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AFLATOXIN EXPOSURE IN CHILDREN AGED 4 MONTHS TO 5 YEARS FROM SUB-SAHARAN AFRICA AND ITS EFFECT ON STUNTED CHILD GROWTH – A TOPICAL REVIEW

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ABSTRACT

The mould species fungi encompass among others the family of *Aspergillus* which produce and secrete a number of compounds toxic to human health, namely the aflatoxins. These mycotoxins are converted to secondary metabolites that exert the often detrimental biological effect, and they are commonly found in various foods such as fruits, nuts, seeds, and grains. Members of the *Aspergillus* family thrive in wet and humid conditions, so they are particularly prevalent in regions with a tropical climate, for example in countries in sub-Saharan Africa. Aflatoxins are a type of mycotoxins considered to be among the most known potent mutagenic and carcinogenic substances. For example, it can cause liver cancer. Importantly, aflatoxins often contaminate weaning foods consumed by small children. This topical review outlines the evidence on the associations between aflatoxin exposure and impaired linear growth (stunting, that is low height/length for age) in small children (aged 4 months to 5 years) from sub-Saharan Africa. Ten studies were retrieved from the database PubMed after considering predefined inclusion and exclusion criteria. Six out of the ten studies found a significant inverse association between increased aflatoxin exposure and stunted child growth. Although aflatoxin exposure and stunting prevalence were high and widespread in most of the included studies, in general, studies conducted in Western Africa had the highest aflatoxin exposure and stunting prevalence. The only included randomized controlled trial found that a diet aimed at reducing aflatoxin exposure significantly improved linear growth in children at 13, but not at 22, months of age. The evidence suggests that aflatoxins have an impact on linear growth among children aged 4 months to five years. Possibly there is a threshold value for aflatoxin exposure before a significant impact on growth can be identified. Raised awareness and management strategies for the high aflatoxin exposure rates in sub-Saharan Africa and its negative health effects, in particular stunted child growth, are warranted.

Key words: Aflatoxin, children, diet, linear growth, stunting, sub-Saharan Africa, topical review

Acronyms: AFB1- aflatoxin B1; FAO-Food and Agriculture Organization; SSA-sub-Saharan Africa; WHO-World Health Organization

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INTRODUCTION

According to the World Health Organization (WHO) and The Food and Agriculture Organization (FAO), aflatoxins are among the most potent mutagenic and carcinogenic substances known [1]. They can be fatal if the exposure is very high during a short period of time, resulting in acute aflatoxicosis [1]. Long-term consequences of high aflatoxin exposure are for example liver cancer and immune dysfunction [2, 3, 4]. A debated consequence of aflatoxin exposure is impaired child growth, but the underlying mechanisms are not fully understood [1, 5, 6, 7, 8, 9]. Aflatoxin levels in food and feed are often well regulated and low in high-income countries, but for example in sub-Saharan Africa (SSA), high aflatoxin exposure is widespread [8, 10, 11, 12, 13].

Aflatoxins are mycotoxins produced as secondary metabolites by specific moulds in the *Aspergillus* family, particularly *Aspergillus flavus*, *Aspergillus parasiticus* and *Aspergillus niger*. These fungi are commonly found in food, especially in dried or drying foods, such as fruits, nuts, seeds, and grains. The fungi are especially prevalent in hot and humid conditions, and when post-harvest storage environment is not optimal [8]. The most toxic type of aflatoxin is aflatoxin B1 (AFB1]). Aflatoxin M1 and aflatoxin M2 are metabolites of AFB1 which can only be found in meat and milk products, for example in human breast milk if the mother is exposed to aflatoxins [8]. After intake of AFB1, it covalently binds to serum albumin. When the albumin-AFB1 complex is degraded in the body, AFB1-lysine is released, yielding a biomarker of aflatoxin exposure. Albumin-AFB1 is measured in serum or plasma samples using enzyme-linked immunoabsorbent assay (ELISA), while AFB1-lysine in serum or plasma can be detected by using isotope dilution mass spectrometry or high-performance liquid chromatography (HPLC). Although the measurements of albumin-AFB1 and AFB1-lysine are closely related, according to Scholl *et al.* [14] they are analytically distinct biomarkers of aflatoxin and AFB1 exposure, respectively. The albumin-aflatoxin and AFB1-lysine biomarkers both represent the exposure of aflatoxin the last two to three months [15]. Scholl *et al.* [14] found that albumin-aflatoxin measured by ELISA, is on average 2.6-fold greater than those of the AFB1-lysine measurements. Therefore, when comparing aflatoxin levels in humans between different studies, it must be considered which measurements and analysis are used [14, 16]. Another method of measuring aflatoxin concentration in blood is by using albumin-normalized AFB1-lysine. People have different amounts of albumin in their blood, and this method normalises the AFB1-lysin to the albumin level in the individual [17].

Maize and groundnuts are among the commodities with an increased risk for contamination by aflatoxins, and these foods often contribute to various diets in low-resource settings, leading to high aflatoxin exposure, particularly in pregnant women

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and their infants [13, 18, 19, 20, 21]. In sub-Saharan Africa, maize and groundnuts are staple household food products, and the hot and humid environment makes the crops easily contaminated with aflatoxins [8, 11, 22]. Even though there are strict regulations on aflatoxin contamination in several sub-Saharan African countries, many people consume maize and groundnuts that have not undergone any inspections. The most affected populations are smallholder farmers who are highly dependent on subsistence farming. In addition, food safety is also a major problem among urban people who mainly live in informal settings such as townships and slums [11, 22]. Low income in these households leads to a low diversity in their diets, which may encourage high consumption of aflatoxin-contaminated maize, maize-based products and groundnuts [8, 11, 12, 13].

Stunting is defined by WHO as a height-for-age z-score two standard deviations or more below the WHO Child Growth Standards median [23, 24], and is considered a marker of chronic undernutrition. Stunted children also have higher mortality rates and impaired development of cognitive and physical health [23, 25]. In a recent review we reported that children in sub-Saharan Africa are highly affected by stunting, with an average prevalence of 41% [25]. Previously published reviews analysing aflatoxin exposure in children and animals found negative associations between aflatoxin exposure and growth [26, 27]. However, no review has apparently considered specifically the aflatoxin exposure in small children in sub-Saharan Africa and the association with stunting. Therefore, this topical review (also denoted a literature review) aimed to summarise studies published between 2000 to 2024 that examined the association between aflatoxin exposure and stunted growth in children aged 4 months to 5 years living in sub-Saharan Africa.

MATERIALS AND METHODS

Search strategy and inclusion and exclusion criteria

A literature search was conducted in the database PubMed on the 11th of April 2024. The search combination of MESH words used: growth disorders OR growth AND aflatoxin AND child OR infant. All the 135 articles were recorded in the EndNote software, where duplicates were removed. The title and abstracts in the articles were examined and articles found non-relevant were excluded. The search did not exclude reviews or meta-analyses, they were excluded when reading the abstract or title. Then a full screening of the remaining 20 articles was conducted, removing articles that did not comply with the inclusion criteria.

Inclusion criteria/study-article selection

The inclusion criteria were original studies published from January 1st 2000 to March 1st 2024 measuring aflatoxin exposure by using an objective aflatoxin biomarker in serum or plasma, and they had to measure stunted growth by using height-for-age

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z-score (HAZ) as defined by WHO. Eligible articles had to include measurements on children aged 4 months to 5 years. The focus was on original articles, hence meta-analyses and reviews were not included as they report on already published data. In addition the studies had to be written in English, and the study region was limited to countries in sub-Saharan Africa defined by the United Nations Statistics Department [28].

RESULTS AND DISCUSSION

Literature search

The literature search identified 135 articles in PubMed. There were ten studies that met the inclusion criteria and were included in this topical review. The selection process is presented in Figure 1.

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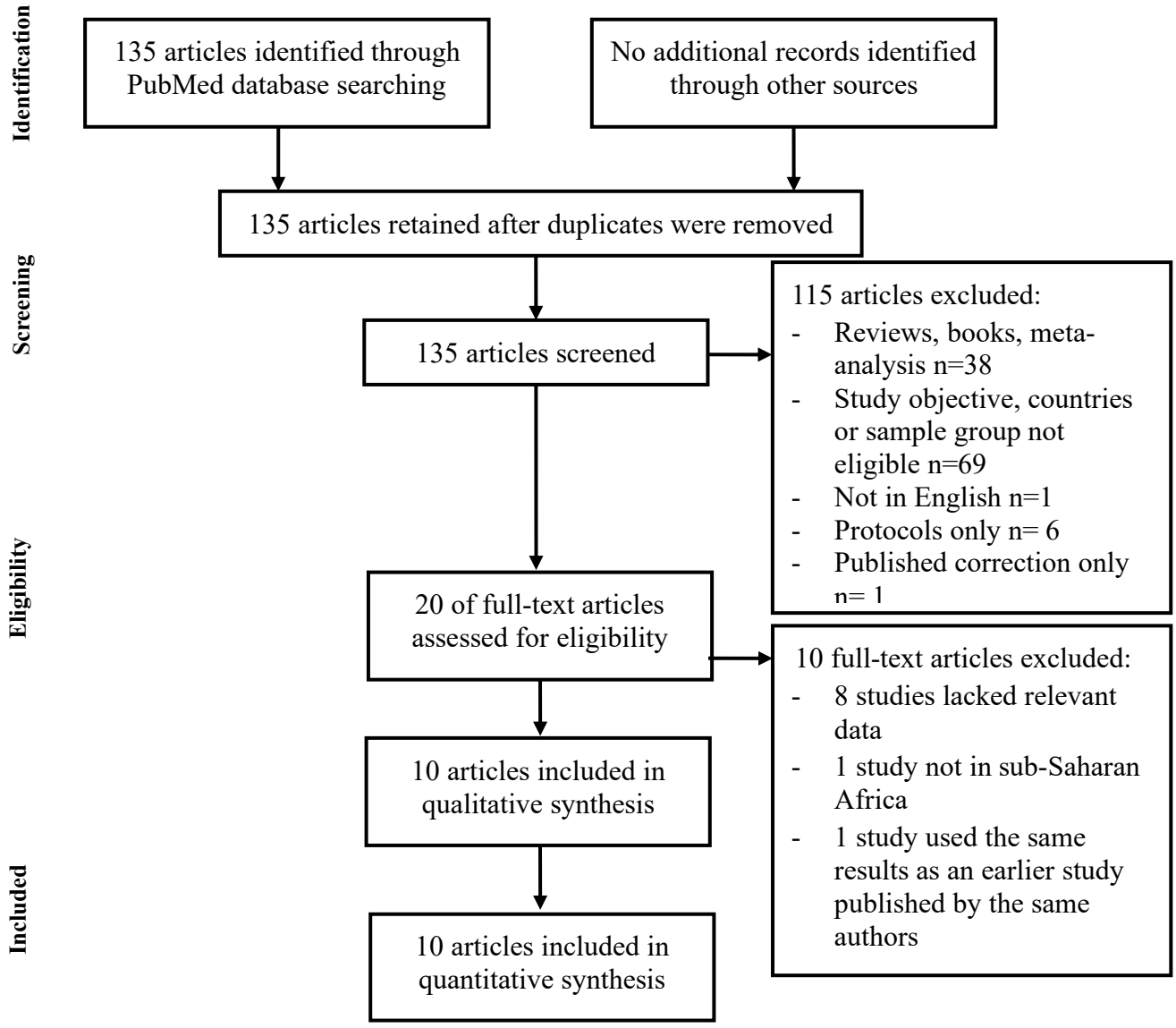

Figure 1: Flow chart illustrating the literature search and selection of articles.

Study characteristics

Among the ten included articles (studies), one was a randomised controlled trial (RCT), 6 were prospective cohorts and 3 were cross-sectional studies. Chen *et al.* [6], Matchado *et al.* [29] and Watson *et al.* [30] are studies using sub-samples of children from bigger trials [Mal-ED, iLiNS-DYAD and ENID], respectively. The oldest study was published in 2002, while the newest was published in 2023 [30, 31]. The studies originated from nine sub-Saharan African countries. All studies stemmed from low-resource settings where the prevalence of stunting varied much [Table 1]. The studies covered different ages of children, from 6 months to 69 months, all including at least one measurement of aflatoxin exposure and HAZ when the

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children were between 6 to 24 months of age. Three of the studies also examined exposure of the mycotoxin fumonisin [29, 32, 33], but those results are not included in this topical review. The study characteristics and extracted results are presented in Table 1.

Aflatoxin exposure

Aflatoxin concentrations in serum or plasma were measured in four of the studies using HPLC [29, 33, 34, 36], two of the studies used isotope dilution mass spectrometry [16, 30], and four studies used ELISA [6, 19, 32, 35]. Three of the studies measured concentrations in plasma samples [6, 16, 29] whereas the remaining studies used serum samples. The studies that used ELISA, reported on average a higher mean level of aflatoxin concentration than the ones using HPLC or isotope dilution mass spectrometry. If the scaling factor of 2.6 was incorporated, the concentration levels in the studies using HPLC or isotope dilution mass spectrometry would be more like those reported in the studies using ELISA. All the aflatoxin concentrations are listed in Table 1.

Gong *et al.* [35] reported the highest mean levels of aflatoxin concentrations in Beninese children. Tessema *et al.* [33] studied Ethiopian children and reported the lowest aflatoxin exposure levels. Only 10% of the children in the study by Tessema *et al.* [33] were exposed to detectable levels of AFB1-lysine in serum, and therefore the median value was below the limit of detection. Because of the low exposure rate, Tessema *et al.* [33] reported the AFB1-lysine concentrations as maximum values found pre- and post-harvest in both the stunted and non-stunted children, as shown in Table 1.

Matchado *et al.* [30] measured AFB1-lysine by isotope dilution mass spectrometry, and showed the results as median and interquartile range. Watson *et al.* [6] did not show the exact concentration for albumin-AFB1 in their study, but showed a figure of the concentrations at different measurement times and the percentage of the children having detectable albumin-AFB1 levels in plasma. Watson *et al.* [6] wrote in their report that the levels were higher than those found by Shirima *et al.* [32] who found albumin-AFB1 levels ranging from 4.7 pg/mg to 23.5 pg/mg, as shown in Table 2. Alamu *et al.* [34] measured both AFB1-lysine and albumin-normalised AFB1-lysine, the albumin normalised AFB1-lys results are used in this review.

In general, the studies conducted in West Africa [6, 16, 19, 35] found higher aflatoxin exposure levels compared with studies in other parts of sub-Saharan Africa [29, 30, 32, 33, 34, 36].

Stunting prevalence

The prevalence of stunting was reported in all, but three studies [30, 33, 35]. The studies found high prevalence of stunting, the lowest prevalence was found in

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Zambia by Alamu *et al.* [34] (19.8% in children aged 6 to 24 months), while the highest was found by Chen *et al.* [16] in Tanzania and McMillan *et al.* [29] in Nigeria (75% in children aged 36 months and 74% in children aged 6 to 48 months, respectively). Note, however, that McMillan *et al.* studied children with known severe acute malnutrition. All the longitudinal and the cross-sectional studies done by Alamu *et al.* [34] reported that the prevalence of stunting increased with increasing age [6, 29, 32, 34, 36].

Association between aflatoxin exposure and stunted growth

The studies adjusted for confounders when analysing the association between aflatoxin exposure and stunting. Watson *et al.* [6] and McMillan *et al.* [16] did not adjust for age and gender. Moreover, all studies adjusted for socioeconomic status or another measurement of wealth status in the household, except McMillan *et al.* [16] and Alamu *et al.* [34]. The four studies conducted in Western Africa (Benin, Gambia, Nigeria and Togo) reported significant inverse associations between aflatoxin exposure and stunting [6, 16, 19, 35].

Cross-sectional studies

All the cross-sectional studies reported a significant inverse relationship between AFB1 levels and stunted growth. Alamu *et al.* [34] found that the children with increased albumin-normalised serum AFB1-lysine were 1.3-fold more likely to be stunted. Gong *et al.* [19] reported that stunted or underweight children had 30-40% higher mean albumin-AFB1 values than the children that were not stunted or underweight. McMillan *et al.* [16] found that AFB1-lysine levels were significantly higher in stunted children than in the control group. That study also reported a weak, but significant correlation between AFB1-lysin and HAZ, but this was no longer significant when adjusted for malnutrition status [16].

Longitudinal studies

Three of the prospective cohorts found a significant association between aflatoxin levels and stunted growth [6, 30, 35], whereas two studies found no such association [29, 32]. One study reported a significant association between aflatoxin exposure levels and stunted growth when all different aflatoxin biomarkers measured in this study were pooled together as a group, but it was no longer significant if only using the AFB1 concentration [33].

In the only randomized controlled trial that was included in this topical review, Hoffmann *et al.* [36] implemented an intervention that aimed at decreasing the aflatoxin contamination in the diet of Kenyan children. They measured HAZ and aflatoxin levels when the children were on average 13 months of age and later when children were on average 22 months of age. They reported a significant increase in HAZ in the intervention group at 13 months of age and stunting prevalence was

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reduced by 7 percentage points, but they found no effect on serum AFB1-lysine levels [36]. Furthermore, they reported that the intervention group had lower AFB1 serum levels than the control group at 22 months of age, but it had no significant effect on the stunting prevalence or HAZ. Notably, this study had a high rate of follow-up loss.

According to the findings in this topical review, there is a possible, yet not verified association with, and impact of, aflatoxin exposure on stunted child growth in sub-Saharan Africa. Six out of the ten studies reported a significant inverse association between aflatoxin levels in serum and stunted growth, while the other four found no significant association. The studies all agreed that (i) aflatoxin exposure is widely prevalent in young children in this region and (ii) that how it affects their linear growth should be studied further [6, 16, 19, 29, 30, 32, 33, 34, 35, 36].

The prevalence of aflatoxin exposure in the studies were generally high, especially in Western Africa. As stated in the introduction, about 40% of the children in sub-Saharan Africa are stunted, but for example Alamu *et al.* [34] and Watson *et al.* [6] had a sample that had a prevalence much lower than this. Some of the studies also reported much higher prevalence than the expected prevalence [16, 29, 32]. All the studies conducted in Western Africa found a significant inverse association between aflatoxin exposure and stunted growth [6, 16, 19, 35]. Aflatoxin exposure depends on geographical location, and these results suggest that children in Western Africa are more vulnerable to aflatoxin exposure than in the rest of sub-Saharan Africa.

Possibly there is a threshold value, but not yet quantified, for aflatoxin exposure before it may result in impaired child growth. The two studies by Gong *et al.* [19, 35] had two of the highest levels of AFB1 measured in the study sample, and both studies found significant associations with stunted growth. Watson *et al.* [6] also reported high exposure levels and found significant associations with impaired growth. Chen *et al.* [29], Shirima *et al.* [32] and Tessema *et al.* [33] reported much lower AFB1 levels in their study samples, and the latter studies reported no significant associations between aflatoxin exposure and stunted growth. If we had included the scaling factor of 2.6 in the studies by Chen *et al.* [29] and Tessema *et al.* [33], because they measured AFB1 using HPLC, the concentrations would still have been much lower than those reported by Gong *et al.* [19, 29, 33, 35]. If adjusting for measurement by HPLC in the study by Hoffmann *et al.* [36] the reported AFB1 levels would have been as high as reported in both studies by Gong *et al.* [19, 35, 36]. Hoffmann *et al.* [36] did not report a significant association between AFB1 exposure and HAZ, even though the aflatoxin exposure reported was high. In contrast, McMillan *et al.* [16] reported a much lower concentration of AFB1 than reported by Hoffmann *et al.* [36], but they did find a significant correlation between AFB1-lysine and HAZ, although weak. Possibly then, the exposure level must reach

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a threshold value before aflatoxin exposure may impair child growth, but it is not in agreement with all the studies discussed in this review, suggesting that other factors may interfere as well.

It is also important to discuss the importance of weaning in this topical review. The reason for choosing children older than 4 months was because it meant that most of them had either started weaning or were fully weaned. Although aflatoxin may be passed on to the infant via breastmilk, breastfeeding generally protects the infant from high aflatoxin exposure through their diet. The most typical weaning foods in sub-Saharan Africa are often maize-based products, and these products are often contaminated by aflatoxins. Therefore, when the children stop to be exclusively breastfed the aflatoxin exposure increases [13, 37]. In sub-Saharan Africa, about 37% of infants under six months of age are exclusively breastfed [38]. Therefore, it is probable that some of the youngest children included in the studies reported herein were either exclusively or partially breastfed. For example, Watson *et al.* [6] reported that only 48% of children at six months of age had detectable albumin-AFB1 in plasma. The percentage increased to 98 already when the children were 12 months of age, reflecting that aflatoxin exposure increased with weaning. Three of the studies included in this topical review adjusted for weaning status, including Watson *et al.* [6, 32, 35].

In the only randomized controlled trial included in this topical review Hoffmann *et al.* [36] studied the impact of reduced aflatoxin exposure in the diet and its effect on child growth. The intervention group ate maize contaminated with a known and safe amount of aflatoxins, while the control group continued with their usual maize-based diet. The authors found a significant increase in HAZ in the intervention group when children were on average 13 months of age, and the stunting prevalence was reduced by seven percentage points. Still, they found no effect of the intervention on serum AFB1-lysine levels. Hoffmann *et al.* [36] did not report the number of children being breastfed. Therefore, since both study groups had relatively low aflatoxin exposure levels it may be explained by children still breastfeeding. Possibly, if the authors have included an intervention that decreased aflatoxin contamination in maize, this may have led to a larger effect when both groups are older and eat maize and maize-based products instead of breast milk. Notably, the AFB1 levels in the intervention group were significantly lower than in the control group when the children were on average 22 months of age [36]. Moreover, at that child age, Hoffmann *et al.* [36] did not find a significant increase in HAZ, as found when the children were 13 months. One would expect the HAZ to increase as the aflatoxin exposure was reduced. The reason for this may be because the measurements were different in nature: AFB1-lysin in serum reflects exposure to aflatoxins the last 2 to 3 months, while the HAZ measurements reflects the linear child growth status when

measurements are conducted. Because of this, linking aflatoxin exposure to child growth can be difficult [14, 36].

Several studies that have examined older children than those included in this topical review, have not found any significant associations between aflatoxin exposure and stunted child growth. For example, one cross-sectional study reported no significant correlation between aflatoxin exposure and HAZ in Kenyan children aged 6 to 12 years [39]. In that study, the exposure was high with a geometric mean of 10.5 pg/mg albumin-AFB1, measured by HPLC, and all children had detectable levels. Another study examined Pakistani children and found that children aged 5 to 11 years had the highest aflatoxin exposure, but no significant associations with HAZ were found [40]. Another study in Gambia among children aged 6 to 9 years found no association with aflatoxin exposure and HAZ [4]. These studies suggest that the older children may not be so vulnerable to the possible negative growth effects of aflatoxin exposure.

With regard to birth outcomes and growth, studies have reported significant associations among children exposed to maternal aflatoxin in utero and aflatoxins through breast milk during the first months of life [21, 41]. For example, Tesfamariam *et al.* [42] found that chronic maternal aflatoxin exposure was associated with lower fetal growth. Turner *et al.* [21] found that infants exposed to higher level of maternal aflatoxin concentrations had a HAZ profile that decreased with age. Turner *et al.* [21] also found that albumin-AFB1 levels in the infants' blood at 16 weeks of age (younger than children included in this review) were significantly negatively related to HAZ, but only 11% of them had detectable albumin-AFB1, probably because most of them were still exclusively breastfed [21]. A study conducted in Uganda found that maternal aflatoxin levels were significantly associated with adverse birth outcomes, such as lower birth weight and smaller head circumference [43]. All these studies showed that aflatoxin exposure early in life and in utero can contribute to impaired growth in children or to other negative health outcomes. In contrast, one study examined Tanzanian mothers and their infants, and they did not find the same significant associations [44]. This may be because the mothers in that study had a median level of AFB1-lysine of 1.4 pg/mg which is much lower than found in the studies by Tesfamariam *et al.* [41] (12.6 pg/mg), Turner *et al.* [21] (40.4 pg/mg) and Lauer *et al.* (5.83 pg/mg) [45]. This raises the question again if there is a threshold value at which the aflatoxin exposure will impair child growth.

There are many different risk factors that may influence child stunting, making it difficult to examine aflatoxin exposure as an individual risk factor. Age is one such known risk factor, and it was earlier reported that children between one to four years of age in sub-Saharan Africa had the highest prevalence of stunting [25]. All the studies in this topical review did adjust for age in their analyses. Reportedly, stunting

prevalence among boys in sub-Saharan Africa is also higher than that of girls [25]. All the studies, except those of Watson *et al.* [6] and McMillan *et al.* [16], adjusted for both age and gender.

Socioeconomic status and household wealth are other known risk factors for child stunting [25]. All studies included in this topical review adjusted for either socioeconomic status or household wealth, except McMillan *et al.* [16] and Alamu *et al.* [34]. Chen *et al.* [29] reported that higher socio-economic status was positively associated with HAZ, but not AFB1-lysine levels. This suggests that socioeconomic status may play an important role in child stunting prevalence, making it difficult to evaluate the significant effect aflatoxin exposure had on child growth reported in the studies that did not adjust for socioeconomic status [16, 34].

Stunting is a marker of chronic undernutrition. Therefore, if a study included participants with a high prevalence of malnutrition, it can be difficult to evaluate the individual impact aflatoxin exposure may have on stunted child growth. McMillan *et al.* [16] studied children of whom about 80% had severe acute malnutrition. Notably, they found a significant correlation between aflatoxin exposure and stunting, but they only adjusted for age. Moreover, when they adjusted for malnutrition status the correlation became insignificant [16]. Among the studies included herein, only Chen *et al.* [29], Shirima *et al.* [32] and Hoffman *et al.* [36] included adjustments of child nutrition status.

The main limitations of this topical review are the lack of randomized controlled trials, the often small sample sizes, and different measurement methods of aflatoxin concentrations. Adding more databases for the literature search may have caused a broader selection of data. The strengths include strict inclusion and exclusion criteria and that the studies were conducted during a long time period (24 years).

CONCLUSION AND RECOMMENDATIONS FOR DEVELOPMENT

This topical review found that child aflatoxin exposure is a highly prevalent problem in sub-Saharan Africa, especially in Western Africa and Kenya, and with a possible impact on stunted growth. The children (aged 4 months to 5 years) participating in the included studies were highly exposed to aflatoxins in the diet and they were more vulnerable to the negative growth impact made by aflatoxin compared with older children, and exposure in utero and as infants. In general, the studies that reported the highest aflatoxin exposure, found a significant inverse association between aflatoxin exposure and stunted child growth. The possibility of aflatoxin exposure having to reach a threshold value before it impacts on child growth, should be studied further. This topical review highlights the importance of raising awareness to this problem, and efforts should be implemented to make it easier for households with little resources to give their children a safer and less aflatoxin contaminated diet.

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This therefore calls for relevant stakeholders to put in place strategies to prevent and control aflatoxin contamination of food products from maize, groundnuts, sorghum, soybean and milk commonly fed to children. Such strategies should include ensuring good agriculture, hygiene and manufacturing practices, as well as use of available aflatoxin decontamination technologies in the country.

Conflict of interest

The authors declare no conflict of interest.

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Table 1: Summary of study characteristics and results from the ten included studies

Reference	Type of study and study site	Age (months) and sample size (n)	Exposure assessment, age at measurements (months), aflatoxin biomarker concentration	Stunting prevalence	Relationship between aflatoxin exposure and stunting outcomes	Adjustments and other observations
Alamu *et al.* [34]	Cross-sectional, Zambia (Chipata and Monze districts)	6-24, 400 (200 from Chipata and 200 from Monze)	HPLC in serum, 6-24, GM (range) Chipata: 4.97 (0.78-201.9) pg/mg albumin-AFB1 Monze: 6.51 (0.92-315) pg/mg albumin-AFB1	Total: • 19.8% Chipata: • 12.81% Monze: • 7.0%	• Children with increased albumin-normalised serum AFB1 were 1.3-fold more likely to be stunted (OR =1.30, p=0.0146). • No significant association between AFB1-lysine and stunted growth (p=0.142).	Sex, sickness, serum albumin and age. • Child sicknesses were more prevalent in Chipata and could explain the higher percentage of stunting. • Higher prevalence of stunting in older children.
Chen *et al.* [29]	Prospective cohort, Tanzania	36, 114	HPLC in plasma, 24, Mean (95% CI): 5.1 (3.5, 6.6) pg/mg albumin-AFB1	24 months of age: • 61% 36 months of age: • 75%	• No correlations found between aflatoxin exposures and stunting (p = 0.51).	Plasma vitamin A, iron, zinc, protein and animal protein, folate, SES index, and child sex. • Higher SES was positively associated with HAZ (p = 0.0392), but not with AFB1-lysine concentrations (p = 0.12) • Sub-study of the MAL-ED trial.
Gong *et al.* [19]	Cross-sectional, Benin and Togo	9-69, 479	ELISA in serum, 9-69, GM: 32.8 (range 5-1064 pg/mg albumin-AFB1	33%	Significant association between increased albumin-AFB1 levels and stunted growth: • Stunted children had 30-40% higher mean albumin-AFB1 levels (Trend test: p = 0.0001).	Age, sex, SES, and agro-ecological zone. • The albumin-AFB1 levels in children increased with age up to 3 years, and within the 1–3-year age group was significantly (p=0.0001) associated with weaning status.

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Gong *et al.* [35]	Prospective cohort, Benin	16-37, 200	ELISA in serum, 25-27, GM: • 37.4 pg/mg albumin-AFB1 June 2001 • 38.7 g pg/mg albumin-AFB1 October 2001 • 86.8 pg/mg albumin-AFB1	Data not given	Significant correlation between HAZ and albumin-AFB1 • February: p = 0.009 • For mean albumin-AFB1 over three survey points: p < 0.0001	Age, child sex, height, weaning status, SES and village: • The highest quartile of albumin-AFB1 was associated with a mean 1.7 cm reduction in growth over 8 months compared with the lowest quartile. • Children who were fully weaned at recruitment had higher albumin-AFB1 than those still partially breastfed (p < 0.0001).
Hoffmann *et al.* [36]	Cluster-randomized controlled trial, Kenya	< 24, 1230	HPLC in serum, 13 months of age at midline and 22 months of age at endline, Mean midline: p>0.05 Mean endline: • 18.1 pg/mg albumin-AFB1	Midline measurement: • All: 23% Endline measurement: Intervention group: • 39.26% Control group: • 35.71%	• The intervention significantly reduced endline serum AFB1-lysine levels (-0.273, 95% CI −0.547 to 0.001; p=0.025), but no, but it had no effect on stunting prevalence. • Intervention had no effect on endline child HAZ (-0.01, 95% CI -0.165-0.146; p = 0.551). • HAZ increased from baseline to midline by 0.16 (95% CI −0.009 to 0.33; p=0.032), but intervention had no effect on serum AFB1-levels.	Household food insecurity, household dietary diversity, household assets, number or adult equivalents in the household, child sex, age, maternal age, height and education and the educational level of the head of the household, when the participation were enrolled in the study (which study enrolment wave). • High rates of follow-up loss. • The study was conducted in 6 enrolment waves, with 4 months apart from when the women were invited to enrol in the study.
Matchado *et al.* [30]	Prospective cohort, Malawi	< 30, 241 mother-infant dyads	IDMS in serum, pg/ul albumin-AFB1 median (IQR) 6: 60% 0.07 (0.01, 0.24) 18: 95% 0.35 (0.19, 0.91)	Data not given	AFB1-lysine at 18 months was significantly associated with persistent impaired linear growth: • 18-months AFB1-lysine was negatively associated with HAZ outcomes at 18, 24, and 30 months (β = –0.18; 95% CI: –0.32, –0.04, β = –0.21; 95% CI: –0.35, –0.07, β = –0.18; 95% CI: –0.32, –0.03 at 18, 24 and 30 months, respectively). All p < 0.05.	Maternal age, pre-pregnancy body mass index, HIV infection, haemoglobin, weight and mid-upper-arm-circumference-for-age z-score gain rate from baseline to 36-week, food security, malaria infection, housing quality, child sex, and age. • Maternal aflatoxin exposure was not associated with impaired child growth • AFB1-lysine at 6 months found non-significant associated with HAZ • Sub-study of iLiNS-DYAD

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McMillan *et al.* [16]	Cross-sectional study, Nigeria	6-48, 58	IDMS in plasma, 6-48, GM (range): Control: 0.8 (0.2-2.9) pg/mg albumin-AFB1 Stunted: 4.6 (0.2-27.6) pg/mg albumin-AFB1 All: 2.4 pg/mg albumin-AFB1	74%	• Concentration of AFB1-lysine was significantly higher in stunted children (o = 0.032) compared to controls. • A weak, but significant correlation between AFB1-lysine and HAZ (Spearman's rho = -0.34, p = 0.0093)	Age. • 81% of the children had severe acute malnutrition at baseline • All children weaned at baseline • Association between AFB1-lysine and stunting was not significant when adjusting for malnutrition status • Small study sample
Shirima *et al.* [32]	Prospective cohort, Tanzania	6-14, 166	ELISA in serum, GM (95%CI) 6-14: • 4.7 (3.9, 5.6) pg/mg albumin-AFB1 After 6 months: • 12.9 (9.9, 16.7) pg/mg albumin-AFB1 After 12 months: • 23.5 (19.9, 27.7) pg/mg albumin-AFB1	First measurement (6-14 months of age): • 44% After 6 months: • 55% After 12 months: • 56%	• Non-significant negative associations between mean albumin-aflatoxin levels from all sampling times and HAZ at 12 months after recruitment (beta = -0.07; 95% CI: -0.27, 0.13; p = 0.257).	Breastfeeding, village, maternal education, family socioeconomic status, sex, baseline age, baseline length and protein and energy intakes. • None of the children were breastfeeding at recruitment. • Small study sample
Tessema *et al.* [33]	Prospective cohort, Ethiopia	6-35, 102 (50 stunted and 52 non-stunted)	HPLC in serum, 21 pre-harvest, maximal AFB1-lysine pre-harvest (August-September) to maximal value post-harvest (February): Stunted: • 167-290 pg/ml Non-stunted: • 182-987.6 pg/ml	Data not given	• Exposure to any of the individual aflatoxins was not associated with linear growth (all p>0.05). • Aflatoxins as a group was significantly associated with HAZ (p < 0.0001).	Age, time of assessment, inflammation, household wealth status, presence of diarrheal illness and sex. • Small study sample

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Watson *et al.* [6]	Prospective cohort, Gambia	Birth 6-24, 374	ELISA in plasma, 6, 12 and 18, Percentage of children with detectable albumin-AFB1 concentrations: 1. 48% 2. 98% 3. 99%	6 months of age: • 5.6% 24 months of age: • 25.9%	Found a small, but significant effect of aflatoxin exposure on child growth. • Inverse associations reported between albumin-AFB1 and HAZ (β - 0.04, p = 0.015) from 6 to 18 months of age. • Albumin-AFB1 at 12 months was associated with changes in HAZ and infant length between 12 and 18 months of age (b = - 0.01, p = 0.003; b = - 0.003, p = 0.02, respectively).	Supplementation group, season of sampling, mother's household quality, age of introduction of non-breast milk foods. • Sub-study from the ENID trial • High prevalence of undetectable albumin-AFB1 in children at 6 months reflects that breastfed infants have lower aflatoxin exposure. • Albumin-AFB1 levels increased when infants got older (p = 0.001).

GM = geometric mean
SES = socioeconomic status
ELISA = enzyme-linked immunosorbent assay
HPLC = high-performance liquid chromatography,
DMS = isotope dilution mass spectrometry
HAZ = height-for-age z-score
OR = odds ratio
IQR = interquartile range

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REFERENCES

1. **World Health Organization.** Safety evaluation of certain contaminants in food: prepared by the eighty-third meeting of the Joint FAO/WHO Expert Committee on Food Additives (JECFA). World Health Organization and Food and Agriculture Organization of the United Nations. 2018(WHO Food Additives Series, No. 74; FAO JECFA Monographs 19 bis).

2. **Xu Y, Moore S, Chen G, Nshe P, Faye-Joof T, Prentice A, Gong YY and M Routledge** Impact of dietary aflatoxin on immune development in Gambian infants: a cohort study. *BMJ Open*. 2021; **11(7):** e048688.

3. **Magnussen A and MA Parsi** Aflatoxins, hepatocellular carcinoma and public health. *World J. Gastroenterol.* 2013; **19(10).**

4. **Turner PC, Moore SE, Hall AJ, Prentice AM and CP Wild** Modification of immune function through exposure to dietary aflatoxin in Gambian children. *Environ. Health Perspect.* 2003; **111(2):** 217-20.

5. **Hoffmann V, Jones K and JL Leroy** The impact of reducing dietary aflatoxin exposure on child linear growth: a cluster randomised controlled trial in Kenya. *BMJ Glob. Health.* 2018; **3(6):** e000983.

6. **Watson S, Moore SE, Darboe MK, Chen G, Tu YK, Huang YT, Eriksen, KG, Bernstein RM, Prentice AM, Wild CP, Xu Y, Routledge MN and YY Gong** Impaired growth in rural Gambian infants exposed to aflatoxin: a prospective cohort study. *BMC Public Health.* 2018; **18(1):** 1247.

7. **Andrews-Trevino J, Webb P, Shrestha R, Pokharel A, Acharya S, Chandyo R, Davis D, Baral K, Wang J-S, Xue K and S Ghosh** Exposure to multiple mycotoxins, environmental enteric dysfunction and child growth: Results from the AflaCohort Study in Banke, Nepal. *Matern. Child Nutr.* 2022; **18(2):** e13315.

8. **Hell K and A Roth** Aflatoxins and Nutrition. Deutsche Gesellschaft fur Internationale Zusmmenarbeit (GIZ) GmbH. 2019.

9. **Mahfuz M, Hasan SMT, Alam MA, Das S, Fahim SM, Islam MM, Gazi MA, Hossain M, Egner PA, Groopman JD and T Ahmed** Aflatoxin exposure was not associated with childhood stunting: results from a birth cohort study in a resource-poor setting of Dhaka, Bangladesh. *Public Health Nutr.* 2021; **24(11):** 3361-70.

10. **Xu Y, Gong YY and MN Routledge** Aflatoxin exposure assessed by aflatoxin albumin adduct biomarker in populations from six African countries. *World Mycotoxin J.* 2018; **11(3):** 411-9.

11. **Wanjiru JW, Njue LG, Okoth MW and GM Karau** Exposure of Kiandutu Slum Residents to Aflatoxins Through Maize-Based Products Consumption. *Afr. J. Food Agric. Nutr. Dev.* 2024; **24(2):** 25508-25524. https://doi.org/10.18697/ajfand.127.23885

12. **Egal S, Hounsa A, Gong YY, Turner PC, Wild CP, Hall AJ, Hell K and KF Cardwell** Dietary exposure to aflatoxin from maize and groundnut in young children from Benin and Togo, West Africa. *Int. J. Food Microbiol.* 2005; **104(2):** 215-24.

13. **Obade M, Andang'o P, Obonyo C and F Lusweti** Exposure of children 4 to 6 months of age to aflatoxin in Kisumu County, Kenya. *Afr. J. Food Agric. Nutr. Dev.* 2015; **15(2):** 9949-9963. https://doi.org/10.18697/ajfand.69.14020

14. **Scholl PF, Turner PC, Sutcliffe AE, Sylla A, Diallo MS, Friesen MD, Groopman JD and CP Wild** Quantitative comparison of aflatoxin B1 serum albumin adducts in humans by isotope dilution mass spectrometry and ELISA. *Cancer Epidemiol. Biomarkers Prev.* 2006; **15(4):** 823-6.

15. **Wild CP, Hudson GJ, Sabbioni G, Chapot B, Hall AJ, Wogan GN, Whittle H, Montesano R and JD Groopman** Dietary intake of aflatoxins and the level of albumin-bound aflatoxin in peripheral blood in The Gambia, West Africa. *Cancer Epidemiol. Biomarkers Prev.* 1992; **1(3):** 229-34.

16. **McMillan A, Renaud JB, Burgess KMN, Orimadegun AE, Akinyinka OO, Allen SJ, Miller JD, Reid G and MW Sumarah** Aflatoxin exposure in Nigerian children with severe acute malnutrition. *Food Chem. Toxicol.* 2018; **111:** 356-62.

17. **Smith JW, Ng DK, Alvarez CS, Egner PA, Burke SM, Chen JG, Kensler TW, Koshiol J, Rivera-Andrade A, Kroker-Lobos MF, Ramírez-Zea M, McGlynn KA and JD Groopman** Assessing the validity of normalizing aflatoxin b(1)-lysine albumin adduct biomarker measurements to total serum albumin concentration across multiple human population studies. *Toxins*. 2022; **14(3)**.

18. **Ismail A, Naeem I, Gong YY, Routledge MN, Akhtar S and M Riaz** Early life exposure to dietary aflatoxins, health impact and control perspectives: A review. *Trends Food Sci. Technol.* 2021; **112**.

19. **Gong YY** Dietary aflatoxin exposure and impaired growth in young children from Benin and Togo: cross sectional study. *BMJ.* 2002; **325:** 20-1.

20. **Andrews-Trevino JY, Webb P, Shively G, Rogers BL, Baral K, Davis D, Paudel K, Pokharel A, Shrestha R, Wang J-S and S Ghosh** Relatively low maternal aflatoxin exposure is associated with small-for-gestational-age but not with other birth outcomes in a prospective birth cohort study of Nepalese infants. *J. Nutr.* 2019; **149:** 1818-25.

21. **Turner PC, Collinson AC, Cheung YB, Gong Y, Hall AJ, Prentice AM and CP Wild** Aflatoxin exposure in utero causes growth faltering in Gambian infants. *Int. J. Epidemiol.* 2007; **36:** 1119-25.

22. **Mahfuz M, Alam MA, Fahim SM, Gazi MA, Raihan MJ, Hossain M, Egner PA, Obong Bessong P, Petri WA, Groopman JD and T Ahmed** Aflatoxin exposure in children living in Mirpur, Dhaka: data from MAL-ED companion study. *J. Expo Sci. Environ. Epidemiol.* 2019; **29:** 655-62.

23. **World Health Organization**. Global nutrition targets 2025: stunting policy brief (WHO/NMH/NHD/14.3). Geneva: World Health Organization 2014.

24. **World Health Organization.** WHO child growth standards: length/height-for-age, weight-for-age, weight-for-length, weight-forheight and body mass index-for-age : methods and development. Geneva: World Health Organization; 2006.

25. **Quamme SH and PO Iversen** Prevalence of child stunting in Sub-Saharan Africa and its risk factors. *Clin Nutr Open Sci.* 2022; **04/01:** 42.

26. **Urugo MM, Teka TA, Lema TB, Lusweti JN, Djedjibegovíc J, Lachat C, Tesfamariam K, Mesfin A, Astatkie T and MA Abdel-Wahhab** Dietary aflatoxins exposure, environmental enteropathy, and their relation with childhood stunting. *Int. J. Food Sci. Nutr.* 2024: 1-14.

27. **Nejad BG, Mostafaei Z, Rezaabad AB, Mehravar F, Zarei M, Dehghani A, Raeisi Estabragh MA, Karami-Mohajeri S and H Alizadeh** A systematic review with meta-analysis of the relation of aflatoxin B1 to growth impairment in infants/children. *BMC Pediatr.* 2023; **23:** 614.

28. **The United Nations Statistics Division.** Standard or area codes for statstical use (M49): United Nations.

29. **Chen C, Mitchell NJ, Gratz J, Houpt ER, Gong Y, Egner PA, Groopman JD, Riley RT, Showker JL, Svensen E, Mduma ER, Patil CL and F Wu** Exposure to aflatoxin and fumonisin in children at risk for growth impairment in rural Tanzania. *Environ. Int.* 2018; **115:** 29-37.

30. **Matchado A, Smith JW, Schulze KJ, Groopman JD, Kortekangas E, Chaima D, Arnold CD, Maleta K, Ashorn, U, Ashorn P, Dewey KG and CP Stewart** Child aflatoxin exposure is associated with poor child growth outcomes: a prospective cohort study in rural Malawi. *Curr. Dev. Nutr.* 2023; **7:** 101962.

31. **Gong YY, Cardwell K, Hounsa A, Egal S, Turner PC, Hall AJ and CP Wild** Dietary aflatoxin exposure and impaired growth in young children from Benin and Togo: cross sectional study. *BMJ.* 2002; **325:** 20-1.

32. **Shirima CP, Kimanya ME, Routledge MN, Srey C, Kinabo JL and HU Humpf** A prospective study of growth and biomarkers of exposure to aflatoxin and fumonisin during early childhood in Tanzania. *Environ. Health Perspect.* 2015; **123:** 173-8.

33. **Tessema M, De Groote H, Brouwer ID, De Boevre M, Corominas AV and BJ Stoecker** Exposure to aflatoxins and fumonisins and linear growth of children in rural Ethiopia: a longitudinal study. *Public Health Nutr.* 2021; **24:** 3662-73.

34. **Alamu EO, Gondwe T, Akello J, Maziya-Dixon B and M Mukanga** Relationship between serum aflatoxin concentrations and the nutritional status of children aged 6-24 months from Zambia. *Int. J. Food Sci. Nutr.* 2020; **71:** 593-603.

35. **Gong Y, Hounsa A, Egal S, Turner PC, Sutcliffe A and AJ Hall** Postweaning exposure to aflatoxin results in impaired child growth: a longitudinal study in Benin, West Africa. *Environ. Health Perspect.* 2004; **112:** 1334-8.

36. **Hoffmann V, Jones K and JL Leroy** The impact of reducing dietary aflatoxin exposure on child linear growth: a cluster randomised controlled trial in Kenya. *BMJ Global Health.* 2018; 3:e000983.

37. **Gong Y, Egal S, Hounsa A, Turner P, Hall A and K Cardwell** Determinants of aflatoxin exposure in young children from Benin and Togo, West Africa: the critical role of weaning. *Int. J. Epidemiol.* 2003; **32:** 556-62.

38. **Victora CG, Bahl R, Barros AJ, França GV, Horton S and J Krasevec** Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *Lancet*. 2016; **387:** 475-90.

39. **Nabwire Wangia-Dixon R, Xue KS, Alcala J, Quach THT, Song X and L Tang** Nutrition and growth outcomes are affected by aflatoxin exposures in Kenyan children. *Food Addit. Contam. Part A Chem. Anal Control Expo. Risk Assess.* 2020; **37:** 2123-2134.

40. **Ashraf W, Rehman A, Ahmad MU, Rabbani M, Mushtaq MH and K Aamir** Assessment of aflatoxin B(1)-lysine adduct in children and its effect on child growth in Lahore, Pakistan. *Food Addit. Contam. Part A Chem. Anal Control Expo. Risk Assess.* 2022; **39:** 1463-73.

41. **Tesfamariam K, Gebreyesus SH, Lachat C, Hanley-Cook GT, Roro M and YG Mengistu** Chronic aflatoxin exposure during pregnancy is associated with lower fetal growth trajectories: a prospective cohort from the Butajira Nutrition, Mental Health, and Pregnancy (BUNMAP) Study in rural Ethiopia. *Am. J. Clin. Nutr.* 2022; **116:** 1634-41.

42. **Tesfamariam K, Argaw A, Hanley-Cook GT, Gebreyesus SH, Kolsteren P and T Belachew** Multiple mycotoxin exposure during pregnancy and risks of adverse birth outcomes: a prospective cohort study in rural Ethiopia. *Environ Int.* 2022; **160:** 107052.

43. **Lauer JM, Duggan CP, Ausman LM, Griffiths JK, Webb P and JS Wang** Maternal aflatoxin exposure during pregnancy and adverse birth outcomes in Uganda. *Matern Child Nutr.* 2019; 15:e12701.

44. **Magoha H, Kimanya M, De Meulenaer B, Roberfroid D, Lachat C and P Kolsteren** Risk of dietary exposure to aflatoxins and fumonisins in infants less than 6 months of age in Rombo, Northern Tanzania. Matern. *Child Nutr.* 2016; **12:** 516-27.

45. **Lauer JM, Natamba BK, Ghosh S, Webb P, Wang JS and JK Griffiths** Aflatoxin exposure in pregnant women of mixed status of human immunodeficiency virus infection and rate of gestational weight gain: a Ugandan cohort study. *Trop. Med. Int. Health.* 2020; **25:** 1145-54.