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Review
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Review

Exposure of Kenyan population to aflatoxins in foods with special reference to Nandi and Makueni counties

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Abstract

Aflatoxins cause acute and chronic health and production effects in humans and animals, respectively. This study reports on the exposure of the Kenyan population to the two mycotoxins in two counties (Nandi and Makueni) using children younger than 5 years as the proxy. A questionnaire was administered, which captured knowledge of the incidences of acute aflatoxin poisoning, food consumption patterns. Analysis of aflatoxin in samples of urine, breast milk, maize, sorghum, and millet was done using cELISA and HPLC. Maize and sorghum are used in the weaning formula of children. The difference in consumption of maize- and sorghum-based diets in children between younger than 1 and between 1 and 5 years was significant with $P = 0.037$ and $P = 0.002$, respectively, in Nandi and Makueni. In children younger than 5 years, the consumption of maize ranged from 0.1 to 0.25 kg per person per day in Nandi and Makueni with an aflatoxin exposure rate of 0.011 and 0.49 μg per kg body weight (bwt) per day, respectively. The exposure to aflatoxin through milk for children younger than 5 years was 4×10^{-4} and 1×10^{-4} μg per kg bwt per day in Makueni and Nandi, respectively. The exposure of nursing children through breast milk was 6×10^{-3} and 1×10^{-6} μg per kg bwt per day in Makueni and Nandi, respectively. Children younger than 30 months in Makueni had 1.4 times higher levels of aflatoxin M1 (AFM1) in urine than those of the same age in Nandi. The stunting and severe stunting rates in Makueni and Nandi were 28.7%, 18.5% and 30.7%, 16.5%, respectively. Thus, there is need for urgent mitigation measures, a constant surveillance program, strict regulations, and awareness creation directed at poor households to reduce dietary exposure to mycotoxins.

Key words: Aflatoxins; Contamination; Exposure; Kenya.

Introduction

Aflatoxins frequently contaminate staple foods of many Kenyans. Aflatoxins are primarily produced by *Aspergillus flavus* and *A. parasiticus*. Humans are exposed to aflatoxins from consumption of cereals, nuts, pulses, roots, and animal source foods from livestock fed contaminated feeds. In Kenya, maize is the major staple

food and it is estimated that annual per capita consumption stands at 97 kg (FAO, 2000) compared to 76 kg per capita consumption in East and Southern Africa, respectively (M'mboyi *et al.*, 2010). Maize contamination with aflatoxins in Kenya has been reported by Lewis *et al.* (2005), Muture and Ogana (2005), Strosnider *et al.* (2006), Probst *et al.* (2010), Daniel *et al.* (2011), and Muthomi *et al.* (2012). Human exposure from milk obtained from animals fed with contaminated feeds has been reported by Kang'ethe *et al.* (2007) and Kang'ethe and Lang'at (2009).

Long-term exposure (2–3 months prior to sampling) to aflatoxins in humans can be assessed by analysis of albumin adducts (Wild and Turner 2002; Gong *et al.*, 2002, 2004) and presence of aflatoxin M1 (AFM1) products of AFB1 breakdown in urine for short-term exposure.

This study aimed at establishing the exposure levels of households to aflatoxins and effects of aflatoxin exposure exemplified by growth indices in children younger than 5 years in the Nandi and Makueni counties of Kenya.

Materials and Methods

Site and household selection

The counties Nandi and Makueni in Kenya were purposely selected based on history of human acute aflatoxicosis in Makueni (Lewis *et al.*, 2005) and high incidences of esophageal cancer in Nandi (Wakhisi *et al.*, 2005). The sub-locations were identified by a team of researchers together with veterinarians, agriculture extension officers, and health officers at the county and ward offices. The selection criterion was based on dairy and maize production. In the first instance, the sub-counties were selected that fitted the criteria based on the records at the county level. Three sub-counties were selected in each county. At the sub-county headquarters, the wards were selected using the same criteria of dairy production and maize growing. At the ward level, the extension officers (veterinarians, agriculture, and public health) selected the sub-location that best fitted the criteria used to select the wards. The households in the sub-location were listed which fitted the criteria of having dairy animals, grew maize and or sorghum and millet, had a child younger than 5 years, and/or if the spouse was pregnant at the time of listing.

Based on the population and number of households in the sub-location and the households that fitted the criteria, a proportion of the households was randomly selected for sampling (Martin *et al.*, 1987), and the household sample size was corrected for finite population according to Daniel (1999).

Nandi falls within the agro-ecological zones of lower humid highlands to upper midland and upper highland zones ((LH2, LH3, UM3 and UM4, Jaetzold *et al.* 2006). It lies between 1300 and 2500 m above sea level and receives about 1200–2000 mm of rain per year. Nandi is best suited for tea cultivation, dairy production, and growing maize, wheat, and barley. Makueni falls within the lower midland agro-ecological zones LM3, LM4, and LM5. It receives between 200 and 1200 mm of rainfall per year, which is unreliable at times with frequent droughts resulting in crop failures.

Sampling of urine

Urine samples were collected from children younger than 5 years in all the households. Clean bottles were supplied to the mothers for collecting the urine. The samples were frozen at –20°C until analysis within 3 months.

Sampling of breast milk

Nursing mothers were given clean bottles into which they expressed milk from their breasts. The samples were frozen at –20°C until they were analysed within 3 months.

Testing for aflatoxin M1 in urine using c-ELISA (helica test kit)

To determine aflatoxin M1 in urine, 5 ml of urine was centrifuged at 743 × g for 10 min. Nine hundred and fifty microlitres of distilled water was pipetted into skatron (SKATRON AS LIER, Norway. CAT. No 7071) tubes and 50 µl of standards or supernatant-urine was added into 950 µl of distilled water in the skatron tubes and mixed by priming pipetting at least five times. Two hundred microlitres of the assay buffer was added into the mixing well per plate and 100 µl of the diluted standards (ranging from 0 to 40 ppt) and urine samples was added into wells of the mixing well per plate. The contents of the mixing well were shaken using a micro-shaker (DYNATECH) for 2 min. One hundred microlitres of the mixture was transferred to the antibody-coated Reaction-Assay Plate (aflatoxin M1 assay for urine, Helica Biosystems Inc, 1527 W. Alton Santa Ana, California, USA). The samples were mixed by shaking for 1 min (DYNATECH Shaker) and incubated at room temperature in the dark for 1 h. The plate was washed three times using phosphate-buffered saline–Tween-20 (0.05%) buffer using the well-wash (Thermo Scientific, Finland) machine with 3-min intervals between the washes. After drying, 100 µl of conjugate was added into each well, mixed gently by tapping, and incubated at room temperature for 15 min in the dark. The plate was then washed and 100 µl of substrate reagent (tetramethylethidine) was added into each well, mixed gently by tapping, and incubated at room temperature for 15 min in the dark. The reaction was stopped by adding 100 µl per well of stop solution and the optical density (OD) read at 450 nm within 15 min of stopping the reaction. The level in each sample was determined using the program from the kit manufacturer, which allowed calculations of the levels based on the absorbance readings.

Testing for aflatoxin M1 in breast milk using cELISA (Ridascreen test kit)

The analysis of milk for AFM1 was carried out using the competitive ELISA method according to manufacturer's instructions (r-biopharm, Germany) with modification adopted from EVIRA (Finnish Food Safety Authority). Analysis was done within 3 months of sample collection. Five milliltres of milk sample was warmed and centrifuged for 15 min at 1011 × g. The upper cream layer was removed and 2.5 ml of the defatted milk transferred to a test tube and 5 ml of ethyl acetate (88.1 g/mol; melting point –83.6°C) added. This was vortexed for 1 min and the mixture centrifuged at 1011 × g for 15 min at room temperature. Three milliltres of the ethyl acetate layer were transferred into a clean test tube and evaporated to dryness using a stream of nitrogen. The sample was diluted with 250 µl of the sample dilution buffer and 30 µl of 70% methanol:water, vortexed, and analysed for AFM1 using a competitive ELISA according to manufacturer's procedure. The limit of detection was 5 ng/l. Levels below this limit were estimated using a company program that determined quantities by extrapolation.

Anthropometric measurements

Height, weight, and age were the anthropometric measures taken from the children enrolled in the study. The height was measured

using a measuring tape to the nearest whole centimetre while the weight was measured to the nearest gram using a Salter weighing scale. Weight for age *Z*-score (WAZ), height for age *Z*-score (HAZ), and weight for height *Z*-scores (WHZ) were calculated according to the median value of the international reference population recommended by the National Centre for Health Statistics (NCHS) and World Health Organization (WHO, 1986).

Household food sources and consumption

A questionnaire was administered to the respondents of 547 households in total. The respondents, mainly spouses, were asked to indicate for each household member the type of food eaten (maize, sorghum, and millet); source of the food type whether it was homegrown, market sourced, gift, or relief; amounts eaten and frequency per day. The amount eaten was indicated by the wife who dished out food for every household member from a template displaying a photograph of the food quantities; she would select the amount which closely estimated the amount served.

Data analysis

The data were entered in spreadsheets and analysed in SPSS Version 19.0. Exploration of the data was by numerical summaries together with graphics. Relations and associations in both the opinion/perceptions and experimental data were further examined using chi-square and *t*-test for the level of significance at 5%.

Results

Food consumption

Cereals

The largest quantity of maize, sorghum, and milk consumed by households was home produced. Maize is the main staple food in Nandi and Makueni. The majority of the households (60–90%) relied on homegrown maize except in Makindu location in Makueni and Kaptumo location in Nandi where homegrown maize contributed about 40% of the household needs. Maize and sorghum was consumed as *ugali* or porridge and *muthokoi* (traditionally dehulled maize, cooked together with beans or pigeon peas). Maize and sorghum in the form of *uji*, *githeri*, and *ugali* were used for weaning children. Children younger than 1 year and between 1 and 5 years ate more sorghum *uji* in Makueni, compared to those of the same age (Figure 1) in Nandi. The difference in consumption between the two age groups was significant with $P = 0.037$ and $P = 0.002$ for younger than 1 year and 1–5 years, respectively. Makueni households were giving *uji* made from maize to older children (1–5 years) while in Nandi this started with children younger than 1 year.

Consumption of maize in the form of flour per person per day differed across the sites and within age groups. For children younger than 5 years, consumption ranged from 0.1 to 0.25 kg per person per day in Nandi and Makueni, respectively. The overall average consumption of maize in the form of flour in Nandi and Makueni in terms of kg per person per day was 0.18, 0.36 and 0.4 for people younger than 5, 6–60, and older than 60 years, respectively. The most vulnerable individuals to aflatoxin poisoning (younger than 5 years and older than 60 years) in Makueni were consuming 0.22 and 0.41 kg of flour per day, respectively, but this was not the case in Nandi where those younger than 5 and older than 60 years consumed 0.13 and 0.37 kg, respectively.

Consumption of milk

Households in both Makueni and Nandi had an average of 5.9 persons and consumed on average 0.38 of milk per person per day in the two sites. The average consumption in Makueni and Nandi was 0.32 and 0.44 of milk per person per day, respectively. The annual per capita consumption was 117.9 and 161.9 in Makueni and Nandi, respectively. Sixty-three per cent of children less than 1 year old were consuming milk as fresh, 16% consumed milk mixed with porridge, while 3.4% drank milk with *ugali* and 8.7% took milk in the form of fermented milk, especially in Nandi.

Aflatoxin M1 in urine

A total of 377 and 362 urine samples were collected from children younger than 5 years in Makueni and Nandi, respectively, and analysed using cELISA for AFM1 in urine. Results showed that 79% and 83% of the urine samples were positive in both Makueni and Nandi, respectively (Table 1). There was a significant difference ($t = 12.64$, P value = 0.00) in the mean of aflatoxin M1 levels in the urine samples collected from the two counties, though there was no significant difference in the values between urine samples from children younger than 30 months and those between 30 and 60 months within the counties. Urine samples (positive and negative) from Makueni had a higher mean level of AFM1 (910.56 [721.31, 1099.78] parts per trillion (ppt)) than those from Nandi (518. 60 [400.58, 636.62] ppt). The children younger than

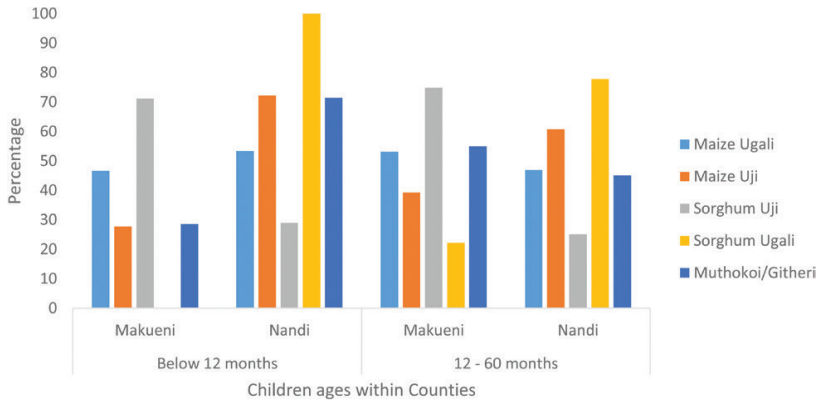

Figure 1. Consumption patterns of diets based on maize, sorghum, and millet by children younger than 60 months in Makueni and Nandi counties. *Ugali*, thick porridge prepared from maize flour; *Uji*, porridge prepared from flour (sorghum, millet, and maize singly or in combinations).

30 months in Makueni had very high levels of AFM1 in urine; being 1.4 times higher than that found in the urine of children of the same age in Nandi. For children between 30 and 60 months in Makueni, the levels of AFM1 in urine were 2.3 times higher than those from Nandi.

Aflatoxin M1 in breast milk

A total of 67 and 98 human milk samples were collected from Nandi and Makueni, respectively, and 86.7% and 56.7% of these samples were positive for aflatoxin M1 in Makueni and Nandi, respectively (Table 2). There was a significant difference ($t = 30.75$, $P < 0.001$) in the mean values of aflatoxin M1 in the breast milk from Nandi and Makueni. The Makueni samples had a higher mean of 8.46 [8.24, 8.68] ppt compared to 0.02 [0, 0.061] ppt in Nandi. The mean level in breast milk samples from Makueni was 423 times higher than the mean level found in the breast milk samples from Nandi.

Exposure through maize and milk

Table 3 shows the exposure to aflatoxins in Makueni and Nandi among the various age groups. Makueni households had a higher exposure rate than the households in Nandi. This is a reflection of higher average levels of aflatoxins in foods from the two sites. Exposure rates to aflatoxins through flour in children younger than 5 years in Makueni were 44 times more than in their counterparts in Nandi.

Growth indices

Stunting

Stunted growth is a reduced growth rate in human development. It is a primary manifestation of malnutrition in early childhood. The stunting and severe stunting rates in Makueni and Nandi were 28.7, 18.5 and 30.7, and 16.5%, respectively. The national average for stunting is 26 and 11% for severe stunting (KDHS, 2015). The levels of aflatoxin in cereals from households with stunted children differed significantly between the two counties (Table 4). The AFM1 levels in urine from the stunted children was not significantly different between Makueni and Nandi.

Underweight

Weight-for-age is a composite index of height-for-age and weight-for-height. It takes into account both acute and chronic malnutrition. Children whose weight-for-age (WAZ) is below –2 standard deviations (SD) are classified as underweight. Children whose weight-for-age is below –3 SD are considered severely underweight. The underweight (WAZ) children were 2.9 and 14.6% in Nandi ($n = 102$) and Makueni ($n = 103$), respectively, while Makueni and Nandi registered a proportion of 3.9% each for severe underweight. The national average for underweight is 11 and 2% for severe underweight (KDHS, 2015). The estimated levels of underweight are 10.2 and 11% in Makueni and Nandi, respectively.

Discussion

The presence of AFM1 in urine indicates recent exposure of children to aflatoxin-contaminated food because the AFM1 is easily excreted from the body. Children younger than 5 years were introduced to cereal-based diets at different times in the two counties and their consumption of maize-based diets ranged from 0.1 to 0.25 kg per person per day in Nandi and Makueni, respectively. Lower consumption of maize-based diets in Nandi is due to consumption of alternative foods from sorghum and finger millet. Nandi compared to Makueni is richer in other types of foods. The proportion of maize contaminated with aflatoxin from both counties was 68.3 and 80.4% in Nandi and Makueni, respectively (Kang'ethe *et al.*, unpublished data), with Makueni having 24.5% of positive samples with aflatoxin levels above 10 ppb level. Sorghum was equally contaminated with aflatoxin, 66.7 and 88.9% of the samples were positive and 37.1 and 29.9% exceeded the limits in Nandi and Makueni, respectively. Feeding on such contaminated foods explains the high levels of AFM1 in urine samples from children in both Nandi and Makueni counties. This finding agrees with Gong *et al.* (2002, 2004) in Benin who noted that the levels of aflatoxin–albumin adjunct levels were approximately two times higher in fully weaned children compared with those receiving a mixture of breast milk and solid food.

Human breast milk samples from Makueni had higher AFM1 levels than the levels in Nandi samples (mean 8.46 [8.24, 8.68] ppt in Makueni compared to 0.02 [0, 0.061] in Nandi). The proportion of samples with levels above the 25 ppt EU limit was 10.2% in Makueni but none exceeded this limit (25 ppt) in Nandi. This is a further reflection of the higher levels of aflatoxins found in cereals consumed in these two areas where more samples exceeded the 10 ppb limit in Makueni (25.05 [18.48, 31.62]) than in Nandi 0.98 ([0.86, 1.10]). In Nigeria Adejumo *et al.* (2013) reported 82% of the breast milk samples were positive and 16% exceeded the 25 ng/kg (ppt) limit for AFM1 by the European Union. The socioeconomic status of the mothers of the children in the Nigerian study was found to influence their dietary exposure and also that of the infants breastfeeding on them. Abdulrazzaq *et al.* (2003) reported that in the United Arab Emirates 92% of the breast milk samples tested were positive, while Sadeghi *et al.* (2009) reported a prevalence of 98.1% positive in the breast milk samples taken in Tehran but only one sample exceeded the EU/USA limit of 25 ng/kg limit. The major source of AFM1 in breast milk, like AFM1 in children urine, is the recent consumption of aflatoxin-contaminated foods by the nursing mothers.

Homegrown maize constituted 75 and 59% of the household consumed maize in Nandi and Makueni, respectively. The homegrown maize had mean aflatoxin levels of 0.98 [0.86, 1.10] and 25.05 [18.48, 31.62] for Nandi and Makueni, respectively (Kang'ethe *et al.*, unpublished data). The other source of high levels of AFM1 in breast

Table 1. Aflatoxin M1 in positive urine samples from children under 30 months and younger than 5 years in Makueni and Nandi counties.

County	Age group	Mean (ppt) (95% CI)	*t*-value	*P*-value
Makueni	Younger than 30 months ($n = 34$)	1182.894 (±637.99)	0.562	0.46
	30–60 months ($n = 47$)	1546.257 (±662.65)		
Nandi	Younger than 30 months ($n = 42$)	857.279 (±625.09)	0.481	0.49
	30–60 months ($n = 54$)	667.294 (±299.06)		

Table 2. Aflatoxin M1 in breast milk samples (cELISA and HPLC) from breast feeding mothers in Nandi and Makueni counties.

Sample	Site	% Positive	% >25 ppt	Range of positive (ppt)	Mean (ppt) [95% CI]	*t*-value	*P*-value
cELISA							
Breast milk	Makueni (*n* = 98)	86.7	10.2	0.215–47.5	8.46 [8.24, 8.68]	19.96	<0.001
	Nandi (*n* = 67)	56.7	0.0	0.003–3.7	0.02 [0, 0.061]		
HPLC							
Breast milk	Makueni (*n* = 18)	22.2	11.1	1.4–152.7	10.83 [6.86, 14.8]	1.4	0.183
	Nandi (*n* = 21)	9.5	0.0	0.5–0.8	0.06 [0.043, 0.077]		

Table 3. Exposure to aflatoxins from consumption of maize and aflatoxins in milk in Makueni and Nandi counties.

Food type	Age in years	Aflatoxins			
		Makueni		Nandi	
		Average consumption (kg per day)	Average exposure (µg per kg bwt per day)*	Average consumption (kg per day)	Average exposure (µg per kg bwt per day)*
Maize flour	<5	0.28	0.49	0.17	0.011
	6–59	0.59	0.26	0.48	0.008
	Over 60	0.60	0.26	0.48	0.008
Milk	<5	0.27	0.0004	0.55	0.0001
	6–59	0.36	0.0001	0.73	0.00004
	Over 60	0.33	0.0001	0.72	0.00004

*The average body weight was based on median measured body weight of 16 500 men and 19 969 women in 13 EU states after applying growth factors. For less than 5 years, the average body weight was the mean of average body weight of children less than 3 years and average body weight of children between 3 and 6 years (van Buuren *et al.*, 2012).

Table 4. Aflatoxin levels in cereals from households with stunted children and Aflatoxin M1 content in urine samples from the stunted children in the same households in Makueni and Nandi counties.

County	Food type	Mean aflatoxin levels in ppb (±95% CI)
Nandi	Maize (*n* = 37)	0.97 [0.67, 1.27]
	Sorghum (*n* = 9)	27.8 [0, 63.88]
	Millet (*n* = 6)	1.50 [1.06, 1.94]
	Urine AFM1 (*n* = 41)	997.3 [0, 17638.82]
Makueni	Maize (*n* = 98)	41.5 [26.65, 56.35]
	Sorghum (*n* = 46)	20.43 [6.94,33.92]
	Millet (*n* = 1)	2.00
	Urine AFM1 (*n* = 106)	1336 [925.33, 1746.67]

AFM1, Aflatoxin M1 in parts per trillion.

milk is consumption of cow milk contaminated with AFM1. The presence of AFM1 in cow milk is a result of feeding AFB1 contaminated feeds to the animals. Spoilt maize was used as animal feed across the two counties (23.9% in Makueni and 77.6% in Nandi, Kang'ethe *et al.*, unpublished data) and this acted as an exposure route for animals to aflatoxins and eventually exposure of breast feeding mothers and children weaned on to cow milk to this mycotoxin. Aflatoxin M1 mean levels in the home-produced milk were 22.3 [16.89, 7.67] ppt in Makueni and 2.7 [1.92, 3.50] ppt in Nandi.

Considering that a child consumes 776 ± 141 g per day of breast milk (WHO/UNICEF, 1998), and taking an average of 776 g per day and a mean for aflatoxin contamination of 10.83 and 0.06 ppt for breast milk obtained from Makueni and Nandi, respectively, determined using HPLC, exposure through this route would be 6×10^{-3} and 1×10^{-6} µg per kg body weight (bwt) per day in Makueni and Nandi, respectively. The levels are lower than the allowed limit of 19 400 ppt per day intake considering a maximum limit of 25 ppt and 776 g of milk intake per day (Ghiasian and Maghsood, 2012). Aflatoxin intake in children from this route would be excreted through urine and is consistent with these intake levels.

This study confirms that malnutrition is a problem in the two counties; Makueni shows a higher percentage of children underweight, 14.6%, and equated to Nandi, 2.9%. Makueni also had a higher percentage of stunted children (28.7%) than Nandi (18.6%). These figures, although lower than those reported for Kenya (16.4% for underweight and 36.2% for stunting; WHO, 2010), the Makueni (in Eastern province) figures are lower for stunting and higher for underweight than those reported for the Eastern province (30.1% stunting and 12.2% underweight, KDHS, 2015). Again, the Nandi (Rift valley province) figures are higher for stunting than the figures given for the entire Rift valley (14.9%) but less than the underweight figure (7.3%) for Rift valley province (Kenya Nutritional profile, FAO, 2005), although these values are high enough to cause concern. These figures are also comparable with the prevalence recorded by Gong *et al.* (2002) in Togo and Benin of 33% for stunting and 29% for underweight. Although the evidence linking aflatoxins to causal of stunting is still weak, aflatoxins are known to contribute to stunting. It was noted in a study in Togo and Benin (Gong *et al.*, 2002) that the degree of stunting and underweight in children younger than 5 years was correlated with high aflatoxin–albumin adducts found in the serum of 99% of the children. In Nandi, children younger than 30 months had higher levels of aflatoxin M1 in urine than those over 30 but less than 60 months old, but this was the reverse in Makueni. The difference in the aflatoxin M1 levels in urine for children younger than 30 months and older than 30 months in Makueni and Nandi counties was not statistically significant ($t = 0.562$, $P = 0.456$; $t = 0.481$, $P = 0.49$, respectively). There was no significant difference for the AFM1 in urine for children younger than 30 months in

Makueni and Nandi (t = 0.67, P = 0.415), but there was a significant difference in the AFM1 levels in urine samples for children older than 30 months and younger than 60 months (t = 6.126, P = 0.015) with those in Makueni having higher levels and double those in Nandi.

The aflatoxin levels in maize, sorghum, and millet from households with stunted children and AFM1 in urine of stunted children from the same households were compared for Makueni and Nandi (Table 4). Households with stunted children but where no samples were analysed for aflatoxins were not taken into account. The mean aflatoxin levels in maize from households with stunted children were significantly different between Makueni and Nandi (t = 10.75, P < 0.01) whereas aflatoxin levels in sorghum and millet from households with stunted children and urine from stunted children were not significant between the two counties. The likelihood of children who were stunted and being exposed to aflatoxins (presence of AFM1 in urine) was 6.8 times more likely to occur in Makueni compared to 2.1 times in Nandi (OR = 6.8 and 2.1, respectively) while the probability of underweight children being exposed to aflatoxins was 2.4 times more likely to occur in Makueni compared to 1.9 times among children in Nandi (OR = 2.4 and 1.9 respectively).

Conclusions

The exposure of people to aflatoxins (or mycotoxins) consequently starts early and continues into adulthood. The results of this study emphasize the need to be more vigilant on aflatoxin and control. It is important to educate the farmers on better cattle feeding practices and mothers on the need to feed the family with good-quality cereals to avoid chronic exposure to aflatoxin through the feed-milk chain. Mothers would be a good target for these capacity building messages as preparation of the family food is the mother's main responsibility.

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Conflict of interest statement. The authors declare that they have no conflict of interest with the publication of this paper.

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