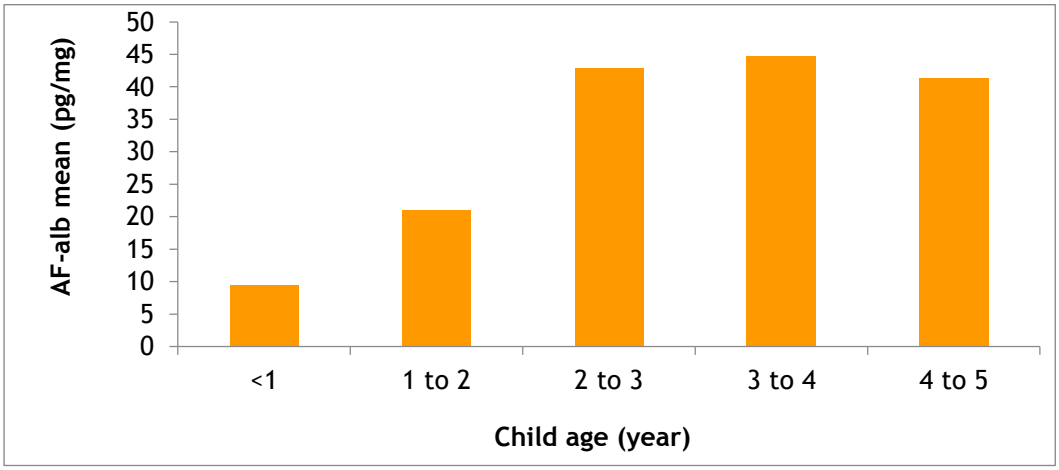


Figure 3: Aflatoxin exposure increases with age in children from Benin.

One study in Benin and Togo shows that children who were weaned had approximately double the mean AF-alb levels than children who were still partially breastfed (Gong et al. 2003). A study carried out in three geographically distant villages from Tanzania highlighted that introducing complementary food and family food significantly increased both aflatoxin and fumonisin exposure in children, with those who were fully weaned having the highest exposure to both mycotoxins (Shirima et al. 2013). This points to the need to consider multi-mycotoxin health risks in children in the design of future programs.

Aflatoxin-Related Nutrition and Health Outcomes

There is compelling evidence that aflatoxin exposure contributes to child stunting. Aflatoxin exposure in West African children aged 9-60 months was shown to be associated with stunting and wasting (Gong et al. 2002; 2003). In a follow-up study in Benin, aflatoxin exposure was associated with reduced height and weight gain over eight months in children aged 16-37 months at the start of the study (Gong et al. 2004). Aflatoxin exposure in utero has also been associated with low birthweight (Abdulrazzaq et al. 2004; Shuaib et al. 2010) and reduced height and weight gain in children under two years old (Turner et al. 2007).

It has also been hypothesized that aflatoxin may contribute to acute malnutrition, particularly protein-energy malnutrition (PEM) characterized by inadequate protein intake (Hendrickse 1984; Bain et al. 2013). Both PEM and aflatoxin exposure are prevalent in African children, particularly during the weaning stage, and prevalence rates are

influenced by season and geographical distribution in a similar way. In addition, the clinical and metabolic manifestations of kwashiorkor and aflatoxin exposure are similar, e.g., hypoalbuminaemia (lowered blood albumin), fatty liver, and immunosuppression (Hendrickse 1984, 1997). But a cause-and-effect relationship has not been confirmed.

It has been proposed that aflatoxin exposure can cause intestinal damage resulting in reduced nutrient absorption (Gong et al. 2008; Smith et al. 2012). It may be postulated, therefore, that the unsatisfactory outcomes from micronutrient supplementation trials may be attributable to aflatoxin exposure. Hence, by reducing aflatoxin exposure, the outcomes of supplementation trials should improve. However, no studies have yet directly examined the impact of aflatoxin on micronutrient supplementation, although a few have explored a possible association between aflatoxin exposure and micronutrient deficiency. Gong et al. (2004) found no significant correlation between vitamin A or zinc with AF-alb in a post-weaning study in Benin.

Turner et al. (2003), in their study of the effect of aflatoxin exposure on immune function in Gambian children aged six to nine years, also investigated the correlation between vitamin A and C with AF-alb levels. Vitamin C was the only micronutrient that demonstrated an inverse relationship with AF-alb. A more recent cross-sectional study of 147 Ghanaian adults found significant correlations between high AF-alb levels and low vitamin A concentrations in plasma samples (Obuseh et al. 2011). Participants with high AF-alb levels (>0.80 pmol/mg albumin) had a 2.6-fold greater risk of having lower vitamin A levels. Tang et al. (2009) also found in a sample of Ghanaian adults a significant negative correlation between aflatoxin B_1 albumin adduct (AF-alb) and vitamin A. However, no conclusive association between aflatoxin and micronutrients can be drawn from these limited data.

Aflatoxin exposure and associated health risks pose a potential threat to international efforts to combat malnutrition. Chronic aflatoxin exposure is evident in the first 1,000 days and throughout the whole lifetime (Wild and Gong 2010). It is, therefore, a reasonable assumption that aflatoxin exposure is an exacerbating factor of malnutrition, and may have hindered nutrition improvements in many donor- and government-supported nutritional intervention programs. It appears that efforts to improve child health through tackling malnutrition may not be successful unless the aflatoxin problem is also addressed (Smith et al. 2012).

Situational Analysis

Background: Child Malnutrition in EAC

Stunting, which reflects the failure to receive adequate nutrition over a long period, is a major childhood nutritional problem across East African. The prevalence of stunting has been unacceptably high and increases with the age of a child up to 23 months, with little change from 24–59 months.

All the EAC countries are listed among countries with the highest burden of childhood stunting in the world. Table 2 summarizes prevalence of stunted, underweight, wasted, and low birthweight children by country. Nationally, stunting prevalence in children under the age of five years is 58 percent in Burundi, 35.2 percent in Kenya, 44.2 percent in Rwanda, 42 percent in Tanzania, and 33.4 percent in Uganda. The corresponding prevalence of children with low weight for age for each country is 28.8, 16.1, 11.4, 15.5, and 13.8 percent, respectively.

However, the levels of acute malnutrition (wasting) are relatively low, ranging from the highest (6.7 percent) in Kenya to the lowest (3.0 percent) in Rwanda.

Table 2: Prevalence of malnutrition in children under five years old.

Country	Stunted	Severe stunting	Underweight	Severe underweight	Wasting	Severe wasting	Low birth weight	Source
Burundi	58.0	27.0	28.8	7.8	5.8	1.4	11	ISTEEBU (2012)
Kenya	35.2	14.3	16.1	3.6	6.7	1.9	6	KNBS and IFC Macro (2010)
Rwanda	44.2	17.0	11.4	2.3	3.0	1.0	7	NISR (2012)
Tanzania	42.0	16.5	15.5	3.6	4.8	1.2	8	NBS and IFC Macro (2011)
Uganda	33.4	13.7	13.8	3.4	4.7	1.5	10.2	UBOS (2012)

Weight of the child at birth is another important determinant of infant and child health and mortality. The prevalence of low birthweight, defined as less than 2.5 kg at birth, is 11 percent in Rwanda, 6 percent in Kenya, 7 percent in Rwanda, 8 percent in Tanzania, and 10.2 percent in Uganda.

For all types of malnutrition, prevalence is substantially higher in rural than urban areas (ISTEEBU 2012; KNBS and ICF Macro 2010; NISR 2012; NBS and IFC Macro 2011; UBOS 2012).

During recent years, there have been decreased trends of prevalence of malnutrition in East African countries, attributed to implementation of strategies for improvement of nutritional status. Nevertheless, the prevailing high rate of stunting remains a major public health concern. A substantial number of children also suffer from other forms of malnutrition, including anemia and vitamin A deficiency which affect 49 and 38 percent, respectively, of children aged 6–59 months in Uganda (UBOS 2012).

The prevailing state of malnutrition demonstrates that nutrition strategies may have not accurately identified and addressed other factors that have an adverse impact on health and nutrition, such as exposure to mycotoxins, especially aflatoxin.

Childhood SStunting

In the last two decades, the prevalence of stunting in children under five years old has decreased elsewhere in the world but remained high in sub-Saharan Africa and South Asia

(Black et al. 2013). Nutrition-specific interventions, including micronutrient supplementation and fortification, as well as complementary feeding, have reduced stunting only to a certain extent (Lutter et al. 2011). It is likely that other factors, including aflatoxin exposure, may play a critical role in child stunting.

It is evident that aflatoxin exposure in humans occurs throughout life, including during gestation (Wild and Gong 2010). Aflatoxin exposure in utero not only effects birthweight but may also play a role in stunted growth in early childhood (up to 24 months). Higher levels of AF-alb in maternal blood are significantly associated with lower weight and lower height gain (Turner et al. 2007). Furthermore, it has been predicted that a reduction in maternal AF-alb level from 110 pg/mg to 10 pg/mg would lead to a 2 cm increase in height and a 0.8 kg increase in weight within the first 24 months of life. A strong negative correlation between levels of AFM1 in cord blood and maternal serum with birth weights was observed in the United Arab Emirates (Abdulrazzaq et al. 2004) and low birth weight was seen in children of Ghanaian women with higher AF-alb level (Shuaib et al. 2010).

Because the first two years after birth is a fast growth period, the impact of aflatoxin exposure on growth is most critical during this period. In a cross-sectional study of 479 children aged between nine months and five years (Gong et al. 2002, 2003), 33 percent, 29 percent, and 6 percent of the children were found to be stunted (height for age Z score HAZ<-2), underweight (weight for age Z score WAZ<-2), and wasted (weight for height Z score WHZ<-2), respectively. These definitions are based on WHO criteria (WHO 2006). Significant negative correlations between AF-alb and each of the growth parameters were observed. Another study by Turner et al. (2003) found that AF-alb levels were weakly associated with wasting, but not with stunting or underweight. Other cross-sectional studies examined this relationship (Mahdavi et al. 2010; Okoth and Ohingo 2004).

Gong et al. (2004) examined the effects of aflatoxin exposure on growth in 200 children from Benin (16-37 months old) followed up for eight months. AF-alb levels inversely correlated with stunting and wasting. There was a difference in height of 1.7 cm over the eight-month period between the highest and lowest AF-alb quartile. This shows a temporal relationship indicating a potential causal effect.

Although the mechanisms that link aflatoxin exposure with impaired child growth are yet to be defined, it has been hypothesized (Gong et al. 2008; Smith et al. 2012) that: 1) aflatoxin-induced intestinal epithelium damage may contribute to EE; 2) aflatoxin-associated immune suppression could increase children's susceptibility to infections such as diarrhea; 3) liver toxicity of aflatoxin may reduce

the production of insulin-like growth factors pathway proteins (IGFs) in the liver, leading to reduced IGFs in circulation and an adverse impact on child growth. Indeed, reduced levels of IGFs due to aflatoxin exposure have been demonstrated by recent data in vitro and in vivo (Castelino et al. 2014a).

It is worth noting that the observed effects of aflatoxin on growth could in part be due to effects of other mycotoxins that co-contaminate crops.

Exposure to Aflatoxin During Pregnancy

Aflatoxin metabolites have been found in serum samples of pregnant women and in cord blood of newborns, suggesting dietary exposure to aflatoxin during fetal development. Studies in Kenya found aflatoxin B_1 and its metabolites in 53 percent (n=125) (De Vries et al. 1989) and 77 percent (n=26) (Yard et al. 2013) of the blood samples from pregnant women. The toxins were also detected in 53 percent of pregnant women at delivery (n=59) and in 37 percent of neonate cord blood (n=101); one study also recorded two stillbirths with aflatoxin detected in maternal and cord blood (De Vries et al. 1989).

In Kenya and Uganda, aflatoxin was detected in serum or urine samples of the adult population (Autrup et al. 1987; McCoy et al. 2008; Asiki et al. 2014). Although the physiological status of female subjects was not stated, the findings imply a potential for aflatoxin exposure during pregnancy. Exposure to aflatoxin during pregnancy has also been shown in Nigeria (Abulu et al. 1997), Sierra Leone (Jonsyn, 1998), The Gambia (Turner et al. 2007), and Ghana (Shuaib et al. 2010).

Exposure of Infants to Aflatoxin in the First Six Months

During the first six months of life, infants may be exposed to AFM1 through breast milk and to other aflatoxins if lactating mothers give their infants foods in addition to breast milk. In a study conducted in Tanzania in 2013, AFM1 was detected in samples of breast milk from lactating mothers of infants less than six months old (Magoha et al. 2014). More than 90 percent of breast milk samples tested in this study were found to contain AFM1 at levels exceeding the European Union (EU) limit of 0.025ng/ml set for AFM1 in infant foods, and more than 76 percent of samples exceeded the EU limit of 0.05ng/ml set for AFM1 in dairy milk and milk products. This suggests that breastfed infants are regularly exposed to aflatoxin through breast milk. Exclusive breastfeeding for the first six months from birth is a global public health recommendation to achieve optimal growth, development, and health (Black et al. 2013), hence efforts should be made to ensure that lactating mothers consume only aflatoxin safe foods. breastfeeding is safer than early weaning because AFM1 in milk is less toxic than AFB1 found in traditional complementary foods (IARC 2002). The rates of early introduction of foods in infants less than six months old are very high in the EAC. These are up to 50 percent in Tanzania (NBS and ICF Macro. 2011), 68 percent in Kenya (KNBS and ICF Macro 2010), 37 percent in Uganda (UBOS 2012), and 31 percent in Burundi (ISTEEBU 2012). In Rwanda, this proportion is lower, at 15 percent (NISR 2012).

Dietary Exposure to Aflatoxin Beyond Six Months

Aflatoxin exposure assessments conducted for children fed with complementary foods in the EAC region suggest that exposure from complementary food is common and increases with age. This is exemplified by a biomarker-based assessment conducted in several rural villages of Uganda and Tanzania, which found AF-alb adducts in plasma samples from young children.

AF-alb levels in blood samples from Tanzanian children at recruitment (aged 6–14 months) and at six–12 months later have recently been reported (Shirima et al. 2013; Routledge et al. 2014). AF-alb was detected in 84 percent of children (n=146) at six months following recruitment and in, almost all (99 percent) at 12 months following recruitment.

This exposure increased with child age and was higher in children who were fully weaned than in those partially weaned (Shirima et al. 2013). Biomarker levels were significantly correlated with dietary exposure levels for these infants (Routledge et al. 2014) confirming that the main source of aflatoxin in the children is the complementary food. In Uganda, AF-alb was detected in blood samples in 96 percent of children aged zero to three years old (n=96). The toxin was also detected in five children who were reported to be on exclusive breastfeeding during the time of survey, although the levels were less than half that of the children who were not exclusively breastfed (Asiki et al. 2014). Among the children who were on complementary feeding, mean AF-alb levels were significantly higher in those who were given maize-based foods. This again suggests that maize is the main source of dietary exposure. The common complementary foods used in East Africa are those locally grown and processed, and prepared at home (Dop and Benbouzid 1999). Since such foods are widely produced and consumed without any external inspection to regulate quality control, and because some of them are prone to aflatoxin contamination, they are a potentially hazardous source of child exposure during the period of complementary feeding. This further strengthens the rationale that exclusive breastfeeding for the first six months and continued breastfeeding during complementary feeding, as recommended by WHO, serve as important measures for reducing aflatoxin exposure in children during the period of complementary feeding. Table 2 summarizes aflatoxin exposure during the first 1,000 days of life in the EAC countries.

Aflatoxin Exposure from Complementary and Family Foods

The degree of aflatoxin exposure is a function of the concentration of aflatoxin in the food and the amount of contaminated food consumed by an individual (Shephard 2008). Maize and groundnuts, two of the most aflatoxin contamination prone crops, (Fung and Clark 2004), are dietary staples in East Africa. In tandem with high levels of on-farm consumption, combined with a majority of small holder subsistence farming and informal trading systems, these commodities are widely consumed without adequate inspection. As a

consequence, aflatoxin exposure throughout the human population is pervasive. Measurements of aflatoxin in maize and groundnuts in Uganda (Kaaya and Warren 2005; Kitya et al. 2010), Kenya (Lewis et al. 2005; Daniel et al. 2011), Tanzania (Kimanya et al. 2008; Rushunju et al. 2013) and Rwanda (Nyinawabali 2013; RBS 2014) revealed contamination levels exceeding maximum limits of 5 µg/kg and 10 µg/kg for AFB1 and total aflatoxin, respectively. Within the East Africa region, maize is the dominant staple food, with a high annual per capita intake of 112.5 kg in Tanzania (Nkonya et al. 1998) and 98.0 kg in Kenya (Kang'ethe 2011). Maize is also a major ingredient of complementary foods for children, widely prepared and consumed in the form of porridges. In Uganda, 89 percent (n=261) of children were reported to eat maize porridge as their usual diet (Kikafunda et al. 2003). Other studies in the region have reported that cereal porridge with maize as an ingredient is commonly used as a complementary food for children (Onyango et al. 1998; Dop and Benbouzid 1999; Harvey et al. 2010; Mamiro et al. 2011). Other ingredients that may often be included in porridge are sugar, cow's milk (fresh or fermented), oil, and groundnut paste/flour, adding two additional aflatoxin prone foods, groundnut and milk, to dietary consumption within the 1,000 days.

Groundnut, typically used in composite flour for porridge, is also added in sauce dishes or eaten as a snack (Dop and Benbouzid, 1999; Kikafunda et al. 2003; WFP 2008).

As maize and groundnuts are the foods with high risk for aflatoxin exposure, the control of aflatoxin contamination in maize and groundnuts and dietary diversification are a key beginning point for minimizing exposure to aflatoxins during the 1,000 days.

Breast milk provides the best nutrition during infancy; it also provides the child a period of protection from highly contaminated family food. Mothers need to be supported and encouraged to practice exclusive breastfeeding in the first six months after birth and continue breastfeeding during complementary feeding period for the purpose of child nutrition and for reducing aflatoxin exposure risk.

Table 3: Aflatoxin exposures in pregnant women, breastfeeding mothers, neonates, infants, and young children in East Africa.

Target group	N	Biomarker	Positive (&) percent)	Exposure range (min-max) unless stated	Country
Women at pregnancy	125	AFM1/ AFM2, AFB1 in blood	53	AFM1/AFM2: 12-1689 pg/ml AFB1: 87-11574 pg/ml	**Kenya** De Vries et al. 1989
Women during delivery	59	AFM1/ AFM2, AFB1 in blood	53		
Neonate	101	AFM1/ AFM2, and AFB1 in cord blood	37	AFM1/AFM2: 17-656 pg/ml AFB1: 86-6819 pg/ml	
Women at pregnancy	26	Aflatoxin B1 lysine in serum	77	AFB1-lysine (<LOD - 10.5 pg/mg albumin	Yard et al. 2013
Lactating mothers	143 121 118	AFM1 in breast milk collected at 1st month 3rd month 5th month after delivery	100 100 100	0.01-0.55 ng/ml 0.01-0.47 ng/ml 0.01-0.34 ng/ml	**Tanzania** Magoha et al. 2014
Infants	118	AFM1 through breast milk	NA	0.81-66.79 ng/kg body wt/day*	
Infants and young children**	148 146	AF-alb in blood (12-20 month) (18-26 month)	84 99	12.9 (9.9-16.7) pg/mg 23.5 (19.9-27.7) pg/mg	**Tanzania** Shirima et al. 2013; Routledge et al. 2014
Infants and young children**	96	AF-alb in blood	96	9.7 (8.2-11.5) pg/mg	**Uganda** Asiki et al. 2014

***Infant exposure to AFM1 was estimated based on level of AFM1 in mother's breast milk and standardized quantity of breast milk intake per day for infants of his/her age.**
****AF-alb levels are in geometric mean with 95 percent confidence interval.**
LOD = Limit of detection.

Table 3 summarizes findings of aflatoxin exposure during the first 1,000 days from the studies conducted in Kenya, Tanzania, and Uganda. It describes evidence of exposure from biomarker studies conducted during pregnancy, at delivery, and in neonates, infants, and young children. Table 3 also shows the total number of samples (n) that were analyzed for each study, the type of aflatoxin exposure biomarkers (AF-alb, AFM1, or AFM2) analyzed, and the percentage of samples detected with the aflatoxin metabolites. Detection of aflatoxin metabolites in biological samples suggests that the studied populations were exposed to the toxin through consumption of contaminated foods.

Other Sources of Aflatoxin Exposure in the EAC

As previously explained, when an individual or animal consumes AFB1-contaminated food, the toxin can be metabolized and excreted in milk as AFM1. In Kenya, AFM1 was detected in 72 percent (n=439) of cow milk samples from Eldoret, Machakos, Nakuru, and Nyeri. About 35

percent of the positive samples had contamination levels exceeding 0.05µg/kg, the FAO/WHO and EU permissible level of AFM1 for milk (Kang'ethe and Lang'a 2009). In Tanzania, 92 percent of samples of fresh cow milk retailed in Dar es Salaam city were found to contain AFM1 (Urio et al. 2006). Contamination of cow's milk with aflatoxin is a serious food safety concern because milk is an important component of child diets (Deveci and Sezgn 2005). In Uganda, 62 percent of children were reported to start complementary feeding with cow's milk (Kikafunda et al. 2003). In Kenya, 90 percent of mothers feed their infants cow's milk during complementary feeding (IYCN 2009). Fresh or fermented milk is also added in porridge.

Since the route of AFM1 contamination in animal milk is through contaminated feeds, measures to control this mode of human contamination should focus on preventiing cattle from consuming contaminated feed. This can be achieved through the establishment and enforcement of standards for animal feeds, as well as appropriate standards for milk and milk products based on high levels of sensitivity to aflatoxins during the 1,000 days and beyond into childhood.

Another important measure is creating awareness of the occurrence of AFM1 in milk and the possible means for its management on the farm. Further, in addition to the contamination of locally produced milk, AFM1 contamination has also been found in processed milk (milk powder and pasteurized milk) in some other countries (van Egmond et al. 2005), underscoring the need for regulating AFM1 in imported milk-based products, especially those intended for infants and young children.

Studies have indicated that aflatoxins are detectable in poultry products, including eggs, gizzard, liver, and muscles (Trucksess et al. 1983; Herzallah 2009; Tchana et al. 2010), demonstrating that these products can be another source of aflatoxin exposure. This emphasizes the importance of interventions to protect against animal exposure to aflatoxin and to monitor the toxin and its metabolites in animal-derived food products. High levels of aflatoxins have been detected in cured fish in Tanzania (Mugula and Lyimo, 1992) and Kenya (Okoth and Ohingo 2005), cassava in Tanzania (Manjula et al. 2009), millet, sorghum, and cassava in Uganda (Kaaya and Warren 2005; Kitya et al. 2010) and Kenya (Okoth and Ohingo 2005). Farmed fish fed with contaminated feed will also contain high levels of aflatoxins. This demonstrates that regulation of aflatoxin levels in a number of frequently consumed commodities other than maize and groundnuts is required.

In addition to maize, groundnuts, and animal milk, a ll highly susceptible to aflatoxin contamination, other common types of complementary foods include banana (popular in Uganda and also in some parts of other EAC countries), potatoes, rice, millet, wheat, cassava, fruits, and vegetables. Also included are meat, poultry, eggs, fish, and beans. As some of these foods are less susceptible to aflatoxin contamination with appropriate on-farm and postharvest management practices, with adequate assessment, they hold the potential

for minimizing aflatoxin exposure through dietary diversification. Since these foods also provide higher levels of the protein and micronutrients essential for child growth, additional nutritional benefits can be realized.

National Initiatives for Aflatoxin Abatement

Aflatoxins are human liver carcinogens, especially in combination with the hepatitis B virus (HBV) (IARC 2002). HBV vaccination is crucial to reduce the global burden of hepatocellular carcinoma (HCC) and since the virus is often transmitted from mother to child, vaccination at an early age is important (Kensler et al. 2003). At present, children in East Africa are immunized against hepatitis B in accordance with the WHO guidelines. Other childhood vaccines are also given to protect against tuberculosis, diphtheria, pertussis, tetanus, polio, measles, and haemophilus influenza. Immunizing children against vaccine-preventable diseases can greatly reduce childhood morbidity, HCC in later life, and other effects associated with aflatoxin exposure. More than 74 percent of children are fully immunized across all East African countries (NBS and ICF Macro 2011; KNBS and ICF Macro 2010; ISTEEBU 2012; NISR 2012; Uganda-EPI 2011). Measures to vaccinate other vulnerable groups, such as women, as well as monitoring the impact of the HBV vaccine in the mitigation of HCC, are worth consideration. As part of their initiative to address aflatoxin issues in the region, recently, the EAC Ministerial Health Council endorsed expanded HBV vaccination programs and the introduction of the hepatitis A vaccine.

In East Africa, there have been several interventions to improve vitamin and mineral nutrition, which are targeted to address nutritional problems of public health significance and emerging challenges that have the potential for being a significant barrier to human development. These interventions are prioritized for children under five years and women of reproductive age due to their high vulnerability to malnutrition.

The interventions being implemented in the EAC region include:
1) vitamin and mineral supplementation for groups at risk to produce rapid improvement of their micronutrient status;
2) food fortification, including salt iodation, and fortifying other selected foods, such as oil, maize flour, and wheat flour with vitamins and minerals;
3) growing nutritionally biofortified crop varieties; and
4) interventions based on dietary practice changes.

These interventions are implemented under established national strategies and policies related to nutrition, food security, food safety, and nutrition and HIV strategies; they can also be important in abating the effects of aflatoxin.

Policy Recommendations

1. Collaborate with the Scaling Up Nutrition movement and Millennium Development Goals policy and program leaders to design and integrate food safety goals and objectives that include aflatoxin abatement measures for the 1,000 days, for both mothers and children.
2. Work with national governments and the EAC to include aflatoxin control activities in SUN, MDG, and other food security and nutrition policies and programs for the 1,000 days population.
3. Continue to promote exclusive breastfeeding for the first six months of life to minimize and delay the premature introduction of harmful levels of aflatoxin in complementary foods.
4. Design and implement beneficiary-specific nutrition-education modules for reduced aflatoxin ingestion during the 1,000 days, working with ministries of health and agriculture.
5. Through ministries of health, train midwives, trained birth attendants, community health aids, nurses, nutritionists, and doctors who are primary care givers for pregnant and lactating women and their young children.
6. Working through ministries of agriculture, livestock and fisheries, provide extension services on good agricultural practices to reduce aflatoxin contamination, especially for on-farm consumption.
7. Develop a special curriculum to train women and caregivers on improved storage and handling practices to reduce postharvest aflatoxin growth, especially for complementary and early foods.
8. Conduct national reviews of current dietary recommendations for vulnerable groups that may be inadvertently advocating consumption of aflatoxin-prone foods during the 1,000 days.
9. Through the EAC five -year communications strategy, promote dietary diversity for pregnant and lactating women as an affordable and rapid response mechanism to reduce the transmission of aflatoxin to infants and unborn children.
10. Develop rapid response programs that coordinate with the donors, nongovernmental and other organizations to provide food assistance to pregnant and lactating women, infants and young children in areas affected by seasonal aflatoxin contamination spikes.
11. Advocate for research to better quantify the impacts of aflatoxin on morbidity and mortality outcomes during the 1,000 days, especially stunting and pediatric AIDS.
12. Facilitate coordination among the One Health initiative, ministries of health, ministries of trade and industry, private sector food processors and importers, and regional economic organizations to ensure that the regulatory environment and standards for food and feed adequately address the special needs of the 1,000 days population and other vulnerable groups.

List of Abbreviations and Definitions

Term	Definition
AF-alb	Aflatoxin albumin adducts
AFB1	Aflatoxin B1
AFM1	Aflatoxin M1
BCC	Behavior change communication
EAC	East African Community
FAO	Food and Agriculture Organization of the United Nations
HAZ	Height for age Z
HBV	Hepatitis B virus
HCC	Hepatocellular carcinoma
HCV	Hepatitis C virus
IARC	International Agency for Research on Cancer
ISTEEBU	Institut de statistiques et d'etudes economiques du Burundi (Institute of Statistics and Economic Studies of Burundi)
IYCN	Infant and Young Child Nutrition
MDG	Millennium Development Goals
NBS	National Bureau of Statistics
NISR	National Institute of Statistics of Rwanda
PEM	Protein Energy Malnutrition
RBS	Rwanda Bureau of Standards
SUN	Scaling Up Nutrition
UBOS	Uganda Bureau of Statistics
UNICEF	United Nations Children's Fund
VAD	Vitamin A deficiency
WFP	World Food Program
WAZ	Weight for age Z
WHZ	Weight for height Z
WHO	World Health Organization

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