Global Veterinaria 14 (4): 560-566, 2015
ISSN 1992-6197

DOI: 10.5829/idosi.gv.2015.14.04.9445

Prevalence of Aflatoxin M1 in Some Milk Products Widely Consumed by Infants and Children, Marketed in Sharkia, Egypt

Magdy Sharaf Elsayed and Eman Nabil Abd El-Fatah

Department of Food Control, Faculty of Veterinary Medicine, Zagazig University, Egypt

Abstract: AflatoxinM1 (AFM1) has been the major mycotoxin frequently found in milk and dairy products. These products are widely consumed by infants and children who are more susceptible to adverse effects of mycotoxins, considering its significant impact on human health. Determination of AFM1 levels in dairy products becomes imperative, so the present study was undertaken to investigate the levels of AFM1 in some milk products consumed widely by infants and children and marketed in Sharkia Governorate markets. For this purpose total of 80 milk product samples were randomly collected. Aflatoxin M1 was detected in 23 (28.75%) out of the 80 samples analyzed in which the levels of AFM1 of the samples were found to exceed the limits (0 ng/kg) allowed by Egyptian regulation. Results of yoghurt examination showed that 3 (12%) of the local processed type contain AFM1 and exceeding Egyptian permissible limits, with a mean value 0.803 ± 0.1322 ng/kg, while processed cheese results also exceeded Egyptian permissible limits in 11 (44%) of examined samples and ranged from 23.2 to 47.1 ng/kg. Of the analyzed Infant formula milk powder samples, 2 (13.3%) samples were contaminated by AFM1. 7 (46.67%) of the UHT milk samples exceeded the permissible limit where the highest recorded level was 33.4 ng/kg. The results of this study indicate that continuous monitoring of AFM1 levels in commonly marketed milk products in Sharkia markets should be regularly done.

Key words: AFM1 · Mycotoxins · Infant Formula Milk Powder · UHT Milk

INTRODUCTION

Mycotoxins are a a group of naturally occurring secondary metabolites which are mainly produced by the filamentous fungi [1]. They represent a major problem for food industries affecting productivity, welfare and health and are also a permanent risk concerning food safety for humans and animals [2].Among mycotoxins, aflatoxins (AFs) are the most toxic and carcinogenic class and are mainly produced by fungi.(Aspergillus flavus, Aspergillus parasiticus and rarely by Aspergillus nomius). They can contaminate food, vegetable, fruits, cereals and cattle feed [3]. The contamination of milk and dairy products with aflatoxins and the concentration of these toxins in milk products may vary according to geographic location, the development level of the country and climatic conditions [4].

Eighteen AFs have been identified up to now, but only four of B1, B2, G1 and G2 are most concerned [5]. Among them, AFB1 is the most toxic and has been designated as group 1 carcinogenic compound by the International Agency for Research on Cancer (IARC) [6].

AFB1 is a contaminant found in feeds that have been improperly dried. Dairy cows consuming AFB1-contaminated feeds accumulate ahydroxylated metabolite of AFB1 known as AFM1 in the milk [7]. It has been reported that there is a linear relationship between AFM1 in milk and AFB1in the feed consumed by the animals with approximately 1 to 6% of the ingested AFB1 appearing as AFM1 in milk [8]. Once AFB1is absorbed into the cow's body, the clearance of AFM1 in milk may take 5 to 7 days depending on the amount and duration of the AFB1consumption [9].The level of AFM1 in raw milk is a concern due to its mutagenic, carcinogenic and teratogenic effects [10]. And is considered as major etiological factor for hepatocellular carcinoma (HCC) [11]. And it is assumed that neither storage nor processing, could destroy the AFM1 toxin [12] and can be detected in dairy products submitted to pasteurization, sterilization process and also in fermented products [13].

Several methods have been described for the determination of AFM1, including thin-layer chromatography (TLC), high-performance liquid

Corresponding Author: Nabil Abd El-Fatah, Food Control Department, Faculty of Veterinary Medicine, Zagazig University, Egypt. E-mail: dr.emannabil@hotmail.com.

chromatography (HPLC) [14] or mass spectrometry [15]. These techniques have high sensitivity and accuracy, but require extensive sample preparation, expensive equipment and well- trained personnel. Recently, ELISA methods have also been described and were mainly used in routine analysis [16]. These methods have been shown to be simple, having the portability of the equipment and hand-holding validation and are reliable for the analysis of a large number of samples [17].

A flatoxin M1 occurrence in milk and dairy products is an important issue because many people consume these products on a daily basis, especially for the growing infant population, which depend on milk as a major nutrient. [18]

Early infants' exposure to carcinogenic aflatoxins is a serious determinant for their health. Infants regularly have a high dietary intake per kg of body weight accompanied by rapid growth. The capability of carcinogens biotransformation is slower in infants than adults; consequently the circulation and exposure time to the toxins is increased in infants' tissues [19]. Even when the levels are within the regulatory limits it cannot prevent the chronic effect of aflatoxins, principally the carcinogenic effect, due to sustained exposure to low levels of aflatoxins [20].

Therefore, current study aimed to investigate the occurrence and levels of AFM1 in various types of milk products consumed widely by infants and children and marketed in Sharkia Governorate, Egypt and compare the positive levels, if any, with those set as permissible by the Egyptian Standard and the European Commission.

MATERIALS AND METHODS

Sample Collection: Total of 80 samples of different types of milk products were collected 25 for each yoghurt and processed cheese samples and 15 for each infant formula milk powder and UHT milk samples from supermarkets located in Sharkia Governorate. An insulating container at 4°C was used for their transportation to the laboratory.

Determination of AFM1 Content: The method used in this study was the enzyme-linked immunosorbent assay (ELISA). Using Ridascreen AFM1 kits (R-Biopharm, Derm-Stadt, Germany), which contained Microtiter plates coated with specific antibodies to AFM1, AFM1 standard solution of (0, 5,10, 20, 40 and 80 ng/l), peroxidase conjugated AFM1, together with substrate chromogen and stop solution. All samples were prepared and defattened using the method outlined in the ELISA kits, as briefly described.

Preparation of Samples

Cheese Samples: cheese samples were prepared according to the method outlined in the ELISA kit. Two grams of a representative cheese samples were added to 40 ml of dichloromethane. The mixture was extracted by shaking for 15 min. The suspension was filtered and 10 ml of the filtrate was evaporated at 60°C under the weak N2 stream. The oily residue was redissolved in 0.5 ml methanol, 0.5 ml phosphate buffer saline and 1 ml of heptane. The mixture was centrifuged at 2700 g/15°C for 15 min. The upper layer of heptane was removed and 100 µl of the aliquot were diluted with 400 µl of kit buffer. 100 µl of the diluted samples were applied in the kit.

Yoghurt Samples: Yoghurt samples were pasteurized by heating them to 80°C in a water bath for 3 min. Then, the samples were cooled down to at room temperature[21]. Ten grams of each yoghurt sample were diluted in100 ml PBS-buffer (pH 7.2). The mixture was homogenized for 2 min a Stomacher Lab-Blender (Interscience) and 100 µl diluted samples were used directly in the test

Infant Formula Milk Powder: Ten grams of powder milk were placed in a flask and 100 ml of deionized water was added. The mixture was stirred for 5 min and then centrifuged at 3500 g for 10 min at10°C temperature. After centrifugation, the upper fatty layer was removed and 100 µl of the skimmed milk was used for ELISA analysis.

UHT Milk: 20 ml of milk were chilled to 10°C and was centrifuged for 10 min at 3500 g. The fatty layer was removed and 100 µl of the defatted milk was applied directly in the ELISA Microtiter plate

AFM1 detection: A 100 µl of each of the AFM1 standard solutions and the previously prepared samples (skimmed milk samples or cheese samples) were added to microtiter wells (100 µl /well) and incubated for 60 min at room temperature in the dark. The supernatant was removed from the wells and the microwell holder was vigorously tapped upside down against absorbent paper to ensure its complete removal from the wells. The wells were then washed three times with 250 ml of washing buffer. 100 µl of the diluted enzyme conjugate (peroxidase conjugate AFM1) were added to each well, gently shaken and incubated for 60 min at room temperature in the dark.

The wells were again washed with 250 ml of washing buffer, as described above and 100 µ of substrate/chromogen were added to each well and incubated for 15 min at room temperature in the dark. 100 µl of the stop solution (1 N H2SO4) were added to wells and mixed. Absorbance was measured at 450 nm using ELISA microplate reader (KC-100. Caretium, Shenzhen, China).

RESULTS AND DISCUSSION

Analysis of milk product samples indicated their contamination to somewhat by AFM1, exhibiting a wide array of hazardous impacts on human health. In the present study total of 80 milk product samples which are consumed widely by infants and children were analyzed for AFM1. The statistical analysis of obtained data (Table 1) revealed that 23 (28.75%) out of 80 analyzed samples were contaminated by AFM1. The contamination level of the yoghurt samples ranged from 0.2 to 1.51ng/kg, with 3 of the 25 (12%) locally produced samples exceeding the Egyptian regulation limit for AFM1 of (0 ng/kg). Concerning the results of processed cheese samples, examination revealed that AFM1 present in 11(44%) samples with a contamination level ranged from 23.2 - 47.1ng/kg with (100%) of the positive samples exceeding the Egyption regulation limit (Table 2).

According to Hosny *et al*. [22] and Temamogullari and Kanici [23] AFM1 was detected in both yoghurt and processed chesse samples in lower percentage, while Iqbal and Asi [24]detected it in both products in higher percentage than our results.

In other investigations, AFM1 was detected in chesse by 78, 64, 64.8 and 80% [18-25-26-27].

Fifteen different IFMP samples from different countries were collected from Sharkia Governorate markets for the analysis of AFM1 (Table 1). The results showed 13.3% of the tested Infant Formula Milk Powder samples were positive for AFM1 (2 out of 15). For UHT milk samples 7 (100%) of the positive samples had AFM1 levels exceeding the Egyptian regulation limits [28] (Table 2) and contamination level of these samples ranged from19. 4 - 33.4 ng/kg.

On the other hand, results in (Table 2) showed that, none of the examined samples either yoghurt, processed cheese, IFMP or UHT milk samples exceeding the European Commission standards EC [29], which recorded (0.025µg//kg) in IFMP samples, (0.25 µg/Kg) for cheese samples and (0.05 µg/Kg) for milk products.

In relation to many previously reported surveys worldwide, for Infant formula milk powder samples examined by Baydar *et al.* [30] AFM1 was found in 100% of examined Infant formula milk powder samples, on the other side AFM1was detected by Meucci *et al.* [31] and Kabak [32] but with lower percentage,while El-Tras *et al.* [33]and Hosny *et al.* [9] detected it by higher percentage and for UHT milk samples examined by Cano-Sancho *et al*. [34], Abdallah *et al.* [35], Temamogullari and Kanici [23] and Hosny *et al*. [9] AFM1 was found by percentages 94.4%, 82.30%, 54.4% and 25% respectively.

In our study the results showed that the yoghurt contamination percentage was the least among all examined products. This decrease in AFM1 was attributed to factors such as low pH, the formation of organic acids

Table 1: Occurrence of AFM1 in samples collected from the local markets in Sharkia.

Dairy products	Examined samples N.	Positive samples N (%)	Min-max (ng/Kg) Or (ng/l.)	Mean ±SE (ng/Kg) Or (ng/l.)
Yoghurt	25	3(12%)	0.2 - 1.51	0.803±0.1322
Processed cheese	25	11(44%)	23.2 - 47.1	31.873±1.460
Infant formula Milk powder	15	2(13.3%)	0.1 - 0.97	0.535±0.1588
UHT milk	15	7(46.67%)	19.4 - 33.4	26.48±1.381

Table 2: Comparing the detected levels of AFM1 (µg/l) in samples of milk products to levels of the existing regulations

		Exceeding Egyptian regulations (a) 0.0 µg//kg		Exceeding EC regulations (b) (0.025µg//kg in IFMP) (0.25 µg/Kg for cheese) (0.05.µg/Kg)for milk products.	
Dairy product	Positive samples	N (%)	Range	N (%)	Range
Yoghurt	3	3(100%)	0.2 - 1.51	0(0%)	0.0-0.0
Processed cheese	11	11(100%)	23.2 - 47.1	0 (0%)	0.0-0.0
Infant formula Milk powder	2	2(100%)	0.1 - 0.97	0 (0%)	0.0-0.0
UHT milk	7	7(100%)	19.4 - 33.4	0 (0%)	0.0-0.0

(a)- [28]. (b)- [29].

or other fermentation by-products, or even to the presence of lactic acid bacteria. The low pH during fermentation alters the structure of milk proteins such as the caseins leading to formation of yoghurt coagulum [36]. The change in casein structure during yoghurt production may affect the association of AFM1 with this protein [37] causing adsorption or occlusion of the toxin in the precipitate.

The research work carried out by Khoury *et al*. [38] cleared the binding ability of AFM1 by Lactic acid bacteria (LAB) such as Lactobacillus bulgaricus and Streptococcus thermophilus and found that they were effective in reducing the extent of free AFM1 content in liquid culture medium and during yogurt processing. Therefore, LAB could be used as a biological agent for AFM1 reduction.

Several studies have been conducted regarding the effect of yogurt manufacturing on AFM1 content. Some authors reported no influence on aflatoxin M1 content [39]. In contrast, Bakirci [40] detected variable increases of AFM1 content in yogurt related to the milk.

Cheese samples in our study have the highest contamination level as they were ranged from 23.2 - 47.1 ng/Kg. Several studies in different countries have reported high or low contamination levels of AFM1 in different categories of cheese samples. These significantly variable AFM1 levels may be due to several influencing factors such as cheese manufacturing procedures and storage [41], types of cheese, conditions of cheese ripening [42], analytical methods and finally the geographical and seasonal effects [12]. In addition, the storage conditions of products including humidity and temperature are also important for toxin production.Cheese is the most potent source of a flatoxin among foods because of the AFM1 associated with the casein fraction in milk [43]. A study revealed that AFM1 levels are 3-5 times higher in cheese compared with that in the corresponding milk [44]. In addition, studies done on AFM1 concentration changes in cheese showed no significant change of concentration even after 3 months of storage [41]. It is important to mention that the stability of AFM1 during processing and storage makes it dangerous.

Results of IFMP and UHT milk samples revealed that 2 (13.3%) and 7 (46.67%) respectively, were contaminated by AFM1. In Egypt, raw milk was recurrently a cause of many public health problems due to the lack of the hygienic measures and investigations [33] and consumers are depending only on heat treatment of this milk; however AFM1 is resistant to thermal inactivation [45]. Studies have shown that there were no significant changes of AFM1 concentration after heat processing (pasteurization or boiling) or Ultra-high temperature processing (UHT) technique [22].

There are some assumptions declare that, AFM1 may be considered as communicable due to its possible transmission from food producing animals to humans and from mother to child. The lactating animal could be regarded as intermediate host also due to the biological transformation of AFB1 to AFM1 inside the animal body. Consequently, the farm animals may be considered as a reservoir for AFM1. Generally, presence of aflatoxins in animal or human bodies cause a disease named Aflatoxicosis, so the presence of AFM1 may be specified as Aflatoxicosis M1. We can presume, there- after, a novel concept to consider AFM1 as an etiological factor for a foodborne zoonosis terming Aflatoxicosis M1. [33] Aflatoxicosis causes anemia, reduction of immune function, hepatotoxicosis, hemorrhage, teratogenesis, carcinogenesis and mutagenesis. The most prevalent symptoms of aflatoxicosis in animals are reduced growth rate and poor intellectual and behavioral performance. The liver is considered a target organ for the toxic and carcinogenic effects of AF [46].

Several studies indicate that exposure to AFM1 has a possible link to an increased risk of developing cancer in humans [47]. The demonstrated toxic and carcinogenic effects of AFM1 lead the WHO-IARC [48] to change its classification from group 2 (possibly carcinogenic to humans) to group 1 (carcinogenic to humans) agents. The carcinogenicity of AFM1 may be in fluenced by the duration and level of exposure. Exposure is most likely to occur through the frequent consumption of milk and milk by-products (infant milk, cheese, butter, yoghurt) [49]. Consequently, many countries have issued strict regulations concerning the maximum permissible AFM1 levels in milk and dairy foods to protect consumers, especially children [18].

In Egypt, the incidence of hepatocellular carcinoma, HCC was doubled throughout the past decade [50]. In most African countries, aflatoxins are widely found in dietary staple foods [51].

In spite of the significance of promoting the sanitary measures of raw milk, the animal feed should be free of fungal growth, especially in current screened area which has high temperature and humidity conditions. The fungal action is activated for producing aflatoxins in hot and humid environments [52].

AFM1 can be reduced through feed decontamination using chemical, physical or biological treatments [53]. Also, using of non-nutritionally inert adsorbents can sequester the aflatoxins and reduce the absorption of toxins from the intestinal tract [54]. Chances of mycotoxin production are very small when dairy products are kept at refrigeration temperatures, Good hygiene practice is very important to fight mold spoilage. Because air is generally an effective vehicle for distribution of mold, filtration of air and even the practice of clean room techniques have been introduced in some places. Vacuum-packaging or modified atmosphere packaging is used to inhibit mold growth and application of chemical inhibitors on wrappings and product surfaces is also used [55].

CONCLUSIONS

The results of the underlying study indicated that AFM1 could be detected in dairy products marketed in Sharkia Governorate.Despite of low incidence of AFM1 in milk and other dairy products compared to other regions worldwide, more emphasis should be given to the determination of AFM 1 in milk and dairy products, because the human intakes of them especially infants and children are of considerable amounts. Therefore, it is recommended in order to ensure safety consumption of milk and milk product daily intake, aflatoxin B 1 contaminated feeding for dairy cattle should be avoided. This seems to be the most practical way. Implementing a food control system, such as the HACCP system, in the food industries is suggested as an efficient means for limiting mycotoxin contamination. Frequent analytical surveillance by food control agencies is highly recommended to control the incidence of mycotoxin contamination especially in dairy products

REFERENCES

1. Iqbal, S.Z., M.R. Asi and A. Ariño, 2011. Aflatoxin M1contamination in cow and buffalo milk samples from the North West Frontier Province (NWFP) and Punjab provinces of Pakistan. Food Additives and Contaminants: Part B, 4: 282-288.
2. Brayden, W.L., 2012. Mycotoxin contamination of the feed supply chain: implications for animal productivity and feed security. Animal Feed Science and Technology, 173: 134-158.
3. Asi, M.R., S.Z. Iqbal, A. Ariño and A. Hussain, 2012. Effect of seasonal variations and lactation times on aflatoxin M1contamination in milk of different species from Punjab, Pakistan. Food Control, 25: 34-38.
4. Van der Spiegel, M., H.J. Van der Fels-Klerx and H.J.P. Marvin, 2012. Effects of climate change on food safety hazards in the dairy production chain. Food Research International, 46: 201-208.
5. Decastelli, L., J. Lai, M. Gramaglia, A. Monaco, C. Nachtmann and F. Oldano, 2007. Aflatoxins occurrence in milk and feed in Northern Italy during 2004-2005.Food Control, 18: 1263-1266.
6. IARC (International Agency for Research on Cancer)., 1993. Some naturally occurring substances: Food items and constituents, heterocyclic aromatic amines and mycotoxins. IARC Monograph on the Evaluation of Carcinogenic Risks to Humans, World Health Organization, Lyon, France, 56: 1–391.
7. Diaz, G.J. and E. Espitia, 2006. Occurrence of aflatoxin M1 in retail milk samples from Bogota, Colombia. Food Additives and Contaminants, 23: 811-815.
8. Fallah, A.A., 2010. Aflatoxin M1contamination in dairy products marketed in Iran during winter and summer. Food Control, 21: 1478-1481.
9. Hosny, G., M. El-sadany and M. Abd Elmottale, 2014. Monitoring of Aflatoxin M1in Some Dairy Products in Local Market of Alexandria, Egypt: Attempts for Detoxification. International Research Journal of Scientific Findings, 1: 053-064.
10. Sassahara, M., N.D. Pontes and E.K. Yanak, 2005. Aflatoxin occurrence in foodstuff supplied to dairy cattle and aflatoxin M1 in raw milk in the North of Parana state. Food and Chemical Toxicology, 43: 981-984.
11. IARC (International Agency for Research on Cancer)., 2002. Some traditional herbal medicines: Some mycotoxins, naphthalene and styrene. IARC Monograph on the Evaluation of Carcinogenic Risks to Humans, World Health Organization, Lyon, France, 82: 1-556.
12. Tajkarimi, M., F. Aliabadi-Shojaee, A. Salah Nejad, H. Pursoltani, A.A. Motallebi and H. Mahdavi, 2008. A flatoxin M1 contamination in winter and summer milk in 14 states in Iran. Food Control, 19: 1033-1036.
13. Picinin, L.C.A., M.M.O.P. Cerqueira, E.A. Vargas, A.M.Q. Lana, I.M. Toaldo and M.T. Bordignon-Luiz, 2013. Influence of climate conditions on a flatoxin M1 contamination in raw milk from Minas Gerais State, Brazil Food Control Journal, 31: 419-424.

14. Unusan, N., 2006. Occurrence of aflatoxin M1 in UHT milk in Turkey. Food and Chemical Toxicology, 44: 1897-1900.
15. Cavaliere, C., P. Foglia, E., Pastorini, R. Samperi and A. Laganà, 2006. Liquid chromato-graphy/tandem mass spectrometric confirmatory method for determining aflatoxin M1 in cow milk comparison between electrospray and atmospheric pressure photoionization sources. Journal of Chromatography, 1101: 69-78.
16. Anfossi, L., M. Calderara, C. Baggiani, C. Giovannoli, E. Arletti and G. Giraudi, 2008. Development and application of solvent-free extraction for the detection of aflatoxin M1 in dairy products by enzyme immunoassay. Journal of Agricultural and Food Chemistry, 56: 1852-1857.
17. Li, P., Q. Zhang and W. Zhang, 2009. Immunoassays for aflatoxins. Trends in Analytical Chemistry, 28: 1115-1131.
18. Elkak, A., O. El Atat, J. Habib and M. Abbas, 2012. Occurrence of a flatoxin M1 in cheese processed and marketed in Lebanon. Food Control, 25: 140-143.
19. WHO- Principals for evaluating health risk from chemicals during infancy and early Childhood. 1986. The need for a special approach. Environmental Health Criteria No. 59, Geneva.
20. Galvano, F., V. Galofaro and G. Galvano, 1996. Occurrence and stability of aflatoxin M1 in milk and milk products: a worldwide review. Journal of Food Protection, 59: 1 079-1090.
21. Akkaya, L., Y.O. Birdane, H. Oguz and M. Cemek, 2006. Occurrence of aflatoxin M1 in yogurt samples from Afyonkarahisar, Turkey. Bulletin of the Veterinary Institute in Pulawy, 50: 517-519.
22. Henry, S.H., T. Whitaker, I. Rabbani, J. Bowers, D. Park, W. Price and F.X. Bosch, 2001. Aflatoxin M1. Joint FAO/WHO Expert Committee on Food Additives (JECFA), pp: 47.
23. Temamogullari, F. and A. Kanici, 2014. Short communication: Aflatoxin M1 in dairy products sold in Şanliurfa, Turkey. J. Dairy Sci., 97: 162-165.
24. Iqbal, S.Z. and M.R. Asi, 2013. Assessment of aflatoxin M1in milk and milk products from Punjab, Pakistan. Food Control, 30 :235-239.
25. Tavakoli, H.R., M. Riazipour, A. Kamkar, H.R. Shaldehi and A.S.M. Nejad, 2012. Occurrence of aflatoxin M1in white cheese samples from Tehran, Iran. Food Control, 23: 293-295.
26. Duarte, S.C., A.M. Almeida, A.S. Teixeira, A.L. Pereira, A.C. Falcão, A. Pena and C.M. Lino, 2013. Aflatoxin M1in marketed milk in Portugal: Assessment of human and animal Exposure. Food Control, 30: 411-417.
27. Mohajeri, F.A., S.A. Ghalebi, M. Rezaeian, H.R. Gheisari, H.K. Azad, A. Zolfaghari, A. Aziz and A. Fallah, 2013. aflatoxin M1contamination in white and Lighvan cheese marketed in Rafsanjan, Iran. Food Control, 33: 525-527.
28. Egyptian Standard 1- 1875. 2007. "Maximum levels of mycotoxin for foods and feeds, part-1: Aflatoxins" Egyptian Organization for Standardization and Quality.
29. European Commission Regulation (EC). 2006. No 1881/2006 of 19 December 2006 setting maximum levels for certain contaminants in foodstuffs. Official Journal of European Union, 364:5-24. downloaded *from http://eur-lex.europa.eu/ LexUriServ/ LexUriServ.do?uri=OJ:L:2006:364:0005:0024:EN:PDF* on 12/8/2014.
30. Baydar, T., P. Erkekoglu, H. Sipahi and G. Sahin, 2007. Aflatoxin B1, M1 and Ochratoxin A Levels in Infant Formulae and Baby Foods Marketed in Ankara, Turkey. Journal of Food and Drug Analysis, 15: 89-92.
31. Meucci, V., E. Razzuolia, G. Soldania and F. Massartb, 2010. Mycotoxin detection in infant formula milks in Italy. Food Additives and Contaminants, pp: 1-26.
32. Kabak, B., 2012. A flatoxin M1 and ochratoxin A in baby formulae in Turkey: Occurrence and safety evaluation. Food Control, 26: 182-187.
33. El-Tras, W.F., N.N. El-Kady and A.A. Tayel, 2011. Novel food-borne zoonosis. Food and Chemical Toxicology, 49: 2816-2819.
34. Cano-Sancho, G., S. Marin, A. J. Ramos, J. Peris-Vicente and V. Sanchis, 2010. Occurrence of aflatoxin M1 and exposure assessment in Catalonia (Spain). Revista Iberoamericana de Micología, 27: 130-135.
35. Abdallah, M.I.M., M.S. Bazalou and M.Z. Al-Julaifi, 2012. Determination of aflatoxin M1 concentration in full fat cow's UHT milk sold for consumption in Najran-Saudi regarding its public health significance. Egypt. J. of Appl. Sci., 27: 40-54.
36. Govaris, A., V. Roussi, A. Koidis and N.A. Botsoglou, 2001. Distribution and stability of aflatoxin M1 during processing, ripening and storage of Telemes cheese. Food Additives and Contaminants, 18: 437-443.

37. Brackett, R.E. and E.H. Marth, 1982. Association of aflatoxin M1 with casein. Zeitschrift fur Lebensmittel Untersuchung und Forschung, 174: 439-441.
38. Khoury A., A. El Atoui and J. Yaghi, 2011. Analysis of aflatoxin M1 in milk and yogurt and AFM1 reduction by lactic acid bacteria used in Lebanese industry. Food Control, 22: 1695-1699.
39. Blanco, J.L., B.A. Carrion, N. Liria, S. Diaz, M.E. Garcia, L. Domingues and G. Suarez, 1993. Behavior of aflatoxin during manufacture and storage yogurt. Milchwissenchaft, 48: 385-387.
40. Bakirci, I., 2001. A study on the occurrence of aflatoxin M1 in milk and milk products produced in Van province of Turkey. Food Control, 12: 45-51.
41. Deveci, O., 2007. Changes in the concentration of a flatoxin M1 during manufacture and storage of white pickled cheese. Food Control, 18: 1103-1107.
42. Oruc, H.H., R. Cibik, E. Yilmaz and O. Kalkanli, 2006. Distribution and stability of a flatoxin M1 during processing and ripening of traditional white pickled cheese. Food Additives and Contaminants, 23: 190-195.
43. Yapar, K., M. Elmali, A. Kart and H. Yaman, 2008. Aflatoxcin M1 levels in different type of cheese products produced in Turkey. Medycyna Weterynaryjna, 64: 53-55.
44. Prandini, A., G. Tansini, S. Sigolo, L. Filippi, M. Laporta and G. Piva, 2009. On the occurr-ence of aflatoxin M1 in milk and dairy products. Food and Chemical Toxicology, 47: 984 -991.
45. Deshpande, S.S., 2002. Fungal toxins. Handbook of food toxicology, In: Ed., Deshpande, S.S. pp: 387-456. Marcel Decker, New York.
46. Buldu, H.M., A.N. Koc and G. Uraz, 2011. Aflatoxin M1 contamination in Cow's milk in Kayseri (Central of Turkey). Turkish Journal of Veterinary and Animal Sciences, 35: 1-5.
47. Wogan, G.N., S.S. Hecht, J.S. Felton, A.H. Conney and L.A. Loeb, 2004. Environmental and chemical carcinogenesis. Seminars in Cancer Biology, 14: 473-486.
48. WHO-International Agency for Research on Cancer (IARC), 2002. Monograph on the evaluation of carcinogenic risks to humans. WHO, 82: 171-275.
49. Caloni, F., A. Stammati, G. Friggè and I. De Angelis, 2006. A flatoxin M1 absorption and cytotoxicity on human intestinal in vitro model. Toxicon, 47: 409-415.
50. Iyer, P., A. Zekri, C. Hung, E. Schiefelbein, K. Ismail, A. Hablas, I.A. Seifeldin and A.S. Soliman, 2010. Concordance of DNA methylation pattern in plasma and tumor DNA of Egyptian hepatocellular carcinoma patients. Exp. Mol. Pathol., 88: 107-111.
51. Wild, C.P. and P.C. Turner, 2002. The toxicology of aflatoxins as a basis for public health decisions. Mutagenesis, 17: 471-481.
52. Lamplugh, S.M., F. Apeagyei, D.D. Mwanmut and R.G. Hendrickse, 1988. Aflatoxins in breast milk, neonatal cord blood and serum of pregnant women. BMJ (Clin. Res. Ed.), 296(6627): 968.
53. Piva, G., F. Galvano and A. Pietri, 1995. Detoxification method of aflatoxins. Rev. Nutr Res., 15: 767-776.
54. Nageswara, R.S.B. and R.C. Chopra, 2001. Influence of sodium bentonite and activated charcoal on aflatoxin M1 excretion in milk of goats. J. Small Rumin. Res., 41: 203-213.
55. Sørhaug, T., 2011. Spoilage Molds in Dairy Products Encyclopedia of Dairy Sciences (2ed.) pp: 780-784.