

Aflatoxin M1 (*Aspergillus parasiticus, flavus*) Occurrences in Milk and Milk Products and Its Possible Health Effects

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Abstract

Aflatoxin M1 (AFM1) occurrence in milk and milk products has been a major concern among food scientists over the past three decades owing to its possible health risk in humans. The well-documented AFM1 associated adverse health outcomes include hepatocarcinogens, mutagens genotoxins, mutagenicity, and teratogen. The paper reviews the occurrence of AFM1 in milk, milk products, and human milk in developing and developed countries, with its safety standards of regulation. The health associated risks with AFM1 are the important methods used in detecting and reducing AFM1 in milk and other milk products are presented.

Keywords

Aflatoxins, Milk, Milk Products, Human Milk, Contamination, Feeds

1. Introduction

Aspergillus parasiticus and *Aspergillus flavus* are sub-groups of fungi that produce Aflatoxins [1]. They sometimes grow on plant products. *Aflatoxin B1 (AFB1)* constitutes a category of powerful and potent hepatocarcinogen mycotoxins that cause contamination to feed especially cereals and grains, particularly across the tropics and sub-tropics. The consumption of feeds infected with AFs affects animal health and ends in economic losses [1] [2] [3] [4]. When AFB1 is consumed by an animal, it's absorbed within the alimentary canal and metabolized into aflatoxin M1 (AFM1) within the liver. This can be excreted in urine and bile but can pass into milk if the animal is lactating [5] [6]. Additionally, AFM1 can even be passed into human milk when AFB1 contaminated food sub-

stances are consumed. It, however, has been documented in previous studies that about 0.3% - 6.2% of AFM1 can pass into milk. The extent of contamination depends on the season, (winter being more than summer), environmental and genetic condition of the animal [6] [7] [8] [9] [10]. Detection of AFM1 derivate in milk ranges from 12 - 24 hours after the ingestion of AFB1, however, there is a decrease in concentration after 72 hours of contact [5] [8].

AFM1 in milk raises concerns on food safety because of its association with adverse health effects in humans [11]. There's a growing incidence of the occurrence of AFM1 in milk and its products hence raising more concerns about food safety and public health attention across the globe [12]. Among AF, AFM1 is one among the foremost widespread and unsafe for human health [13]. Evidence from the "International Agency for Research on Cancer" (IARC) proves that AFM1 is a possible carcinogen in humans [14]. Milk and its products are an important part of the human diet particularly for infants and young children hence increasing the exposure to AFM1 can be detrimental to these age populations [15]. Nevertheless, some countries have set a limit of AFM1 acceptable in milk and other milk products that are expected to be safe [16] [17]. The uppermost limits of AFM1 in milk and other milk products set by the "European Community" are 0.05 µg/L [18] and the regulatory limit for the USA is 0.5 µg/L [19]. However, there are some nations in Africa with no specific regulation regarding AFM1 [20]. There are efforts to decrease or detoxify AFs from food and food products over the years. Literature available states that the exposure to high temperature, radiation, and pasteurization reduces the strength of AFs, however, this reduction isn't always significant [17] [21]. Conversely, results from studies conducted by Turkoglu and Keyvan, [22] demonstrated that milk that has undergone pasteurization had significantly lower concentrations of AFM1 compared to unprocessed milk. Pasteurization kills harmful bacteria and other microorganisms [22] [23]. The appropriate solution is to reduce the consumption of AFs by eating food with low levels, guaranteed by the regulatory limits of food products [24].

The purpose of this review article is to 1) explore the occurrence of AFM1 in milk, milk products, and human milk in both developing and developed countries, and the safety standard of regulation; 2) the health associated health risks with AFM1 are examined, and 3) also the important methods used in detecting and reducing AFM1 in milk and other milk products are presented.

2. Methodology

Scopus and PubMed search were conducted from January to March, of which a total of 308 original articles were found. Keywords used include; Aflatoxin, Milk, Human Milk, Milk products, Contamination, and Feeds however, only 77 articles including government documents were included in this review. The three reviewers independently screened abstracts of studies and disagreement were settled through consents. Eligible studies (studies that assessed AFM1 occur-

rence in milk, methods used to reduce AFM1, associated health risks etc.) were extracted independently by reviewers. Non-English, review articles were and articles older than 2010 were excluded from the study. The handful review papers were however referenced for general knowledge.

3. AFM1 Occurrence in Milk and Milk Products

3.1. Global Occurrences

3.1.1. Developing Countries

Although AFM1 occurrence is not limited to any country, Asia has the highest prevalence of AFM1. According to a study conducted by Nile *et al.* 2016, [25] cow's milk had the highest concentrations of AFM1 (62%) when compared with sheep, goat, and buffalo milk. The concentration of AFM1 found was said to be higher than the regulatory limit in the EU. Also, the study found season variation and location to be associated with AFM1 contamination in milk [25].

Furthermore, Lin *et al.* 2019 [26] examined the amount of AFM1 in other unprocessed milk products of buffalo. A total of 62% of AFM1 were detected in raw milk compared to other products that had undergone high-temperature short time (HTST). The quantity of AFM1 identified in the different products was greater than the EU standard but below China's (500 ng/kg) limit. Likewise, a study carried out in Iran by Fereshteh, Hadi, and Lane showed that the amount of AFM1 contained in cow's milk was higher than the levels of both the EU and Iran [27].

Studies have also reported differences in AFM1 levels according to the weather conditions [11] [25]. However, it is stated that there is also a strong link between the different agricultural practices and modes of feeding systems of livestock and aflatoxin infection [16]. Li *et al.*, Akbar *et al.*, and Shokri and Toabi, for instance, identified higher quantities of AFM1 in milk and other milk products during winter compared to other seasons [15] [28] [29]. It is documented that, during summer, fresh feeds such as weed, pasture, and fodder are available in South Asia hence animal graze. However, during winter, they are unavailable, and animals are fed with stored cereals, soya bean, cotton seeds, etc. which are thought to be concentrated in AFM1. The storage of fodder results in the feed being contaminated in winter [30]. More so, other reports from Iran indicate a higher amount of AFM1 in cow milk than other milk products. The amounts found in the various products by all the studies were found to be higher than that of the EU standard [6] [31] [32].

The occurrence of AFM1 has also been reported in some African countries. Kenya is the only country that constantly reports on AFM1 occurrence although others report seldomly [33]. In Kenya, studies by Kuboka *et al.* 2019, Lindahl *et al.* 2018, and Anyango *et al.* 2018 reported the presence of AFM1 in milk and milk products. The AFM1 levels reported exceeded both the US and the EU limits (mean of 290.3 ± 663.4 ng/kg) with 7.5% the US limit, 50% exceeding EU limit and 26.4% exceeding the EU respectively [28] [29] [34]. The occurrence of

AFM1 remains significantly high in most developing countries. Although these countries have higher contamination levels due to their weather conditions, most of these countries do not have a standard of regulation.

3.1.2. Developed Countries

AFM1 has been reported in some countries in Europe, the US, Canada, and other developed countries. Results from studies by Tsatskiris *et al.* 2013 demonstrated the occurrence of AFM1 in milk and milk products in Greece. The study sampled a total of 196 milk and milk products (raw milk and UHT) and AFM1 was analyzed using the ELISA method. AFM1 ranged from 5 and 10 ng/l and leading to a total of 46.5% of AFM1 detection. However, the level of AFM1 detected was way below the EUP limits except for two samples. The lower incidence of AFM1 among this population could be attributed to their adoption of the EUP standards of regulations [35]. Similarly, AFM1 has been detected in milk and other milk products in Spain. The study used ELISA for detecting AFM1 and the results indicated that a higher concentration of AFM1 in UHT milk and yogurt 94.4% and 2.8% respectively but not cheeses. Findings from this study are similar to that of Tsakiris *et al.* as the AFM1 levels detected in both studies were below the EUP standards [35]. AFM1 contamination in milk and its products occurs in both developed and developing countries however, developing countries have a higher amount of AFM1 contamination compared to developed countries. The low incidence of AFM1 contamination in developed countries is a result of higher standards of regulations particularly for the European countries [33].

3.2. The Occurrence of AFM1 in Breastmilk and Commercial Infant Formula

The occurrence of AFM1 has also been reported in breastmilk as well as infant formula milk and this has been associated with an adverse health outcome in infants and young children. The exposure to AFM1 at an early age has been documented to be associated with poor growth outcomes, as well as immunosuppression among infants and young children [36].

3.2.1. Breastmilk

According to the findings from a recent study, a prevalence of AFM1 was detected among breastfeeding mothers in the South-Eastern part of Nigeria. Both frequency and levels of AFM1 in breastmilk were determined using High-Performance Liquid Chromatography (HPLC) with fluorescence. The average amount of AFM1 amount in human breast milk was 4.02 ± 1.12 ng/L and 100%. The occurrence of AFM1 was highly associated with the intake of cereals and grains, dry fruits, cassava, and peanut oil ($p < 0.05$) following post-harvest. Levels of AFM1 detected were below the international limits there but there is the need for practices to reduce AFM1 contamination among these population groups (breastfeeding mothers) since they are vulnerable [37]. Results from the

study above are consistent with that of Ortiz *et al.* 2018 [38]. Furthermore, a study conducted in Serbia assessed AFM1 in a total of 60 breastmilk samples both colostrum and matured breastmilk (4 - 8 months). Samples were assessed using ELISA and daily AFM1 ingestion was measured using 60 mL/kg body weight (b.w.) per day for colostrum breast milk. While only 36.4% of colostrum milk was contaminated, all matured breastmilk samples were highly contaminated beyond the maximum acceptable limits in human milk 25 ng/L per the “Regulation on health safety of dietetic products” [39]. Additionally, a cross-sectional study analyzed 150 breast milk samples from breastfeeding mothers in Egypt found that (65.3%) of the breast milk was contaminated with AFM1. The occurrence of AFM1 in these breast milk samples was 0.2 and 19.0 mg/l (mean: 7.1 ± 5.0 mg/l) which is higher than the EU limits [40]. Lastly, Bogalho *et al.* 2019 examined the occurrence of AFM1 in maternal breast milk. A total of 67 breast milk was sampled and examined results found that 32 out of 67 samples contained AFM1 constituting (32.8%). The level of AFM1 found exceeded the limit of the EU standard ranging from 5.1 and 10.6 ng/L (7.4 ± 1.9 ng/L) [41]. The higher levels of AFM1 found in matured breast milk require both food safety measures as well as public health attention. Evidence from a large meta-analysis is consistent with the results of all the studies above [42].

3.2.2. Commercial Infant Formula

Several studies have documented the occurrence of AFM1 in commercial infant formula just like in other milk and milk products [43] [44] [45]. Hassan *et al.* 2018 sampled a total of 69 commercial infant formula in Qatar and assessed for AFM1 contamination using HPLC. The results indicated that a total of 33% of infant formula was contaminated with AFM1 and the concentration of AFM1 was above the EU maximum acceptable limits [45]. This is an indication that infant formula in Qatar could pose health risks to infants and young children. Moreover, Akhtar *et al.* 2017 quantitatively analyzed AFM1 levels in commercial infant formula using the ELISA method among the Pakistani population. Out thirteen samples that were taken AFM1 present were present in 53.84% and 30.76% were found to contain AFM1 level above the EU tolerable limits and this can result in toxicity among infants [43]. This finding is like that of Tonon *et al.* 2018 who indicated that 2 samples out of 38 exceeded the EU acceptable limits [44]. Additionally, a cross-sectional study surveyed the effects of the storage of infant formula on AFM1 concentration in Tripoli-Libya. Formula and other milk products were assessed for AFM1 using ELISA (RIDASCREEN). Interestingly, the results of the study showed that AFMI increased with increasing storage on the shelf. Expired products had higher concentrations compared to newly manufactured products >60.18 ng·kg⁻¹ [46]. The results from studies assessing the occurrence of AFM1 in infant formula consistently show that some sample contains AFM1 levels beyond the EU. There is a need to ensure proper regulation of commercial infant formula to reduce contamination as well as toxicity in infants and young children.

4. Methods of AFM1 Detection in Milk and Milk Products

There are various methods for detecting AFM1 in milk and other milk products. The most common methods recently used include; Enzyme-linked immunosorbent assay (ELISA), and liquid chromatography (LC) with fluorescence detection (FLD). However, other methods include; Thin-layer chromatography (TLC), lateral flow Immunoassays, ultra-performance liquid chromatography-tandem mass spectrometry (UHPLC/MS/MS), and gel-based immunoassays. The TLC is an old technique and the use of HPLC with FLD is the most effective in AFM1 detection and widely used these days [47].

5. Stability, Toxicity, and Health Risk of AFM1 in Milk and Milk Products

5.1. Stability

Dragan *et al.* 2019 investigated the stability of AFM1 from milk to cheese and found that 60% and 40% of AFM1 accumulates or remains in whey milk and fresh cheese respectively [48]. Moreover, another study by Skribic *et al.* 2015 studied the stability of AFM1 in fifty-four cheese (white and hard) samples in the Serbian market. Thirteen percent of the cheese samples analyzed were found to be contaminated with AFM1 in concentrations above the EU standard. The results from this study are evidence that the AFM1 fungi are stable to both higher temperatures and acidification [49]. Similarly, Fernandes *et al.* 2012 evaluated how AFM1 was carried from milk to whey and Minas Frescal cheese. The results demonstrated that AFM1 levels found cheese was about 2.5 times higher than the levels found in milk. The level AFM1 in whey and curd ranged from 0.099 to 0.215 ng·ml⁻¹ and 0.199 to 0.416 ng·g⁻¹ respectively. However, the study found no effects between the storage time of cheese of about 30 days and AFM1 levels and starter culture in cheese production did not have any impact on AFM1 stability. The level of AFM1 found in cheese was below the tolerable limits in Brazil [50]. The results from this study support other findings indicating a higher concentration of AFM1 in cheese than the milk used in producing the cheese. A study conducted by Cavallarin *et al.* 2014 [51] used naturally contaminated milk samples with AFM1 concentration values of 5.30, 4.52 and 5.30 ng/kg to produce cheese using both primosale, robiola, and macagno techniques. In all techniques, milk samples were heated up to 40, 25, and 38 degrees Celsius in primosale, robiola, and macagno technique respectively. In robiola technique, the AFM1 levels in whey samples were 30% and 65% while the AFM1 levels in whey samples of both primosale and macagno had higher concentrations of 60% - 82%. Despite the different techniques employed, the study found a higher concentration of AFM1 in cheese compared to the milk used in making the cheese. Also, during the 3 months maturation period of macagno (hard) cheese, there was a 6-fold increase in AFM1 levels. Change in the levels of AFM1 in milk compared to cheese is more related to moisture loss during processing than the technology used [51].

Conversely, according to findings from a study on stability and distribution of AFM1 in cheese and yogurt by Mari *et al.* 2013, [52] AFM1 were detected in 13 (76%) of UHT with ranges from 8 to 215 ng/L and 9 - 437 ng/L. Pasteurized milk samples and infant formula were free from AFM1. The study found a mean reduction of 6.4% in AFM1 concentrations after fermentation. Again, there was a reduction in AFM1 by 28.9%, 13.5% and 34.2% in three trials during cheese production. Reduction in AFM1 level was associated with cheesecloth used, the environmental conditions of home-produced cheese or analytical recoveries. Similarly, a study by Chavarría *et al.* 2017 found a higher proportion of AFM1 in whey compared to fresh cheese [53]. The cheese was produced with milk contaminated with two levels AFM1 (0.5 and 1.5 $\mu\text{g}\cdot\text{L}^{-1}$) to determine the carryover AFM1 from contaminated milk to fresh cheese and whey. For both concentrations there was a higher level of AFM1 was found in whey (0.5 $\mu\text{g}\cdot\text{L}^{-1}$: mean value [70.4% \pm 22.0%]; 1.5 $\mu\text{g}\cdot\text{L}^{-1}$: mean value [73.1% \pm 14.0%]) than in cheese. AFM1 and protein stability were monitored during cheese storage. The concentrations of AFM1 in cheese were measured on days 0, 7, 14, 21, and 28 by triplicate. On day 0, there was an average $0.42 \pm 0.26 \mu\text{g}\cdot\text{L}^{-1}$ of AFM1 in cheese manufactured. Following 28 days of storage, between 10.1% and 70% of AFM1 remained in cheese. The reduction in AFM1 levels is directly correlated with both nonfat milk solids and proteins in the starter milk p values < 0.05 ($r = 0.747$) and ($r = 0.769$) respectively [53]. The results from this study indicated higher levels of AFM1 in whey rather than cheese. This finding is consistent with that of Cateneo *et al.* 2013 [54].

Findings from the above studies demonstrate inconsistency regarding the stability of AFM1 in milk and other dairy products. Hence further research is needed on this subject.

5.2. Toxicity and Associated Health Risk

Studies indicate that the presence of AFM1 in milk is of a public health concern since milk forms a very important part in human diets across nations. AFM1 has been classified in the group 1 carcinogens by the IARC, thus, the health risk associated with it cannot be overlooked. AFM1 has also been associated with teratogenic and mutagenic activities particularly regarding liver health [55]. Another issue of concern is the fact that AFM1 found in milk can contaminate other milk products during processing and even storage products [50]. Tsakiris *et al.* 2013 examined hazard index in AFM1 contaminated milk and found that the highest risk of AFM1 was associated with children 1 - 3 years of age, but this is not significant statistically [35].

A recent study by Daou *et al.* 2020 sampled 868 raw, UHT, and pasteurized milk to determine its AFM1 content. A food frequency questionnaire was used to determine milk consumption. The study also evaluated AFM1 exposure and associated liver cancer risk among the Lebanese population. The average everyday exposure to AFM1 from each element was summarized to get the total eve-

ryday exposure to AFM1 (ng/kg body weight/day) from milk and dairy in Lebanon. Contamination of AFM1 ranged from, 0.013 - 0.219 µg/L, 0.015 - 7.350 µg/L, and 0.011 - 0.440 µg/l pasteurized, UHT, and raw milk respectively. AFM1 concentrations detected in these exceeded the EU limits by 28%, 54.5%, and 45.5% in raw, pasteurized and UHT respectively. Liver cancer risk was calculated based on “JEFCA estimates that the intake of 1 ng per kg of body weight per day of aflatoxins will induce 0.0083 liver cancer cases per year per 100,000 persons in non-Europeans” [56]. The study showed that intake of AFM1 was correlated with an estimated 0.0041 cancer cases per 100,000 persons per year [57]. According to the results of this study, milk and milk product ingestion among the Lebanese population can be considered harmful and of serious public health concern.

Furthermore, a recent study investigated AFM1 contamination in Italy on hepatocarcinoma outcomes. The study sampled and analyzed a total of 31,702 milk. “Estimated daily intake (EDI), the Hazards Index (HI)”, and the percent of cases of hepatocarcinoma (HCC) from exposure to AFM1 in different age categories were also determined. AFM1’s EDI in the different age categories was within the range of 0.025 - 0.328 ng·kg⁻¹ body weight (BW) per day, based on mean intake levels and calculated mean milk toxicity across the duration of the study. The results of HI among infants and young children was 1.64 and 1.4. The estimated proportions of HCC reported cases attributed to AFM1 exposures were 0.005 and 0.004 cases per 100,000 individuals in age categories of 0 - 0.9 and 1 - 2.9, respectively [58]. While the study found a small HCC, risk associated with other age groups, the risk in infancy was higher so further studies, as well as preventive measures, are needed.

Additionally, Gao *et al.* 2016 studied the toxicity of cells individually and in combination with AFM1, ochratoxin A (OTA), zearalenone (ZEA), and α -zearalenol (α -ZOL) in human Caco-2 cells. Their results showed that the toxicity of cells of AFM1 in human colon adenocarcinoma cells (Caco-2) was comparable or even greater. The prevailing mechanism for the cytotoxic effect of AFM1 led to induced cell death in a combination of OTA, ZEA, and α -ZOL [59]. Lastly, Alireza and Sepehr 2019 assessed the harmful risk associated with AFM1 contamination in milk. Risk assessment was done in five main stages; 1) hazard identification, 2) dose-response determination, 3) exposure assessment, 4) risk characterization, and 5) risk management. The cancer associated risk was evaluated by “multiplying lifetime average daily exposure by cancer slope factor”. Findings from this study indicated that carcinogenic risk was not high due to low milk intake. In this analysis, however, levels of AFM1 were not measured in milk or milk products instead, the analysis was based on the secondary data from previous findings [60]. A recent study [61] together with several studies [25] [27] [29] found higher limits of AMF1 in milk and other dairy products exceeding the EU limits. However, due to ethical issues concerning studies of this nature, there is no single experimental study in a human study on this subject matter. Hence, only associations can be made but not causalities.

6. Treatment of AFM1 in Milk and Milk Products

While some studies have demonstrated that the stability of aflatoxin to heat [48], evidence from literature indicates that AFM1 can be treated using microorganisms like lactic acid bacteria (LAB), yeast specifically *Saccharomyces cerevisiae* (SC), enzymes and some other techniques like ultra-violet light among others [62]. According to the findings from a study by Corasim *et al.* 2013, the use of microorganisms SC and LAB was effective in the treatment of AFM1 in skim milk [63]. In this study, commercially available SC (SAFLAGERW37/70, *Fermentis*) was used together with three different strains of LAB. The results of the study indicated that the use of both SC and LAB significantly decreased AFM1 by 100% after 30 - 60 minutes due to the binding of AFM1 to the microorganisms ($P < 0.05$). However, the use of either SC or LAB alone resulted in a reduction of 90.3% - 92.7% and 11.5% - 11.7% respectively. The results of this study are consistent with findings of Ismail *et al.*, 2017 [64] who evaluated the impact of different levels of AFM1 on the binding capacity of microorganisms. Their results indicated 100% of AFM1 in milk reduced to undetectable levels by SC or in combination with LAB at a level of 0.05 $\mu\text{g/L}$ and 92% by SC at 10^{10} cell mL^{-1} at a level of 0.1 $\mu\text{g/L}$. Similarly, recent studies show the effectiveness of LAB and SC in the reduction of AFM1 [65] [66]. There is also evidence that the use of enzymes is potent in the reduction of AF. However, the literature supporting this is quite old and more common in AFB1 rather than AFM1 [67]. Nevertheless, the use of fermentation microorganisms such as LAB and SC remains very effective and popular in controlling AFM1 contamination in milk and other dairy products.

7. Legislation or Regulation on AFM1 in Milk and Milk Product

Due to the toxicity associated with AFM1, certain nations have introduced legislation on amounts of AFM1 in milk and milk products to protect consumer health [13]. According to Škrbić *et al.* 2014, the regulatory limit for AFM1 in milk should consider the rate of conversion of total AFs in a dry matter to AFM1 in milk [68]. Nevertheless, it is documented that when lactating animals ingest feed infected with 30 $\mu\text{g/kg}$ of total AFs, approximately 50 ng/L is released as AFM1 into the milk and this can also be passed during the processing of other milk products [5] [50]. For this reason, The Codex Alimentarius Commission and the European Commission have set an acceptable limit of AFM1 for infants' either raw, UHT pasteurized milk at 50 ng/L [18] [69] [70]. The following countries France, Kenya, Belgium, Turkey, Iran, Switzerland, Germany, Sweden, Argentina, and Honduras have adopted this standard hence uses the same standard of regulation for their milk and milk products [34] [47] [71] [72] [73] [74]. However, the United States of America together with other countries like China, Serbia, Czech Republic, Kuwait, and Bulgaria have their acceptable limits as the standard level for AFM1 which is 500 ng/ [68] [71] [75] [76] [77]. Also, some

countries have made their regulations specific to infant formula, for instance, in Austria and Switzerland, the maximum level of AFM1 allowed is strictly 10 pg/mL.

8. Future Trends of AFM1 in Milk and Milk Products

Milk and milk products are a major animal source food for people of all ages particularly infants and young children [15]. Moreover, it is estimated that there is a need to increase the production of milk by two-folds from 2000 to 2050 to satisfy population needs [47]. Although there is a growing demand for these products, the high risk of contamination of milk and milk products with AFM1 has been associated with increased health risk [55]. It is not surprising that this subject matter remains of interest to many researchers in the field of food science. There have been advances in both techniques for detecting and controlling AFM1 in milk and milk products over the past few years, but these techniques do not completely degrade AFM1 [62]. Higher incidence and prevalence of AFM1 in milk and milk products remain significantly high in some developing countries like Iran [15]. Good harvesting and post-harvest practices, proper storage of animal feed as well as a higher standard of regulation are needed to reduce the occurrence of AFM1 in these populations. Although regulations limiting the level of AFM1 in milk and milk products claim those levels are safer, it is, however, difficult to calculate the levels of carcinogens in these contaminated products. More so, there remains a challenge in quantifying the level of carcinogens safer for specific population groups like an infant and young child as well as pregnant women [33]. Future studies should consider a large prospective longitudinal (cohorts) where participants exposed to AFM1 in milk and milk products will be followed for years to evaluate the risk of adverse health outcomes. Besides, there is a challenge in the treatment of AFM1 in milk and milk products. While a method like the use of peroxide has been reported to reduce AFM1 by 100%, it uses a higher dose of hydrogen peroxide and residue of this has adverse health consequences. Again, reports indicate that many treatment methods alter the organoleptic properties of milk and milk products making them less desirable [62]. Future research should, therefore, consider a new technique that will be potent in controlling AFM1 toxin without adverse health outcomes as well as retaining the original flavor of milk and milk products.

9. Conclusion

High contamination of animal fed by AF significantly results in the contamination of milk and dairy products. Additionally, the occurrence of AFM1 has been reported in both human milk and commercial infant formula. The occurrence of AFM1 in milk and milk products is high especially in Asia while Europe remains relatively at low risk due to high regulatory standards. Available evidence shows a seasonal variation in the occurrence of AFM1 with winter having the highest number of contaminations due to poor storage conditions of feeds. Therefore, it

is necessary to ensure constant monitoring of animal feeds especially during the winter season to control these toxins. Although some studies in the human population associate AFM1 consumption with health risks like hepatitis B and certain cancers, evidence or studies are sparse. Also, all studies were observational hence could not effectively establish a causal link. Enzyme-linked immunosorbent assay is the most common technique of AFM1 detection, however, the use of HPLC with FLD is proven to be the most effective and currently becoming popular among other techniques. Evidence of stability of AFM1 to heat is inconsistent, with some studies indicating a significant reduction while others indicate no effect. Furthermore, AFM1 has been demonstrated to have carcinogenic properties. There are various techniques for treating AFM1, but the use of yeast and LAB remain the two most common. The combination of the two is very effective in decreasing up to 100% of AFM1 in milk and dairy products. In summary, good harvesting and post-harvest practices should be adopted in addition to strict regulatory standards to avoid or decrease the contamination of milk and its products for safe human consumption.

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Conflicts of Interest

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

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