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Review Article

The Scourge of Aflatoxins in Kenya: A 60-Year Review (1960 to 2020)

Timothy Omara,[1,2,3] **Ambrose K Kiprop**,[1,2] **Phanice Wangila**,[4] **Alex Paul Wacoo**,[5] **Sarah Kagoya**,[6] **Papias Nteziyaremye**,[1,2] **Mark Peter Odero**,[1,2] **Caroline Kiwanuka Nakiguli**,[1,2,7] **and Samuel Baker Obakiro**[1,2,8]

[1]*Department of Chemistry and Biochemistry, School of Sciences and Aerospace Studies, Moi University, Uasin Gishu County, P.O. Box 3900, Eldoret, Kenya*
[2]*Africa Center of Excellence II in Phytochemicals, Textiles and Renewable Energy (ACE II PTRE), Moi University, Uasin Gishu County, P.O. Box 3900, Eldoret, Kenya*
[3]*Department of Quality Control and Quality Assurance, Product Development Directory, AgroWays Uganda Limited, Plot 34-60, Kyabazinga Way, P.O. Box 1924, Jinja, Uganda*
[4]*Department of Physical Sciences, University of Kabianga, P.O. Box 2030, Kericho, Kenya*
[5]*Department of Medical Biochemistry, School of Biomedical Sciences, College of Health Sciences, Makerere University, P. O. Box 7026, Kampala, Uganda*
[6]*Department of Quality Control and Quality Assurance, Sweets and Confectionaries Section, Kakira Sugar Limited, P.O. Box 121, Jinja, Uganda*
[7]*Chemistry Department, Faculty of Science, Mbarara University of Science and Technology, P.O. Box 1410, Mbarara, Uganda*
[8]*Department of Pharmacology and Therapeutics, Faculty of Health Sciences, Busitema University, P.O. Box 1460, Mbale, Uganda*

Correspondence should be addressed to Timothy Omara; prof.timo2018@gmail.com

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Aflatoxins are endemic in Kenya. The 2004 outbreak of acute aflatoxicosis in the country was one of the unprecedented epidemics of human aflatoxin poisoning recorded in mycotoxin history. In this study, an elaborate review was performed to synthesize Kenya's major findings in relation to aflatoxins, their prevalence, detection, quantification, exposure assessment, prevention, and management in various matrices. Data retrieved indicate that the toxins are primarily biosynthesized by *Aspergillus flavus* and *A. parasiticus*, with the eastern part of the country reportedly more aflatoxin-prone. Aflatoxins have been reported in maize and maize products (*Busaa*, *chan'gaa*, *githeri*, *irio*, *muthokoi*, *uji*, and *ugali*), peanuts and its products, rice, cassava, sorghum, millet, yams, beers, dried fish, animal feeds, dairy and herbal products, and sometimes in tandem with other mycotoxins. The highest total aflatoxin concentration of 58,000 μg/kg has been reported in maize. At least 500 acute human illnesses and 200 deaths due to aflatoxins have been reported. The causes and prevalence of aflatoxins have been grossly ascribed to poor agronomic practices, low education levels, and inadequate statutory regulation and sensitization. Low diet diversity has aggravated exposure to aflatoxins in Kenya because maize as a dietetic staple is aflatoxin-prone. Detection and surveillance are only barely adequate, though some exposure assessments have been conducted. There is a need to widen diet diversity as a measure of reducing exposure due to consumption of aflatoxin-contaminated foods.

1. Introduction

Mycotoxins constitute a family of secondary metabolites biosynthesized by fungi from genera *Penicillium*, *Aspergillus*, and *Fusarium* [1]. They contaminate various agricultural commodities prior to or after harvest [2]. Deoxynivalenol (DON), aflatoxins (AFs), ochratoxins, zearalenone (ZEA), T-2 toxins, and fumonisins (FUMs) are some of the

mycotoxins of toxicological priority in foods [3, 4]. In developing countries, AFs and FUMs pose the greatest threat [4, 5]. At least 4 billion people in Third World countries are recurrently exposed to AFs [6], and the statutory standards for AFs in foods negatively impact the economic growth of such nations [7–9].

Aflatoxins are a group of mycotoxins produced by at least 20 fungal strains of *Aspergillus* section Flavi, Nidulantes, and Ochraceorosei [10, 11]. Their discovery and recognition are traced back to 1960 in which Turkey "X" disease was reported in England with several poults lost to the toxins after feeding on a contaminated peanut ration [12, 13]. AFs were eventually recovered in East Africa (Kenya and Uganda) in peanut rations that caused substantial losses in ducklings [14, 15]. AFs are chemically polysubstituted coumarins with very similar chemical structures [16]. About 20 different types have been reported, and aflatoxin B_1 (AFB_1), aflatoxin G_1 (AFG_1), aflatoxin M_1 (AFM_1), aflatoxin B_2 (AFB_2), and aflatoxin G_2 (AFG_2) are of demonstrated toxicological importance [17]. The B-type AFs are pentanone derivatives that show strong blue fluorescence under UV light. On the other hand, the G-series AFs are six-membered lactones that fluoresce yellow-green under UV light and thus the B and G nomenclature [2, 18]. Aflatoxin G_2 and B_2 are congeners of G_1 and B_1 which lack the 8,9-double bond in the furan ring and therefore usually only encountered in tandem with the latter [19]. Aflatoxins M_1 and M_2 are metabolic derivatives of B_1 and B_2 that exhibit blue-violet fluorescence and are usually detected in urine and milk of animals served AFB_1-contaminated rations [20, 21].

Aflatoxins are mutagenic, genotoxic, immunosuppressive, carcinogenic, and teratogenic [17] in the order $AFG_2 < AFB_2 < AFG_1 < AFM_1 < AFB_1$ [22–24] (Figure 1). This order reflects the role of epoxidation of the 8,9-double bond and the unique potency of cyclopentenone ring in B-type AFs [25]. The mutagenic and carcinogenic effects of AFB_1 and other AFs possessing double bonds between C_8 and C_9 in the furan ring have been ascribed to their hepatic bioactivation to the intermediate metabolite (AFB_1-8,9-epoxide) [26–30]. The reaction is catalyzed by polymorphic cytochrome P450 enzymes [28, 31]. AFB_1-8,9-epoxide is an unstable mutagen [32] that covalently interacts with nucleophilic sites of cellular macromolecules such as nucleic acids (principally DNA and RNA), inducing irreversible signaling and genetic, metabolic, and cell configuration dysregulations [26, 33–36]. Details of how AFs induce mutagenicity and carcinogenicity have been discussed in sufficient details in previous studies [28, 37].

Mutegi et al. [38] published a review on the prevalence and strategies for mitigation of AFs in Kenya from 1960 to 2018. Since then, more than 15 studies on AFs have been undertaken in Kenya. The current review digests the scourge of AFs in Kenya from 1960 to the present, highlighting the progresses in the occurrence, detection, quantification, and exposure assessment. Prevention and control measures as well as evidence-based management strategies are discussed.

2. Occurrence of Aflatoxins in Kenya

2.1. Causative Fungi and Prevalence of Aflatoxins. Aflatoxins in Kenya are predominantly produced by *Aspergillus parasiticus* and *A. flavus* [39–49]. The latter is a universal fungus known to produce AFB_1 and AFB_2 along with aspergillic, cyclopiazonic, and kojic acids [50]. *A. parasiticus* produces both B and G AFs plus kojic and aspergillic acids [50–52]. *A. niger*, *A. terreus*, and *A. versicolor* were reported in soils and mill dust in Eastern Kenya [40]. Further, the occurrence of *A. alliaceus*, *A. tamarii*, and *A. caelatus* in Kenya has been echoed [44, 53, 54]. A genetic profiling study reported that *A. minisclerotigenes* in Eastern Kenya exhibited a higher AF biosynthesis potential than *A. flavus* [42]. Though both the L- and S-strain morphologies of *Aspergillus* section Flavi have been reported, probing aetiological studies revealed that aflatoxicoses associated with maize consumption in Kenya have been due to a novel S-morphology fungus previously implicated for the 2004–2006 aflatoxicosis outbreaks [39, 55, 56]. Overall, *A. flavus* is considered the primary producer of AFs in agricultural commodities with an optimal growth temperature of 25°C and a minimum water activity of 0.75. AF biosynthesis, however, starts at 10–12°C [57].

Kenya possesses an erratic tropical climate characterized by periodic droughts, high humidity, and high temperatures preceding harvests [58]. The climate is tropical along the coast, temperate inland, and arid in the north and northeast. The country has distinct seasons: March to June with long rains and October to December with intermittent rains. It has four marked climatic zones, further partitioned into agroecological zones contingent on the rain and temperature conditions suitable for the staple crops. The Central Highlands and the Rift Valley are blessed with fertile soils, rainfall of up to 3000 mm per annum, and temperatures of 21°C to 26°C. On the other hand, the western part of the country is hot and remains wet throughout. Rain received is more than 1000 mm per annum with temperatures of 27°C to 29°C. North and eastern parts are relatively hot with annual rains lower than 510 mm and temperatures which occasionally reach 39°C in some areas [58].

Poor grain conditioning before storage, use of propylene storage bags, drying of grain on bare grounds, insect infestation, poor storage structures (stores with leaking roofs), poor transportation, and handling of produce as well as chronic poverty have been incriminated for the aflatoxigenic contamination of Kenyan foods [3, 59–68]. Contamination has also been due to the cultivation of maize in ecologically predisposed regions of the country [69–73]. Biophysical factors (including soil, plant genetic constitution and susceptibility, and fungal community), low education levels, inadequate sensitization, and gender have also favoured the spread of AFs in Kenya [60, 67, 74–76]. On toxicological studies, AFB_1 is by far the most studied AF in Kenya, followed by AFM_1 [38]. Thus, most studies reported on the levels of AFB_1, AFM_1, or total AFs. It is worth noting that because of the aflatoxicoses that dawned several times on the country, a number of

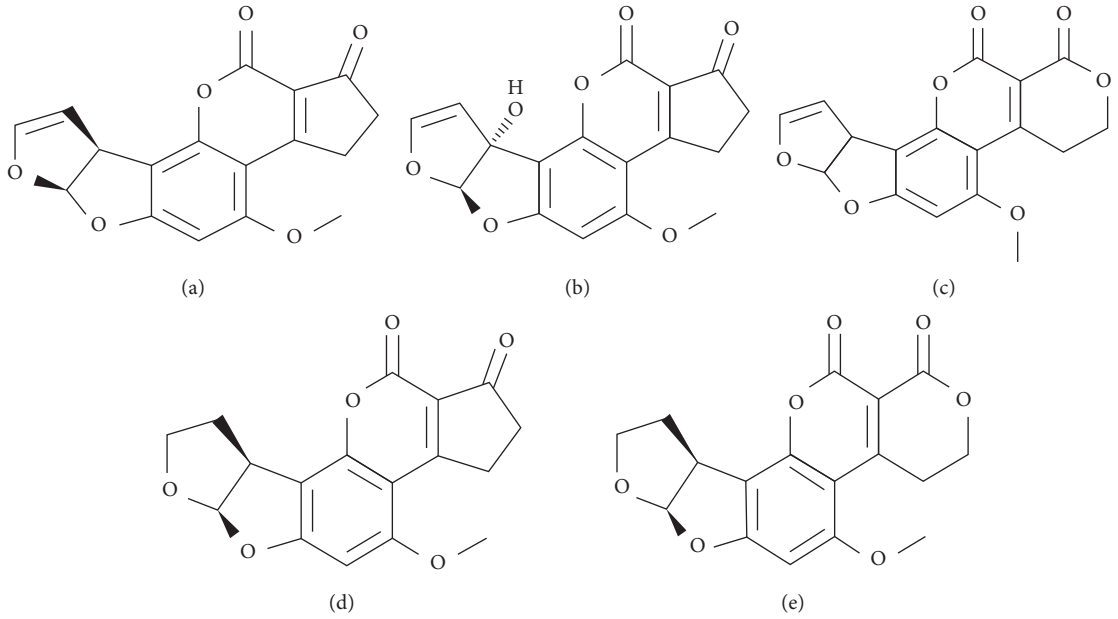


Figure 1: Structure of major AFs of toxicological concern: (a) AFB_1; (b) AFM_1; (c) AFG_1; (d) AFB_2; (e) AFG_2.

investigations have been undertaken, with often alarming AF levels reported [38, 63, 70, 77–79].

2.2. Commodities Contaminated. Aflatoxins in Kenya have been reported to contaminate staple foods such as maize (*Zea mays* L.) and its products (*Busaa, chan'gaa, githeri, irio*, *muthokoi, uji,* and *ugali)* [8, 40, 41, 45, 54, 63, 66, 77, 80], sorghum (*Sorghum bicolor* L.) [66, 76, 80], millet (*Eleusine coracana*) [76, 81], pigeon peas and their local products [80, 82, 83], peanuts and its products [53, 61, 62, 80, 84], cassava, rice, and dried silverfish (*Rastrineobola argentea,* locally called *omena*) [80, 85], animal feeds [73, 86], dairy products (milk, yoghurt, and *Lala*) [57, 66, 87–90], and herbal products [91]. Research on AFs in Kenya has concentrated mostly on maize, peanuts, animal feeds, and dairy products, particularly milk [92]. Despite their ubiquitous presence in foods, food processing techniques cannot completely destroy AFs in precontaminated foodstuffs owing to their heat-proof nature [93].

2.2.1. Cereals and Cereal-Based Products. Maize, millet, and sorghum are Kenyan staples for specific regions. Maize is the main dietary staple [94, 95]. Subsistence farmers stock maize under various suboptimal conditions for more than 3 months prior to use or sale [96]. Maize is often for home consumption as flour or used for making *irio* and *githeri* (a traditional dish of maize mixed with legumes or pulses such as beans, pigeon peas, and cowpeas, usually cooked whole), though some may be sold [8]. Approximately 60% of the maize are processed for consumption using hammer mills [40, 97]. It was previously echoed that maize consumption is the primary route through which Africans have been chronically exposed to AFs [98–100]. On the other hand, sorghum and millet are grown primarily in the semiarid regions of the country and are consumed mainly as flours used for the preparation of thick porridge (*ugali*) and thin porridge (*uji*). *Uji* is an ingredient of infant weaning foods and diet for children [9].

In Kenya, maize meal consumption is estimated at 400 g/person/day with an average total AF content of 0.132 μg/kg and has been incriminated for all aflatoxicoses recorded [71, 101]. In one of the pioneering studies, Kenji et al. [81] reported very high total AFs of 1,120 μg/kg in malted maize with an 86% incidence of AFB_1. AFB_1 ranged from 0 to 260 μg/kg in malted millet from Thika market (Kenya) though no AFB_2 and AFG_1 were detected. On the other hand, maize flour had AFB_1 ranging from 0 to 160 μg/kg (from Nairobi) and traces (from Thika) with undetectable AFB_2. In another study, 68% of a maize-based traditional brew (*Busaa*) in the slums of Nairobi was declared to contain AFs in concentrations above 5 μg/kg, 17% of which were above 50 μg/kg [102]. Likewise, the magnitude of AF contamination of 480 maize grains, maize flour, and dehulled dry maize-*muthokoi* (362 random environmental samples, 26 cases, and 92 controls) samples from Makueni, Kitui, Machakos, and Thika districts was assessed [103]. It was reported that 46.4% of the environmental samples, 15% of cases, and 29.3% of controls were within the then threshold of 20 μg/kg, implying that 54.6% of the samples could not be used for human consumption. Further, 6.9% of the environmental samples, 57.7% of cases, and 21.7% of controls had AF concentrations above 1000 μg/kg. The overall AF contamination of the samples ranged from 0 to 58,000 μg/kg [103]. Further, Sirma et al. [76] recorded total AFs of 0.17–5.3 μg/kg from 67% of maize from the Rift Valley region. About 92% of millet and 50% of sorghum samples collected in the study were positive for AFs in the ranges of 0.14–6.4 μg/kg and 0.21–210.1 μg/kg, respectively.

Later, Muthomi et al. [40] reported that samples of whole maize, mill dust, and semiprocessed maize in Machakos,

Eastern Kenya, had more than 20 μg/kg AFB_1 threshold allowed by then in Kenya. The highest AFB_1 level (160 μg/kg) was recorded in whole grains. The dust registered the highest AF content, probably due to dehulling operations and the continuous availability of maize products which are potential substrates for *A. flavus* proliferation. As expected, semiprocessed grains had the lowest contamination levels and this was speculated to be so due to dehulling of the grains as reported elsewhere [104].

Similarly, a random study appraising market maize contamination as well as the relationship between market maize AFs and aflatoxicosis outbreak was conducted [71]. A total of 65 markets were surveyed, 243 maize vendors were interviewed, and 350 maize and maize products samples were drawn from the most affected districts as per the previous history of aflatoxicoses. About 55% of the samples had AFs in levels above the then advisory threshold of 20 μg/kg, 35% had levels >100 μg/kg, and 7% had levels >1,000 μg/kg (Table 1). Makueni district which had the highest number of aflatoxicosis case-patients had evidently higher market maize AF concentrations than Thika district (which had the fewest case-patients) with a geometric mean of 52.91 μg/kg versus 7.52 μg/kg. In addition, maize from local farms in the affected areas was more likely to have AFs in concentrations >20 μg/kg vis-à-vis those from other regions or countries. Because it was understood that contaminated homegrown maize in the affected areas infiltrated the distribution system, wild AF contamination of market maize was inevitable, and the contaminated market maize bought after homegrown maize supplies were exhausted was cited as the reason for persistent exposure to AFs. The authors stressed that efforts to meaningfully evade AF exposure during an aflatoxicosis epidemic should take into account the role of the market chain [71].

Probst et al. [55] reported that, in Eastern province (Kitui and Mukueni), Coast (Makueni, Kwale, Kilifi, Tana River, and Taita Taveta), and Rift Valley (Marakwet, Keiiyo II, Kajiado, Baringo, Nakuru, and Laikipia), total AFs in maize ranged from 219.6 to 426.3 μg/kg, 0.1–120.4 μg/kg, and below detection limit (BDL) to 13.4 μg/kg, respectively. Indeed, the Aflacontrol Project [105] also reconfirmed this observation. Maize grains sampled between January 2010 and May 2010 from fields (preharvest), stores (postharvest), and wholesalers, retailers, and open-air vendors were declared to be contaminated with AFs. The highest level of AFs in preharvest maize ($n = 281$) was 1,455 μg/kg from Mbooni East (Eastern Kenya). No appreciable differences were noted between samples from Western and Eastern Kenya. For example, samples from Homa Bay and Rongo had 37 μg/kg and 54 μg/kg of total AFs vis-à-vis 21 μg/kg reported in Makueni, 25 μg/kg reported in Mbeere North, and 44 μg/kg reported in Mbooni East. Matter-of-factly, more samples from the Western sites were unfit for human consumption (had total AFs >10 μg/kg) than those from the eastern sites. For 241 postharvest samples, 38% from the eastern region had AF levels above 10 μg/kg. The plague was most acute in Makueni where 87% of samples were unfit for human consumption and the maximum AF level was 1,777 μg/kg. In Mbooni East and Mbeere North, the proportion of maize with levels above 10 μg/kg was 29% and 7%, respectively. In entirety, the proportion of maize unfit for human consumption was higher in the eastern sites than in the western sites, but there was considerable variation across the different areas sampled.

Another study reaffirmed the foregoing. For 306 maize samples collected from markets in Upper Eastern Kenya ($n = 101$), Lower Eastern Kenya ($n = 87$), Homa Bay/Rongo ($n = 102$), and Kisii Central ($n = 21$), majority (206) had AF levels below 10 μg/kg. However, the eastern side had more samples with AFs >10 μg/kg, with a maximum of 1,633 μg/kg recorded. In another concerted study, Collins et al. [106] reported that maize from Homa Bay and Rongo had mean AF levels of 37.0 μg/kg and 54.0 μg/kg compared to 21.0 μg/kg, 25.0 μg/kg, and 44.0 μg/kg in Makueni, Mbeere North, and Mbooni East, respectively.

In consonance with the aforementioned, Muthomi et al. [107] evaluated the distribution and contamination levels of *Aspergillus* species (spp) and AFB_1 in soil, maize, and maize-based products. Maize grain ($n = 256$), semiprocessed grain ($n = 56$), flour ($n = 52$), hammer mill dust ($n = 11$), and soils ($n = 117$) had *A. flavus* in all the samples, though the fungi were prevalent in the grain. AFB_1 was undetected in samples from the humid regions but was present in concentrations in excess of 10 μg/kg in 20% of the samples, with maxima of 136 μg/kg for semiprocessed maize, 77 μg/kg for whole grain, and 41 μg/kg for flour in open bags. Incidental high temperature and periodic droughts prevalent in the semiarid regions were incriminated for the higher levels of *A. flavus* and AFB_1 recorded. A study report recapitulated in [16, 108] indicated that Kenyan maize was the most contaminated in East Africa with a mean of 131.7 μg/kg (Table 2).

A total of 54 processed and unprocessed (brands A and B) cattle feed from agricultural and veterinary stores and 96 human foods (unprocessed and processed maize, polished and unpolished rice, peanut seeds, and flour) samples collected from open market traders in Nairobi County were analyzed [86]. The awareness of the traders on AFs and the associated health effects were assessed using questionnaires. Total AF concentrations recorded were 120.9 ± 27.2 μg/kg (processed feed), 77.6 ± 16.0 μg/kg (brand A), 48.6 ± 12.0 μg/kg (brand B), 49.7 ± 14.7 μg/kg (unprocessed maize), 101.20 ± 21.30 μg/kg (maize flour), 38.2 ± 10.5 μg/kg (unpolished rice), 63.9 ± 14.5 μg/kg (polished rice), 54.6 ± 14.8 μg/kg (peanut seeds), and 120.9 ± 27.2 μg/kg (peanut flour). Higher AF levels were reported in processed foods (mean: 95.0 ± 12.7 μg/kg) than in nonprocessed foods (mean: 47.5 ± 7.6 μg/kg), and this implied that some food processing techniques used predisposed the foods to aflatoxigenic contamination. Roughly 56.6% of the traders were aware of AF contamination; cattle feed traders were more conversant with AFs (40%) than human food traders (17%). A very small portion of food traders (3.7%) and feed traders (8%) were aware of the health effects of AFs in humans and animals, respectively. Because the mean AF levels in both feeds and foods were above statutory limits, the author recommended the need for creating traders' awareness on AFs, their effects, and practices that favour AF proliferation.

Aflatoxin exposure via intake of maize and its products was evaluated through analysis of 20 samples each of maize

Table 1: Distribution of AFs in maize products from some Kenyan districts following the 2004 aflatoxicosis.

District	Number of samples[a]	Total aflatoxin concentration[b]			
		≤20 μg/kg (%)	21–99 μg/kg (%)	100–1,000 μg/kg (%)	>1,000 μg/kg (%)
Makueni	91	32 (35)	12 (13)	36 (40)	11 (12)
Kitui	73	28 (38)	15 (21)	23 (32)	7 (10)
Machakos	102	50 (49)	26 (25)	23 (23)	3 (3)
Thika	76	50 (66)	13 (17)	10 (13)	3 (4)
Total	342	160 (47)	66 (19)	92 (27)	24 (7)

Excerpted from Lewis et al. [71]. Values shown were for samples with AFs and the percentage of total samples within the district. [a]Number of samples analyzed for AFs, did not include samples that were collected but not analyzed. [b]Acceptable upper limit for AFs in grains by then was 20 μg/kg.

Table 2: AFs contamination of foods in Eastern Africa.

Food	Country	Per capita intake (g/person/day)	Average AF level (μg/kg)
Maize	Kenya	405	**131.7**
	Tanzania	69	**49.7**
	Uganda	400	9.7
Groundnuts (peanuts)	Uganda		**25.1**
	Tanzania		**15.0**
	Burundi	65	**12.5**
Cassava chips	Uganda		0.5
	Tanzania	214	0.9
Sorghum	Tanzania	40	3.0
Milk	Kenya	750 ml	**0.8**
	Tanzania	750 ml	**0.9**
	Rwanda	750 ml	Not detected

Values in bold indicate exceedance of East African thresholds.

kernels, *muthokoi*, and *githeri* haphazardly drawn from households in the eastern district of Kibwezi of Makueni County [109]. Uncertainty and variability in dietary exposure were modelled quantitatively. AFs were recorded in 45% of maize (range: 18–480 μg/kg), 20% of *muthokoi* (range: 12–123 μg/kg), and 35% of *githeri* (range: 6–30 μg/kg). The mean dietary exposure to AFs in maize kernels, *muthokoi*, and *githeri*, respectively, was 292 ± 1567, 27 ± 154, and 59 ± 62 ng/kg bw/day. The amount and frequency of consumption of the foods were cited as the requisite factors for dietary exposure to AFs. Moreover, some maize ($n = 268$), sorghum ($n = 62$), and millet grains ($n = 39$) from households and markets in villages of Nandi County were subjected to AF analysis [76]. Computed 67.7% (72/106), 73.3% (44/60), and 65.7% (67/102) of maize from Laboret, Kilibwoni, and Chepkongony sublocations were contaminated with AFs (range: 0.17–5.3 μg/kg); 92.9% (13/14), 100% (9/9), and 87.5% (14/16) of millet from Laboret, Kilibwoni, and Chepkongony had AFs in the range of 0.14–6.4 μg/kg. However, only 50% (9/18), 36.4% (8/22), and 27.3% (6/22) of sorghum drawn from Laboret, Kilibwoni, and Chepkongony, respectively, had AFs above 10 μg/kg (range: 0.15–210.1 μg/kg).

To check for chronic inadvertent exposure to AFs, maize ($n = 75$) and maize flour ($n = 27$) from different parts of Kenya were collected and analyzed [95]. Striking differences in the AF levels of maize grain between the regions and stores from which samples were drawn were reported. Eastern Kenya had the highest contaminated samples with a mean of 22.54 ± 4.94 μg/kg, while those from Nairobi had the lowest contamination (7.92 ± 1.57 μg/kg). No appreciable differences were observed for total AFs in maize flours from the regions. AFs in maize flours were marginally above the European Union (EU) limit of 5 μg/kg, and most of the samples had AFs lower than the statutory limit of 10 μg/kg. The authors attributed this to adherence to good manufacturing practices by the millers. The highest AF level in maize flours from Eastern Kenya was 6.98 ± 0.53 μg/kg [95]. Recently, Obonyo and Salano [63] echoed that maize grain in Eastern Kenya from May harvest had lower AF levels with variation (5.68 ± 6.31 μg/kg, 100% AFB_1) than those from October-December rains (10.77 ± 10.14 μg/kg, 72% AFB_1). From the two seasons, the authors hinted that 16% and 44% of the samples had total AFs above the statutory limit of 10 μg/kg. Another group [110] undertook a random study in three agroecological zones: Kitui (semihumid to semiarid), Nakuru (semihumid), and Kitale (subhumid to semihumid) to determine the prevalence and AFs in 130 stored maize samples and the aflatoxigenicity potential of *A. flavus* in stored maize. The authors put forward that aflatoxigenic contamination between the sampled sites was markedly distinct with the highest mean total AFs of 9.68 μg/kg reported in Kitale district. *A. flavus* was detected in 91 (70%) samples and the colonies with exorbitant aflatoxigenicity stemmed from Nakuru County with a recorded mean total AF of 239.7 μg/kg.

Recently, maize from smallholder farmers' fields in Eastern and Southwestern Kenya ($n = 789$) was analyzed for AFB_1 [94]. The authors detected AFB_1 (range: 0.01–9,091.8 μg/kg; mean: 67.8 μg/kg) in 274/416 samples

from Eastern Kenya. In the Southwest, the toxins were quantified in 233/373 samples (range: 0.98–722.2 μg/kg; mean: 22.3 μg/kg). Of these, 153 (55.8%) from Eastern and 102 (43.8%) from Southwest had AFB_1 surpassing the threshold of 5 μg/kg. The probable daily intake (PDI) of AFB_1 in the East was 0.07 to 60,612 ng/kg bw/day (mean 451.8 ng/kgbw/day), while for the Southwest, it varied from 6.53 to 4,814.7 ng/kgbw/day with a mean of 148.4 ng/kgbw/day. The mean PDI for the regions surpassed AFB_1 tolerable daily intake and therefore poses health concerns. As such, it was advanced that control of preharvest AF contamination should be emphasized in the bid to reduce exposure to AFs through maize consumption [94].

The prevalence and levels of AFs in freshly harvested maize and freshly milled maize flour ($n = 338$) from households in Siaya and Makueni Counties were evaluated by Nabwire et al. [77]. All (100%) of the samples had detectable AFs, which ranged from 2.14 to 411 μg/kg. The geometric mean of total AFs in samples from Makueni and Siaya Counties was reported as 62.5 μg/kg and 52.8 μg/kg, respectively. This revalidated the fact that AFs are prevalent in maize and its products in the area. Taken together, regional disparities in AF levels of Kenyan maize have been reported with the Eastern provinces registering the highest level of 58,000 μg/kg [70, 103, 109] vis-à-vis the other provinces that registered 4,500 μg/kg as the maximum [72, 78]. Recently, the Kenya Bureau of Standards (KEBS) banned a number of maize flour products on the market because of high AF levels [8]. As per the current study, reluctancy to dehull maize has been recognized as a plausible explanation for the exorbitant AF concentrations recorded in Kenyan maize flour.

2.2.2. Peanuts (Arachis hypogaea L.) and Its Products. Peanuts (groundnuts) are the only affordable dietary protein source in Kenya [65]. It is grown predominantly in the western but sold and consumed countrywide [61, 111]. Peanut productivity has over the years declined due to unpredictable rainfall, lack of disease-resistant peanut varieties, poor agronomic practices, and poor institutional support accorded to farmers [112–114]. It is grown for local consumption but is also exported through the World Food Programme [115, 116]. Peanut is rich in proteins, fats, carbohydrates, zinc, sodium, potassium, calcium, magnesium, iron, phosphorous, and vitamins E and B [117]. In tandem with maize, they are the major portions of the gruel used to make weaning foods in Kenya and these have been shown to be a route of AF exposure [118, 119]. Most of the peanut samples tested in the country had AF levels above recommended regulatory limits set by the KEBS [120]. Fortunately, its consumption is as low as 1.1 g/person/day [92].

In one of the earlier surveys [61], baseline data on AF levels as well as 384 and 385 peanut samples from Busia and Homa Bay districts of Western Kenya were collected and analyzed. Total AFs ranged from 0 to 2,688 μg/kg and 0 to 7,525 μg/kg in samples from Busia and Homa Bay, respectively. Out of all the samples drawn ($n = 769$), 87.01% contained <4 μg/kg of AFs, 5.45% were in the range ≥4 and 20 μg/kg while 7.54% surpassed the advisory threshold of 20 μg/kg. There was a highly strong association between the districts and the analytical total AFs recorded, which was further corroborated by a significant correlation between total AF levels and agroecological zones. Logistic regression unveiled that peanuts from Busia were 2.6 times at risk of contamination vis-à-vis those from Homa Bay and that planting improved cultivars could lower the odds of contamination to half those for local landraces. In the continuity of the foregoing, the authors [44] reported that the total AF content of 436 peanut samples drawn from Busia and Homa Bay districts varied from BDL to 2,687.6 μg/kg and BDL to 1,838.3 μg/kg in about 32% of the samples with detectable AFs. Both the incidence and the number of colonies of *A. flavus* S-strain were significantly and positively correlated with the total AF content of the samples. Up to 99.3% of the samples containing <10 μg/kg of total AFs did not have *A. flavus* S-strain. This corroborated a previous report which confirmed the presence of *Aspergillus, Rhizopus, Fusarium*, and *Penicillium* spp in peanuts with AF contents spanning beyond 100 μg/kg [84].

In another survey by Mutegi et al. [62], peanut and their products from supermarkets and informal markets were analyzed. The authors announced that raw podded peanuts had the lowest AF contamination, with 96% having levels of less than 4 μg/kg and only 4% having more than 10 μg/kg. Irrespective of the provenance, 69% of the samples and 75% of spoilt nuts had total AFs exceeding 10 μg/kg. Though most samples (59%) had AF levels below 4 μg/kg, only 4% of these were acceptable under the KEBS but could be rejected under EU regulations. Of these, 37% of the peanuts were found to be unfit for human consumption as per KEBS and EU regulatory limits. Further, the team [64] evaluated the effect of storage bags, temperature, and relative humidity on the quality and AF content of peanut kernels of Homa Bay local, Valencia red, ICGV-SM 12991, and ICGV-SM 99568 varieties stored for 6 months in jute, polypropylene, and polyethylene bags. Moisture content, physical damage, rancidity, and AF levels were determined before storage and after every 30 days during storage. Moisture content changed remarkably from 3.3 to 6.9% with samples stored in different bag types recording mean values of 5.1% (polypropylene), 5.2% (polyethylene), and 5.3% (jute). Physical damage (range: 0.1 to 9.8%) was influenced by storage temperature and relative humidity and the type of storage used. Rancidity ranged from 0.8 to 5.3 and increased with storage duration from a mean of 1.5 before storage to a peak of 2.5 after 5 months of storage. There was a reported variation in total AFs (range: 0 to 47.8 μg/kg) of nuts stored in polyethylene bags having 7.3% and 13.4% more contamination than those stashed in polypropylene and jute bags.

Accordingly, it was hypothesized that the processing of peanuts in the cottage industry could facilitate their contamination by AFs. As such, Ndung'u et al. [53] assessed the AF content of raw and roasted peanuts and peanut butter marketed in Nairobi and Nyanza Provinces of Kenya. Marketers and processors of these were also interviewed on

the source of groundnuts and the incidence of *Aspergillus* section Flavi was determined. The authors stressed that the percentage of defective nuts among all unsorted nuts ranged from 0 to 26.3%. The mean percent of defective nuts was higher for Nairobi (imported from Malawi) than for Nyanza (homegrown) samples. Total AFs in the samples ranged from 0 to 2,377.1 μg/kg with higher mean total levels in raw samples from Nairobi than from Nyanza (Table 3). The source of groundnuts and defective nuts was positively associated with AF levels. *A. flavus* (L- and S-strains), *A. parasiticus*, *A. niger*, *A. tamari*, *A. alliaceus*, *A. caelatus*, and *Penicillium* spp were isolated from the samples.

The prevalence and diversity of fungal spp and aflatoxigenic contamination of 228 marketed peanut samples (from 140 formal and 88 from informal markets) in Kericho and Eldoret towns of Kenya were established [115]. *A. flavus* (L- and S-strains), *A. parasiticus*, *A. tamarii, A. caelatus, A. alliaceus* (members of *Aspergillus* section Flavi), and *A. niger* as well as *Penicillium*, *Mucor*, *Fusarium*, and *Rhizopus* spp were encountered. Total AFs in the nut products were 0 to 2,345 μg/kg in raw, 0 to 382 μg/kg in roasted, and 0 to 201 μg/kg in roasted decoated peanuts. Altogether, AFs occurred in higher concentrations in samples from informal (mean = 97.1 μg/kg) than from formal (mean = 55.5 μg/kg) markets. Meanwhile, a positive and strong correlation was cited between AF levels and the major aflatoxigenic fungi in raw peanuts from formal markets of Eldoret. Further, AFs in raw nuts from informal markets in Kericho positively and strongly correlated with the population of *A. flavus* (both strains). In roasted coated peanuts sampled from Eldoret formal markets, AFs correlated positively and significantly with *A. flavus* S-strain.

Another investigation [121] which compared the oil content and total AF level of peanuts in Busia and Kisii Central districts reported that Valencia red, Uganda local, Homa Bay local, and Local red peanut varieties from Busia had lower levels of total AFs except the Local red variety which had the highest total AF of 267 μg/kg with the lowest average oil content of 42.7% (Table 4). Peanuts from Kisii Central had higher AF levels and low oil contents. Summed up, there was an increase in total AF levels with decreasing oil contents except for Uganda local red from Kisii. In the continuity of the foregoing study, Menza and Muturi [49] reported the occurrence of five causative *Aspergillus* fungi: *A. flavus* (L- and S-strains), *A. parasiticus*, *A. niger*, and *A. tamari*. Overall, the occurrence of *A. flavus* (both strains) was significantly higher than other aflatoxigenic spp identified in the nuts. *A. flavus* L-strain was the most common isolate (58.8%) in samples from Busia while the S-strain dominated (60.2%) in peanuts from Kisii Central. All in all, *A. flavus* S-strain was the most dominant with a mean prevalence of 45.1%.

From the prevenient reports, it is notable that relatively higher AF concentrations have been detected and quantified in Kenyan peanuts. A plausible explanation advanced has been that aflatoxigenic fungi infect the shells, testa, and seeds as the pods in the soil grow. Further, mechanical damage while harvesting, drying, and storing aggravates the risk of invasion by the toxigenic fungi and aflatoxin biosynthesis. This is corroborated by a Tanzanian report which unveiled that grains and oilseeds borne on aerial generative structures had comparatively lower AF levels vis-à-vis those borne in geocarpic structures of Bambara and peanuts [122].

2.2.3. Cassava (Manihot esculenta Crantz). Cassava is a revered food crop with edible carbohydrate-rich tuberous roots and proteinaceous young leaves [123, 124]. However, it houses 2 cyanogenic glucosides: linamarin and lotaustralin (methyl linamarin) inherently synthesized for protection against predation. These cyanogens are spread in the whole plant but occur in higher quantities in the leaves and root cortex [125]. Cyanide is an inhibitor of aerobic respiration through blockage of mitochondrial electron transport and oxygen uptake. In addition, cassava is also prone to mycotoxins, particularly AFs.

In a study, dried cassava chips ($n = 13$) and cassava flour ($n = 26$) sourced from Nairobi and Mombasa markets were assessed for hydrogen cyanide, AF, and moisture contents [126]. Hydrogen cyanide ranged from 27.20 to 42.92 mg/kg and 21.45–37.77 mg/kg in cassava chips and 21.53 to 64.63 mg/kg and 21.70 to 70.03 mg/kg in flour from Nairobi and Mombasa, respectively. These were all above 10 mg/kg recommended by the East African standards (EAS 739: 2010 and EAS 740: 2010, respectively). AFs were detected in 2 flour samples from Nairobi (mean levels of 6.60 and 8.89 μg/kg) and a sample from Mombasa (mean level of 2.84 μg/kg). Moisture content ranged from 8.62 to 9.98% and 8.85 to 11.57% in cassava chips and 8.50 to 12.51% and 7.30 to 11.0% in flour samples from Nairobi and Mombasa, respectively. The study revealed that marketed cassava flour though of good aesthetic quality could be mycotoxigenically unsafe for consumption.

There are no reports in the open literature on plant products such as sugarcane, spices, beans, wheat, and barley in Kenya. *A. flavus* was not identified in soils with sugarcane grown which reportedly had *Phanerochaete chrysosporium*, *A. niger*, *Trichoderme viride*, and *Fusarium equiseti* [46]. Sugarcane is one of the daily consumables in the form of sugar and has been previously reported to house AFs [127]. In addition, no recent studies have reported on the AF content of commercial beers consumed by Kenyans despite it being one of the widely consumed foods that evidently utilize cassava and cereal food crops (sorghum, barley, and maize). Beers are essentially continuous mixed-culture fermentation products. As such, brewing could be the innocuous route for AF exposure as it offers auspicious conditions in which toxigenic fungi thrive and may be an excellent avenue for use of aflatoxin-contaminated raw materials as the end-users cannot directly notice [16]. Similarly, no reports exist on AFs in beans. In the neighbouring Uganda where sometimes Kenya imports beans, AFs were earlier recorded in excess of 1,000 μg/kg [128].

2.2.4. Animal Products. Aflatoxin-contaminated animal products such as blood, eggs, ghee, meat, milk, and dairy products present food safety concerns [129]. In Kenya, AFM_1 in bovine milk is the most studied. A list was

Table 3: AF content of peanuts and peanut butter from market outlets in Nairobi and Nyanza Provinces, Kenya.

Source	Sample	Sample type	AF level (μg/kg)		Aflatoxin-positive samples (%)		
			Range	Mean	≤4.0 μg/kg	≤10.0 μg/kg	≥10.0 μg/kg
Cottage industry	Raw peanuts	Pink regular ($n = 3$)	BDL-52.4	18.3	60	80	20
		Red regular ($n = 1$)	NA	5.0			
		Red small ($n = 1$)	NA	BDL			
	Roasted peanuts	Red regular ($n = 8$)	2.4–297.7	54.8	25	50	50
	Peanut butter	Paste ($n = 11$)	BDL-2,377.1	318.3	18	27	73
Nairobi wholesale outlets	Unsorted peanuts	Pink regular ($n = 11$)	BDL-364.7	111.2	22	26	74
		Red regular ($n = 12$)	BDL-276.1	89.1			
	Sorted peanuts	Pink regular ($n = 4$)	BDL-82.4	24.0	36	82	18
		Red regular ($n = 5$)	2.0–9.2	5.3			
		Red small ($n = 2$)	6.0–7.8	6.9			
Nyanza retail outlets	Unsorted nuts	Pink large ($n = 3$)	3.7–128.8	71.6	71	75	25
		Pink regular ($n = 9$)	BDL-229.8	44.9			
		Red regular ($n = 9$)	BDL-14.0	1.9			
		Red mixed ($n = 3$)	NA	BDL			

Adapted from [53]. BDL: below method detection limit of 0.5 μg/kg; NA: not applicable.

Table 4: Oil content and total aflatoxins of peanuts from Busia and Kisii Central districts of Kenya.

District	Variety	Mean oil content (%)	Mean total AFs (μg/kg)
Busia	Valencia red	47.2	2.3
	Uganda local red	46.7	2.4
	Homa Bay local	43.2	2.8
	Local red	42.7	**267.0**
Kisii Central	Valencia red	46.6	**93.0**
	Uganda local	45.7	**405.0**
	Homa Bay local	40.6	**101.5**

Adapted from [121]. Values in bold indicate exceedance of the permissible limit of 10 μg/kg.

developed [58] of the regions in Kenya that are at risk of AF outbreaks from milk consumption, and this encompassed all the milk production areas of Kenya. The dairy industry in Kenya is dominated by crosses between dairy and zebu breeds with more than 70% contribution to total national milk production. The feeds used are natural forage, cultivated fodder, and crop byproducts such as maize stalks and stover. Supplements such as dairy meal, maize germ, maize bran, cottonseed cake, wheat pollard, and wheat bran are also sometimes used [74].

A correlative study conducted in four urban centers by Kang'ethe and Lang'a [89] analyzed 613 milk and 830 feed samples for AFM_1 and AFB_1. About 86% (353/412) of the feed samples from farmers were positive for AFB_1 and 67% (235/353) of these exceeded the FAO/WHO limit of 5 μg/kg. About 81% (197/243) of the feeds from feed millers and 87% (153/175) from agrochemical shops were AF positive, with 58% (115/197) and 66% (92/153) of these samples exceeding permissible limits, respectively. Approximately 72% (315/439) of the milk from dairy farmers, 84% (71/85) from large- and medium-scale farmers, and 99% (88/89) of the pasteurized marketed milk were positive for AFM_1, and 20%, 35%, and 31% of positive milk from dairy farmers, medium- and large-scale farmers, and market outlets, respectively, exceeded the WHO/FAO limits of 0.05 μg/kg. On the one hand, 67% of the urban smallholder dairy farmers had knowledge that milk could be contaminated with AFM_1 but did not know the possible exposure mitigation strategies. Feed millers, on the other hand, knew about AFB_1 in grains and its excretion as AFM_1 in milk but were not alleviating exposure to animals [89] (Table 5). Similarly, Sirma et al. [130] surveyed 286 households in 37 villages representing four agroecological zones (semiarid, temperate, subhumid, and humid). They drew 280 samples of bovine milk which were subjected to AFM_1 analysis. AF levels were from 0 to 0.359 μg/kg. Generally, 58.9% of the milk sampled had AFM_1 levels BDL though 9.3% exceeded the WHO/FAO limit of 0.05 μg/kg (Table 6).

Aflatoxins were detected and quantified in fresh and sun-dried *Rastrineobola argentea* (*Dagaa* fish) collected from various markets in Luanda, Rongo, Kisumu, Ahero, and Maseno of the Winam Gulf of Lake Victoria [85]. Fresh samples had no detectable AFs, but the dried samples had mean total AF levels of 0.34 ± 0.09, 0.21 ± 0.00, 0.25 ± 0.06, 0.53 ± 0.11, and 0.11 ± 0.00 μg/kg wet weight, respectively. It was asserted that the occurrence of AFs in processed *Dagaa* fish (*omena*) could have been due to the fact that the samples were collected from the markets in July 2010 when there were rains and drying was incomplete, and thus, the sun-dried *Dagaa* fish were packed in sacks when they were incompletely dried which favoured the growth of moulds.

In a bid to assess the AF status of marketed raw milk and associated risk factors in periurban Nairobi, raw milk retailers in Dagoretti division were interviewed and milk

Table 5: Synopsis of aflatoxins in animal feeds and bovine milk in some Kenyan municipalities (from [89]).

	Source/municipality	% AF positive	>5.0 μg/kg (%)	Mean	Range
Aflatoxin B_1 (feed samples)	*Urban smallholder dairy farmers*				
	Nyeri ($n = 118$)	68.6	49.2	136.0 ± 10.0	4.0–63.0
	Eldoret ($n = 108$)	98.1	61.1	23.2 ± 23.2	4.2–178.2
	Machakos ($n = 99$)	94.9	73.3	27.7 ± 74.9	3.6–595.0
	Nakuru ($n = 87$)	80.5	58.6	17.4 ± 11.1	1.8–58.0
	Feed manufacturers				
	Nyeri ($n = 14$)	100.0	42.9	6.4 ± 4.9	1.9–15.8
	Eldoret ($n = 18$)	88.9	66.7	13.9 ± 12.8	1.9–49.0
	Machakos ($n = 1$)	100.0	100.0	43.8 ± 0.0	43.8
	Nakuru ($n = 171$)	77.8	43.3	26.0 ± 44.5	0.9–280.0
	Nairobi ($n = 390$)	84.6	56.4	13.0 ± 15.9	0.9–280.0
	Agrochemical shops				
	Nyeri ($n = 19$)	89.5	31.6	8.9 ± 8.5	1.9–28.7
	Eldoret ($n = 58$)	93.1	72.4	17.0 ± 34.6	1.8–238.0
	Machakos ($n = 29$)	79.3	43.3	17.6 ± 19.6	2.0–64.4
	Nakuru ($n = 69$)	84.1	43.5	46.0 ± 8.4	2.0–46.2
Aflatoxin M_1 (milk samples)	*Urban smallholder dairy farmers*				
	Nyeri ($n = 120$)	60.8	3.3	33.8 ± 68.7	5.0–46.0
	Eldoret ($n = 107$)	68.2	10.3	39.9 ± 39.7	5.4–228.0
	Machakos ($n = 99$)	82.8	24.2	99.7 ± 168.9	5.1–780.0
	Nakuru ($n = 110$)	77.3	20.9	83.3 ± 129.3	5.2–550.0
	Medium- and large-scale farmers				
	Nyeri ($n = 25$)	76.0	0.0	20.2 ± 29.0	5.2–50.0
	Eldoret ($n = 16$)	68.8	12.5	115.6 ± 202.7	5.5–560.0
	Machakos ($n = 7$)	100.0	50.0	52.2 ± 34.7	10.9–102.5
	Nakuru ($n = 27$)	89.9	55.6	65.1 ± 36.7	5.3–165.0
	Nairobi ($n = 10$)	100.0	50.0	99.8 ± 97.3	10.0–245.0
	Marketed milk				
	Nyeri ($n = 10$)	100.0	30.0	129.3 ± 198.8	16.5–600.0
	Eldoret ($n = 18$)	100.0	22.2	36.4 ± 24.5	5.8–74.0
	Machakos ($n = 18$)	94.4	16.7	33.1 ± 17.0	11.0–67.0
	Nakuru ($n = 19$)	100.0	36.8	36.1 ± 22.9	8.0–71.0
	Nairobi ($n = 24$)	100.0	41.7	64.9 ± 76.4	7.9–300.0

n = number of samples. Means were presented with errors as standard deviations.

Table 6: AFM_1 contamination of bovine milk in some selected agroecological zones of Kenya (from [130]).

County	Agroecological zone	Number of samples	AFM_1-positive samples (%)		
			<0.002 μg/kg	≤0.002–0.05 μg/kg	≥0.05 μg/kg
Tharaka-Nithi	Humid	64	34	41	25
Kwale	Subhumid	29	76	17	7
Bungoma	Temperate	64	53	41	6
Kisii	Temperate	63	65	30	5
Isiolo	Semiarid	60	77	22	2

samples were drawn and tested for AFM_1 [131]. The business types encountered were dairy shops, kiosks, street or mobile vendors, and grocery stands. Milk was primarily from dairy farms (59%) or intermediate distributors (35%). Although 58% of the retailers had known of AFs and many were in agreement that AFs could occur in milk, only 29% supported that "milk safety cannot be solely judged by sight or taste" and only 6% supported that "milk is not completely safe even after boiling". Analysis of the milk samples recorded mean AFM_1 of 0.1287 μg/kg (median = 0.0499 μg/kg; maximum of 1.675 μg/kg). In entirety, 55% of the samples exceeded the EU maximum level of 0.05 μg/kg and 6% exceeded the recommended maximum level of the US FDA of 0.5 μg/kg. Vis-à-vis milk from street vendors, a significantly higher AFM_1 concentration was detected in milk from kiosks and dairy shops, especially when the milk was sourced from farms without an intermediate distributor. Similarly, it was reported that 156 samples out of 185 (150 raw milk and 35 processed milk and milk products) from Bomet County were positive for AFM_1 with an overall prevalence of 84.32% [132]. About 43.8% of these were above 0.05 μg/kg, with raw milk compared to processed milk (52% vs. 8.6%) having more contamination.

In the same manner, AFM_1 was detected in 291 samples of raw, pasteurized, and UHT milk, yoghurt, and *Lala* [90]. Monthly samples were drawn over a period of 1 year, just as

a consumer would purchase them from retailers and traders in a low-income area (Dagoretti) and a major supermarket in a middle/high-income area (Nairobi). More than 50% of the samples had AFs exceeding 0.05 μg/kg, though only 3 exceeded 0.5 μg/kg and the geometric mean AFM_1 level was 0.0619 μg/kg in the 135 samples from Dagoretti while it was 0.0361 μg/kg in 156 samples from Nairobi. The levels varied significantly depending on the time of year, with the lowest levels reported in January. UHT milk had the lowest AF levels, and more expensive milk had lower AFM_1 levels [90].

In a recent study [129], it was pointed that exposure to AFM_1 in milk and the health risks associated with it are not clearly understood and monitored in Kenya. Thus, the team assessed the awareness, knowledge, and practices of urban and periurban farmers about AFs and evaluated the levels of AFs in on-farm milk in Kasarani subcounty, Nairobi County. In total, 84 milk samples were analyzed, and 90% (83/84) were analytically declared to be contaminated with AFM_1 (mean value of 0.084 μg/kg). About 64% of the samples had AFM_1 levels well above the EU limit of 0.05 μg/kg. Though 80% of the farmers had knowledge of AFs, no correlation existed between the farmers' knowledge and gender with AFM_1 prevalence.

Kang'ethe et al. [87] reported that 45.5% and 98.6% of bovine milk and animal feeds in Kenya were positive for AFs. About 49% and 83% of these had AFM_1 above 0.05 μg/kg and AFB_1 above 10 μg/kg, respectively. Similarly, an AF risk mapping study from milk consumption using biophysical and socioeconomic data [58] reported a mean AFB_1 content of 9.25 μg/kg in animal feeds and a mean AFM_1 content of 0.0265 μg/kg in bovine milk. Higher mean of the logarithmic AFB_1 concentrations was reported in areas with historical aflatoxicosis outbreaks compared to those without outbreak history, a phenomenon that was not true for the mean logarithm of AFM_1 when compared between areas with and those without a history of aflatoxicosis outbreaks. Analogously, a cross-sectional study of aflatoxigenic contamination of bovine milk and dairy concentrates was done in five counties of Kenya representing the agroecological zones: Kwale, Isiolo, Tharaka-Nithi, Kisii, and Bungoma [133]. Concentrates and milk were collected twice (during the dry season and rainy season) from 285 farmers in the five counties and analyzed for AFB_1 and AFM_1. Between 0 and 68% used concentrates, which had AFB_1 ranging from <1 μg/kg to 9,661 μg/kg with 47.8 to 90.3% positive samples. About 33.3% to 87.5% of the concentrates had more than 5 μg/kg AFB_1 (83.3% to 100% from retailers and 28.6% to 100% from manufacturers). AFM_1 prevalence in milk was lowest in Kwale (13.6%) and highest in Tharaka-Nithi (65.1%). About 3.4% (Kwale) to 26.2% (Tharaka-Nithi) of milk samples had AFM_1 above the WHO/FAO threshold of 0.05 μg/kg, with the highest contamination of 6.999 μg/kg. The study was in consonance with preceding studies which indicated that AFs are prevalent in Kenyan dairy rations and milk.

In Kisumu, Anyango et al. [134] reported that 97 randomly selected dairy farmers primarily fed cows on forage and concentrates. AFM_1 levels in milk collected from these farms ranged from BDL to 0.151 μg/kg (mean of 0.02967 μg/kg) and 26.4% of these exceeded the EU limit. Concentrate feeding was associated with higher AFM_1 levels so that farms feeding concentrates were more likely to record milk AF levels above 0.05 μg/kg [134]. Further, the prevalence of AFM_1 in 96 samples of informally marketed milk from Nairobi, the knowledge of milk traders on AFs, and the effects of boiling and fermentation on AFM_1 were assessed [135]. By and large, all samples had detectable AFM_1 (limit of detection = 0.005 μg/kg) with a mean of 290.3 ± 0.663 μg/kg. About 64% of the samples had AFM_1 above 0.05 μg/kg while 7.5% exceeded 0.5 μg/kg. Majority of the traders had low (69.8%) or medium (30.2%) knowledge of AFs. The educated and female traders were more knowledgeable, and fermentation of milk to *Lala* (a traditional fermented drink) or yoghurt significantly reduced AFM_1 levels by 71.8% (in *Lala* after 15-hour room temperature incubation) and 73.6% in yoghurt after incubation at 45°C for 4 hours. Boiling, however, had no appreciable effect on AFM_1 levels [135].

According to Sirma et al. [9] using a quantitative risk model, an equivalent of 5 hepatocellular cancer cases and deaths and the disability-adjusted life years of 255 for Kenya in 2016 were estimated as due to exposure to AFs in milk. Other than milk, there are no reports in the open literature on AF content of other products of animal origin such as blood, eggs, ghee, and meat in Kenya.

2.2.5. Animal Feeds. As pointed earlier, farming is one possible exposure route to AFs. For example, maize which is known to be highly susceptible to AF contamination in Kenya is also a major component of livestock and poultry feeds, and therefore, regular indirect human exposure through the consumption of animal products that contain AF residues cannot be underrated. Elevated levels of AFB_1 have been recorded in Kenyan animal feeds [88, 89]. The situation is exacerbated by dairy farmers' habit of utilizing spoilt (pest- or mould-damaged and rotten) grains for the formulation of dairy rations [74, 92]. A study carried out on animal feeds in Nairobi Province revealed that AFs ranged from 5.13 μg/kg to 1,123 μg/kg, with the largest proportion lying between 11 μg/kg and 99 μg/kg [78].

Further, 81 fish feeds sourced from 70 farms and 8 feed manufacturing establishments located in Nyeri, Kenya, were subjected to AF analysis by Mwihia et al. [73]. Fish were also sampled from 12 farms for gross and microscopic pathological investigation. About 84% of the feeds were AF-positive (range 1.8–39.7 μg/kg, mean of 7.0–8.3 μg/kg, and median of 3.6 μg/kg). About 18.5% of the feeds sampled registered total AFs above the statutory limit of 10 μg/kg. Meanwhile, homemade and tilapia feeds had evidently higher AF levels than commercial and trout feeds. Maize bran-based feeds and fish meal recorded higher AF levels than those devoid of these constituents. Microscopy revealed that five trout farms (41.7%) had fish with swollen abdomens, enlarged livers with white or yellow nodules, and large dark basophilic hepatic cells with hyperchromatic nuclei in irregular cords. As such, the authors inferred that aflatoxigenic contamination of fish feeds is a scourge in Nyeri which if left unchecked may cause detrimental health effects in edible fish in the area.

2.3. Co-Occurrence of Aflatoxins with Other Mycotoxins. It is now established that mycotoxins can coexist in foods [136]. In 1995, Muriuki and Siboe [45] analyzed 40 samples of flour packed in 90 kg bags, 58 samples of *Ugali* brand, and 74 samples of *Jogoo* brand drawn from Nairobi, Kenya. The samples were analyzed for resident mycoflora, and some mycotoxins associated with key fungal spp. *Aspergillus flavus*, *A. sulphureus*, *Fusarium moniliforme*, *Penicillium stoloniferum*, and *P. cyclopium* were the reported fungal spp in the samples. Ochratoxin A was the most prevalent mycotoxin, and all the flour brands had AFB_1 and AFB_2 (0.4–20 μg/kg), ochratoxin A (50–1,500 μg/kg), and ZEA (2,500–5,000 μg/kg). The authors recommended the need for rigorous countrywide monitoring of mycotoxins in maize at both farm and market levels. The foregoing was substantiated by a report by Kedera et al. [137] who reported the presence of *Fusarium* fungi and fumonisin B_1 (FB_1) in maize kernel samples from smallholder farm storages in Bomet, Bungoma, Kakamega, Kericho, Kisii, Nandi, Siaya, Trans-Nzoia, and Vihiga districts in the tropical highlands of Western Kenya. Later, Mbugua and Gathumbi [138] affirmed the occurrence of AFB_1, FB_1, ZEA, and DON in 36 Pilsner and 39 Tusker beer samples sourced from Nairobi and the surrounding satellite towns. All the samples were negative for AFB_1; the prevalence of DON and ZEA were 100% in both brands while FB_1 incidence was 72%, with incidences in Tusker (76.9%) being markedly higher than in Pilsner (66.7%). The mean values of contamination were 3.29 and 3.57 ng/mL for DON, 0.28 ng/mL and 0.32 ng/mL for FB_1, and 7.84 and 8.50 pg/ml for ZEA in Tusker and Pilsner brands, respectively. A positive correlation was reported between DON and FB_1, and DON and ZEA, affirming their co-occurrence to be from *Fusarium* spp. This communication suggested that there were some but safe exposure to *Fusarium* mycotoxins by lager beer consumers of Kenya.

Fumonisin B_1 and AFB_1 in symptomless and rotten maize harvested at different harvest time points after physiological maturity (HTPAPM) from Malava and Tongaren were evaluated [139]. *Fusarium verticillioides* dominated at all HTPAPM though *F. graminearum*, *F. subglutinans*, *A. flavus*, *A. parasiticus*, and *Stenocarpella maydis* were also encountered. FB_1 concentrations in symptomless maize ranged between 22 and 1,348 μg/kg with mean levels of 56, 80, and 317 μg/kg, respectively, at 4, 8, and 12 weeks HTPAPM for Malava in the year 2001. In Tongaren during the same year, mean FB_1 levels of 41, 179, and 590 μg/kg were recorded at 4, 8, and 12 weeks HTPAPM, respectively. The concentration of FB_1 in rotten maize ranged from 39 to >5,000 μg/kg and increased with HTPAPM. The highest AFB_1 level was 17.0 μg/kg in rotten maize. The authors hinted that the isolation of *F. subglutinans* and *F. graminearum* was an indication that other mycotoxins (DON, ZEA, and moniliformin) associated with infertility and hypoestrogenism could be inevitable in the samples.

In a study scrutinizing commodities, feeds, and feed ingredients from Middle East and Africa [140], 48% (12/25) samples from Kenya were positive for B-trichothecenes (mean: 422 μg/kg; maximum: 3859 μg/kg), none had A-trichothecenes, 76% (19/25) had FUM (mean: 956 μg/kg; maximum 10,485 μg/kg), 56% (14/25) had ZEA (mean: 67 μg/kg; maximum: 167 μg/kg), 78% (21/27) had AFs (mean: 52 μg/kg; maximum: 556 μg/kg), while 50% (1/2) had ochratoxin A (mean: 2 μg/kg). A gluten sample from Kenya presented the highest level of FUM found in the whole survey (10,485 μg/kg).

Similarly, maize samples were collected from 30 markets in diverse agroecological zones of Meru, Machakos, and Kitui counties during the 2013 harvest [54]. *Fusarium* and *Aspergillus* spp were isolated from the samples. Total AFs in Meru, Kitui, and Machakos samples were beyond the threshold of 10 μg/kg. Meru had both the highest and lowest levels of AFs detected (115.7 μg/kg and 0.3 μg/kg, respectively). FUMs were reported in levels above the acceptable limits in Meru, and though detected in Kitui and Machakos, the contamination levels were within acceptable limits. Utilizing a near-infrared single kernel sorting machine, removal of AF and FUM-contaminated kernels was perfected with up to 97.8% efficacy for AFs and 60.8% for FUM. The accepted fractions had statistically lower mycotoxin levels than the rejected maize [54].

The prevalence of AFs and FUM was investigated in maize intended for immediate human consumption in Eastern Kenya. Samples were collected from people who brought their maize for processing at local commercial mills [75]. Interviews and sampling of maize flours were done for 1,500 people who processed maize at 143 mills in 10 administrative districts. Mycotoxin analysis revealed that 39% and 37% of the samples, respectively, had AFs and FUM in levels above tolerable limits. Samples with AFs above 10 μg/kg were 22–60% across the districts. A higher occurrence of AFs was associated with smaller maize farms, lower grain yield, and monocropping systems. A larger magnitude of the toxin was observed in the subhumid agroecological zone, in samples with more broken kernels and less maize ear damage at harvest. Further scrutiny of paired grain samples (visually sorted and unsorted) showed that sorting reduced FUM by 65% to below the advisory threshold of 1,000 μg/kg. Sorting did not, in essence, have any effect on AF concentration [75].

Besides, the presence of AFs, FUM, and DON in *Busaa* (a maize-based traditional beer) in Bomet County, Kenya, was reported [141]. Of the 61 samples obtained from homesteads involved in brewing in the northeastern part of Bomet East constituency, 93%, 9.8%, and 23%, respectively, were contaminated with AFs (mean: 5.2 ± 0.2 μg/kg; range: 2.8–11 μg/kg), FUM (mean 1460 ± 188 μg/kg; range: 280 to 4000 μg/kg), and DON (mean: 259 ± 5.2 μg/kg; range: 200–360 μg/kg). About 65.6% of these had AFs above the EU limit of 4 μg/kg, but FUM and DON concentrations were all within the tolerable limits of 4,000 μg/kg and 1,750 μg/kg, respectively. AFs & FUM, AFs & DON, and AFs, FUM, & DON co-occurred in 9.8%, 23%, and 3.3% of the samples, respectively [141].

Comparably, Mutiga et al. [72] evaluated AFs and FUM in maize from Western Kenya. The study covered 3 agroecological zones, taking samples of milled maize from 985 patrons of 26 hammer mills. AFs were detected in 49% of the

samples, with 15% of these being above 10 μg/kg. An estimated 65% of the samples from a drought-prone area were above acceptable limits. In Bungoma County, the authors assessed both AFs and FUM in four maize varieties at harvest and after 2 and 4 months of storage. For this, storage shed grain and milled samples were solicited. Mean AFs were identical for storage sheds and mills at 2.3 μg/kg. About 41% of the samples from mills had detectable AFs, 4% of which were above 10 μg/kg, while 87% had detectable FUM, with 50% above 1,000 μg/kg limit permitted in Kenya. Mean contamination levels did not vary during storage. As such, maize varieties reportedly differed in FUM contamination, with the most popular varieties spotted to be vulnerable to AFs, FUM, and weevils. It was concluded that thorough mycotoxin surveillance is vital for all parts of Kenya, irrespective of the past history of mycotoxin poisoning [72].

Samples of 74 animal feeds and 120 milk samples were simultaneously collected from individual cows and actors in the informal subvalue chains of rural and periurban dairy systems in Nakuru County, Kenya [142]. AFB_1 was detected in 56 % (41/74) of the feeds in levels above the EU limit of 5 μg/kg (range: BDL to 147.86 μg/kg) while DON was identified in 63% (27/43) of the feeds (range: BDL to 179.89 μg/kg). In the periurban dairy system, 48.5% (33/68) of the milk samples were contaminated with AFM_1 in levels exceeding EU threshold of 0.05 μg/kg (range: 0.017 to 0.083 μg/kg). Surprisingly, all milk samples from the rural dairy system had AFM_1 in levels below the EU limit of 0.05 μg/kg (range: BDL to 0.041 μg/kg). Linear regression depicted that there was a correlation between abiotic factors, viz., pH, water activity, and moisture content of feeds with AFB_1 and DON contamination.

Herbal preparations were sampled from Eldoret (14 liquid, 2 oil, and 34 powder samples) and Mombasa (12 liquid, 1 capsule, 3 oil, 6 tablets, and 28 powder samples) towns and analyzed for total AFs and FUMs [91]. Reported 32% of herbal products from Eldoret had AF levels less than 0.25 μg/kg, while 34% had AFs between 0.38 and 24 μg/kg. FUM occurred in very low concentrations in more than half of the samples. Samples drawn from Mombasa had AFs in levels lower than those from Eldoret, but the number of AF-contaminated samples was higher. About 32% of the samples had <0.25 μg/kg with 14 μg/kg being the highest. About 80% had <0.25 μg/kg, and the highest was >20 μg/kg. Six out of 14 (42.9%) liquid herbal samples from Eldoret were contaminated with AFs and 3 of the 6 were also contaminated with FUMs. All the 12 (100%) liquid samples taken from Mombasa were contaminated with both AFs and FUMs. A total of 27 out of 34 (79.4%) powders from Eldoret were contaminated, 23 with both mycotoxins and 4 with AFs only, while all the tablets (15 samples) and powders (19 samples) from Mombasa were contaminated with both mycotoxins; however, all capsules were free of mycotoxin contamination. All oily herbal samples ($n = 3$) from Mombasa were contaminated with both AFs and FUM, while only 1 oil sample from Eldoret was contaminated with FUM [91].

Into the bargain, a survey covering 116 push-pull and 139 non-push-pull cropping systems was conducted to determine the socioeconomic and agronomic factors that influence farmers' knowledge on incidence and contamination of maize by ear rots and associated mycotoxins in Siaya, Kakamega, Kisumu, Migori, and Vihiga counties of Western Kenya [143]. Data from smallholder farmers (23–80 years, 50% being female) were collected using questionnaires, and 10–20 maize cobs, depending on the size of cob, were collected from the standing crop in the field of each interviewed farmer and analyzed for AFs and FUMs. The authors reported that few farmers had knowledge of AFs and ear rots in maize. Overall, less than 20% of maize samples had AFs co-occurring with FUM, but more samples were contaminated with FUMs (range: 145.3–50,769.2 μg/kg) than AFs (range: BDL-242.3 μg/kg) with maize containing the mycotoxins in levels above permissible limits (10 μg/kg for AFs and 1,000 μg/kg for FUMs) being lower in samples from push-pull cropping system. Age of farmer and county of residence were significantly and positively associated with knowledge of AFs on the one hand. On the other hand, cropping system, county of residence, and level of education were positively associated with knowledge of maize ear rots. In addition, a strong correlation between knowledge of maize ear rots and knowledge of AFs was witnessed. The concentration of the mycotoxins was significantly and positively associated with the use of diammonium phosphate fertilizer at planting. AF levels were also positively associated with stem borer pest damage, though agronomic practices were not ideally different between push-pull and non-push-pull farmers [143].

2.4. Geographical Distribution of Aflatoxins in Kenya. Kenya was one of the hotspots of AFs first recorded [15, 144] with countries such as Uganda and Brazil [26, 145]. Kenya is partitioned into about 7 agroecological zones: humid, subhumid, transitional, temperate, semiarid, arid, and per-arid [146]. AFs tend to be detected in samples from all the different zones [105, 110, 130]. This can be attributed to the similarity in the agronomic and pre-, peri-, and postharvest handling practices and the interregional marketing of foods [61, 92, 111, 147]. However, the eastern part of the country is more aflatoxin-prone, possesses the most toxigenic *Aspergillus* spp, and has been the epicenter of aflatoxicoses recorded in Kenya [46]. Eastern Kenya experiences hotter and drier climatic conditions in comparison with Western Kenya. For this reason, it received characterization as semihumid to semiarid while Western Kenya is classified as subhumid to semihumid agroecological zone [110]. Environmental conditions have been demonstrated to influence the ability of *Aspergillus* fungi to infect, colonize, and survive on crops as well as produce mycotoxins. Further, fluctuations in these conditions also affect the quantities as well as community compositions of aflatoxin-producing fungi [148]. The prevalence of AFs in Eastern Kenya therefore is in

congruence with a previous emphasis that mycotoxin infectivity is always multifactorial, but climate is the most important [149].

3. Capacity for Detection and Quantification

Detection and quantification of AFs are key to their mitigation because their distribution in samples is often skewed [150]. The first step for accurate detection and quantification of AFs is sampling, i.e., sampling/subsampling is the largest source of error in AFs analysis [151]. For this reason, a representative sample ought to be drawn from the sample lot. For the over 50 KEBS listed laboratories for monitoring mycotoxins in foods, Gafta methods (No. 130, 24:1) and EAS 79 are used as the sampling protocols. However, some clients do the sampling themselves, in which case the testing laboratories do not question the actual reason for sampling, or where and how the samples were taken. In addition, data from such analyses are always confidential, which do not enhance evidence-based decision making by policymakers [97].

Another emerging challenge in analyses of food toxins in Africa, Asia, America, and Europe is "masked mycotoxins" as they are not often identified and detected by the usual analytical techniques [152]. Masked (matrix-associated) mycotoxins are those that are biosynthesized by the toxigenic fungi and later undergo biomodification by plant enzymes during the infection stages. They may be housed in the vacuoles in the soluble form or bound to macromolecules and thus remain undetectable [153]. Unfortunately, these modified toxins can hydrolyze and revert back into their toxic forms during processing or digestion [154–156]. A way to circumvent this analytical problem has been to hydrolyze the modified forms (using enzymes, alkaline, or acidic pretreatments) [157–159] into their free forms which can then be detected [157, 160]. For this reason, there is a paucity of data on masked AFs as usually detection and quantification are done for free AFs in matrices.

The methods for the detection of AFs used by studies in Kenya are outlined in Table 7. On the whole, AF research in Kenya used laboratory-based enzyme-linked immunosorbent assays (ELISAs), high-performance liquid chromatography (HPLC), thin-layer chromatography (TLC), fluorimetry, liquid chromatography-tandem mass spectrometry (LC-MS/MS), tandem quadrupole mass spectrometry (TQMS), and ultra-high-pressure liquid chromatography (UHPLC). Lateral flow immunochromatography (LFI) has also been used. There has been a shift in instrumentation for AF analysis, as evidenced by advancement from nondifferential TLC in 1973 to relatively fast and differential UHPLC, hyphenated with triple quadruple mass spectrometry (UHPLC-TTQS) in 2017–2020. Overall, the most employed method has been ELISA, which itself has undergone several advancements in the past few years. This could be because it is practically inexpensive, easy to use, is highly sensitive for routine analysis of food products, demands minimum sample clean up, and poses no inherent health hazards as it uses enzyme labels. In addition, concurrent analysis of several samples on a 96-well assay platform is possible, and thus, it has a high sample throughput with low sample volume requirement which offers obvious advantages [28]. In addition, ELISA has lower detection limits than most instrumental techniques which are used for AF determination [28].

However, the drawbacks of the foregoing standard methods are that they are unsuitable for rapid and real-time applications in food and feed sample analyses as they are relatively tedious and require technical know-how to operate. Rapid and robust methods such as polymerase chain reaction (PCR) and nondestructive methods based on fluorescence/near-infrared spectroscopy (FS/NIRS) and hyperspectral imaging (HSI) have emerged for quick and easy detection of AFs [161]. Some studies in Kenya [41–43, 46, 47, 110, 162] have utilized PCR in their analyses. It is of interest to note that, at industrial level, agroprocessing entities monitor grain total AFs utilizing single-step lateral flow immunoassays utilizing Reveal Q+ test strips that are developed and read on AccuScan Gold readers [163]. The bright greenish-yellow fluorescence (BGYF) or the black light test, which can aptly identify commodities presumed to be contaminated with AFs, has been reported in Kenya [54]. This test is relatively cheap and simple especially for detecting AFs in maize where kernels are viewed under an ultraviolet lamp at 365 nm for characteristic bright greenish-yellow fluorescence which indicates a possible presence of aflatoxigenic fungi or the mycotoxin itself [164]. This could be adopted by regulatory agencies for the purpose of AFs surveillance.

4. Exposure Assessment

4.1. Exposure to Aflatoxins in Kenya. Exposure to AFs occurs via periodic ingestion of contaminated plant or animal products such as meat, eggs, blood, and milk of livestock previously served AF-contaminated rations [20]. Farmers and their workers may also inhale dust generated during the processing of contaminated crops and feeds or the toxins may permeate through their skin [165, 166]. Exposure to AFs such as AFM_1 may also be through their endogenous production [32]. In point of fact, AFs are known to cross the placenta, so that exposure to them may start *in utero* and continues in the postnatal period through breastfeeding [92, 167–169].

It is now established that detecting and quantifying food AF levels are not always adequately reflective of the extent of exposure because the quantities in foods are not directly the same as those ingested. For this reason, epidemiological biomarkers are often used to assess exposure. Biomarkers are quite exact in evaluating the magnitude of AF exposure because of their nonsubjectivity and faculty that allows the estimation of internal and biologically effective doses. Popular AF biomarkers are urinary AF-N^7-guanine (for assessing previous day's exposure) and breast milk AFM_1 which indicate exposure levels in the past 24 hours and plasma/serum aflatoxin-albumin (AF-alb) adduct with a half-life of about 2 months enhancing the evaluation of chronic and routine exposure [170]. Albumin is the sole serum protein that can get bound to AFB_1, yielding

Table 7: Analytical methods used by aflatoxin investigations in Kenya.

Methods of analysis	Samples/matrices	Mycotoxins analyzed	Years	Authors
ELISA	Maize grain	Total AFs	2020	Marete et al. [178]
Fluorimetry, PCR	Soil	Total AFs	2020	Monda et al. [46]
UHPLC	Maize grain (fresh), maize flour	AFB_1, AFG_1, AFB_2, AFG_2	2020	Nabwire et al. [77]
UPLC, PCR	Maize grain	AFB_1, AFG_1, AFB_2, AFG_2	2019	Oloo et al. [42]
Quantitative PCR (qPCR), TLC, HPLC	Maize tissues/grain	*A. flavus* biomass, AFB_1, AFG_1, AFB_2, AFG_2	2019	Mitema et al, [43]
ELISA	Bovine milk	AFM_1	2019	Kagera et al. [129]
ELISA	Maize	AFB_1	2019	Mahuku et al. [94]
ELISA	Maize	Total AFs, FUM	2019	Njeru et al. [143]
ELISA	Bovine milk	AFM_1	2019	Kuboka et al. [135]
PCR	Soils	*A. flavus* genotyping	2018	Islam et al. [47]
LC-MS/MS, UHPLC-TTQS, PCR	Maize samples	AFB_1, AFG_1, AFB_2, AFG_2, *Aspergillus* spp genotyping	2018	Okoth et al. [48]
PCR, HPLC	*Kimere* (a fermented milk product)	AFB_1	2018	Nduti [179]
LFI	Maize grain, human sera (children)	Total AFs, AFB_1 (lysine adducts)	2018	Hoffmann et al. [180]
ELISA	Raw, pasteurized and UHT milk, yoghurt, *Lala*	AFM_1	2018	Lindahl et al. [90]
ELISA, TLC, HPLC	Maize grain	Total AFs, AFB_1	2018	Obonyo and Salano [63]
ELISA, PCR	Maize kernels	Total AFs	2018	Gachara et al. [110]
ELISA, LC-HRMS/MS	Fish feeds	Total AFs	2018	Mwihia et al. [73]
ELISA, HPLC	Urine, breast milk, maize flour, sorghum, millet	AFM_1	2017	Kang'ethe et al. [169]
LFI	Maize grain and maize flour	Total AFs	2017	Nduti et al. [95]
ELISA	Herbal products	Total AFs, FUMs	2017	Keter et al. [91]
HPLC, UPLC-MS/MS, LC-MS/MS	Human urine, human blood	AFM_1, AFB_1 (lysine adducts)	2017	Awuor et al. [181]
ELISA	Dairy cattle feeds, bovine milk	AFB_1, AFM_1	2016	Ochungo et al. [58]
ELISA	Bovine milk	AFM_1	2016	Kirino et al. [131]
ELISA	Animal feeds and bovine milk	AFB_1, DON, and AFM_1	2016	Makau et al. [142]
ELISA, HPLC, LC/MS	Maize grain, urine	AFB_1, AFM_1	2016	Nduti et al. [182]
ELISA	Dairy cattle concentrates, bovine milk	AFB_1, AFM_1	2016	Senerwa et al. [133]
ELISA	Bovine milk (raw and processed), dairy products	AFM_1	2016	Langat et al. [132]
ELISA	Maize, sorghum, and milk	Total AFs and AFM_1	2016	Kiarie et al. [66]
HPLC	Peanuts	Total AFs	2016	Menza et al. [121]
ELISA	Maize grain	Total AFs	2016	Kirui [183]
ELISA	Cassava (chips and flour)	Total AFs	2015	Gacheru et al. [126]
ELISA	*Omena*, maize, sorghum, rice, peanuts, cassava	AFB_1, AFM_1	2015	Obade et al. [80]
ELISA	Maize (grain and flour)	Total AFs, FUM	2015	Mutiga et al. [72]
ELISA	Maize, sorghum, millet	Total AFs	2015	Sirma et al. [76]
HPLC	Human sera (women)	AFB_1 (lysine adducts)	2015	Leroy et al. [60]
ELISA, qPCR	Human sera (children)	AFB_1 (albumin adducts)	2015	Castelino et al. [162]
TLC, HPLC	Fresh and sun-dried fish (*Rastrineobola argentea*)	Total AFs	2015	Orony et al. [85]
ELISA	Cattle feeds, rice, maize, peanuts	Total AFs	2014	Nyangaga [86]
ELISA	Maize grain	Total AFs, FUM	2014	Mutiga et al. [75]
TLC, HPLC	Maize grains, *githeri*, *muthokoi*	Total AFs	2014	Kilonzo et al. [109]
ELISA	Bovine milk	AFM_1	2014	Sirma et al. [130]
ELISA, BGYF	Maize grain	Total AFs, FUM	2014	Murithi [54]
LFI	*Busaa* (a local brew)	Total AFs, FUM, DON	2014	Kirui et al. [141]
ELISA	Peanuts (raw and roasted)	Total AFs	2013	Nyirahakizimana et al. [115]
ELISA	Peanuts	Total AFs	2013	Mutegi et al. [64]
ELISA	Peanuts and peanut products	Total AFs	2013	Mutegi et al. [62]
ELISA	Peanuts (raw and roasted), peanut butter	Total AFs	2013	Ndung'u et al. [53]
TQMS	Human sera	AFB_1 (lysine adducts)	2013	Yard et al. [184]
ELISA	Peanuts	Total AFs	2012	Mutegi et al. [44]
LC-MS/MS, PCR	Maize kernels	AFB_1, AFG_1, AFB_2, AFG_2	2012	Okoth et al. [41]

Table 7: Continued.

Methods of analysis	Samples/matrices	Mycotoxins analyzed	Years	Authors
TLC	Maize (grains, flour), milled maize-cereal products, dairy cattle feed, oil seed cake	Total AFs	2012	Okoth and Kola [78]
ELISA	Human plasma (children)	AFB_1 (albumin adducts)	2012	Gong et al. [185]
ELISA	Maize (grains, flour, semiprocessed), soil, mill dust	Total AFs	2012	Muthomi et al. [107]
Fluorimetry	Maize grain	Total AFs	2011	Daniel et al. [70]
LC-MS, HPLC	Commodities, feeds, and feed ingredients	Total AFs, FUM, ZEA, trichothecenes (A&B), ochratoxin A	2011	Rodrigues et al. [140]
ELISA	Ground maize, soil	Total AFs	2010	Probst et al. [55]
ELISA	Milk, animal feeds	AFM_1, AFB_1	2009	Kang'ethe and Lang'a [89]
ELISA	Maize, soils, mill dust	AFB_1	2009	Muthomi et al. [40]
ELISA	Peanuts	Total AFs	2009	Mutegi et al. [61]
ELISA	Maize grain	AFB_1, FB_1	2009	Alakonya et al. [139]
HPLC/fluorimetry	Maize grain	AFB_1	2007	Probst et al. [39]
ELISA	Peanuts	Total AFs	2007	Mutegi et al. [65]
HPLC	Maize kernels, maize flour, *muthokoi*	Total AFs	2005	Lewis et al. [71]
Fluorimetry	Maize grain	Total AFs	2005	Azziz-Baumgartner et al. [69]
Fluorimetry	Maize grain and maize products	Total AFs	2005	Muture and Ogana [103]
ELISA	Pilsner and Tusker beers	AFB_1, FB_1, DON, ZEA	2004	Mbugua and Gathumbi [138]
TLC	Peanuts	Total AFs	2004	Gachomo et al. [84]
TLC	Weaning foods	Total AFs	2004	Okoth and Ohingo [118]
TLC, HPLC	Malted millet, maize flour	AFB_1, AFB_2	2000	Kenji et al. [81]
ELISA, HPLC-FS	Human sera	AFB_1 (lysine adducts)	1990	Wild et al. [186]
HPLC	Breast milk, human sera, neonatal cord blood, blood (pregnant women)	AFB_1, AFB_2, AFG_1, AFG_2, AFM_1, AFM_2, aflatoxicol	1989	Maxwell et al. [187]
HPLC	Human urine	AFB_1 (guanine adduct)	1987	Autrup et al. [188]
TLC	Local beer, food (maize, millet, sorghum, pigeon peas, and yam components)	Total AFs	1973	Peers and Linsell [82]

Years are those in which the data joined scientific literature with most data gathered and analyzed in more than 3 months before publication. *Muthokoi* are maize kernels with the outer hull removed. *Lala* also called *Maziwa Lala* or *Mala* is a locally fermented milk product. *Busaa* is a sociocultural maize-based traditional brew mostly consumed during events such as male circumcisions, weddings, and funerals, made from raw maize flour and semiground finger millet malt [141]. LC-HRMS/MS: liquid chromatography high-resolution mass spectrometry. LFI: Hoffmann et al. [180] used Romer AgraStrip rapid test; Kirui et al. [141] used Envirologix Quick Tox kits.

biomarker adducts [171]. Human blood and urine AF-alb can be used to determine the biologically effective dose of ingested AFB_1. Though both AFG_1 and AFB_1 can covalently interact with albumin to form AF-8, 9-epoxide [98], the adduct levels are taken to reflect the amount of the latter as the former is infrequently encountered in foods [172]. The AF-alb adduct is the most frequently utilized AF biomarker because it is easily detectable using ELISA (Table 7) [173]. Additionally, the determination of AFB_1-Lys levels in proteolytic digests of serum with LC-MS/MS and HPLC-FS has recently been appraised [174, 175].

Biopsy material was first utilized in 1967 to illustrate that the Kamba ethnic community of Kenya had a frequency of liver cancer that was doubling that of the Kikuyus [176]. This is partly supported by the fact that subsequent aflatoxicoses were witnessed in Eastern Kenya where the Kamba are the main inhabitants [71, 177]. A dietary AF-liver cancer study in Murang'a district of Kenya reassessed the correlation between AF and the disease incidence rates based on a total of 7 years of cancer registration [82]. The results of the study were however interpreted in combination with a study later done in Swaziland. With consideration of males and females separately, the pooled results of the studies hinted that there was a high degree of positive correlation between the calculated ingestion levels of AFs (X) and the adult incidences of hepatocellular carcinoma (Y) for the two studied populations and for both males and females. With the assumption of a wet intake diet of 2 kg/day and a mean body weight of 70 kg, the relationship for adult females was $Y = 4.14 \log_{10} X - 0.80$. With a further assumption of a daily intake of native beer of 2 liters/day, the regression equation for adult males was $Y = 21.96 \log_{10} X - 11.17$. The regression data were reported to corroborate those reported by previous researchers [82].

To validate the assertion, another team [188] evaluated if there were any synergistic effects of hepatitis B virus and AFB_1 on the incidences of hepatocellular cancer. The study encompassed various parts of Kenya with different liver

cancer incidences so as to establish the rate of exposure to AFs and the prevalence of hepatitis infections. It turned out that of all the tested participants, 12.6% were positive for AF exposure as shown by urinary excretion of AF-N^7-guanine adduct and the highest exposure to the toxins was in the Western Highlands and Central Province. The incidence of hepatitis infection nationwide as measured by the presence of the surface antigens was 10.6% with a marked regional variation. Execution of multiplicative and additive regression analysis suggested that the two were not a synergetic combination in the etiology of liver cancer, though a moderate degree of correlation between AF exposure and liver cancer was observed when the study was limited to certain ethnic groups [188].

Further, Maxwell et al. [187] undertook a study in Kenya, Sudan, Ghana, and Nigeria to evaluate the extent of AF exposure by breastfed infants and to investigate the possibility that AFs cross the human placental membrane. In this study, breast milk, cord blood, and maternal blood were analyzed for AFs which were detected in 28% of 191 Kenyan, 37% of 99 Sudanese, and 34% of 510 Ghanaian breast milk samples (Table 8). Blood drawn from 101 babies in Kenya, 282 babies in Ghana, and 78 babies in Nigeria had AFs in 37%, 31%, and 12% of the samples, respectively. In Kenya, the rate of detection was higher in the wet season (52%) than in the dry season (23%). Maternal blood sampled at delivery in 83 Kenyan cases and 77 Nigerian cases recorded AFs in both maternal and cord blood specimens in 14 Kenyan and 7 Nigerian instances. These confirmed that infantile exposure to AFs occurs and demonstrated the ability of AFs to cross the human placental membrane [187].

Differently, a survey which recruited adults from Kenya, Thailand, The Gambia, and France was used to validate the measurement of AF-albumin adducts by three methods [186]. Levels of 7 to 338 pg AF/mg alb were observed in the first three countries while no adducts were detected in samples from France. Another cross-sectional serosurvey in Kenya confirmed regional influence on AF exposure patterns [184]. Randomly selected 600 serum specimens stratified by province from a 2007 Kenya AIDS Indicator Survey were analyzed for AFs. About 78% of the sampled group had exposure to AFs and this varied by province. The highest were in Eastern (median = 7.87 pg/mg alb) and Coast (median = 3.70 pg/mg alb) provinces, while Nyanza (median ≤limit of detection) and Rift Valley (median = 0.70 pg/mg alb) provinces recorded the lowest exposures. According to the authors, age group, sex, marital status, religion, and socioeconomic characteristics did not influence exposure.

In another study, random samples of weaning flours were obtained from 242 households with 3- to 36-month-old children (43.6% males and 53.4% females) in Kisumu district, Kenya, and analyzed for AFs [118]. The types of weaning foods, handling, and storage of the foods were captured. The nutritional status of the children was also determined along with heights and lengths. About 29% (70/242) of the samples were positive for total AFs (range: 2–82 μg/kg). Malnutrition was 34% for stunting, 30% for underweight, and 6% for wasting. About 53.8% of the wasted children were being fed on AF-contaminated weaner flour vis-à-vis 27.7% of the normal children. The contaminated flours ($n = 70$) were being stored in plastic containers (63%), polyethylene bags (20.4%), metal buckets (3.7%), manila sacks (1.9%), earthen pots (1.9%), and reed baskets (7.4%) for 1 day to 2-3 weeks. These all had effects on aflatoxigenic contamination as those with AFs had higher mean moisture content (13.6%) than those devoid of AFs (12.5%). *Aspergillus* spp (including *A. flavus* and *A. parasiticus*), *Fusarium, Mucor, Rhizopus nigricans*, *Trichoderma viride*, and *Candida* spp were isolated from the flour samples [118].

Agreeably, Leroy et al. [60] collected socioeconomic data to quantify the extent to which socioeconomic characteristics could explain the differences in serum AFB_1-lysine adduct levels in 100 women from the Eastern province of Kenya. The correlation between serum AFB_1 level and a number of households, farm, and individual characteristics was assessed for 884 mothers (pregnant or with a child under 24 months). AF was detected in all women with a median level of 7.47 pg/mg alb. Higher exposure levels were correlated with poverty: predicted serum AF levels in women living in the worst socioeconomic conditions were 4.7–7.1 times higher than those with the best socioeconomic status.

Further, samples of *Rastrineobola argentea* ($n = 50$), polished rice ($n = 31$), peanuts ($n = 22$), cassava ($n = 37$), maize ($n = 41$), and sorghum ($n = 28$) were collected from Kibuye wholesale, Kibuye open-air, Ahero, Oile, and Mamboleo markets in Kisumu County and analyzed for AFs [80]. Processed bovine milk samples ($n = 50$) were collected from supermarkets along with raw bovine milk samples ($n = 30$) from 3 market milk bazaars in the same markets. Analytical results indicated that AFs ranged from 0 to 34.5 μg/kg AFB_1 in the solid foods, 0.012 to 0.127 μg/kg AFM_1 in processed milk, and 0.0002 to 0.013 μg/kg AFM_1 in raw milk. Only cassava among the scrutinized food items had detectable AFs below the regulatory limit of 10 μg/kg AFB_1 by then. Daily AF consumption ranged from 4.43 ng/kg bw/day in a combination of maize flour and milk to 110.4 ng/kg bw/day in a combination of sorghum and raw milk for 6-month-old children (average weight: 7.9 kg) with a daily consumption of 60 g of mixed cereal flour and 500 ml of milk per day. These results emphasized that weaning children in Kisumu County are chronically exposed to high AF levels for the fact that the analyzed food items are common ingredients of weaning foods in the area [80]. In addition, the calculated AF consumption of 0.6 ng/kg bw/day for a child at 6 months weighing 7.9 kg was higher than that indicated by the Codex Alimentarius Committee (0.1 ng/person/day) AFM_1 through milk for the Africa region. The weighted mean concentration of 0.05 μg AFM_1 in milk and a consumption of 0.25 ng/kg bw/day have been associated with a prevalence of between 3.2 and 20 cancer cases/year/10^6 [189]. The exposure was much higher than estimated because most children in Kenya are breastfed until at least the latter part of the second year and yet they begin to receive cereal-based gruel before the age of 3 months [190]. Further, the results of the study corroborated a previous report which estimated that about 40% of foods from farmers in the Nyanza province had AF levels above the statutory limit of 10 μg/kg [65].

Table 8: AF content of breast milk and cord blood from Kenya, Sudan, Ghana, and Nigeria.

Sample	Country	Number of samples	Number of AF-positive samples	Positive samples (%)
Breast milk	Kenya	191	53	28
	Sudan	99	37	37
	Ghana	510	163	32
Cord blood	Kenya	101	37	37
	Nigeria	78	9	12
	Ghana	282	86	30.5

Adapted from [187]. AFM_1 was detected in 121 milk samples (range: 5–1379 ng/L), AFM_2 in 103 (range: 3–6368 ng/L), AFB_1 in 41 (range: 150–55,792 ng/L), AFB_2 in 10 (range: 49–623 ng/L), AFG_1 in 4 (range: 1890–5180 ng/L), AFG_2 in 3 (range: 10–87 ng/L), and aflatoxicol in 6 (range: 14–270 ng/L). In cord blood, AFM_1 (range: 25–8942 ng/L) and AFM_2 (range: 15–732 ng/L) were detected frequently in 63 and 47 samples; AFB_1 (range: 185–43822 ng/L) and AFB_2 (range: 10–925 ng/L) were detected in 20 and 19 samples. AFG_1 was detected 4 times (range: 611–2086 ng/L), AFG_2 once (37 ng/L) and aflatoxicol thrice (177, 214, & 280 ng/L).

Another cross-sectional study was undertaken involving 204 low-income households randomly selected in two low-income areas (Korogocho and Dagoretti), Nairobi, Kenya [66]. Demographics, a 24-hour dietary recall, and anthropometric measurements were conducted in children aged 1–3 years. Height-for-age Z-scores (HAZ), weight-for-age Z-scores (WAZ), and weight-for-height Z-scores (WHZ) were calculated for each child using the WHO growth standard reference data. Maize ($n = 99$ and 87), sorghum ($n = 53$ and 36), and milk ($n = 76$ and 52) samples from the households or retailers from Korogocho and Dagoretti, respectively, were analyzed for total AFs and AFM_1. As a whole, 98% of food samples collected were AF positive (maize: mean: 6.7, range: 0.0–88.83; sorghum: mean: 8.07, range: 0.1–194.41; milk: mean: 0.132, range: 0.007–2.56 for samples from Korogocho; maize: mean: 2.97, range: 0.0–20.0; sorghum: mean: 2.59, range: 0.2–14.47; milk: mean: 0.093, range: 0.002–0.64 for samples from Dagoretti). About 41% of the children had stunted growth; boys were more stunted than girls ($p = 0.057$), and Korogocho had more stunted children than Dagoretti ($p = 0.041$). The average AF exposure was 21.3 ng/kg bw/day. Exposure to AFM_1, location, and sex was significantly associated with HAZ, with boys and children from Korogocho having lower HAZ, and AFM_1 was negatively associated with HAZ ($p = 0.047$), suggesting that AFM_1 was associated with stunting. No correlation was statistically found between total AFs and HAZ, WAZ, and WHZ. The authors reiterated that there was a high prevalence of malnutrition (stunting) in the studied low-income urban sites, and this was most pronounced in the high-density area. It was stressed that the association between AFM_1 and growth impairment warranted further investigations [66].

Kang'ethe et al. [169] reported that, with a maize consumption of 0.1 to 0.25 kg/person/day in Nandi and Makueni counties, an AF exposure rate of 0.011 and 0.49 μg/kg bw/day, respectively, was recorded in children younger than 5 years. Exposure to AFM_1 through milk consumption in this study was 4×10^{-4} and 1×10^{-4} μg/kg bw/day, respectively. Breast milk nursed children on the other hand had exposure of 6×10^{-3} and 1×10^{-6} μg/kg bw/day in Makueni and Nandi, respectively. Children younger than 30 months in Makueni had 1.4 times higher levels of AFM_1 in urine than those of the same age in Nandi. The stunting and severe stunting rates in Makueni and Nandi were 28.7% and 18.5%, and 30.7% and 16.5%, respectively.

In a recent study [180] which enrolled 1230 unborn children, 881 (72%) were included in LAZ and 798 (65%) in the serum AFB_1 analysis. A cluster randomised controlled design was used (28 intervention and 28 control clusters). The intervention arm received a swapping (contaminated maize was replaced with safe maize) and a stockist intervention (households were encouraged to purchase from a stockist supplied with clean maize). Women in the fifth to the final month of pregnancy were invited to enroll in the study. Outcomes were child LAZ, the prevalence of stunting, and child serum AFB_1-lysine adduct level 24 (end-line, primary outcomes) and 11 to 19 months (midline, secondary outcomes) after trial commencement, respectively. The intervention was reported to considerably reduce end-line ln serum AFB_1-lysine adduct levels (intervention effect was 0.273, 95% CI 0.547 to 0.001; one-sided $p = 0.025$) but had no effect on end-line LAZ or stunting (mean LAZ at end-line was −1.64). At midline, the intervention increased LAZ by 0.16 (95% CI −0.009 to 0.33; one-sided $p = 0.032$) and reduced stunting by 7% points (95% CI −0.125 to −0.007; one-sided $p = 0.015$) but had no effect on serum AFB_1 levels [180]. It was inferred that the midline analysis suggested that AFs may affect linear growth at younger ages.

Overall average estimation of exposure rates based on annual consumption, as is appropriate for cancer risk because of the cumulative nature of this response, indicates that AF exposure was 3.5 to 14.8 ng/kg/day in Kenya for about 67% of the population [92, 191]. No study in Kenya has examined the relationship between AFM_1 in breast milk samples and growth impairment in infants.

4.2. Coexposure to Aflatoxins with Other Mycotoxins. Aflatoxin poisoning could be compounded by the occurrence of AFs in combination with other mycotoxins such as FUM, trichothecenes, ochratoxins, ZEA, and DON [16, 192, 193]. This is supported by the occurrence of mycotoxin producing fungi simultaneously in the same batch of food/matrix and the faculty of some toxigenic fungi to produce more than one mycotoxin in a given matrix. For example, *Fusarium* (*F. verticillioides*, *F. proliferatum*, and *F. oxysporum*) [54] and *Penicillium* spp were reported with

Aspergillus fungi in Kenya [41, 45, 53, 84, 115, 194, 195], sometimes in soils and mill dust around maize stores [40]. *Fusarium* spp are known for the production of FUMs [196].

The current review did not identify any reports evaluating coexposure to AFs in combination with other mycotoxins and the potential adverse health outcomes. There have been developments in both mycotoxin-specific and multimycotoxin methods developed for biological matrices [197]. For example, mycotoxin-specific biomarkers for FUM in maize and DON in wheat have been developed and validated [198, 199], though none has been applied in Kenya. In neighbouring Tanzania, coexposure to AFs with other mycotoxins utilizing individual biomarkers was recently investigated. Children (6–14 months old) were recruited at a maize harvest season and followed up twice at 6-month intervals. The children were reported to be chronically exposed to AFB_1, FB_1, and DON [200, 201]. Blood AF-alb [200] and urinary DON levels [201] steadily increased over the 12 months, which likely corresponded to increased food intake that is possible as the child grows. A linear trend was not apparent for urinary FB_1 as the mean level at 6 months was significantly lower than mean levels at recruitment and at 12 months [200]. It was deduced that the lower exposure levels observed 6 months postharvest could be reflective of reduced maize stocks, resulting in lower maize consumption [200]. Though no significant correlation was appreciated between AF exposure and stunted child growth, increased FUM exposure was evidently associated with reduced length-for-age Z-scores [200].

In addition, coexposure to mycotoxins *in utero* is also wanting, as observations elsewhere reported AF-alb in 36% of the blood samples with urinary AFM_1 and DON present in 47% and 68% of samples from pregnant women in their third trimester coexposed to AFs and DON [202]. About 41% of the pregnant women were concurrently exposed to both AFs and DON. Thus, assessment of coexposure to AFs in Kenya with other mycotoxins is warranted.

5. Infantile Stunting due to Aflatoxin Exposure in Kenya

The first 1,000 days of life (from conception to about 36 months) is a critical window for healthy growth and development. Dietary intake of AFs during pregnancy plays a fundamental role in the child's future health status [187, 197]. In sub-Saharan Africa, and particularly Kenya, malnutrition and child growth impairment are major public health burdens [80, 118, 180]. Intake of low, daily doses of AFs over long periods result in chronic aflatoxicosis expressed as impaired food conversion, stunting in children, immunosuppression, cancer, and reduced life expectancy [6, 203–205]. The WHO defined stunting as a height-for-age Z-score (HAZ), of <−2, being underweight as a weight-for-age Z-score (WAZ), of <−2, and wasting as a weight-for-height Z-score (WHZ), of <−2 [206]. Stunting of infants in some aflatoxin-prone areas of Kenya is shown in Table 9.

It was advanced that AF exposure may disrupt the insulin-like growth factor (IGF) pathway through liver toxicity. In a study in Kenya [162], AF-alb concentrations were inversely associated with IGF1 levels ($p = 0.039$) and IGF binding protein 3 levels ($p = 0.046$) in a sample of 199 school children from Yumbuni in the west and Matangini (Lower Mangalete) in the east. A path analysis showed that lower IGF1 levels explained about 16% of the effect of AFs on child height ($p = 0.052$). Both IGF1 and IGFBP3 were significantly associated with child height and weight ($p < 0.01$). Children in the highest tertile of AF-alb exposure (>198.5 pg/mg) were shorter than those in the lowest tertile (<74.5 pg/mg), after adjusting for confounders ($p = 0.043$). To further investigate this putative mechanistic pathway, human hepatocyte line 16 (HHL-16) cells were treated with AFB_1 at 0.5, 5.0, and 20.0 μg/mL for 24–48 hours. IGF1 and IGFBP3 gene expression measured by quantitative PCR and protein in culture media showed a significant down-regulation of IGF genes and reduced IGF protein levels. The study concluded that AF-induced changes in IGF protein levels could contribute to growth impairment where AF exposure is high [162].

Aflatoxin-child growth impairment may be explained by the immunosuppressive effect of AFs that aggravates neonatal infection susceptibility, thereby impairing nutritional status through inappetence and diminished nutrient absorption [207]. It is also argued that exposure to AFs may initiate intestinal damages by dysregulation of protein synthesis culminating in diminished nutrient absorption and impaired growth [208]. AFs have also been implicated in the etiology of other liver diseases including jaundice, cirrhosis, and hepatomegaly [185, 209, 210]. A study in Kenya by Gong et al. [185] reported that the prevalence of hepatomegaly, a firm form of liver enlargement, increased in children with higher AF exposure. This is in complete agreement with the knowledge that the liver is the target metabolic organ for AFs.

6. Aflatoxicoses in Kenya

Since the discovery of AFs, Kenya has been one of the countries with devastatingly severe human exposure to AFs [109, 211, 212]. Exposure to AFs is chiefly via intake of contaminated food. Ingestion, however at concentrations greater than 6000 mg/kg in food, degenerates into liver failure and can be lethal after 1-2 weeks of exposure (acute aflatoxicosis) [213]. Aflatoxicosis is typified by oedema, convulsions, vomiting, jaundice, abdominal pain, sudden liver failure, and lastly death [214]. In practice, acute toxicities associated with exposure to elevated AF levels are not very common globally; cases occur and are concentrated in high-risk regions such as the Makueni County of Kenya [77] (Table 10). In humans, acute toxicity due to exposure to high dietary doses of AFs (2,000–6,000 μg/day) in contaminated maize was reported in Western India in 1974 with a case fatality rate of 10% [215, 216]. In Taiwan, 26 members of 3 families were victims of consumption of about 200 μg/kg of AFs in mouldy rice. Three of the victims died [217]. In neighbouring Uganda, a 15-year-old boy also succumbed to death following ingestion of cassava containing 1,700 μg/kg of AFs, leaving behind a brother and a sister who survived very narrowly [218]. Recently, consumption of AF-

Table 9: Aflatoxin levels in foods and stunting in some aflatoxin hotspots of Kenya.

County	Stunting (%)	Highest reported AF levels in foods (μg/kg)	Authors
Urban Nairobi	22.7	4,593.93 (maize and maize products), total AFs	Okoth and Kola [78]
Nairobi (Korogocho and Dagoretti)	41.0	88.83 (maize), 194.41 (sorghum), total AFs	Kiarie et al. [66]
Kisumu	33.1	82.0 (cereal-based weaning foods), total AFs	Okoth and Ohingo [118]
Homa Bay	37.0	1,000 (peanuts), total AFs	Mutegi et al. [65]
Makueni	33.5	5,400 (maize), total AFs	Lewis et al. [71]
Kitui	47.4	25,000 (maize), total AFs	Lewis et al. [71]
Machakos	31.3	3,800 (maize), total AFs	Lewis et al. [71]
Embu	23.7	21.0, total AFs	Collins et al. [106]
Kakamega (Malava)	34.2	17.0 (rotten maize), AFB_1; FB_1 >5,000 μg/kg	Alakonya et al. [139]
Tongaren (Bungoma)	52.1	17.0 (rotten maize), AFB_1; FB_1 was >5,000 μg/kg	Alakonya et al. [139]
Kisii South	35.3	3,442; total AFs	Collins et al. [106]

Adapted from Obade et al. [80].

Table 10: Aflatoxicosis outbreaks reported in Kenya since the discovery of aflatoxins in 1960.

Affected group	Case-patients/ number affected	Area	Toxin source	Recorded effects	Years	Authors
Humans, dogs	None confirmed	Eastern Kenya (29 districts)	Suspected contaminated maize	Price spiral down, grain trade breakdown, unconfirmed dog deaths in Nairobi	2010	Muthomi et al. [236]
Humans	5	Kibwezi, Kajiado, Mutomo	Maize	3 hospitalized, 2 deaths	2008	Muthomi et al. [40]
Humans	4	Kibwezi, Makueni	Maize	2 deaths in Makindu town of Mukueni County	2007	Wagacha and Muthomi [3]
Humans	20	Makueni, Kitui, Machakos, Mutomo	Contaminated maize	Acute poisoning, 10 deaths in Mutomo and 9 in Makueni	2006	Daniel et al. [70, 103]
Humans	75	Machakos, Makueni, Kitui	Maize	Acute poisoning, 75 cases, 32 deaths	2005	Azziz-Baumgartner et al. [69, 70]
Humans	331	Eastern/Central Machakos, Kitui, and Makueni areas	Contaminated maize	Acute poisoning, 125 deaths	2004	Lewis et al. [237]
Humans	6	Thika	Mouldy maize	6 deaths	2003	Onsongo [238]
Poultry/ dogs	Large numbers	Coast	Contaminated feed	150 deaths	2002	Njapau and Probst [239]
Humans	3, 26	Meru North, Maua	Mouldy maize, contaminated maize	Severe liver damage, 16 deaths	2001	Probst et al. [39]
Humans	3	Meru North	Maize	Acute effects, 3 deaths	1998	Mutegi et al. [38]
Poultry	Large numbers	Kenya	Imported maize	Deaths	1984/ 1985	[240, 241]
Humans	12	Machakos	Poorly stored maize	Deaths	1981	Ngindu et al. [177]
Poultry/ dogs	Large numbers	Nairobi, Mombasa, Eldoret	Poorly stored feed	Deaths	1977/ 1978	Muraguri et al. [241, 242]
Ducklings	16,000	Rift Valley	Peanut ration	Deaths	1960	Peers and Linsell [82]

Years are those in which the aflatoxicoses occurred rather than the years the data were published. Data are from [38, 92, 97]. A case report is also filed of a possible aflatoxicosis of a 17-year-old schoolboy [214].

contaminated maize triggered aflatoxicosis in humans with a case fatality rate of 50% in Tanzania [219].

In Kenya, aflatoxicosis was first witnessed in 1960 which recorded the death of at least 16,000 ducklings [82]. In 1981, Kenya witnessed its first serious recorded outbreak of human aflatoxicosis [177]. It was found that after 7 days of consumption of maize grain containing 3.2–12 mg/kg of AFB_1, symptoms of abdominal discomfort, anorexia, general malaise, and low-grade fever were exhibited in 20 cases of patients between 2.5 and 45 years of age. Hepatic failure developed in 12 of the 20 patients, all of whom eventually died 1–12 days following hospital admission. The most unprecedented episode of human aflatoxicosis in history was witnessed in Kenya in 2004 with 317 reported cases of which 125 were fatal [39]. This outbreak, which occurred in the Eastern Province, recorded a case fatality rate of 39%, and out of the 308 patients for whom age data were available, 68 (22%) were <5 years, 90 (29%) were 5–14 years, and 150 (49%) were >15 years. Children younger than 14 years, representing 51% of the children population, were thus

presumed to have had a greater predisposition to aflatoxicosis risk. The case fatality rate was significantly higher in Makueni district than in Kitui district [69, 70, 177, 220, 221].

Since 2004, outbreaks among subsistence farmers have recurred annually in Eastern Province and it is right to assert that the magnitude of exposure to AFs could be higher than reported due to the dearth of robust monitoring systems [63, 109]. Table 10 summarizes some of the fatal aflatoxicoses recorded in the history of Kenya since the discovery of AFs. It is worth noting that several studies on AF poisoning in humans have shown that low-level chronic intake may be more devastating than one-time high-level intake (that leads to aflatoxicosis) as it is linked to the development of hepatocellular carcinoma [30, 82, 128, 222–231]. During the aflatoxicosis outbreak that occurred in 2010, the levels of AFB_1 sera reported in Kenya were among the highest ever recorded in the world [232]. As can be traced from Table 10, most areas that have been hit by aflatoxicosis in Kenya are in the Eastern and some Central parts of the country.

7. Prevention and Control

7.1. International, Regional, and Statutory Efforts. Appreciable efforts have been advanced towards AF control in Kenya through countrywide awareness creation [97, 233]. The regional mycotoxin facility at the Kenya Agricultural and Livestock Research Institute (KALRO) in Katumani offers training to people from both the public and private sectors.

After the fatal aflatoxicosis in which dogs fed on contaminated rations died between 1970 and 1980s, KEBS came up with a standard for dog feeds in 1985. Standards for maize grain, other grains, and their products that have been in existence were also revised. For example, total AFs were initially at 20 μg/kg; this has been revised to 10 μg/kg, with 5 μg/kg as the threshold for AFB_1 in 2007 [234]. At least 25 standards aimed at regulating AFs have been drafted and are in full use and encompass key parameters such as moisture, mouldy grains, pest damage, filth, broken kernels/seeds, foreign matter, and discoloured grains. Most of these standards have been harmonized with the East African Standards by the Eastern Africa Grain Council (EAGC) in collaboration with KEBS through the Eastern Africa Grain Institute with its Kenyan headquarters at Nairobi, Kenya [235]. Between 2015 and 2018, the duo has trained maize exporters, traders, farmer-based organizations, and warehouse handlers on understanding the integrated East African maize standard (EAS 2:2013), food standardization, comparison of East African standards with international standards, standard maize sampling methods, maize grading, mycotoxins, and the available methods for mycotoxin analysis [235].

Following its launch way back in 2006, EAGC has been among the lead in the fight against AFs in East Africa as a whole. It has advanced several interventions to reduce the incidence of AFs, including (1) harmonization of AF control measures and improving the regulatory environment, (2) launch of AF control training programs, (3) furnishing moisture meters and waterproof sheets for drying, fumigation, and storing grains, (4) outsourcing portable kits for detecting and quantifying AFs, (5) farmer-based assessment of AFs prevalence, (6) collaborating with East African Community to expand AF testing and surveillance in maize, and (7) laying strategies of the Partnership for Aflatoxin Control in Africa (PACA) strategy 2013–2022 and revising EAC AFs communication strategy [235]. In addition, AF surveillance and capacity has been enhanced through the PACA Curated Africa Aflatoxin Information Management System (Africa-AIMS) in seven member states: Kenya, Malawi, Nigeria, Senegal, Tanzania, The Gambia, and Uganda.

Kenya Agricultural and Livestock Research Organization in connection with the International Institute of Tropical Agriculture (IITA) in 2018 developed a farmer-centered manual for the management of AFs in maize and peanuts [233]. The manual gives a general overview of AFs (structures, health, and economic effects), how to control AFs, drying, threshing, sorting, and some of the farming practices that favour AF growth. It was particularly drafted to provide ample guidance on the best practices for limiting AF contamination of maize and peanuts and to raise the value of these dietary staples.

Further, there are some projects running in the country to handle the plague of mycotoxins and these include the Aflacontrol Project and Purchasing for Progress (P4P) Programme. The Aflacontrol Project strives to minimize the ravage of AFs in maize and peanut value chains and is spearheaded by International Food Policy Research Institute (IFPRI). In addition, it seeks to increase the understanding of the economic and health impacts of AF contamination and identify and promote cost-effective methods and technologies available to reduce contamination of foods and feeds. The project, funded by Bill and Melinda Gates Foundation, has a partnership with the International Maize and Wheat Improvement Center (CIYMMT), University of Pennsylvania (USA), United States Uniformed Health Services, Kenya Agricultural Research Institute (KARI), and Agricultural Cooperative Development Initiative (ACDI-VOCA). The project has been experimented in Mbere (Embu), Makueni, Homa Bay, Kisii, and Rongo at the household level [97]. So far, it has released policy briefs and held inceptions, and one-year national workshops to disseminate information on AFs. These are targeted at the Ministries of Agriculture and Public Health, who are the key players in mitigating AFs. On the other hand, the Purchasing for Progress Programme is led by the World Food Programme that purchases maize from local farmers, usually ensuring strict adherence to AF limits in the grains. The grains are procured at fairer prices, encouraging the farmers to adhere to good pre-, peri-, and postharvest practices [97]. Several partnerships are currently running in the country; some are with FAO and CDC to mitigate AFs in Kenya. These have been discussed in sufficient details in a previous review by Mutegi et al. [38].

7.2. Scholarly Efforts. Earlier reports on the fate of AFs during the processing of maize into *muthokoi*, a traditional Kenyan food, revealed that traditional maize preparation

methods such as fermentation and dehulling in Eastern Kenya reduced AFs by up to 71% [83]. The findings of this study indicate that exposure to acute AF levels could be minimized during food processing and preparation. Generally, these processing techniques have been traditionally used for increasing the palatability of different food recipes but should also be promoted as strategies capable of reducing AF contamination of grains [97].

Intermediate processes such as sorting and dehusking were shown to reduce AF in peanuts [243]. Soaking peanuts in water, *magadi*, sodium hypochlorite, and ammonium persulphate significantly reduced AF levels by 27.7%, 18.4%, 18.3%, and 1.6%, respectively, while boiling in *magadi*, local ash, baking powder, and water reduced AF levels by 43.8%, 41.8%, 28.9%, and 11.7%, respectively. Similarly, Kirui [183] while assessing the levels of residual AFs following various treatments using physicochemical and traditional cooking methods for maize and maize products reported that boiling maize reduced total AFs from 83.1 ± 0.3 to $7.0 \pm 3.9\,\mu$g/kg, dry decortication reduced the level from 51.3 ± 15.3 to $9.6 \pm 0.8\,\mu$g/kg, while boiling with *magadi soda* (or maize wood ash) reduced the level from 59.5 ± 3.82 to $13.4 \pm 0.42\,\mu$g/kg. Solar irradiation (for 18 hours) reduced the levels from 60.8 ± 1.8 to $13.7 \pm 0.1\,\mu$g/kg while ultraviolet irradiation (for 18 hours) reduced the levels from 81.7 ± 0.5 to $61.4 \pm 4.5\,\mu$g/kg. The author reiterated that only the dry decortication method and boiling with *magadi soda* followed by washing with water and boiling reduced AFs significantly to below the maximum advisory limit of $10\,\mu$g/kg.

In the same struggle, a probiotic yoghurt was formulated with AFB_1 binding *Streptococcus thermophilus*, *Lactobacillus rhamnosus* GR-1, and *Weissella cibaria* NN20 isolated from fermented *Kimere*, a dough food product made from millet [179, 182]. Forty primary school children, with maize being a regular part of their diet, were randomly assigned to consume 200 ml of yoghurt or control milk daily for 7 days, followed by a 7-day washout and another 7-day treatment. After both 7-day treatment periods, AF concentration in urine samples was significantly lower than baseline in the probiotic group ($p > 0.01$) but increased in the milk group. This suggested that locally produced probiotic yoghurt could reduce AF poisoning in Kenyan children, corroborating previous observations in our laboratory [244, 245]. Similarly, fermentation of milk into *Lala*, a traditional fermented drink, and yoghurt significantly reduced AFM_1 levels by 71.8% (in *Lala* after 15-hour room temperature incubation) and 73.6% in yoghurt after incubation at 45 C for 4 hours [135].

In another intervention survey, the use of a calcium montmorillonite clay (calcium silicate 100, popularized as ACCS100) in food reduced the bioavailability of AFs [181]. It was reported to be palatable, effective, and acceptable, though further evaluation in the AF-endemic parts of Eastern Kenya as well as its efficacy to ameliorate AFs to levels incapable of triggering poisoning yet remains to be established.

Another study [43] screened maize lines resistant to *A. flavus* infection, together with a biocontrol strategy. Two African maize lines (GAF4 and KDV1) were reported to have different fungal loads for the aflatoxigenic isolate (KSM014), fourteen days after infection, with no significant variation in *A. flavus* biomass between diseased and non-diseased maize tissues for GAF4. Meanwhile, KDV1 had a significantly higher *A. flavus* biomass ($p < 0.05$) in infected shoots and roots compared to the control. The biocontrol strategy using an atoxigenic isolate (KSM012) against the toxigenic isolate (KSM014) showed aflatoxin production inhibition at the coinfection ratio, 50:50 for both maize lines (KDV1 > 99.7% and GAF 69.4%), as confirmed by bioanalytical techniques. It was indicated that the maize lines, which exhibited resistance to *A. flavus* with the appropriate biocontrol strategy, could reduce aflatoxicosis outbreaks.

In a 2020 report [46], the possibility of using *Pseudomonas* and *Bacillus* bacterial spp was explored in soils from Eastern Kenya (semiarid) and Western Kenya (subhumid-semihumid) [110]. *Pseudomonas (n = 7)* and *Bacillus* ($n = 5$) were identified in the two regions, though Western Kenya recorded a higher occurrence of *Bacillus*. Because these bacterial spp have been frequently associated with biological control of several plant pathogens including *Aspergillus* spp, a regression analysis was done to ascertain if there were any associations between the occurrence of *Aspergillus* spp and these bacterial spp in the studied regions. Weak relationships between the occurrence of *A. flavus* and *Pseudomonas* spp in the western region ($R^2 = 0.03693$) and the eastern region ($R^2 = 0.06126$) as well as occurrence of *Bacillus* spp in the western region ($R^2 = 0.196$) and in the eastern region of Kenya ($R^2 = 0.03693$). The same observation was made for the relationship between the occurrence of *Trichoderma viride* in both eastern ($R^2 = 003406$) and western ($R^2 = 0.2266$) regions of the country. As a consequence, the authors deduced that the occurrence of the bacterial spp had little influence on the occurrence of *A. flavus* in the two regions. To ascertain their assertion, an *in vitro* preliminary assay to determine the inhibitory potential of both *Pseudomonas* and *Bacillus* spp against *A. flavus* proliferation was done. Unfortunately, none of the bacterial strains from either spp had an inhibitory effect on *A. flavus* proliferation [46].

8. Suggested Management Strategies

8.1. Preharvest Management. Staple food crop varieties that are disease-, drought-, and pest-tolerant or less susceptible to fungal growth could be adopted. This approach is so far the best for the reduction of effects of AFs-producing fungi [246]. Valencia red (a peanut variety) was reported to be the least contaminated with AFs and had higher oil content than Uganda local, Homa Bay local, and Local red [121]. Food oils and microorganisms are viable inhibitors of AF biosynthesis [6] through interference with signal transduction regulatory chains in AF gene expression, inhibiting AF biosynthetic cytosolic enzymes and downregulating fungal genes of the oxidative stress defence system [247]. Further, host and parasite macro- and micromolecular trafficking which targets circumvention of AF scourge through the utilization of cross species RNA interference have been attempted in maize and peanuts [248, 249]. Particularly, UBI, COH, 26s,

ATP, PPK, IMP, ABC, and aflM were recommended as the suitable genes for RNAi silencing of *A. flavus in vivo* [248, 249]. This may, however, be impeded by the current policy on genetically modified organisms in the country.

Prompt harvest of mature food crops as well as selective disposal of broken kernels or cobs is a recommended AF mitigation measure [250]. Sorting, winnowing, and dehulling can lower AF levels in grains by 40–80% [151, 251]. Sorting is more practical in groundnuts [250, 252–254] and cassava chips mechanically or utilizing clean water. Soaking or cooking in *magadi soda*, malting, and roasting are other strategies reported to lower food AF levels [83, 251, 255, 256]. *Magadi soda* and wood ash are used by the Kalenjin of the Rift Valley region and Nyanza and Western provinces to increase food palatability and offer convenience as it reduces cooking time but also reduces phytates and increases the availability of niacin [257].

Pest control is another measure of AF management. This may be affected using ash for maize [258, 259] and essential oils which are broad-spectrum bioinsecticides [260–263]. Competitive exclusion is another underexplored AF control measure. A shift of strain profile from toxigenic to atoxigenic is possible and Kenya already approved a potential biocontrol product (Aflasafe KE01) [8, 233]. A typical example is a product constituted by a rhizosphere-competent nonaflatoxigenic *Aspergillus* strain possessing competitive saprophytic potential [264, 265]. For peanuts, an atoxigenic *A. flavus* strain (NRRL 21882) is commercialized as Afla-Guard® in the US [266]. Additionally, pseudomonads and *Trichoderma* spp inhabiting rhizospheres of various plants have been targeted against toxigenic *A. flavus*. Recently, various *Streptomyces*, *Pseudomonas*, and *Trichoderma* spp have been isolated, evaluated, and validated to possess antagonism towards *A. flavus* [267]. However, their efficacy is a theme that could be further explored under Kenyan conditions as was done in some African and Asian countries with up to 79% reduction of AFs reported [268].

8.2. Postharvest Management. Proper drying of produce to moisture contents between 12 and 14%, preferably 12.5% or below, is recommended. Fresh harvests should be shelled and cleaned prior to storage to minimize pest infestation that may initiate mould growth [269]. Further, proper ventilation of storage facilities is a requisite to avoid attaining temperatures between 25°C and 32°C and sustained relative humidity of 65% favourable for mould growth [11]. Moisture of 12–13% and temperatures below 18°C do not favour the growth of *Aspergillus* fungi [270].

Following good agricultural, good storage, and good manufacturing practices as well as the use of advanced agricultural technologies can reduce AF contamination [152]. Novel food processing techniques such as use of ozone, pulsed light, electrolyzed water, electron beam, microwave, cold plasma, and gamma and ultraviolet irradiation can reduce AF concentrations in agricultural foods. Food additives such as citric acid have been reported to sequester AFs in combination with moisture at high pressures and temperatures [271].

Clays notably Novasil Plus can ably bind AFs [272], reducing their available concentration. Other compounds like curcumin can afford alteration of AFB_1 microsomal activation, thereby augmenting its detoxification. Chemoprotection from ingested AFs is also feasible [273]. It makes use of compounds such as esterified glucomanoses and other yeast extracts that accelerate the detoxification process or otherwise block bioactivation of AFs to AF-epoxide, inhibiting AFB_1-induced hepatocarcinogenesis. Oltipraz and chlorophyll have reported use in this regard [273].

The management strategies suggested each have their advantages and limitations. Thus, biocontrol measures in tandem with physicochemical approaches could be adopted to manage the plague of AFs in Kenya.

9. Conclusion

Aflatoxin exposure is ubiquitous in Kenya, and the different commodities have relatively high levels of AFs, usually above statutory compliance limits by several folds. Maize, peanuts, and their products are the most contaminated food crops in Kenya. Variations in AF exposure are evident between the different regions of the country and are fundamentally a function of diet and economic status. Large-scale, evidence-based interventions are required to reduce exposure. More exposure assessments including coexposure with other mycotoxins alongside routine monitoring of AFs should be adopted.

Data Availability

This article is a review article and no raw data were collected. Any data used and/or analyzed are within this article.

Conflicts of Interest

The authors declare that there are no conflicts of interest regarding the publication of this paper.

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