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Internal Exposure to Mycotoxins and Associated Nutritional Risk in Young Children Consuming Maize Porridge in South Kivu, Democratic Republic of Congo: A Pilot Biomonitoring Study

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Abstract

Background: Mycotoxin contamination of maize-based foods is an important but under-characterized exposure pathway for young children in sub-Saharan Africa. Evidence on internal exposure to multiple mycotoxins and its relationship with nutritional status in South Kivu, Democratic Republic of Congo (DRC), remains limited.

Objective: This pilot study assessed internal exposure to multiple mycotoxins among young children consuming maize porridge, characterized co-exposure patterns and exposure determinants, and explored associations between mycotoxin exposure and nutritional status.

Methods: A cross-sectional biomonitoring study was conducted among 70 children aged 11-23 months from rural and urban households in South Kivu. Serum samples were analyzed by liquid chromatography-tandem mass spectrometry (LC-MS/MS) for multiple mycotoxins and metabolites, together with screening for the aflatoxin B1 (AFB1)-lysine adduct. Household

characteristics, maize storage and processing practices, and child feeding practices were recorded. Nutritional status was assessed using height-for-age (HAZ), weight-for-age (WAZ), weight-for-height (WHZ) Z-scores, and mid-upper arm circumference (MUAC). Bayesian regression models were used to investigate exposure determinants and associations with nutritional outcomes.

Results: Daily maize-porridge consumption was more frequent among rural than urban children (46.9% vs. 31.6%; $p < 0.001$), whereas food-washing practices were more common in urban households (63.2% vs. 3.1%; $p < 0.001$). Stunting affected 33.3% of children and was more prevalent among rural than urban children (37.5% vs. 28.9%; $p = 0.048$) and among boys than girls (47.2% vs. 18.1%; $p = 0.023$). Underweight and wasting affected 18.8% and 10.1%, respectively. Citrinin (CIT), ochratoxin A (OTA), and cyclopiazonic acid (CPA) were detected in serum, with higher median CIT and OTA concentrations among rural than urban children (1.3 vs. 0.1 ng/mL and 0.25 vs. 0.10 ng/mL, respectively), while CPA concentrations did not differ significantly by residence or sex; however, CPA detection was higher among rural than urban children (31.2% vs. 5.3%; $\chi^2 = 3.38$, $p < 0.05$). CIT and OTA exposure increased with frequent maize-porridge consumption, while AFB1-lysine exposure was associated with maize-porridge consumption and positively associated with stunting ($\log[\text{OR}] = 1.70$; 95% CrI: 1.02–18.6).

Significance: These findings provide preliminary evidence of multiple mycotoxin exposure among young children in South Kivu and identify maize-porridge consumption as an important potential exposure pathway. The association between AFB1-lysine exposure and stunting warrants confirmation in larger longitudinal studies.

Impact Statement

This study provides rare biomonitoring evidence of multiple mycotoxin exposure among young children in South Kivu. Linking serum biomarkers with dietary practices and nutritional indicators highlights maize-porridge consumption as a potentially important exposure pathway and supports integrated food-safety, biomonitoring, and nutritional interventions to reduce mycotoxin exposure during early childhood.

Keywords: Mycotoxins, Nutritional and health status, Biomonitoring, Citrinin, Ochratoxin A, AFB₁-lysine, Cyclopiazonic acid, sub-Saharan Africa

1. Introduction

Mycotoxins are toxic secondary metabolites produced by fungi, which frequently contaminate staple crops such as maize in tropical and subtropical regions. Chronic dietary exposure to mycotoxins has been associated with impaired immune function, disrupted nutrient metabolism, growth impairment, and carcinogenicity. Consequently, mycotoxins represent a major public health concern in low- and middle-income countries (LMICs) where monitoring is limited and exposures often exceed international reference values (Gichohi-Wainaina *et al.*, 2023; Li *et al.*, 2025; Gemedo, 2025; Miderho *et al.*, 2026).

In sub-Saharan Africa (SSA), mycotoxin contamination of maize-based complementary foods is widespread, and studies conducted in the Democratic Republic of Congo (DRC), Tanzania, Kenya, and Nigeria have documented frequent co-occurrence of aflatoxins (AFs), fumonisins (FUMs), deoxynivalenol (DON), and other toxins in food ingredients intended for infants and young children. Limited dietary diversity and a high reliance on maize-based porridge as complementary food further increase the risk of dietary exposure during early life (Ojuri *et al.*, 2018; Ezekiel *et al.*, 2022; Mollay *et al.*, 2022; Chilaka & Mally, 2020; Ngure *et al.*, 2023; Miderho *et al.*, 2026).

Epidemiological evidence from SSA links chronic mycotoxin exposure to impaired linear growth stunting (HAZ), wasting (WHZ), and underweight (WAZ) in young children. Although the underlying mechanisms remain poorly understood, existing studies suggest that disruptions in nutrient absorption, immune activation, and metabolic pathways contribute to adverse anthropometric outcomes in exposed populations (Chilaka & Mally, 2020; Chen *et al.*, 2018; Gichohi-Wainaina *et al.*, 2023).

Most mycotoxin research in the region has focused on food frequency questionnaires or urinary biomarkers. While these approaches provide valuable insights into exposure sources and excretion patterns, they do not fully capture internal systemic dose, i.e. the amount of toxin that is absorbed and capable of exerting biological effects. In contrast, blood-based biomarkers such as AF-lysine adducts and other circulating toxin or metabolite measures integrate dietary intake, absorption efficiency, metabolism, and clearance, making them more directly relevant for assessing biologically effective exposure linked to health outcomes (Gong *et al.*, 2016; Xu *et al.*, 2018; Arce-López *et al.*, 2020; Turner & Snyder, 2021).

In the DRC, maize constitutes a primary complementary food for infants (up to 11-month old) and young children (12 to 23-month old). However, region-specific data on internal mycotoxin exposure in this population remain exceptionally sparse, with most available studies being limited. Most available studies have focused on food handling practices and contamination prevalence, and, to date, none has characterized multi-mycotoxin serum biomarkers in this high-burden setting. In a previous study, we reported the occurrence of multiple mycotoxins in maize flour used for the preparation of complementary foods for these young children (Miderho *et al.*, 2026).

The present pilot study addresses a critical gap by assessing exposure to multiple mycotoxins in young children aged 11-23 months in South Kivu Province, a region characterized by a high prevalence of stunting (48-53%) (DHS, 2007; Kismul *et al.*, 2017) and frequent consumption of maize-based porridge. Systemic exposure was assessed through liquid chromatography tandem mass spectrometry (LC-MS/MS) based serum biomonitoring, alongside detailed dietary intake and anthropometric indicators (e.g. weight-for-height (WHZ) and height-for-age (HAZ)). By linking internal exposure biomarkers with health and nutritional status across rural and urban health zones within eastern DRC, this study provides a novel assessment of multi-mycotoxin co-exposure patterns during a critical complementary feeding period. Furthermore, it offers proof-of concept evidence to inform targeted mycotoxin mitigation strategies, support food safety policy discussions, and guide future large-scale exposure assessments relating internal dose to child health outcomes in high-risk populations.

2. Material and Methods

2.1. Study area

The study was conducted in Miti-Murhesa (rural) and Bagira (urban) health zones in South Kivu Province, eastern DRC (2°30'-3°30'S, 28°50'-29°50'E) (Figure 1). The region has a tropical highland climate with two rainy seasons, annual rainfall of 1,200-1,600 mm, and mean temperatures of 18-24 °C, conditions that favor fungal growth and mycotoxin accumulation in maize (pre- and post-harvest).

The two study zones were selected to capture contrasting environmental and socio-economic contexts that may influence dietary mycotoxin exposure. Miti-Murhesa, a predominantly rural area, is characterized by smallholder maize-based agriculture, traditional post-harvest handling,

and household-level storage practices. In contrast, Bagira, an urban “commune” within Bukavu, the provincial capital city, represents a setting where households primarily depend on market-purchased maize flour. These contrasting contexts provide a robust framework for evaluating environmental and socio-economic determinants of internal mycotoxin exposure among young children.

2.2. Study design and population

This study employed a cross-sectional pilot biomonitoring design to quantify internal exposure to multiple dietary mycotoxins in young children aged 11-23 months, corresponding to the critical complementary feeding period when maize-based porridges are actively consumed (Alvito & Pereira-Da-silva, 2022; Miderho *et al.*, 2026). Eligible participants were children who had resided in the study area for at least one year and whose primary caregivers provided written informed consent. Participants were randomly selected from the general population within the same study areas as a prior external exposure survey (Miderho *et al.*, 2026). Although some households may have overlapped those included in the previous survey, participant selection for this study was conducted independently to ensure population representativeness while maintaining randomness.

Children with severe acute illness, chronic medical conditions, or recent blood transfusions were excluded to ensure safety and avoid confounding of biomarker measurements.

The sample size was calculated using Fisher’s formula (Fisher *et al.*, 1991), for estimating a single population proportion, as described by Lwanga *et al.*, (1991):

$$n = \frac{Z^2 P(1-P)}{d^2} \quad (1)$$

where n represents the required sample size, Z is the standard normal value corresponding to a 95% confidence level (1.96), P is the anticipated prevalence of detectable mycotoxin biomarkers, and d is the desired absolute precision (margin of error).

The anticipated prevalence (P) was informed by previous biomonitoring studies among infants and young children, which have reported a high frequency of detectable aflatoxin biomarkers. In the AflaCohort Study conducted among children aged 18-22 months in Nepal, detectable aflatoxin biomarkers were frequently observed, demonstrating substantial exposure among young children (Andrews-Trevino *et al.*, 2022). Based on these findings, an expected prevalence of 80% was selected as a conservative estimate for sample size calculation.

The desired precision (d) was set at 10%. According to Lwanga *et al.*, (1991), precision represents the acceptable deviation between the estimated sample proportion and the true population value and directly influences the required sample size. Given that this investigation was designed as a pilot biomonitoring study, the objective was not to generate definitive population prevalence estimates but rather to obtain preliminary information on mycotoxin exposure levels, variability, and the feasibility of biomarker assessment. Therefore, a moderate precision level was considered appropriate, balancing the need for informative estimates with practical and financial constraints related to participant recruitment, serum collection, and LC-MS/MS laboratory analyses. This approach aligns with pilot study recommendations emphasizing feasibility assessment, estimation of preliminary parameters, and refinement of methodologies for future adequately powered epidemiological studies (Thabane *et al.*, 2010; Montgomery, 2025).

Using these assumptions, the minimum calculated sample size was 62 children. To compensate for potential losses due to incomplete questionnaires, inadequate biological samples, or analytical

failures during laboratory processing, a 10% allowance was added, resulting in an adjusted sample size of approximately 69 children. Consequently, 70 young children were enrolled, including 38 children from Bagira (urban setting) and 32 from Miti-Murhesa (rural setting).

The study therefore focused on characterizing biomarker occurrence, describing exposure patterns, assessing variability, and evaluating the feasibility of biological sample collection and laboratory procedures.

2.3.Sampling and recruitment

A two-stage cluster sampling approach was employed to recruit the participants. In the first stage, 50% of health areas within each health zone were randomly selected. In the second stage, eligible households were randomly selected from the general population within these areas. One child per household was randomly selected. In households with multiple eligible children, a lottery method using numbered slips was applied, while households with a single eligible child automatically enrolled for that child.

Community health facilitators informed eligible households about the study and invited mothers/caregivers to bring their children to designated health facilities (“Centre de Santé de Karhanda” in Miti-Murhesa, and “Hôpital Général de Référence de Bagira” in Bagira) for interviews and blood sampling.

Structured interviews were conducted using KoBoCollect, a standardized questionnaire approved in English and translated into French. During administration, facilitators often used local languages (Kiswahili and, occasionally, Mashi) to ensure comprehension. The interview collected data on

demographics, socio-economic status, infant feeding practices, and household maize storage and preparation practices, which are critical determinants of mycotoxin exposure.

2.4. Anthropometric assessment

Anthropometric measurements were obtained according to the World Health Organization (WHO)-recommended protocols (WHO, 2006). Weight was measured using a digital scale calibrated daily and recorded to the nearest 0.1 kg. Recumbent length was measured with a portable infantometer to the nearest 0.1 cm. All measurements were taken in duplicate, and the mean of the two readings was used for analysis. Anthropometric data allowed to determine the nutritional status. Z-scores were calculated for height-for-age (HAZ), weight-for-age (WAZ), and weight-for-height (WHZ) using WHO growth standards. Malnutrition states were defined as stunting (HAZ < -2 SD), wasting (WHZ < -2 SD), underweight (WAZ < -2 SD), moderate acute malnutrition (MAM, WHZ between -3 and -2 SD), severe acute malnutrition (SAM, WHZ < -3 SD), and low mid-upper arm circumference (mid-upper arm circumference, MUAC < 12.5 cm). Implausible values (HAZ < -6 or > +6; WAZ < -6 or > +5; WHZ < -5 or > +5) were treated as missing. These standardized indicators were subsequently used as binary outcomes in statistical analyses to explore associations with internal mycotoxin exposure, enabling the evaluation of both chronic and acute nutritional effects.

2.5. Blood sample collection and handling

Trained medical personnel collected 2-3 mL of venous blood from each child using sterile serum separator tubes (BD Vacutainer®, Becton Dickinson) with no added anticoagulant and standard aseptic techniques. Immediately after collection, the samples were allowed to clot and were

centrifuged at $1,500 \times g$ for 10 min at room temperature, to separate serum. Samples were labeled with unique participant identification codes. The specimens were then stored at $-80\text{ }^{\circ}\text{C}$ under controlled conditions to ensure sample integrity.

Only serum was retained for multi-mycotoxin LC-MS/MS analysis, as it provides a stable, integrative measure of systemic exposure reflecting ingestion and absorption into the systemic circulation. Plasma was avoided due to potential interference from anticoagulants in LC-MS/MS detection (Arce-López *et al.*, 2020 ;Owolabi *et al.*, 2023). Serum aliquots were maintained in a cooled container during transport to the local laboratory (the Center for Tropical Diseases and Global Health, CTDGH, of the Catholic University of Bukavu). Approximately 1 mL of serum was aliquoted into labeled cryotubes (Nunc™ CryoTubes, Thermo Fisher Scientific) and stored at $-80\text{ }^{\circ}\text{C}$ to preserve analyte stability for subsequent analysis until shipment for analysis. The frozen serum samples were subsequently shipped on dry ice (solid CO_2) to the Centre of Excellence in Mycotoxicology and Public Health (CEMPH), Ghent University, Belgium, for multi-mycotoxin quantification and AFB₁-lysine screening.

2.6. Laboratory analysis

- Target biomarkers

The external exposure survey published by Miderho *et al.*, (2026), reporting the multi-mycotoxin concentrations of maize porridge using LC-MS/MS, served as a general foundation for selecting the target mycotoxin analytes for serum analysis in the present study. The multi-mycotoxin panel used for serum analysis encompassed a total of 39 mycotoxins and metabolites, including AFs (AFB₁, AFB₂, AFG₁, AFG₂, AFM₁), DON, its metabolites deepoxy-deoxynivalenol (DOM), DON-3-glucoside (DON-3-G) and 3/15-acetyl-DON (3/15-ADON), FUMs (FB₁, FB₂, FB₃, HFB₁), nivalenol (NIV), neosolaniol (NEO), fusarenon-X (FUS-X), diacetoxyscirpenol (DAS), T-2 toxin (T2), HT-2 toxin (HT2), citrinin (CIT), ochratoxin A (OTA) and ochratoxin alpha (OT α), enniatins (ENN A, ENN A1, ENN B, ENN B1), beauvericin (BEAU), alternariol (AOH), alternariol monomethyl ether (AME), sterigmatocystin (STERIG), roquefortine C (ROQ-C), tenuazonic acid (TeA), cyclopiazonic acid (CPA), zearalenone (ZEN), zearalanone (ZAN), α/β -zearalanol (α -ZAL, β -ZAL) and α/β -zearalenol (α -ZEL, β -ZEL). In addition, all serum samples were screened for the presence of AFB₁-lysine, the established biomarker of chronic AFB₁ exposure. The multiple reaction monitoring (MRM) transitions and retention times (RT) were optimized for each compound and are summarized in supplementary information (S1 for the multi-mycotoxin analysis and S2 for AFB₁-lysine screening).

- Multi-mycotoxin analysis in serum

Simultaneous quantification of mycotoxins was adapted from the protocol of Delgado-Povedano *et al.*,(2025). Serum aliquots (300 μ L) were spiked with 20 μ L ¹³C-labeled internal standards (i.e.,

¹³C-AFB₁, ¹³C-CIT, ¹³C-DON, ¹³C-HT2, ¹³C-FB₁, ¹³C-OTA, ¹³C-T2) and subjected to liquid-liquid extraction (LLE) with 300 µL ice-cold acetonitrile (ACN). Supernatants were evaporated under nitrogen at 40 °C and reconstituted in 75 µL injection solvent (60/40 v/v of mobile phases A/B). Mobile phase A comprised of Ultrapure water (UP-H₂O)/methanol (MeOH)/acetic acid (94/5/1 v/v/v) and mobile phase B of MeOH/UP-H₂O/acetic acid (97/2/1 v/v/v), both containing 5 mM ammonium acetate. Chromatographic separation used a Waters Acquity UPLC HSS T3 C₁₈ column (1.8 µm, 2.1 × 100 mm) with a pre-column, at 0.3 mL/min flow rate and 40 °C, coupled to a Xevo TQ-XS triple quadrupole mass spectrometer for analyte detection. The gradient ranged from 95% A to 99% B over 13 min, followed by re-equilibration to 95% A until 18 min. Detection was performed in both electrospray ionization (ESI)+ and ESI- modes using optimized capillary voltage, gas flow, and collision energy. Quantification was achieved using matrix-matched calibration curves and TargetLynx® v4.2.

- Screening for AFB₁-lysine adduct in serum

Serum pretreatment and LC-MS/MS determination of AFB₁-lysine followed an optimized protocol adapted from Phillips *et al.* (2025). Briefly, 150 µL serum aliquots were vortexed and incubated with 375 µL Pronase E solution (6.5 mg/mL in phosphate buffered saline (PBS)) overnight at 37 °C in a thermoshaker (300 rpm) to hydrolyze albumin-bound AFB₁-lysine adducts. Each analytical batch included study samples and certified quality control materials provided by the Centers for Disease Control and Prevention (CDC, Atlanta, Georgia, USA).

After enzymatic digestion, samples were purified using Oasis MAX 1cc solid-phase extraction cartridges item no. 186000366 (Waters corporation, Antwerp, Belgium), as described by Phillips *et al.*, (2025). Eluates were evaporated to dryness under nitrogen (0.5 mL/min) at 40 °C and

reconstituted in 150 μ L UP-H₂O/ACN/acetic acid (94/5/1, v/v/v). Following sonication and centrifugation (15,000 $\times$ g, 5 min), supernatants were transferred to vials for analysis.

Chromatographic separation and analyte detection was performed as mentioned in *2.6 Multi-mycotoxin analysis in serum*. Detection was performed using ESI+. Gradient elution employed mobile phase A (UP-H₂O with 0.01 % acetic acid) and mobile phase B (ACN with 0.01 % acetic acid) at a flow rate of 0.3 mL/min. The analysis was conducted as a qualitative screening based on absolute peak areas processed in TargetLynx® v4.2. Sample signals were evaluated relative to quality control samples at 1 and 2.5 ppb AFB₁-lysine.

2.7. Data Analysis

All data analyses were conducted in R (version 4.3.2). Categorical variables were summarized as frequencies and percentages (%), and continuous variables as means $\pm$ standard deviations (SD) or medians with interquartile ranges (IQR) for skewed distributions.

Serum samples tested positive for AFB₁, CIT, OTA, CPA, ENN B and AFB₁-lysine. Among these, only CIT and OTA were detected above the lower limit of quantification (LLOQ) and were therefore suitable for quantitative analysis and sequential regression modeling. The remaining analytes (AFB₁, CPA and ENN B) were clearly detectable but below the LLOQ, indicating that the analytical sensitivity of the method was insufficient for reliable quantification. These analytes were therefore evaluated qualitatively, just like the results of the AFB₁-lysine screening.

Values below the limit of detection (LOD) were set to zero. Values between the LOD and the LLOQ were imputed at the midpoint of the LLOQ range (0.05 ng/mL), and reported ranges were replaced by their midpoints. Missing values were coded as NA. This approach ensured all

283 measurable signals contributed to descriptive statistics and allowed consistent binary classification
284 of high versus low exposure.

285 A co-exposure variable representing the number of mycotoxins detected per child was created and
286 categorized as low (0-1), medium (2-3), or high (≥ 4). Serum concentrations of CIT and OTA were
287 right-skewed and log-transformed using log1p to approximate normality, with high exposure
288 defined as values above the 75th percentile. Descriptive statistics for mycotoxins were visualized
289 using boxplots stratified by gender and residence, and group comparisons were performed with
290 the Wilcoxon rank-sum test.

291 Nutritional indicators including stunting, wasting, underweight, MAM, SAM, and MUAC; were
292 classified as binary outcomes using the Anthro R package (version 1.1.0). The prevalence of each
293 condition was reported stratified by gender and residence, and differences between groups were
294 evaluated using Pearson's Chi-square tests. Given the small sample size ($n=70$) and sparse
295 exposure categories, Bayesian logistic regression was used to identify demographic and dietary
296 determinants of elevated mycotoxin exposure. Separate models were fitted for high exposure to
297 citrinin (CIT) (CIT_high), high exposure to ochratoxin A (OTA) (OTA_high), and AFB₁-lysine
298 detection. Predictors included child age, gender, residence, maize porridge consumption
299 frequency, maize source, grain sorting and washing practices, and visible mold/insects in stored
300 maize (Table 1).

301 Regression coefficients were assigned weakly informative normal priors with mean 0 and SD of 2
302 (Normal(0, 2)). The Bayesian logistic regression model was specified through a Bernoulli
303 likelihood, whereby each binary outcome (CIT_high, OTA_high, or AFB₁-lysine detection) was
304 assumed to follow a Bernoulli distribution conditional on the covariates. The probability of

305 exposure was linked to predictors using a logit function. Posterior distributions were then obtained
 306 by combining the likelihood and prior distributions according to Bayes' theorem and were
 307 approximated using Markov Chain Monte Carlo (MCMC) sampling implemented via the
 308 MCMClogit function in the MCMCpack package in R. Four independent chains were run for 4,000
 309 iterations each, including a burn-in (warm-up) of 1,000 iterations, with thinning applied where
 310 necessary to reduce autocorrelation.

311 The Bayesian logistic regression model was defined using a Bernoulli likelihood (Gelman *et al.*,
 312 2013). For each child i , the binary outcome Y_i (representing CIT_high, OTA_high, or AFB₁-lysine
 313 detection) was assumed to follow a Bernoulli distribution conditional on the predictors according
 314 to equation 2:

$$Y_i | \pi_i \sim (\text{Bernoulli}(\pi_i)) \quad (2)$$

315 where Y_i is the binary outcome for child i , π_i the probability of exposure. The probability of high
 316 exposure was modeled using a logit link function according to equation 3:

$$\text{logit}(\pi_i) = \beta_0 + \sum_{k=1}^K \beta_k x_{ik} \quad (3)$$

317 where Y_i is the binary outcome for child i , π_i the probability of exposure, x_{ik} the k predictor
 318 variables, and β_k the regression coefficients. Regression coefficients were exponentiated to obtain
 319 odds ratios (ORs) with 95% credible intervals (CrI). Posterior probabilities quantified the strength
 320 of associations, and associations were considered credible when posterior probability ≥ 0.90 .
 321 Regression coefficients were exponentiated to obtain odds ratios (ORs) with 95% CrI. In addition,

322 results are reported both on the odds ratio scale and on the log scale to improve interpretability,
 323 according to equation 4:

$$\log(OR) = \ln(OR) \quad (4)$$

324 The 95% credible intervals (CrI) were transformed accordingly to equation 5:

$$\log(CrI) = [\ln(CrI_{lower}), \ln(CrI_{upper})] \quad (5)$$

325 Posterior probabilities were used to quantify the strength of associations, and effects were
 326 considered credible when the posterior probability of a non-zero was ≥ 0.90 .

327 Autocorrelation among successive samples was evaluated and reduced through thinning. Posterior
 328 predictive checks indicated that the models adequately reproduced the observed data patterns.
 329 Finally, sensitivity analyses using frequentist logistic regression demonstrated substantial
 330 instability in conventional estimates, further supporting the robustness of the Bayesian approach.

331 **3. Results**

332 **3.1. Sociodemographic, child and maternal characteristics**

333 Significant urban-rural differences were observed in maternal education and occupation (Table 2)
 334 Maternal education was markedly higher in urban households: 44.7% of urban mothers had
 335 completed secondary education compared with 28.1% of rural mothers, and 7.9% of urban mothers
 336 had higher education, which was absent in rural mothers. Conversely, absent formal education was
 337 reported in 37.5% of rural mothers versus 13.2% of urban mothers ($\chi^2 = 9.1$, $p = 0.028$). Occupation
 338 patterns also differed substantially: 65.6% of rural mothers were farmers compared with only

10.5% of urban mothers, while housewifery predominated in urban settings (50%) but was uncommon in rural households (6.3%) ($\chi^2 = 41.2$, $p < 0.001$).

Furthermore, boys represented 39.5% of urban children and 65.6% of rural children, while girls accounted for 60.5% and 34.4%, respectively ($\chi^2 = 4.4$, $p = 0.036$). In contrast, the number of children under 5 per household did not differ significantly across both settings ($\chi^2 = 5.4$, $p = 0.068$), with most households having 2-3 children (urban 57.9%, rural 65.6%).

3.2. Maize storage, food handling, and infant feeding practices

Household maize handling and consumption differed between urban and rural settings (Table 3). Maize storage location varied significantly between the two groups ($\chi^2 = 9.38$, $p = 0.031$). The majority of households stored maize on the ground, a practice observed in 81.6% of urban and 65.6% of rural households. Granary storage was reported by 10.5% of urban and 21.9% of rural households, while hermetic Purdue Improved Crop Storage (PICS) bags were used by 7.9% of urban and 12.5% of rural households. Median storage duration was similar in both settings (urban: 12.0 days, IQR 7-14; rural: 7.0 days, IQR 3-14; $W = 482.5$, $p = 0.242$). There was no significant difference in storage cleaning frequency (weekly cleaning: 78.9% urban, 81.3% rural) or in the presence of visible mold or insects (urban: 47.4%; rural: 34.4%).

Grain sorting practices differed significantly by settings ($\chi^2 = 7.18$; $p = 0.028$), with a higher proportion of urban households reporting always sorting maize (68.4%) compared with rural households (46.9%). In the same vein a lower proportion of urban versus rural households ((10.5% vs 37.5%, respectively) reported to never practice sorting. Maize porridge consumption frequency also differed ($\chi^2 = 13.84$; $p < 0.001$), with daily consumption in 31.6% urban and 46.9%

in rural households; 4-6 times/week in 2.6% urban and 21.9% in rural; 1-3 times/week in 36.9% urban and 28.1% in rural and rarely in 28.9% urban households and 3.1% rural. Food washing was significantly more frequent ($\chi^2 = 9.81$; $p < 0.001$) in urban (63.2%) than in rural households (3.1%). Maize source differed ($\chi^2 = 21.01$; $p < 0.001$), with most rural households growing maize (96.9%) and urban households mainly buying (52.6%) or both buying and growing (31.6%). Processed food consumption was higher in urban (71.1%) than rural (50.0%) households, though not statistically significant ($\chi^2 = 2.72$; $p = 0.09$).

3.3. Child nutritional status

As shown in Table 4, stunting was the most frequent form of undernutrition, affecting 33.3% of children. A significant rural-urban disparity was observed ($\chi^2 = 5.75$, $p = 0.048$), with stunting prevalence higher in rural children (37.5%) than in urban children (28.9%). Stunting also differed significantly by gender, with a higher prevalence among boys (47.2%) than girls (18.1%) ($\chi^2 = 8.82$, $p = 0.023$). These results indicate that chronic undernutrition is widespread in the study population and disproportionately affects rural children and girls.

In contrast, wasting, which reflects acute malnutrition, showed a much lower overall prevalence (10.1%). Although wasting appeared more frequent in rural areas (15.6%) than in urban areas (5.3%), the difference was not statistically significant ($\chi^2 = 1.18$, $p = 0.277$). Similarly, the prevalence among boys (13.9%) was higher than among girls (6.1%), but this difference was also not significant ($\chi^2 = 1.38$, $p = 0.503$).

Underweight's overall prevalence was 18.8%, moderate acute malnutrition (MAM; 2.9%) and severe acute malnutrition (SAM; 1.4%) were rare, and low MUAC was observed in 4.3% of

children overall, but each of these nutritional status there was no statistically significance between urban vs. rural, or between boys and girls.

3.4.Internal mycotoxin exposure profile

The external exposure survey on maize porridge published by Miderho *et al.*, (2026) served as a general foundation for selecting the target mycotoxin analytes for serum analysis in the present study. The multi-mycotoxin panel used for serum analysis encompassed a total of 39 mycotoxins and metabolites, while a screening was performed for AFB₁-lysine, as mentioned in more detail in section 2.6. From those target analytes, serum samples tested positive for AFB₁, CIT, OTA, CPA, ENN B and AFB₁-lysine. Figure 2 shows the serum concentrations of these mycotoxins as well as the detection rates, with data grouped by gender, whereas Figure 3 shows the same data grouped by residence (urban vs. rural).

Median CIT concentrations were 0.75 ng/mL (IQR: 0.10-1.50 ng/mL) in boys and 0.50 ng/mL (IQR: 0.05-1.20 ng/mL) in girls, with no significant difference ($p = 0.518$). CIT detection frequencies were 62.2% and 57.6% in boys and girls, respectively ($\chi^2 = 0.02$, $p = 0.883$). CIT concentrations were higher in rural samples (median = 1.3 ng/mL) compared with urban samples (median = 0.1 ng/mL, $p=0.018$). Rural samples also showed a wider interquartile range (IQR = 0.8-2.2 ng/mL), indicating greater variability, whereas urban values were mostly close to zero (IQR = 0-0.3 ng/mL). CIT was detected in 62.5% of rural samples and 57.9% of urban samples; however, this difference was not statistically significant ($\chi^2 = 0.02$, $p = 0.883$) (Figure 3).

OTA concentrations were also similar between boys (0.18 ng/mL; IQR: 0.13-0.30 ng/mL) and girls (0.14 ng/mL; IQR: 0.09-0.26 ng/mL) ($p = 0.483$), with similar detection frequencies of 54.1%

and 48.5%, respectively ($\chi^2 = 0.12$, $p = 0.821$). OTA concentrations were higher among rural participants compared with urban participants. The median OTA concentration in rural samples was 0.25 ng/mL (IQR: 0.20-0.34 ng/mL), whereas urban samples had a median of 0.10 ng/mL (IQR: 0.07-0.16). The difference in concentrations between rural and urban participants was statistically significant ($p < 0.001$). OTA detection prevalence was 56.2% in rural participants and 47.4% in urban participants, with no significant difference ($\chi^2 = 0.25$, $p = 0.617$).

CPA concentrations showed a median of 0.008 ng/mL (IQR: 0.000-0.046 ng/mL) in boys and 0.050 ng/mL (IQR: 0.040-0.056 ng/mL) in girls and was detected in 24.3% of boys and 9.1% of girls, with no significant difference in concentration or detection rate between gender. CPA concentrations were similar between rural and urban participants. The median CPA concentration was 0.040 ng/mL (IQR: 0.000-0.050 ng/mL) in rural samples and 0.025 ng/mL (IQR: 0.015-0.038 ng/mL) in urban samples (not statistically significant; $p = 0.123$). A significantly higher CPA detection prevalence was observed in rural participants (31.2%) than in urban participants (5.3%) ($\chi^2 = 3.38$, $p < 0.05$).

Serum AFB₁-lys detection of AFB₁-lysine adducts was 56.8% among boys and 48.5% among girls, with no significant difference ($\chi^2 = 1.81$, $p = 0.651$). Serum detection rate of AFB₁-lysine adducts was similar ($\chi^2 = 0.08$, $p = 0.778$) among children living in rural areas (56.2%) and those living in urban areas (50.0%). AFB₁ was detected in only 2.5% of serum samples, indicating a low frequency of detection in the study population.

The distribution of co-exposure differed significantly by child gender ($\chi^2 = 7.88$, $p = 0.019$), with boys more frequently classified in the high exposure group (60.9%) compared with girls (20 %) (Figure 4). In contrast, no significant association was observed between co-exposure categories and residence (proportion of high exposure 55% in rural, 30.4% in urban children; $\chi^2 = 3.20$, $p = 0.202$).

3.5.Determinants of internal mycotoxin exposure

To identify factors associated with elevated mycotoxin exposure among infants and young children, Bayesian logistic regression models were fitted for three exposure outcomes: CIT_high, OTA_high, and detection of AFB₁-lysine. Results are presented as log[OR], corresponding OR = exp(log[OR]), 95% CrI, and posterior probabilities (Table 5).

For CIT_high exposure, increasing age showed a modest positive association with exposure (log[OR] = 0.157; OR = 1.17; 95% CrI: 0.00-0.33; posterior probability = 1.00). Maize porridge consumption also showed positive trends, with young children consuming porridge 4-6 times per week exhibiting higher estimated odds of elevated CIT exposure (log[OR] = 2.59; OR = 13.33; 95% CrI: 0.53-6.09; posterior probability = 0.92). Daily porridge consumption showed a similar direction of effect (log[OR] = 3.28; OR = 26.58; 95% CrI: -2.81-9.60; posterior probability = 0.90), although the wide CrI indicates substantial uncertainty. In contrast, urban residence was consistently associated with lower CIT exposure (log[OR] = -3.91; OR = 0.02; 95% CrI: -8.52 to -0.07; posterior probability = 1.00).

For OTA_high, frequent maize porridge consumption (4-6 times/week) showed the strongest positive association (log[OR] = 16.57; posterior probability = 1.00), although the large estimate

443 should be interpreted cautiously because of the limited sample size. Rare porridge consumption
444 ($\log[\text{OR}] = 5.25$; posterior probability = 0.97), household maize cultivation ($\log[\text{OR}] = 1.54$;
445 posterior probability = 0.97), and female gender ($\log[\text{OR}] = 1.21$; posterior probability = 0.96)
446 also showed positive trends, but their CrI overlapped zero, indicating considerable uncertainty.
447 Conversely, urban residence remained consistently associated with lower OTA exposure ($\log[\text{OR}]$
448 $= -1.72$; $\text{OR} = 0.18$; 95% CrI: -3.00 to -1.02; posterior probability = 1.00). Visible mold or insects
449 in stored maize also showed a negative association ($\log[\text{OR}] = -1.91$; $\text{OR} = 0.15$; 95% CrI: -5.81
450 to -0.26; posterior probability = 1.00), although this unexpected finding should be interpreted
451 cautiously and may reflect unmeasured storage or household characteristics.

452 For AFB₁ exposure assessed by AFB₁-lysine detection, porridge consumption frequency was again
453 associated with biomarker detection. Daily porridge consumption showed higher estimated odds
454 of AFB₁-lysine detection ($\log[\text{OR}] = 1.97$; $\text{OR} = 7.17$; 95% CrI: 0.50-3.33; posterior probability
455 = 0.99), while rare porridge consumption also showed increased estimated odds ($\log[\text{OR}] = 2.36$;
456 $\text{OR} = 10.59$; 95% CrI: 0.75-3.29; posterior probability = 0.99). These findings suggest that dietary
457 patterns may influence aflatoxin exposure, although the underlying mechanisms remain unclear
458 and warrant further investigation, including possible substitution with other contaminated foods
459 such as peanut-based products and potential regional differences in infant feeding practices.
460 Visible mold or insects showed a positive but uncertain association with AFB₁-lysine detection
461 ($\log[\text{OR}] = 0.92$; $\text{OR} = 2.51$; 95% CrI: -0.38-2.36; posterior probability = 0.92).

462 Several predictors demonstrated high posterior probabilities and consistent directions of effect,
463 whereas many 95% CrIs included zero or were relatively wide, indicating substantial uncertainty
464 in the estimated effect sizes. In addition, some observed associations, including the inverse

relationship between visible mold or insect infestation and OTA exposure and the higher estimated AFB₁ exposure among children reporting infrequent porridge consumption, did not follow the expected biological patterns. Therefore, these findings should be interpreted as exploratory associations that identify potential exposure pathways rather than definitive causal determinants and require confirmation in larger prospective studies.

3.6. Association between internal mycotoxin exposure and child undernutrition indicators

Table 6 summarizes the Bayesian associations between internal mycotoxin biomarkers and child undernutrition indicators. Regression coefficients (β) represent changes in the log-odds of each outcome, while ORs indicate the corresponding multiplicative change in odds. Results are presented together with their 95% CrIs.

Given the relatively small sample size and sparse observations for several nutritional outcomes, these analyses should be regarded as exploratory and hypothesis-generating rather than confirmatory.

High CIT exposure demonstrated positive estimated associations with several undernutrition indicators. For wasting, the estimated effect was $\beta = 2.15$ (95% CrI: -1.05 to 6.00; OR = 8.56, 95% CrI: 0.35-405), while underweight showed $\beta = 0.742$ (95% CrI: -1.82 to 3.88; OR = 2.10, 95% CrI: 0.16-48.3). A modest positive trend was also observed for stunting ($\beta = 0.364$; 95% CrI: -2.108 to 2.933; OR = 1.44, 95% CrI: 0.12-7.79). However, all corresponding CrIs were broad and included the null value, indicating substantial uncertainty regarding both the magnitude and

direction of these associations. Extremely large estimates observed for MAM, SAM, and low MUAC likely reflect sparse-data instability and were therefore not interpreted further.

High AFB₁-lysine exposure also showed positive estimated associations with wasting, underweight, and stunting. Wasting was estimated as $\beta = 1.46$ (95% CrI: 0.12 to 2.91; OR = 1.03, 95% CrI: 0.50 to 3.47), while underweight showed $\beta = 1.327$ (95% CrI: 0.30 to 3.65; OR = 1.59, 95% CrI: 0.05 to 8.60). Stunting demonstrated a positive estimated association ($\beta = 1.55$; 95% CrI: 0.715 to 2.923; OR = 1.70, 95% CrI: 1.02 to 18.6), although the wide CrI suggests considerable uncertainty in the magnitude of the effect. Very large coefficients estimated for MAM and SAM were considered indicative of sparse observations rather than precise biological effects and were not interpreted further.

High OTA exposure showed positive estimated associations with wasting ($\beta = 1.98$; 95% CrI: 0.73 to 2.15), underweight ($\beta = 1.11$; 95% CrI: -1.71 to 4.24), and stunting ($\beta = 0.437$; 95% CrI: -2.311 to 3.344), while negative coefficients were estimated for MAM, SAM, and low MUAC. These estimates were generally accompanied by wide CrIs or extreme coefficient values, limiting confidence in their interpretation.

Among the adjustment variables, increasing child age consistently showed negative estimated associations with several forms of undernutrition, including underweight ($\beta = -0.307$; 95% CrI: -0.507 to -0.143; OR = 0.74, 95% CrI: 0.60 to 0.87), low MUAC ($\beta = -0.746$; 95% CrI: -1.77 to -0.013; OR = 0.47, 95% CrI: 0.17 to 0.99), and stunting ($\beta = -0.311$; 95% CrI: -0.495 to -0.155; OR = 0.73, 95% CrI: 0.61-0.86). Female gender demonstrated mixed estimated associations across nutritional outcomes, with most CrIs including the null value.

4. Discussion

The aim of the present study was to assess internal exposure to major mycotoxins and to examine their potential association with nutritional status among infants and young children in a region where malnutrition prevalence is known to be high. The study involved children aged 11-23 months old, consuming maize porridge in South Kivu, DRC. In addition to measuring mycotoxin concentrations in children's serum, the sociodemographic characteristics of mothers were recorded since they may influence complementary feeding practices, diet diversity, and food safety behaviours, which in turn affect both nutritional adequacy and mycotoxin exposure from maize-based porridges.

4.1. Sociodemographic characteristics

Socio-demographic characteristics differed between urban and rural households. Maternal education was significantly higher in urban households, a pattern consistent with broader evidence from SSA showing persistent rural-urban inequalities in maternal education, which remain strongly linked to child health and nutrition outcomes (Abuya *et al.*, 2012; Amaha & Woldeamanuel, 2021; Nyamasege *et al.*, 2021).

Maternal education is widely recognized as a key determinant of child feeding practices and nutritional status. In the context of the present study, these educational differences provide an important contextual framework for interpreting variability in household feeding behaviors and food hygiene practices. Although the present analysis did not formally test mediation pathways, previous studies have shown that higher maternal education improves dietary diversity, caregiving

practices, and reduces stunting risk in early childhood (Ekholuenetale *et al.*, 2022 ;Mporanyi *et al.*, 2025).

Maternal occupation also differed substantially between study areas, with farming predominating in rural households and housewifery in urban settings. This reflects the strong link between livelihood systems and household food sourcing strategies observed in agricultural settings across SSA (Nankinga *et al.*, 2019). Agricultural livelihoods have been associated with greater reliance on home-produced staple crops, particularly maize, which may be vulnerable to post-harvest contamination under suboptimal storage conditions (Adedokun & Yaya, 2021;Mollay *et al.*, 2022). In the present study, this contextual pattern is supported by the high proportion of rural households cultivating maize and the widespread use of ground storage, although causal relationships between occupation and contamination risk were not directly assessed.

The observed sociodemographic structure is important for interpreting downstream nutritional and exposure outcomes. Rural residence was associated with higher prevalence of stunting and higher levels of certain mycotoxins, while urban households demonstrated more diversified food sourcing practices. These patterns are consistent with evidence linking rural residence and low maternal education to increased vulnerability to both undernutrition and dietary toxin exposure in low-income settings (Mollay *et al.*, 2021; Ngure *et al.*, 2023).

4.2. Maize sourcing, storage, food handling, and infant feeding practices

Maize sourcing differed by residence, with rural households relying predominantly on homegrown maize, while urban households combined purchased and own sources. Similar patterns are observed across SSA, where rural populations consuming farm-grown maize face higher

547 mycotoxin exposure due to limited food safety surveillance (Nji *et al.*, 2022). Home-produced
548 maize in Zimbabwe and other SSA regions is frequently contaminated with *Fusarium* and
549 *Aspergillus* species, increasing exposure risk (Akello *et al.*, 2021). Subsistence maize is
550 particularly susceptible to fungal contamination due to inadequate agronomic practices and
551 suboptimal storage conditions (Alberts *et al.*, 2019). However, market maize is not always safer;
552 in Kenya, maize from informal markets sometimes contained higher aflatoxin levels than home-
553 grown maize, reflecting poor storage and climatic stress (Joutsjoki & Korhonen, 2021).

554 Maize storage practices also differed significantly between urban and rural households, consistent
555 with observations across West and East Africa. In Ghana, smallholder farmers typically rely on
556 traditional granaries and open storage, whereas peri-urban households more frequently use
557 improved bags or containers; yet these differences do not always result in shorter storage durations
558 or reduced mold prevalence (Sugri *et al.*, 2015; Adams *et al.*, 2025). Similarly, in Kenya, hermetic
559 PICS bags were more commonly adopted near urban centers, but inconsistent storage and cleaning
560 routines limited the effectiveness of these interventions in reducing contamination (Rono *et al.*,
561 2018). Dry, off-ground storage, more common in better-resourced contexts, is associated with
562 lower mycotoxin levels and reduced exposure risk (Magembe *et al.*, 2016; Arefaynie *et al.*, 2024).

563 Maize porridge consumption frequency differed markedly by residence, with rural children
564 consuming it more frequently than urban children. In the present study, this pattern is consistent
565 with the greater reliance on subsistence maize production in rural households, where most families
566 cultivate maize and depend on it as a primary complementary food. In contrast, urban households
567 exhibited more diversified food sourcing, including greater reliance on purchased foods and a

568 wider range of complementary feeding options, which may reduce the frequency of maize-based
569 porridge consumption.

570 Similar patterns have been reported in other East African settings, where maize remains a dominant
571 component of infant complementary feeding, particularly in rural areas. For example, studies in
572 Tanzania and Kenya have shown that over 90% of infants consume maize-based porridges, which
573 often constitute the main or sole staple in complementary diets (Ngure *et al.*, 2023 ;Mollay, 2025).
574 Such maize-dominated feeding systems have been associated with low dietary diversity and
575 increased vulnerability to nutrient deficiencies and dietary mycotoxin exposure in early childhood.

576 Urban households reported significantly higher grain washing frequencies than rural households,
577 probably reflecting disparities in water access and hygiene knowledge. Postharvest handling
578 factors interact to determine contamination risk (Binamungu *et al.*, 2023; Mollay *et al.*, 2021). In
579 South Kivu, limited clean water and low food-handling knowledge in rural areas contribute to
580 suboptimal hygiene practices. While washing removes dust and debris, it alone does not reliably
581 reduce mycotoxin contamination, which requires integrated interventions including sorting,
582 thorough drying, and clean storage (Miderho *et al.*, 2025; Kamala *et al.*, 2017)

583 Frequent consumption of inadequately processed maize porridge amplifies cumulative exposure
584 to fungal toxins; monotonous maize-dominant diets have been associated with higher AFs and
585 FUMs levels and impaired linear growth (Mollay, 2025). Across SSA, high maize porridge intake
586 is widespread, yet better hygiene and more diversified complementary feeding in urban households
587 correlate with lower mycotoxin exposure and improved nutritional outcomes (Magembe *et al.*,
588 2016; Mollay *et al.*, 2021; Galani *et al.*, 2022). Persistent reliance on maize- and groundnut-based

porridges in rural settings underscores ongoing vulnerability to chronic dietary mycotoxin exposure and undernutrition.

4.3. Child nutritional status

The present study indicates a high burden of chronic undernutrition among children in South Kivu, with stunting affecting approximately one-third of the sample and showing clear disparities by residence and gender. Higher prevalence among rural children and boys reflects persistent inequalities in child growth outcomes within the study setting (DHS, 2014). These findings are consistent with national estimates from eastern DRC, where stunting remains highly prevalent, reaching approximately 43% in some areas (Kandala *et al.*, 2011; Luzingu *et al.*, 2022). Similar levels of chronic undernutrition have been reported across East Africa, where average stunting prevalence is estimated at 33%, ranging from 22% in Kenya to over 50% in Burundi and Rwanda (Tesema *et al.*, 2021; Ngaruye *et al.*, 2023). Across SSA, stunting is driven by a complex interplay of structural and immediate determinants, including maternal education, household poverty, low birth weight, inadequate breastfeeding practices, poor sanitation, diarrhoeal disease, and rural residence (Akombi *et al.*, 2017). The higher vulnerability observed among boys is also consistent with extensive epidemiological evidence suggesting greater biological susceptibility to growth faltering in male children. Meta-analytical and multi-country demographic and health surveys (DHS) evidence indicates higher stunting and wasting risk among boys, with mean HAZ scores consistently lower in boys than girls (Wamani *et al.*, 2007; Dembele, 2025). Large gender disparities have also been reported in the Great Lakes region, with differences exceeding 8% in some settings (Dembele, 2025).

In contrast, acute malnutrition levels were relatively low in the present study. Wasting prevalence was 10.1% and did not differ significantly by residence or gender. This pattern is broadly consistent with national DRC estimates of approximately 6.1% and typical SSA ranges of 5-10% (Akombi *et al.*, 2017; Luzingu *et al.*, 2022), as well as findings from West African contexts such as Senegal (10.3%) (Tamir *et al.*, 2025). However, deviations from this pattern have been reported elsewhere, with higher odds of wasting among boys (Murarkar *et al.*, 2020; Marume *et al.*, 2025). Similarly, meta-analyses confirm that boys are generally at higher risk of wasting, although effect sizes vary across settings (Thurstans *et al.*, 2020; Sahiledengle *et al.*, 2023). Consistent with low wasting prevalence, moderate and severe malnutrition were rare in the present study (MAM 2.9%, SAM 1.4%). According to WHO, wasting prevalence below 5% is considered of ‘very low’ public health significance (De Onis *et al.*, 2018). Substantial heterogeneity exists across the region, with wasting prevalence reported to range from 1% to 58% across 11 African countries (Riwa *et al.*, 2025). Although gender differences in MAM and SAM were not statistically significant in the present study, SAM occurred only among boys (2.8%), which is consistent with broader evidence. Large-scale DHS analyses covering over 700,000 children have reported higher male-to-female risk ratios for wasting and concurrent wasting-stunting (Garenne *et al.*, 2022), while systematic reviews confirm higher odds of wasting among boys across diverse settings (Thurstans *et al.*, 2020).

Rural-urban patterns observed in this study were also broadly consistent with regional evidence, although differences were modest. For example, previous studies have reported slightly higher SAM prevalence in rural compared to urban settings (Fagbamigbe *et al.*, 2020). In the present study, low-MUAC prevalence was relatively low (4.3%), compared with other contexts where MUAC-defined moderate acute malnutrition may reach approximately 10% (De Costa & Duggan,

2021; Patra *et al.*, 2024). Underweight prevalence (18.8%) showed no significant differences by gender or residence. This finding is consistent with evidence that underweight is a composite indicator reflecting both acute and chronic nutritional deficits and therefore tends to be less specific in distinguishing underlying causal pathway (Akombi *et al.*, 2017).

4.4. Internal mycotoxin exposure

In this serum-based pilot study, CIT, CPA, and OTA were detected at measurable levels. The detection of CIT and OTA in serum is consistent with evidence of widespread co-exposure in SSA, where complementary foods frequently exceed safety limits (Chen *et al.*, 2018; Ojuri *et al.*, 2019; Chilaka & Mally, 2020; Nji & Mwanza, 2024). No significant gender differences were observed in serum concentrations or detection frequencies of CIT and OTA, suggesting that biological gender plays a limited role when early-life diets are largely shared.

Comparable findings have been reported in Cameroonian children, where multi-mycotoxin exposure (OTA, DON, AFM₁, FB₁) was frequent, with 73% of urine samples containing multiple toxins, without clear gender-specific patterns (Njumbe *et al.*, 2013). Although higher urinary DON or FB₁ has been observed in boys in other settings, such differences are more likely driven by dietary practices rather than biological susceptibility (Njumbe *et al.*, 2013; Thenuwara *et al.*, 2024). Nevertheless, boys (60.9%) were more frequently classified than girls (20%) in the high-exposure group, indicating potential differences in exposure intensity, consistent with evidence of gender-related variation in environmental toxicant exposure (Ashraf *et al.*, 2022; Thenuwara *et al.*, 2024).

653 Evidence from serum- and plasma-based biomonitoring further confirms widespread chronic
654 exposure across SSA. Aflatoxin exposure assessed using the aflatoxin-albumin adduct biomarker
655 has been consistently detected across multiple countries, demonstrating sustained exposure in
656 vulnerable populations such as children and adolescents (Xu *et al.*, 2018). In Kenya, a national
657 biomonitoring study reported AFB₁-lysine adducts in 78% of serum samples, confirming
658 widespread exposure across geographic and socioeconomic groups (Yard *et al.*, 2013). Systematic
659 evidence further indicates that this exposure is largely driven by contamination of staple foods
660 such as maize and groundnuts, contributing to a substantial public health burden in Africa (Meijer
661 *et al.*, 2021).

662 In addition, human biomonitoring studies show that AFB₁-lysine and OTA are among the most
663 frequently detected mycotoxin biomarkers in blood, plasma, and serum, with consistently high
664 positivity rates across populations (Arce-López *et al.*, 2020). Early evidence on OTA also confirms
665 that serum/plasma concentrations are widely detectable and commonly used as indicators of
666 continuous exposure, although urinary biomarkers may better reflect short-term intake (Scott,
667 2005). These patterns are largely explained by OTA toxicokinetics, including its long half-life,
668 strong binding to serum albumin, renal reabsorption, and enterohepatic recirculation; urinary
669 excretion represents only a small fraction of total exposure, making serum a more stable marker
670 of chronic intake (Studer-Rohr *et al.*, 2000; Scott, 2005; Malir *et al.*, 2016; Malir *et al.*, 2019;
671 Muñoz *et al.*, 2010; Han *et al.*, 2013). CIT may exhibit partially overlapping behavior, although
672 its serum kinetics remain less well characterized.

673 Marked rural-urban disparities were observed, with higher CIT detection among rural children
674 compared to urban children (56.2% vs 15.8%), alongside elevated OTA concentrations in rural

settings. These findings are consistent with evidence from SSA showing that rural populations experience higher mycotoxin exposure due to greater reliance on maize-based diets and suboptimal storage conditions. For example, high prevalence of aflatoxin-albumin adducts (84%) and fumonisin biomarkers (96%) has been reported among children in similar settings (Shirima *et al.*, 2013). Similarly, rural adult populations have shown frequent detection of OTA (86%) and CIT (37%) (Ezekiel *et al.*, 2025), while multi-mycotoxin exposure including AFB₁, FB₁, DON, OTA, and CIT has also been documented in infants, highlighting early-life co-exposure in highly vulnerable groups (Ayeni *et al.*, 2024).

Thus, exposure levels in this pilot study appear moderate compared with high-burden regions, where detection rates for OTA and CIT may approach or reach 100% in some populations (Lombard, 2014). The combination of moderate but frequent co-exposure, higher rural burden, and absence of strong gender differences suggests that environmental factors particularly maize-based diets, storage conditions, and food handling practices are the primary drivers of early-life mycotoxin exposure in SSA (Shirima *et al.*, 2013; Chilaka & Mally, 2020; Wangia-Dixon *et al.*, 2022).

A difference was observed between direct AFB₁ detection and AFB₁-lysine adduct positivity. AFB₁-lysine showed a higher detection frequency compared with free AFB₁, suggesting that biomarker-based assessment of internal exposure may capture a broader window of aflatoxin intake than direct toxin measurement. This discrepancy is consistent with the toxicokinetics of aflatoxin B₁, where rapid metabolism and short half-life of parent compounds limit their detectability in biological matrices, whereas AFB₁-lysine reflects cumulative exposure over time due to its binding to serum albumin and longer persistence in circulation.

4.5. Determinants of internal mycotoxin exposure and child growth outcomes

Frequent maize porridge consumption (4-6 times/week) strongly predicted OTA ($\log[\text{OR}] = 16.57$) and CIT ($\log[\text{OR}] = 2.59$), consistent with similar studies in Tanzania, Zimbabwe, and Cameroon (Magoha *et al.*, 2014; Hove *et al.*, 2016; Abia *et al.*, 2013a). Contamination by *Aspergillus*, *Fusarium*, and *Penicillium* is promoted by high temperature, humidity, rainfall, poor agronomic/storage practices, and limited regulation (Misihairabgwi *et al.*, 2019; Kebede *et al.*, 2020). In Tanzania, 50-87% of household maize contained AFs and FB₁ exceeding guidance (Kamala *et al.*, 2015; Suleiman & Rosentrater, 2015; Njumbe *et al.*, 2014; Stafstrom *et al.*, 2025), while Zimbabwean children <5 years exceeded the FB₁ provisional maximum tolerable daily intake (PMTDI) by 196-272% (Hove *et al.*, 2016). Cameroon surveys detected 69 metabolites with co-exposure to up to five mycotoxins (Ngoko *et al.*, 2001; Abia *et al.*, 2013b; Njumbe *et al.*, 2013; Fapohunda & Adewunmi, 2019), and infant AFM₁ (0.01-0.55 ng/mL) reduced WAZ and HAZ Z-scores (Magoha *et al.*, 2014; Doğan *et al.*, 2022). Co-occurring mycotoxins pose additive risks (Borowsky *et al.*, 2024), underscoring the need for nixtamalization, improved storage, farmer education, risk communication, and dietary diversification (Schaarschmidt & Fauhl-Hassek, 2019; Maris *et al.*, 2025; Ntsoli *et al.*, 2024).

Urban infants had lower CIT ($\log[\text{OR}] = -3.91$) and OTA ($\log[\text{OR}] = -1.72$) risks, consistent with Ghana and Malawi, where markets provide better-dried grains and dietary diversity (Kpodo *et al.*, 2000; Matumba *et al.*, 2015). Rural post-harvest practices increase OTA exposure (Wacoo *et al.*, 2014; Kamika *et al.*, 2014), and low AFs awareness requires farmer training and good agricultural practices (GAPs) (Kampete *et al.*, 2025; Warnatzsch *et al.*, 2020; Mabruki *et al.*, 2022). CIT risk increased with age ($\log[\text{OR}] = 0.157$) and girls had higher OTA odds ($\log[\text{OR}] = 1.21$) (Castelino *et al.*, 2015; Piekkola *et al.*, 2012).

In this study, the Bayesian models suggested a potential positive association between high AFB₁ exposure (AFB₁-lysine) and stunting (OR = 1.70; 95% CrI: 1.02-18.6). However, this result should be interpreted with caution, as the CrI is wide, reflecting considerable uncertainty in the estimate and indicating that the evidence remains preliminary rather than confirmatory. Despite this limitation, the direction of the association is consistent with a hypothesized link between aflatoxin exposure and impaired linear growth in children. This observation aligns with epidemiological evidence from LMICs reporting associations between AFs exposure and reduced child growth outcomes (Watson *et al.*, 2017; Chen *et al.*, 2018; Visser *et al.*, 2020; Rasheed *et al.*, 2021; Watson *et al.*, 2018; Tessema *et al.*, 2021; .

Mechanistically, AFs exposure may impair child growth by inhibiting protein synthesis, disrupting hepatic production of insulin-like growth factor-1 (IGF-1), and damaging intestinal integrity, which may contribute to environmental enteric dysfunction and reduced nutrient absorption during critical growth periods (Castelino *et al.*, 2015; James *et al.*, 2021; Tessema *et al.*, 2021; Urugo *et al.*, 2024). Consistent with this proposed mechanism, aflatoxin exposure may have a stronger relationship with chronic growth indicators such as height-for-age compared with acute malnutrition outcomes. This could tentatively suggest that, in the present exploratory study, AFB₁ exposure was more closely associated with stunting than with wasting, underweight, or low MUAC (Nejad *et al.*, 2023).

Evidence from various studies in Africa supports this relationship. In Benin and Togo, children aged 9 months to 5 years with mean serum AFB₁-lysine concentrations of 32.8 pg/mg albumin had lower height-for-age scores (Gong *et al.*, 2003). Cohort studies in Gambia reported growth impairment at exposure levels of 5-30 pg/mg albumin (Turner *et al.*, 2007) and 52.3 pg/mg albumin

(Watson, *et al.*, 2018), while Tanzanian infants with 4.7 pg/mg albumin showed reduced linear growth (Shirima *et al.*, 2015). Similar associations were reported in Zambia (Alamu *et al.*, 2020), Kenya (Castelino *et al.*, 2015; Wangia-Dixon *et al.*, 2022). These findings reinforce the role of AFs exposure as a potential contributor to chronic growth impairment and highlight the need for integrated food safety and nutrition interventions in high-exposure settings.

Child age was protective against underweight (OR = 0.74; 95% CrI: 0.60-0.87), stunting (OR = 0.73; 95% CrI: 0.61-0.86), and low MUAC (OR = 0.47; 95% CrI: 0.17-0.99), consistent with patterns observed across SSA (Tamir *et al.*, 2024). Child age was positively associated with OTA exposure (OR = 2.14 per month), consistent with cumulative intake and OTA's slower clearance, while female gender showed lower, non-significant odds, echoing findings that dietary factors outweigh biological gender in determining exposure (Abia *et al.*, 2013a; Abia *et al.*, 2024). Co-exposure to many toxins was more frequent among rural children and boys, reflecting complex dietary patterns. Nigerian infant studies detected at least ten mycotoxins in urine including OTA, CIT, DON, AFM₁, and FB₁, highlighting widespread mixed exposure from cereal foods (Ayeeni *et al.*, 2024). Comparable adult data from Cameroon show OTA and CIT detection in 95% and 87% of urine samples, respectively, often with more than five co-occurring toxins (Abia *et al.*, 2024).

Several observed associations were not fully consistent with expected biological pathways. In particular, the inverse association between visible mold or insect infestation and OTA exposure and the elevated AFB₁ exposure estimated among children reporting infrequent maize porridge consumption may reflect residual confounding, exposure misclassification, or dietary substitution patterns. Children consuming maize porridge less frequently may rely more heavily on alternative complementary foods, including peanut-based products, which are recognized as sources of AFs

exposure. Regional differences in infant feeding practices may also contribute to these patterns. Given the relatively small sample size and the wide credible intervals observed for several predictors, these findings should be considered exploratory and hypothesis-generating rather than evidence of definitive causal associations. Furthermore, several predictors exhibited very large effect estimates accompanied by wide CrIs, suggesting that the Bayesian model identified potential exposure signals rather than precise effect sizes. These findings warrant confirmation in larger longitudinal studies.

4.6. Study limitation and future work

This pilot study had a small sample size and a cross-sectional design, limiting causal inference. Pilot sample sizes as small as approximately 12 per group are frequently recommended to generate initial estimates of variability and inform the design of larger studies, and broader guidance supports range scales consistent with the current study design (Julious, 2005). Only CIT, OTA and CPA were quantitatively detected despite analyzing for 39 mycotoxins, and dietary data relied on caregiver recall, which may introduce bias. Other factors affecting growth, such as infections or micronutrient deficiencies, were not fully accounted for. Larger, longitudinal studies are needed to better assess mycotoxin exposure over time and its impact on child growth. Broader mycotoxin screening, evaluation of complementary foods, and interventions targeting maize contamination and dietary diversification should be pursued.

Conclusion

This study assessed internal exposure to multiple mycotoxins in young children aged 11-23 months in eastern DRC, with a primary focus on individual biomarkers including OTA, CIT, and AFB₁-lysine, alongside an exploratory assessment of co-exposure patterns. The findings indicate frequent

exposure to several mycotoxins and suggest that maize-based feeding practices and household storage conditions may contribute to this exposure profile. Nonetheless, these results provide baseline evidence of early-life mycotoxin exposure in this setting and its potential relevance for child growth. However, the co-exposure component was exploratory and limited in scope, and further studies using larger sample sizes and dedicated multi-toxin exposure models are needed to fully characterize combined exposure patterns and their health implications. These findings nevertheless offer important insights to inform targeted nutrition and food safety interventions in this vulnerable region.

Ethics Approval

The study protocol received ethical clearance from the Comité d'Éthique Institutionnel de l'Université Catholique de Bukavu (UCB) under approval number UCB/CEI/PB/016/2025. Written informed consent was obtained from all caregivers before enrollment. Data and biological samples were collected only after consent, and personal information was maintained with strict confidentiality and securely managed throughout collection, processing, and transfer.

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Conflict of Interest

The authors declare that they have no competing interests or conflicts of interest that could have influenced the work reported in this manuscript.

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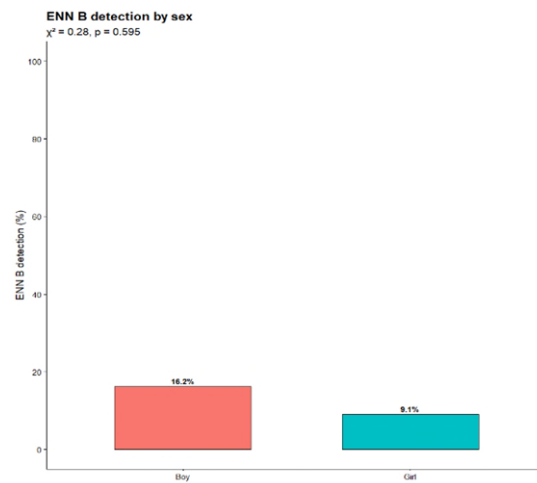
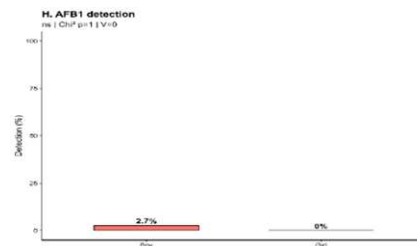
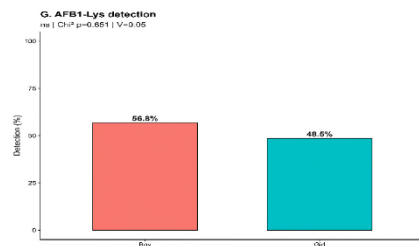
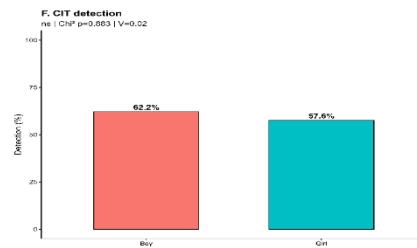
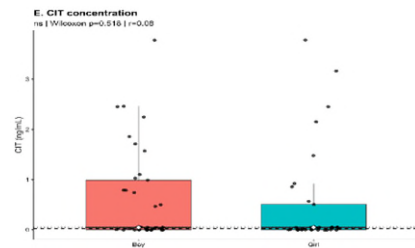
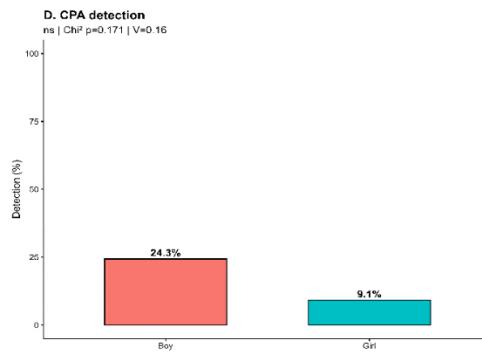
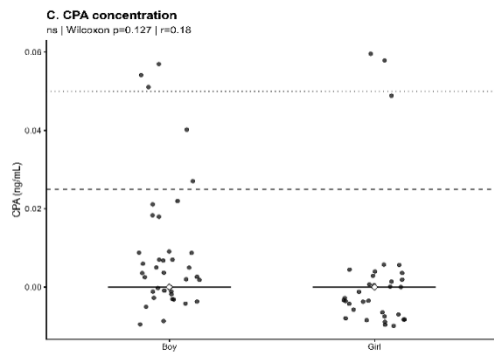
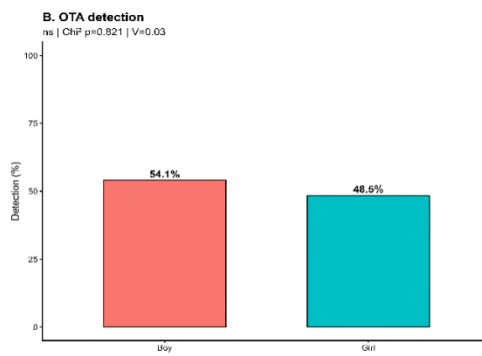
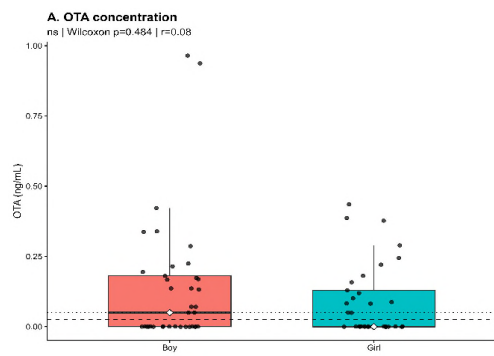


Figure 2. Serum concentrations and detection rates of mycotoxins grouped by gender.

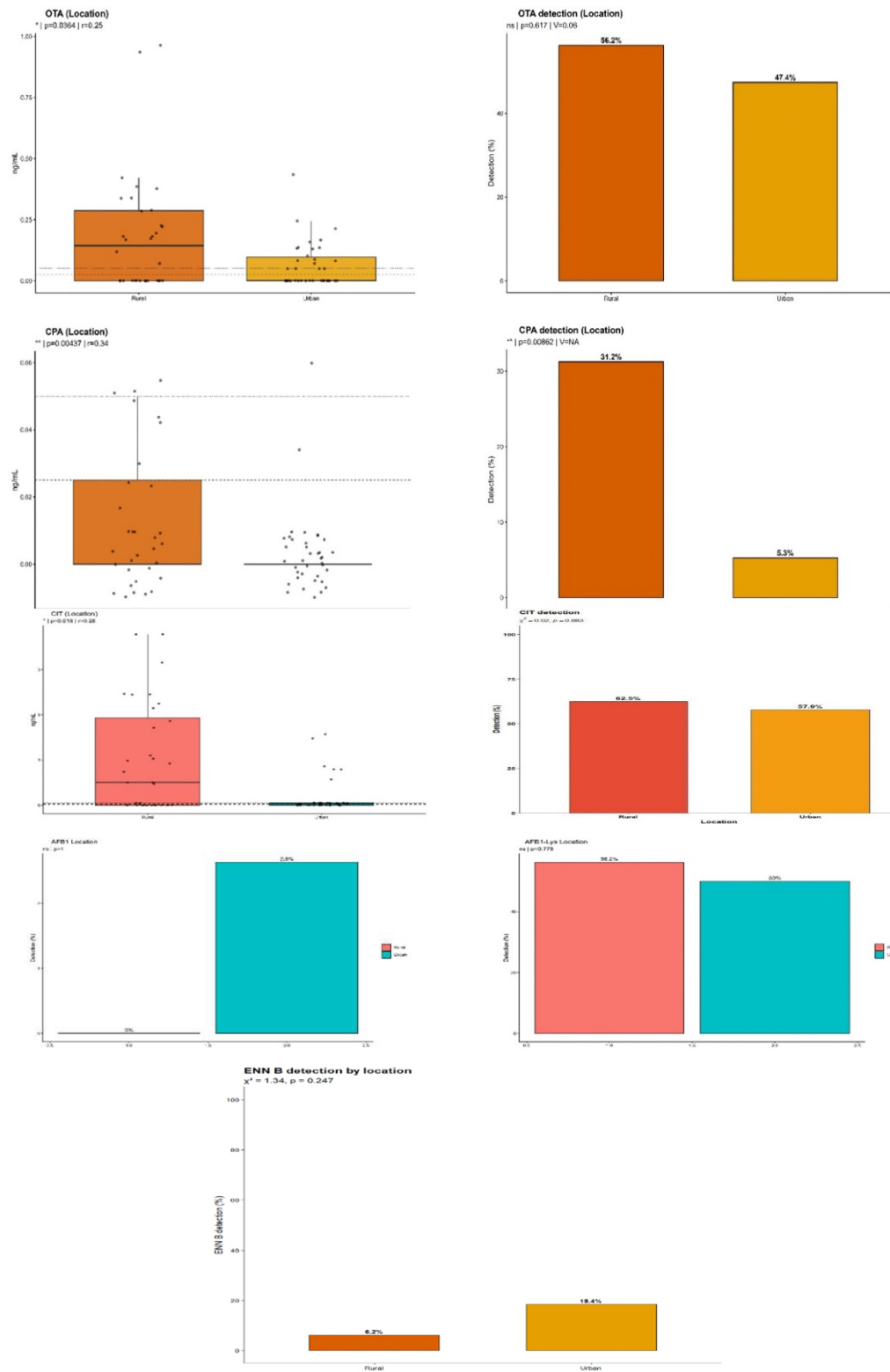


Figure 3. Serum concentrations and detection rates of mycotoxins grouped by residence (urban vs rural)

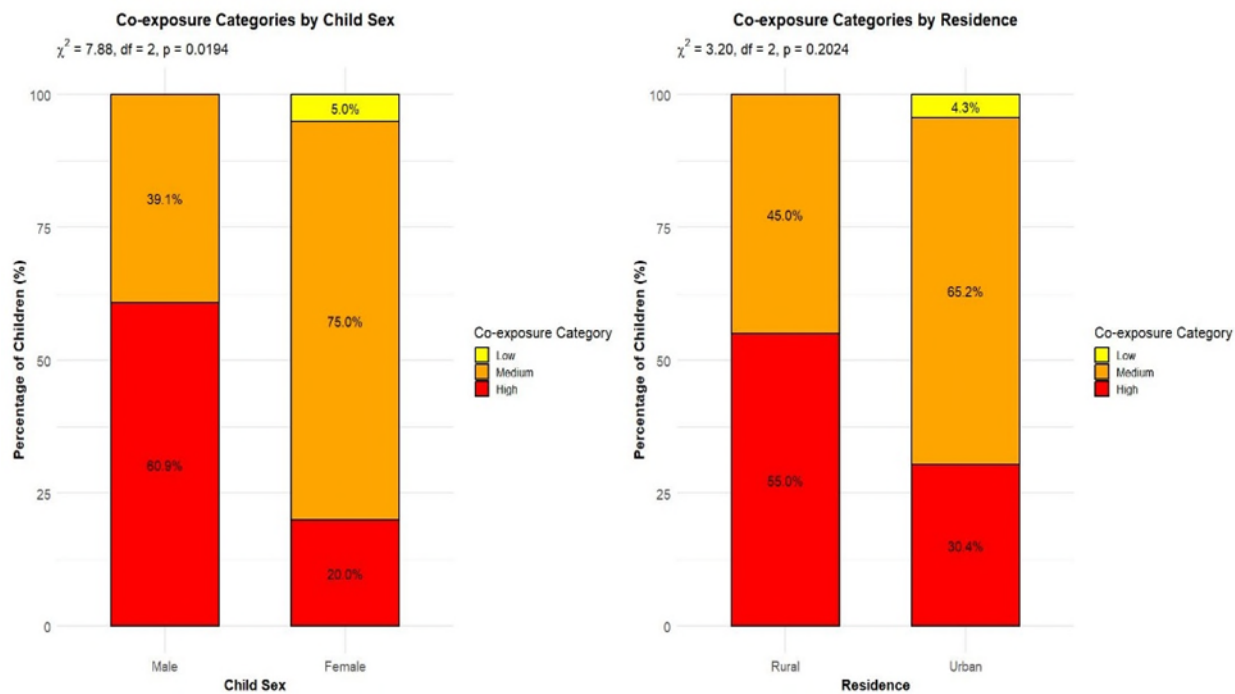


Figure 4. Distribution of mycotoxins co-exposure levels by gender or by residence (rural vs. urban)

Table 1. Definition of variables included in Bayesian regression models

Variable	Description	Type
Dependent variables		
CIT_high	High citrinin exposure	Binary outcome
OTA_high	High ochratoxin A exposure	Binary outcome
AFB1_detected	Detection of aflatoxin B1-lysine	Binary outcome
Stunting	Height-for-age z -score (HAZ) < -2 SD	Binary outcome
Wasting	Weight-for-height z-score (WHZ) < -2 SD	Binary outcome
Underweight	Weight-for-age z-score (WAZ) < -2 SD	Binary outcome
MAM	Moderate acute malnutrition (WHZ between -3 and -2 SD)	Binary outcome
SAM	Severe acute malnutrition (WHZ < -3 SD)	Binary outcome
Low_MUAC	Mid-upper arm circumference < 12.5 cm	Binary outcome
Independent variables		
Age	Infant age at sampling (months)	Continuous
Gender	Infant gender (boy/girl)	Binary
Location	Household residence (urban/rural)	Binary
Grain sorting	Frequency of maize sorting before cooking	Categorical
Food washing	Washing of maize grains before preparation	Binary
Porridge frequency	Frequency of maize porridge consumption	Categorical
Maize source	Source of maize consumed	Categorical
Visible mold/insects	Presence of mold or insects in stored maize	Binary

Table 2: Household and maternal characteristics of study participants stratified by residence (urban vs. rural), with statistical comparisons.

Characteristic	Urban (n=38)	Rural (n=32)	Test	p-value
Gender of the child				
Boy	15 (39.5%)	21 (65.6%)	$\chi^2= 4.4$	0.036*
Girl	23 (60.5%)	11 (34.4%)		
Number of children <5				
1	10 (26.3%)	10 (31.2%)	$\chi^2= 5.4$	0.068 ^{ns}
2-3	22 (57.9%)	21 (65.6%)		

≥4	6 (15.8%)	1 (3.1%)		
Maternal education level				
None	5 (13.2%)	12 (37.5%)	$\chi^2 = 9.1$	0.028*
Primary	13 (34.2%)	11 (34.4%)		
Secondary	17 (44.7%)	9 (28.1%)		
Higher education	3(7.9%)	0 (0.0%)		
Maternal occupation				
Farmer	4 (10.5%)	21 (65.6%)	$\chi^2 = 41.2$	<0.001***
Entrepreneur	9 (23.7%)	8 (25%)		
Housewife	19 (50%)	2 (6.3%)		
Student	2 (5.3%)	1 (3.1%)		
None	4 (10.5%)	0 (0.0%)		

Significance levels are shown as $p < 0.05$ (*), $p < 0.01$ (**), $p < 0.001$ (***), and not significant (ns).

Table 3: Food handling, storage and child feeding practices among study participants, stratified by place of residence (urban vs. rural), with corresponding statistical comparisons.

Characteristic	Urban (n=38)	Rural (n=32)	Test	p-value
Maize storage location				
Hermetic bags (PICS)	3 (7.9%)	4 (12.5%)	$\chi^2 = 9.38$	0.031*
On the ground	31(81.6%)	21 (65.6%)		
Granary	4 (10.5%)	7 (21.9%)		
Food storage duration, days	12.0 (7.0-14.0)	7.0 (3.0-14.0)	W = 482.5	0.242 ^{ns}
Storage cleaning frequency				
Weekly	30 (78.9%)	26 (81.3%)	$\chi^2 = 8.4$	0.038*
Monthly	6 (15.8%)	1 (3.1%)		
Rarely	1 (2.6%)	4 (12.5%)		
Never	1 (2.6%)	1 (3.1%)		
Visible mold or insects				
No	20 (52.6%)	21 (65.6%)	$\chi^2 = 0.73$	0.392 ^{ns}
Yes	18 (47.4%)	11 (34.4%)		
Grain sorting practice				
Always	26 (68.4%)	15 (46.9%)	$\chi^2 = 7.18$	0.0276*
Sometimes	8 (21.1%)	5 (15.6%)		
Never	4 (10.5%)	12 (37.5%)		
Maize porridge frequency				
Daily	12 (31.6%)	15 (46.9%)	$\chi^2 = 13.84$	<0.001***
4-6x/week	1 (2.6%)	7 (21.9%)		

Table 5. Determinants of high citrinin (CIT_high), high ochratoxin A (OTA_high) and detection of aflatoxin B₁ (AFB₁)-lysine) exposure.

Citrinin (CIT_high)				
Variable	Log[OR]	OR = exp(log[OR])	95% CrI (log)	Posterior probability
Age	0.157	1.17	0.00 - 0.33	1
Porridge 4-6/week	2.59	13.33	0.53 - 6.09	0.92
Porridge daily	3.28	26.58	-2.81 - 9.60	0.90
Urban residence	-3.91	0.02	-8.52 - -0.07	1.00
Ochratoxin A (OTA_high)				
Female child	1.21	3.35	-0.05 - 2.78	0.96
Porridge 4-6/week	16.57	1.57×10^7	2.38 - 29.37	1
Rare porridge	5.25	190.6	-0.83 - 11.57	0.97
Maize cultivated	1.54	4.66	-0.38 - 4.32	0.97
Urban residence	-1.72	0.18	-3.00 - -1.02	1.00
Visible mold/insects	-1.91	0.15	-5.81 - -0.26	1.00
Aflatoxin B ₁ (AFB ₁)-lysine detection)				
Porridge daily	1.97	7.17	0.50 - 3.33	0.99
Rare porridge	2.36	10.59	0.75 - 3.29	0.99
Visible mold/insects	0.92	2.51	-0.38 - 2.36	0.92

Estimates are log odds ratios (log[OR] = ln[OR]) with 95% credible intervals (CrI) on the log scale and posterior probabilities. CIT_high, OTA_high, and AFB₁-lysine indicate elevated citrinin, ochratoxin A exposure, and detection of the aflatoxin B₁-lysine biomarker.

Table 6. Bayesian associations between mycotoxin exposure biomarkers and child undernutrition indicators.

[illegible]

CIT_ high	2.15 (- 1.05, 6.00)	8.56 (0.35- 405)	0.742 (- 1.82, 3.88)	2.10 (0.16- 48.3)	17.8 (17.8, 17.8)	5.16e7 (5.16e7, 5.16e7)	-108 (- 108, - 108)	0 (0, 0)	6.09 (- 6.37, 19.4)	440 (0- 2.7e8)	0.364 (- 2.108; 2.933)	1.44 (0.12; 7.79)
AFB ₁ - Lysine high	1.46 (0.12, 2.91)	1.03 (0.5- 3.47)	1.327 (0.3, 3.65)	1.59 (0.05- 8.6)	16.9 (16.9, 16.9)	2.14e7 (2.14e7, 2.14e7)	62.2 (62.2, 62.2)	9.92e26 (9.92e26, 9.92e26)	5.54 (- 8.48, 20.1)	254 (0- 5.4e8)	1.55 (0.715; 2.923)	1.70 (1.02; 18.6)
OTA_ high	1.98 (0.73, 2.15)	0.14 (0- 8.58)	1.11 (- 1.71, 4.24)	3.04 (0.18- 69.8)	-3.26 (-3.26, -3.26)	0.04 (0.04, 0.04)	-90.2 (- 90.2, - 90.2)	0 (0, 0)	-7.79 (- 21.2, 5.89)	0 (0, 362)	0.437 (- 2.311; 3.344)	1.55 (0.10; 8.34)
Child age	-0.224 (- 0.554, 0.034)	0.80 (0.57- 1.03)	-0.307 (- 0.507, - 0.143)	0.74 (0.60- 0.87)	-0.319 (- 0.319, - 0.319)	0.73 (0.73, 0.73)	-38.7 (- 38.7, - 38.7)	0 (0, 0)	-0.746 (- 1.77, - 0.013)	0.47 (0.17- 0.99)	-0.311 (- 0.495; - 0.155)	0.73 (0.61; 0.86)
Female	-0.493 (- 2.32, 1.39)	0.61 (0.10- 4.00)	0.244 (- 0.907, 1.39)	1.28 (0.40- 4.03)	0.444 (0.444, 0.444)	1.56 (1.56, 1.56)	-2.92 (- 2.92, - 2.92)	5e-2 (5e- 2, 5e-2)	-0.402 (- 4.73, 3.00)	0.67 (0.01- 20.1)	0.526 (- 0.575; 1.752)	1.69 (0.56; 5.77)
Co- exposure high	0.903 (- 1.22, 2.93)	2.47 (0.29- 5.7)	-0.76 (- 2.31, 0.659)	0.47 (0.10- 1.93)	-16.6 (-16.6, -16.6)	0 (0, 0)	44.3 (44.3, 44.3)	1.72e19 (1.72e19, 1.72e19)	-4.16 (- 12.4, 2.36)	0.02 (0- 10.6)	-0.462 (- 1.849; 0.841)	0.63 (0.16; 2.32)