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Commentary

EFFECT OF MATERNAL AFLATOXIN EXPOSURE ON GROWTH AND MORBIDITY OF INFANTS 0 TO 3 MONTHS

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Effect of Maternal Aflatoxin Exposure on Growth and Morbidity of Infants 0 to 3 months

Aflatoxin exposure has been implicated in adverse human health outcomes in areas where the toxin is prevalent in foods [1]. Consequently, many people may be chronically exposed to large amounts of the toxin worldwide. The effect of aflatoxin exposure is felt more in developing countries and this may be attributed to high levels of malnutrition coupled with chronic exposure to high levels of the toxin in their diets [2]. Consumption of high levels of aflatoxins has been associated with aflatoxicosis resulting in death, while chronic dietary exposure to aflatoxins is associated with liver and other cancers and poor growth outcomes in young children [3]. Exposure of infants to aflatoxins may be detrimental to their normal growth and other aspects of health later in life. Early linear growth retardation is associated with poor cognitive development, reduced physical productivity, a greater risk of poor pregnancy outcomes later in life, including low birth weight babies and a greater risk of obesity, coronary heart disease, diabetes and hypertension later in life [3]. Therefore, protection of fetuses, infants and young children from the effects of aflatoxins through maternal exposure would not only reduce the risk of them not realizing their cognitive potential, but also contribute to reducing the risk of non-communicable diseases whose prevalence is on the rise, not just in developed countries, but also in developing countries.

Most early studies on aflatoxin focused on its association with cancer [4] and were mainly conducted in developed countries where exposure to the toxin is low. In developing countries, where exposure ranges from low to high, attention to effects of aflatoxins has majorly focused on aflatoxicosis, which involves extreme, acute exposure to the toxin over a short period of time. This is mainly because aflatoxicosis may result in immediate severe outcomes including death, hence is likely to attract attention. Chronic exposure to aflatoxins and its effects has received little attention, and only recently, have concerns that were raised by researchers such as Wild *et al.* [4], been given due attention, especially in developing country contexts. This notwithstanding, evidence has pointed to the need for concern about exposure to aflatoxins in developing countries where it is evident that populations are exposed to varying levels of the toxins, for extended periods of time and not just high exposure for short periods of time resulting in aflatoxicosis.

Effect of Maternal Aflatoxin Exposure on Infant Growth

Young children may be at risk of aflatoxin exposure right from the time of conception through to complementary food period [5]. Exposure of the women to the toxin may, therefore, be a risk factor for infant aflatoxin exposure through breast milk. Most young children in developing countries are weaned onto maize

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and other cereal based porridge, which may expose them to aflatoxins through diet early in life. Evidence from Kenya [6] and Tanzania [7] reveals that children are breastfed until at least the latter part of the second year, but begin to receive cereal-based gruel before the age of 3 months. This means that infants risk double exposure to aflatoxins; from maternal milk as well as from early cereal-based complementary feeds. Studies indicate that children are more vulnerable to effects of aflatoxin exposure than their mothers and are also more likely to die from aflatoxin poisoning compared to adults [8]. This has been linked to lowered capacity for biotransformation of carcinogens in infants than in adults, which may result in longer circulation time of the chemical. Findings from the study carried out in Kisumu, Kenya, indicated that infants of women exposed to aflatoxin weighed less, and were shorter than infants of non-exposed women; and were at higher risk of stunting in the first 3 months of life [9, 10]. This points to a possible relationship between maternal aflatoxin exposure and infant growth.

Effect of aflatoxin Exposure on Infant Stunting (LAZ), Underweight (WAZ), and Wasting (WLZ)

Several studies have supported a possible association between aflatoxin exposure and reduced stunting, underweight and wasting in young children: a study carried out in Benin and Togo confirmed a dose-response relationship between aflatoxin exposure and the degree of stunting and underweight in children <5 years old [11]; a prospective study carried out in Tanzania to investigate the association between child growth and aflatoxin exposure revealed a prevalence of stunting of 44%, 55% and 56% at recruitment, 6th and 12th months after recruitment, respectively [12]; a longitudinal study by Obade and colleagues in Kisumu County established an effect between maternal aflatoxin exposure and infant growth during the critical period of child growth in the first 3 months of life [9, 10]. The study found a higher risk of stunting and underweight, but not wasting, in infants of women who were exposed to aflatoxin, compared to infants who were not exposed. Most studies have found a relationship between aflatoxin exposure and stunting, but not with underweight and wasting. There are conflicting findings reported on association between aflatoxin exposure and infant growth, especially underweight and wasting, suggesting for more research in that area.

Effect of Aflatoxin Exposure on Infant Morbidity

A report by WHO Expert Group Meeting in 2005 indicated that exposure to aflatoxins may be a causative factor in neurological impairment, immunosuppression and child mortality [13]. Aflatoxin exposure is also associated with delayed rate of recovery from protein malnutrition, due to its effect on protein synthesis and increased infections in young children[14]. It has also been reported

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that children who suffer from acute malnutrition, have been found to be prone to the hazards of dietary aflatoxins [15].

Exposure to aflatoxin in contaminated food results in suppression of the cell-mediated immune responses, due to the effect of the toxins on factors responsible for production of lymphokines and antigen processing by macrophages [16]. Macrophages play a major role in host defenses against infection by presenting antigen to lymphocytes during the development of specific immunity and serve as supportive accessory cells to lymphocytes. Macrophages also increase their phagocytic activity and release various active and reactive intermediates, to carry out nonspecific immune responses. In vitro studies have shown mycotoxins to inhibit phagocytic cell function in normal human peripheral blood monocytes, resulting in reduced immunity [17].

It has also been established that the status of micronutrients in the human body may be compromised by aflatoxin exposure [2]. Vitamin A has been found to play a protective role in human lymphocytes by inhibiting generation of aflatoxin B_1 (AFB_1)-induced reactive oxidative species. This may result in lower levels of retinol in individuals who are exposed to aflatoxin due to detoxification of aflatoxin derivatives that are potentially carcinogenic, by the micronutrient. The resulting vitamin A deficiency may increase the biological exposure to aflatoxin contamination [2], increasing liver AFB_1 microsomal activity, conversion of AFB_1 to its reactive metabolite and formation of DNA adducts. A study carried out among Ghanaian participants recorded a significant negative correlation between aflatoxin B_1 albumin adducts and vitamin A and vitamin E levels [18]. Such exposure may also affect micronutrient status in infants resulting in reduced immunity and increased morbidity. Research findings indicate that vitamins A and E supplementation significantly reduces aflatoxin induced toxicity and carcinogenesis, compromising the intended purposes of the nutrients [18]. Aflatoxin exposure may also have an effect on availability of dietary zinc and selenium [19] which have antioxidant properties and are also essential for healthy immune function. Given the probable low-to-high exposure to aflatoxin in Kenya and other developing countries, efforts to improve micronutrient status of young children such as vitamin A supplementation and diet diversification may be compromised by aflatoxin exposure. Maternal exposure to aflatoxin, therefore, exposes the infant to the toxins, affecting the health of the infant by interfering with micronutrients status in the body.

Kisumu County Integrated Development Plan (KCIDP) 2013-2017, highlights main causes of sicknesses in the County as malaria, fever, common cold (flu) and diarrhea [20]. Malaria is a common illness among infants and young children in developing countries and an endemic disease in the African region, and is a leading

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cause of morbidity and mortality, especially in children [21]. Exposure to aflatoxins has been associated with increased risk of diarrheoa and pneumonia in children [21], but not malaria. Results of crude bivariate analysis from the longitudinal study carried out in Kisumu County showed that maternal aflatoxin exposure was associated with malaria and diarrhea, but not with other infections [9].

The mechanism by which aflatoxin affects infant morbidity is not clear, while data on clinical aflatoxicosis in humans is still limited, there is substantial evidence on human exposure in many areas of the world [15]. Aflatoxin exposure may result in altered intestinal integrity through cell toxicity or immunosuppression, making the body susceptible to infections resulting in intestinal malabsorption resulting and reduced food efficiency in infants [11]. A study carried out in the Gambia, revealed that aflatoxin exposure led to a reduction in salivary secretory immunoglobulin A (sIgA) in children resulting in alteration of the mucosal barrier and affecting resistance to intestinal infections [16]. Exposure to aflatoxin may also cause impairment of the intestinal mucosa, which may affect proper absorption and utilization of food in the body resulting in cases of diarrhea and other infections. Aflatoxin exposure has been associated with environmental enteropathy (EE), a sub clinical condition of the small intestine characterized by reduced absorptive capacity and increased intestinal permeability, which may be a cause of stunting [22].

The aforementioned views reveal the possible effect of aflatoxin exposure on infant morbidity and the need for more research and interventional measures. Therefore, although the role of aflatoxin in morbidity remains unclear, there are potential mechanisms that may support such a role. Further, data on association between aflatoxin exposure and infant morbidity is inadequate globally, in Kenya and in Kisumu County.

The study by Obade and colleagues [10] concluded that maternal exposure to aflatoxin increases risk of reduced weight, length, as well as stunting and underweight in infants, but not risk of wasting. The study also established that maternal aflatoxin exposure increases risk of malaria and diarrhea, but not other infections. Therefore, there is need to identify and address sources of aflatoxin contamination in foods in Kenya and the world at large. Further, stringent routine surveillance of foods in the market outlets should be enhanced to ensure quality of foods consumed by all individuals to protect populations, and especially mothers and infants from the negative health effects of aflatoxins.

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