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Full Length Research Paper

Mycotoxines and/or aflatoxines in milk and milk products: Review

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This review was conducted to evaluate micotoxin and or aflatoxines in milk and milk products through critical review. Mycotoxins are secondary metabolites produced by the three major fungus genera Aspergillus, Penicillium and Fusarium. The contamination of food and feeds by Mycotoxin remains a worldwide problem, according to FAO estimation up to 25% of the world's food crops are significantly contaminated with mycotoxin. Aflatoxin is one of aclasses of mycotoxin. The major aflatoxins are afflation B1, B2, G1 and G2. Aflatoxin M1 (AFM1) is the hepatic hydroxylated metabolites of AFB1. AFM1 is found in milk and milk products obtained from livestock that have ingested AFB1contaminated feed. In lactating animals the conversion rate of AFB1 to AFM1 ranges between 0.5 and 6%. Several research workers reported that there is a linear relationship between the amount of AFM1 in milk and AFB1 in feed which is consumed by dairy cattle. Aflatoxin M1 in milk and milk products is considered to pose certain hygienic risks for human health. These metabolites are not destroyed during the pasteurization and heating process. Many countries standards limits of Aflatoxins M1 ranged between 0 to 0.5 ppb, in milk and dairy products.

Key words: Mycotoxin, aflatoxin, milk, milk products.

INTRODUCTION

Mycotoxins are toxic secondary metabolites naturally produced by molds (Aspergillus, Fusarium and Penicillium spp.) (Reddy et al, 2010). It is well known to cause toxicities to humans and animals (Shetty and Jespersen, 2009). After infesting crops, fungi synthesize the toxins, which will be transmitted to the final food products. The classes of mycotoxins with relevance to health are: aflatoxins, ochratoxins, trichothecenes, zearalenone, fumonisins, tremorgenic toxins and ergot alkaloids (Bunaciu, 2010). Aflatoxins are a group of structurally-related toxic compounds produced by certain strains of the fungi Aspergillus flavus and A parasiticus (Ayhan et al, 2010). Since the toxin-producing mold was identified as Aspergillus flavus in 1960 and the toxin was given the name Aflatoxin by virtue of its origin (A. flavus--> Afla). Aflatoxins have sub-acute and chronic effects such as liver cancer, chronic hepatitis, jaundice, hepatomegaly and cirrhosis in humans (Hampikyan et al, 2010). Over 20 naturally occurring aflatoxins are known, the moulds Aspergillus flavus and A. parasiticus produce exclusively aflatoxin B1, B2, G1 and G2, and all the other aflatoxins are derivates of these four. While AFB1 is the most toxic in the group and the toxicity is in the order of B1 > G1 > B2 > G2 (Dorner, 2004). Aflatoxin B1 (AFB1) present in feed of lactating animals gets transformed to 4-hydroxylated metabolite in liver and is excreted in milk as aflatoxin M1 (AFM1). About 1-3% ingested AFB1 is converted into AFM1 (Ali et al., 1999), but it varies from animal to animal, from day to day and from one milking to the other. The presence of AFM1 in milk poses a major risk for humans, especially children, as it can have immunosuppressive, mutagenic, teratogenic, and carcinogenic effects (Sefidgar et al., 2011).

If milk is sold, commercially should be checked for aflat oxinM1. When aflatoxinM1 is found at concentrations of 0 .5 parts per billion (ppb) or greater, the milk is discarded because it cannot be used for products that go in to the human food supply (.Ovidiu et al 2013). The AFM1 could be detected in milk 12-24 h after the first AFB1 ingestion, reaching a high level after a few days. When the intake of

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AFB1 is finished, the AFM1 concentration in the milk decreases to an undetectable level after 72 h (Hampikyan et al, 2010).

Therefore to know more about the effect of AFM1 in milk and milk products, this review is made from different scientific papers to contribute scientific knowledge for the world.

MYCOTOXIN/AFLATOXINE / IN MILK AND MILK PRODUCTS

What is Mycotoxins?

In the 1960s more than 100,000 young turkeys on poultry farms in the United Kingdom died in a period of a few months from an unidentified disease, which was named "turkey × disease". Ducklings and other poultry animals were also affected, and high mortalities were observed. A careful survey of the inputs and environment of the affected farms indicated that the disease was associated with feeds and specifically with peanut meal imported from Brazil. A disease with symptoms typical of turkey × disease was reproduced when animals were fed the same peanut meal. Intensive investigations were then carried out on the suspected ingredient to identify the nature of the toxin, which was soon found to be of fungal origin. The toxin-producing fungus was identified as *Aspergillus flavus* (Nesbitt *et al.*, 1962) and the toxin was accordingly called aflatoxin/Mycotoxin. While all mycotoxins are of fungal origin, not all toxic compounds produced by fungi are called mycotoxins.

The term Mycotoxin is derived from the Greek word ‘mycos’ meaning fungus(mould), and the Latin word ‘toxicum’, which means poison (Jouany et al,2009). Mycotoxins are produced by fungi through their secondary metabolism. Mycotoxin concentration can therefore be independent of the growth of the fungi, which is associated with the primary metabolism. The diversity of the compounds formed and the specificity of the fungal strain for mycotoxin production result from the secondary metabolism, which is usually activated by signals from the environment (cold, heat, dryness, fungicide, etc.). Among the numerous mycotoxins, several groups have been identified, produced by the three major fungus genera Aspergillus, Penicillium and Fusarium (Jouany et al, 2009). According to Jouany et al. (2009), the same mycotoxin can be produced by several different fungi, and the same fungus can generate several mycotoxins. Mycotoxins present in food products and animal feeds are an important problem concerning food and feed safety and significant economic losses are associated with their impact on human and animal health (Shundo et al., 2009b). In addition to being acutely toxic, some mycotoxins are now linked with the incidence of certain types of cancer and it is this aspect which has evoked global concern over feed and food safety, especially for milk and milk products.

What is Aflatoxin?

Aflatoxins belong to the class of mycotoxins and it is a group of approximately 20 related fungal metabolites generally produced by Aspergillus species, namely A. flavus, A. parasiticus, A. ochraceoroseus, A. bombycis, A. nomius, A. fumigitus and A. pseudotamari (Cheraghali et al., 2007). These fungi belong to the class Hyphomycetes, subdivision Deuteromycotina and family Aspergillaceae (Hamid, 2011).

Aspergillus flavus and A. parasiticus are economically important moulds that produce exclusively aflatoxin B1, B2, G1 and G2, and all the other aflatoxins are derivates of these four (Felicia Wu, 2011). The four major naturally produced aflatoxins are known as B1, B2, G1, and G2. “B” and “G” refer to the blue and green fluorescent colors produced under UV light on thin layer chromatography plates, while the subscript numbers 1 and 2 indicate major and minor compounds, respectively (Felicia Wu, 2011). Aflatoxin B1, the most toxic of the aflatoxins, is the most potent naturally occurring chemical liver carcinogen known. Specific P450 enzymes in the liver metabolize aflatoxin into a reactive oxygen species (aflatoxin-8,9-epoxide), which may then bind to proteins and cause acute toxicity (aflatoxicosis) or to DNA and induce liver cancer (Wu and Khlangwiset, 2010).

Aspergillus flavus and A. parasiticus colonize a wide variety of food commodities including maize, oilseeds, spices, groundnuts, tree nuts, milk, and dried fruit (Strosnider et al. 2006). Whether these fungi produce aflatoxin depends on drought stress and rainfall, suitability of crop genotype for its climate, insect damage, and agricultural practices (Wu and Khlangwiset, 2010). These fungi can also produce aflatoxin in “postharvest” conditions: storage, transportation, and food processing. Aflatoxin contamination is a particular problem in maize, oilseeds, spices, peanuts, tree nuts (almonds, pistachios, hazelnuts, pecans, Brazil nuts, and walnuts), milk (in the form of aflatoxin B1’s metabolite aflatoxin M1), and dried fruit (Shephard, 2008). Maize and peanuts are the main sources of human exposure to aflatoxin because they are so highly consumed worldwide and unfortunately are also the most susceptible crops to aflatoxin contamination (Wu and Khlangwiset, 2010). Figure 2 (Wu 2010) depicts the pathway by which aflatoxin accumulates in food crops and contributes to various adverse human health effects.

The International Agency for Research on Cancer (IARC, 2002) has classified aflatoxin B1 as a group 1 carcinogen (that means carcinogenic to humans) since 1987, and a group 1 carcinogenic agent since 1993 due to the exposure to hepatitis B virus (Castegnaro and McGregor, 1998). AFB1 is the most prevalent aflatoxin usually found in cases of aflatoxicosis, and is responsible for acute toxicity, chronic toxicity, carcinogenicity, teratogenicity, genotoxicity and immunotoxicity. AFM1 is a metabolic derivate of AFB1, and AFM2 is a metabolic derivate of AFB2; both come from the metabolism of some animals, and are normally found in milk and urine(Strosnider et al., 2006).

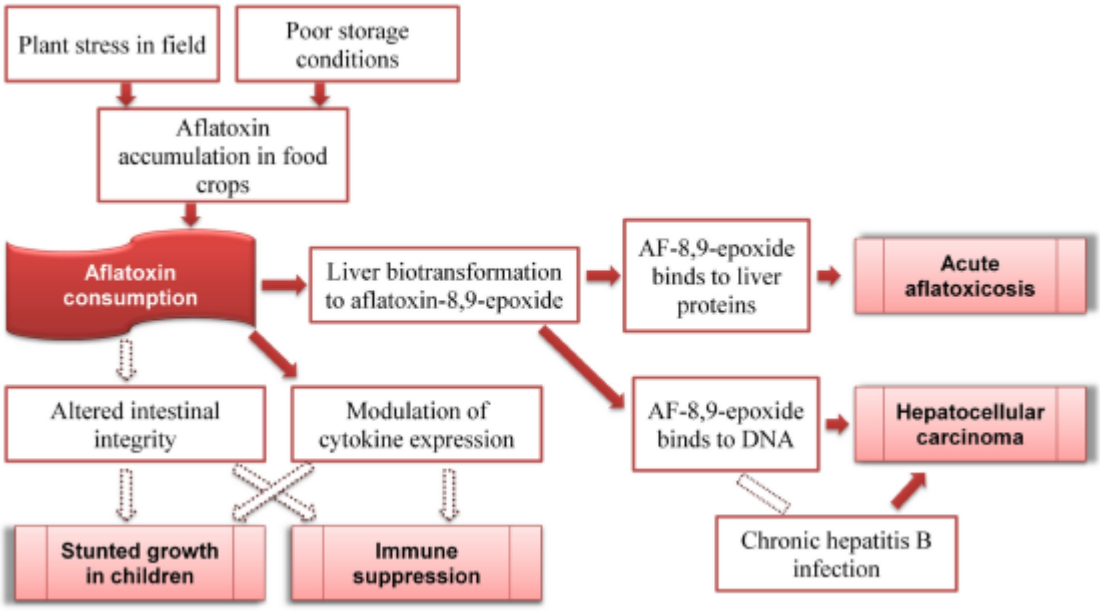


Figure 1. *Aflatoxin and disease pathways in humans.*
Source: Wu (2010). The darker arrows in Figure 1 denote linkages that have been well-established in agricultural and toxicological research, while the white arrows denote linkages that have been relatively less well-established (Wu, 2010).

FM1 is the most economically important AFB1derivative that found in milk and milk products.

Changing of AFB1 to AFM1

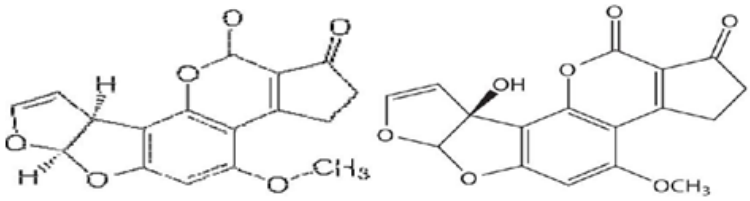
Aflatoxin B1 (AFB1) present in feed of lactating animals gets transformed to 4-hydroxylated metabolite in liver and is excreted in milk as aflatoxin M1 (AFM1). The AFM1 could be detected in milk 12-24 h after the first AFB1 ingestion, reaching a high level after a few days. When the intake of AFB1 is finished, the AFM1 concentration in the milk decreases to an undetectable level after 72 h (Hampikyan et al, 2010). According to the report of Gimeno, (2004) and Özdemir, (2007), cows can transform AFB1 into AFM1 within 12-24 hours after ingestion of contaminated food. Even at six hours after ingestion, AFM1 residues can appear in milk, and the highest levels are reached after a few days. When the intake of AFB1 is stopped, the AFM1 concentration in the milk decreases to an undetectable level after 72 hours. Gimeno (2004) and Özdemir (2007), observed that there was a linear relationship between AFB1 dose and excretion of AFM1 into ewes' milk.

About 1-3% ingested AFB1 is converted into AFM1 (Ali et al., 1999), but it varies from animal to animal, from day to day and from one milking to the other. Gimeno (2004) reported that in dairy cows, the relationship between the concentration of AFB1 in the final consumed ration and AFM1 excreted in breast milk could be 300:1; nevertheless this relation is only an approximation because the range is from 34:1 to 1600:1. In Holstein dairy cows consuming final rations with 80, 86, 470, 557, 1493 and 1089 µg of AFB1/Kg (ppb) on dry substance, there were found in milk AFM1 concentrations of 1.5, 0.245, 13.7, 4.7, 12.4 and 20.2 mg/L (ppb) respectively. On the other hand, when diet of Brindle cows was contaminated with 540 ppb of AFB1, 0.92 ppb of AFM1 was produced. In other cows, the values of contamination in the diet ranged between 64 and 1799 ppb of AFB1 giving some residues in milk between 0.35 and 14.2 ppb of AFM1.

For high-yielding dairy cows producing up to 40 kg of milk per day, Veldman *et al.* (1992) found a carry-over percentage as high as 6.2%. Changes in the plasma-milk barrier and the consumption of large amounts of concentrated feeds in high-merit dairy cows may explain the increase in the carry-over rate of AFM1 in milk. According to Pettersson (1998) AFB1 and AFM1 has a strong correlation, and he proposed the following equation ($r2 = 0.915$) to estimate the transfer of AFM1 in milk:

AFM1 (ng/kg milk) = 10.95 + 0.787 × (µg AFB1 intake per day)

This equation indicates that the animals must ingest less than 50 and 25 µg AFB1 per day to comply with the European regulatory levels of contamination in milk set at



Aflatoxin B1 Aflatoxin M1

Figure 2. Chemical Structur of Aflatoxin B 1 and Aflatoxin M1 .

0.05 and 0.025 µg/kg of milk for adults and infants, respectively. Thus cows must ingest less than 10 and 5 kg of feed contaminated at the maximum authorised level (5 µg AFB1/kg feed for dairy cattle) to maintain a safe level of AFM1 in milk.

What is Aflatoxins M1

AFM1 is a metabolite of Aflatoxin B1 (AFB1) and originally discovered in milk of humans and animals fed on moldy grains containing AFB1 (Qais**,** 2012). With an intake of AFB1 for 2-60 mg / cow / day, AFM1 residues in milk could range between 1 and 50 ppb. There have been found differences between the amounts of AFM´s produced by different bovine species.

The AFM1 distribution in some dairy foods made from contaminated milk is approximately: 40-60% in cheese, 10% in butterfat and <2% in buttermilk. AFM1 is highly soluble in water, so it is incomprehensible why this toxin is deposited in the cheese but not in the milk whey (Yusef & Marth, 1989).

The carcinogenicity of AFM1 is about ten times less than that of AFB1, and for this reason has been included in the class 2B by International Agency for Research on Cancer. The quantity of AFM1 in the milk depends on the concentration of AFB1 in the contaminated feed. It has been reported that milk is one of the main risk factors of human exposure to AFM1. Infants are the foremost milk consumers, which make them more susceptible to the adverse effects of mycotoxins (Shundo et al., 2009a). 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. The milk could be established as a major carrier of AFM1 which affects the human health.

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. The main target organ in mammals is the liver so Aflatoxicosis is primarily a hepatic disease. Aflatoxins also cause decreased milk production. The occurrence of AFM1 in milk and milk products is a serious problem of food hygiene.

Aflatoxin contamination in milk and products is produced in two ways. Either toxins pass to milk with ingestion of feeds contaminated with Aflatoxin, or it results as subsequent contamination of milk and milk products with fungi. AFM1 has been reported to cause certain hygiene difficulties in milk and milk products used for food. During the obtaining of cream, AFM1 disperses heterogeneously in milk. AFM1 is not destroyed during the pasteurization process or in yoghurt and cheese making.

AFM1 in Milk

Milk is the most important source of calcium and phosphorus of human body and due to having essential amino acids, has an important status in supplying the body's protein needs. Studies have shown that there is a close relationship between consumption of milk and health status of people in terms of efficiency, Intelligence quotient (IQ), reducing the risk of infectious diseases, regulation of metabolic activities, decreasing blood pressure, increasing beneficial blood lipids (High-density lipoprotein), preventing from colon cancer and osteoporosis (Hjartaker et al., 2002)

Milk is a key contributor to improve nutrition and food security particularly in developing countries and play a significant role in reducing poverty and malnutrition (Kazemi et al, 2013). However milk, as a liquid, is a highly variable product that rapidly loses its quality and spoils if not to be treated. Since milk may be processed in numerous ways, the effects of storage and processing on stability and distribution of AFM1 are of great concern. Kiermier and meshaley (1977) reported the effect of cold treatments. They observed that detectable AFM1 decreased by 11 to 25% after 3 days at 5ºC, 40% after 4 days at 0ºC, and 80% after 6 days at 0ºC. Whereas, McKinney et al. (1973) revealed that freezing at -18ºC for 30 days resulted in an apparent loss of 14%, with 85% lost after 53 days. Stoloff et al. (1981) suggested less degradation of AFM1 at -18ºC with insignificant loss after 53 days. As to the effect of heating contradictory data have been reported. Kiermeier and Mashaley (1977), reported that various heat–time treatments caused reductions in the AFM1 concentrations of milks between

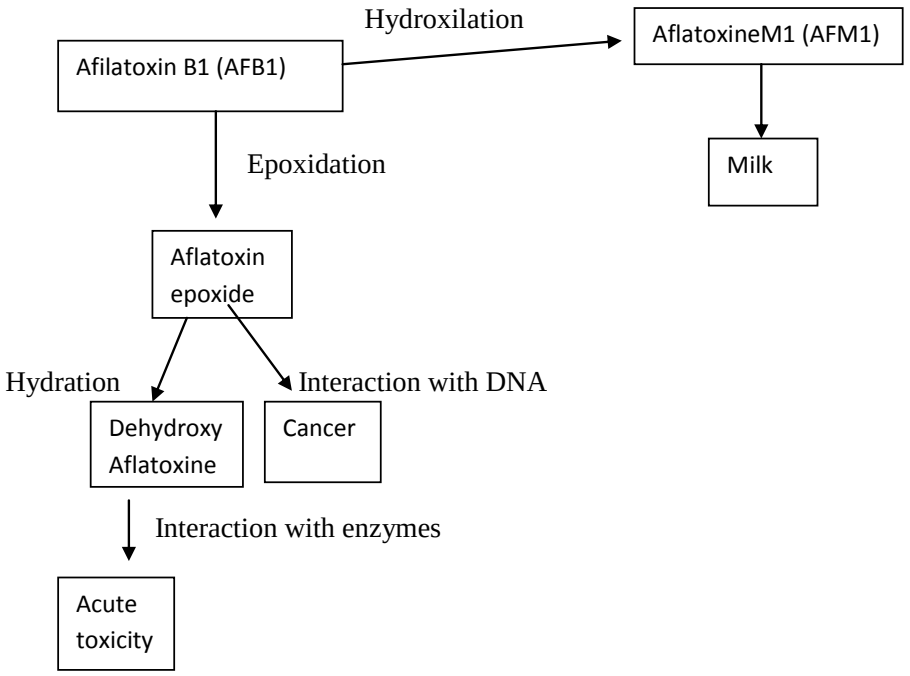


Figure 3. *Some metabolic products from AFB1*

12% and 40%. Choudhary et al. (1998) studied the effect of various heat treatments on AFM1 content of cow's milk and reported that sterilization of milk at 121 °C for 15 min caused 12.21% degradation of AFM1, whereas boiling decreased AFM1 by 14.50%. They concluded that destruction of AFM1 depends on time and temperature combination of the heat treatment applied. In an investigation Conducted by Bakirci (2001), it was observed that pasteurization caused a decrease in the level of AFM1 at the rate of 7.62%. Deveci (2007) showed that pasteurization can partially reduce the amount of AFM1 in milk. However, some reports showing that aflatoxins are stable during heat-treatments such as pasteurization and sterilization (Govaris et al. 2001). AFM1 distribution in milk is not homogeneous. Cream separation can affect AFM1 distribution, since 80% is partitioned in the skim milk portion (Grant and Carlson, 1971) because of AFM1 binding to casein (Brackett, 19982a). An amount of 30% of AFM1 is indeed estimated to be associated with the non fat milk solids and in particular with casein. Contradictory data have been reported on the influence of milk concentration on AFM1. Kiermeier (1973) reported no losses of AFM1, whereas some authors observed losses ranging from 60 to 75% following milk concentration (Moreau, 1976). Data from the studies on the occurrence of AFM1 in milk since the 1990s are reported in Table 1.

Many authors showed that seasonal effect influences concentration of aflatoxin M1. They reported higher concentration of AFM1 in cold seasons as compared to hot seasons (Bilandzic et al., 2010), the reason being in winters mostly milking animals are fed with compound feeds and thus concentration of aflatoxin B1 increases which in turn enhances AFM1 concentration in milk. Moreover, temperature and moisture contents also affect the presence of aflatoxin B1 in feeds. A. flavus and A. parasiticus can easily grow in feeds having moisture between 13% and 18% and environmental moisture between 50% and 60%, furthermore, they can produce toxin (Jay, 1992). Another reason of low AFM1 level in summer may be attributed to out-pasturing of milking cattle.

Aflatoxin M1 (AFM1) in milk is considered to pose certain hygienic risks for human health. These metabolites are not destroyed during the pasteurization and heating process. A total of 85 pasteurized milk samples were analyzed for AFM1 with the ELISA technique, from these samples seventy-five samples (88.23%) were found to be contaminated with AFM1 and 48 samples (64%) exceeded the legal level of AFM1 in milk according to the Turkish Food Codex and Codex Alimentarius limit (50 ng/kg). In Iran, 98 samples were positive for AFM1 with an overall mean level of 0.053 µg/L. Levels of the toxin were also higher in winter and spring than in summer and autumn (Tajkarimi et al., 2007) while in Sarab City of Iran, 77% (total 111 raw milk samples) were contaminated with AFM1 levels (range between 0.015 and 0.280 µg/L) and 40% of the positive

Table 1. Occurrence and content of AFM1in Milk Samples.

Year	Region	Milk type	Samples	+ samples	Range(ng/liter)	Reference
1991	Kazakhstan	C	*	0	NA	Nikov et al.
1992	Cuba	C	85	22	>500	Margolles et al.
1993	Japan	C	37	0	NA	Tabata et al.
1994	USA	D	10	4	95	Kawamura et al.
	China	D	28	21	102.8	
	Italy	D	14	0	NA	
	New Zealand	D	3	0	NA	
	Poland	D	3	1	85	
1995	India	R	504	89	100-3.500	Rajan et al.
1996	Italy	UHT	161	125	<1- 23.5	Galvano et al.
		D	92	49	<1- 79.6	
1998	Kuwait	C	9	5	*	Srivastava et al
		R	7	5	*	
1999	Portugal	R	31	25	*	Martins
1999	Portugal	UHT	70	60	*	Martins
1999	Argentina	R	56	6	Dec-30	Lopez et al.
		PW	5	4	Oct-14	
		P	16	8	Oct-17	
1999-2000	Iran	R	186	119	≤ 10-410	Ghiasian et al.
2001	Turkey	R	90	79	12.5-123.6	Bakirci
2001	Iran	R	111	85	15-280	Kamkar
2002-2003	Brazil	R	22	13	*	Shundo and Sabino
2002-2003	Brazil	P	43	32	*	Shundo and Sabino
2002-2003	Brazil	UHT	34	34	*	Shundo and Sabino
2002	Greece	R	54	*	*	Grigoriadou et al.
2003	Iran	P	624	624	*	Alborzi et al.
2004	Turkey	P	3	2	*	Gurbay et al.
		UHT	24	14	*	
2004	Iran	R	319	172	15.4[a]	Tajkarimi et al
2003-2004	Iran	R	98	*	53[a]	Tajkarimi et al.
2003-2004	Italy	R	208	36	5-36.1	Virdis et al.
*	Turkey	P	85	75	*	Celik et al.
2004-2005	Italy	R	344	5	*	Decastelli et al
2004-2005	Brazil	P	12	7	11-161	Oliveira and Ferraz
		UHT	12	10	11-161	
2005	Pakistan	R	168	168	10-700	Hussain and Anwar
2005	Iran	P	128	128	31-113	Oveisi et al.
2005-2007	Kuwait	R	177	176	4.9-67.8	Dashti et al.
2006	Iran	R	*	*	43-59	Mohammadi et al.
2006	Iran	P	110	110	Aug-89	Karimi et al.

Table 1. Cont.

2006	*Iran*	*PW*	*42*	*42*	*51-914*	*Kamkar*
2006	*Iran*	*P*	*	*	*178.8-253.5*	*Sefidgar et al.*
2006-2007	*Iran*	*R*	*240*	*226*	*12.56[a]*	*Mohammadian et al.*
		P	*32*	*31*	*12.43[a]*	
2007	*Iran*	*P*	*	*	*23.22[a]*	*Mohammadi et al.*
2007	*Iran*	*UHT*	*	*	*19.53[a]*	*Mohammadi et al.*
2007	*Pakistan*	*B*	*55*	*19*	*13[a]*	*Hussain et al.*
		C	*40*	*15*	*14[a]*	
		G	*30*	*6*	*2[a]*	
		S	*24*	*4*	*2a*	
		Ca	*20*	*0*	*0*	
2008	*Iran*	*P*	*50*	*50*	*	*Movassagh Ghazani*
		UHT	*49*	*49*	*	
2007-2008	*Serbia*	*C*	*3*	*	*Oct-50*	*Polovinski- Horvatović et al.*
2007-2008	*Iran*	*UHT*	*210*	*116*	*8-249*	*Heshmati and Milani*
2007-2008	*Iran*	*C*	*75*	*59*	*60.1[a]*	*Rahimi et al.*
		B	*75*	*29*	*31.9[a]*	
		Ca	*40*	*5*	*19.0[a]*	
		S	*51*	*19*	*28.1[a]*	
		G	*60*	*19*	*30.1[a]*	
2008	*Spain*	*R*	*72*	*68*	*9.69[a]*	*Cano-Sancho et al.*
2010	*Iran*	*C*	*88*	*74*	*13-394*	*Fallah*
		G	*65*	*28*	*13-55*	
		S	*72*	*43*	*15-102*	
2011	*Iran*	*P*	*91*	*66*	*13-250*	*Fallah et al.*
2009	*Croatia*	*R*	*61*	*	*0.6-58.7*	*Bilandzic et al.*
2009	*Sudan*	*R*	*44*	*42*	*220-6900*	*Elzupir and Elhussein*
		PW	*12*	*8*	*11-161*	

P: pasteurized; D: Dry milk; PW: Powdered Milk; R: Raw Milk; C: Cow milk; B: Buffalo milk; S: Sheep milk; G: Goat milk; Ca: Camel milk; NA: Not applicable
* Not reported
a Average of contaminate

samples exceeded the tolerance limit of 0.050 µg/L (Kamkar, 2005). In Turkey, 47% of the 129 analyzed samples contained AFM1 levels exceeding the EU accepted limit (Unusan, 2006). In North African countries, randomly selected samples of raw cow milk were contaminated with AFM1 (rang between 30 and 3130 ng/L) (Elgerbi et al., 2004). In India, the incidence of contamination of AFM1 ininfant milk, milk based cereal weaning food and liquid milk samples was almost in the magnitude of 87% (Rastogi et al., 2004), with 99% of contaminated samples exceeding the EU/Codex recommended limits. Hussain *et al.* (2008) reported 42.5% and 52.5% milk samples were found contaminated from buffalo and cow, respectively, with the toxin. AFM1 levels in almost all the contaminated milk samples exceeded 50 ng/kg. From 11,831 processed samples from the territory of the European Union, 280 were from individual small farms, the concentration of aflatoxin M1 in all the samples greater than 0.05 µg/l was detected in only 0.06% of samples, which indicates that the incidence of this mycotoxin are extremely low (EFSA, 2004).

Occurrence of aflatoxin in cheese can be owing to three possible causes:

- AFM1 present in raw milk as a consequence of carryover of AFB1 from contaminated animal feed to milk.

- Synthesis of aflatoxin (B1, B2, G1, and G2) by fungi that grow on
- Cheese (although the low level of carbohydrate does not make it a very suitable substrate)
- The use of powdered milk contaminated with AFM1 for cheese production.

Contrasting data have been reported on the influence of cheese preparation on AFM1 recovery.

Studies performed in the early years showed variable losses of AFM1 during cheese production: 65%, 47%, <20% and <15% according to Stubblefield and Shannon (1974), respectively. In contrast, later investigations of several authors (Deveci, 2007) reported increases in AFM1 concentration in cheese as a function of cheese type, technologies, and the amount of water eliminated during processing. For example, Mohammadi et al. (2008) investigated some factors, which are involved in the process of making Iranian white brine cheese. They reported that some factors such as renneting temperature, press time, and saturated brine pH affected the amount of water eliminated and in turn the content of AFM1 in the cheese curds. However, many results have been drawn from experiments in which the processed milk contained the toxin at high levels, which seldom appear in the practice. Therefore, additional investigations should verify the influence of cheese making on AFM1 occurrence to avoid uncertainty in actual practice when the concentration of the toxin in the processed milk is at around the maximum permissible level of 0.05 mg/kg that is frequently recorded in monitoring programmes.

The increase in AFM1 concentration in cheese has been ascribed to the affinity of AFM1 for casein (Brackett and Marth (1982a) suggested that since is possible to extract AFM1, it must not be covalently bound but linked by hydrophilic interactions hydrophobic areas of the casein. According to Dosako et al. (1980), AFM1 is a water-soluble component and due to the hydrophobic sides of the casein molecule, AFM1 has affinity to casein of milk. Therefore, they defined a factor named “Enrichment Factor” (EF) for cheeses.

Further surveys should be done to find as for cheese manufacture influences on AFM1 distribution. Some tests have been carried out on several kinds of cheeses as to overall stability of AFM1 during ripening and storage. Dragacci et al. (2001) reported that the concentrations of AFM1 in Camembert cheese were higher at the beginning than at the later time of ripening. These results were in agreement with studies by Govaris et al. (2001). Such results however, conflict with reports of earlier studies that indicate different behaviour of AFM1 in various other types of cheeses. Thus, in Camembert , Cheddar and Brick cheeses stored for 3, 14 and 6.5 months, respectively, the concentration of the toxin increased during the early stage of their ripening to decrease thereafter to reach about its initial concentration at the beginning of ripening (Brackett et al., 1982). On the other hand, the concentration of AFM1 in Parmesan cheese started high at the beginning of the ripening period, decreased until about the fifth month and then slowly increased up to the tenth month of storage (Brackett and Marth 1982d). In contrast, the AFM1 content of Mozzarella remained almost constant during storage of 4.5 months (Brackett and Marth, 1982d). Additionally, studies by Huseyin Oruc et al. (2007) showed that the amount of AFM1 in white pickled and Kashar cheeses did not significantly affect over the storage. Kaniou-Grigoriadou et al. (2005) found that the final ripened cheese was free of aflatoxin M1.

These different profiles of AFM1 in various cheese products may be the result of several factors such as heat treatment (Yousef and Marth 1989), proteolysis (Brackett and Marth 1982d, Brackett et al. 1982, Yousef and Marth 1989), exposure of contaminated milk to light (Yousef and Marth 1989), and especially to an inadequate method of analysis (Yousef and Marth 1989). Some results of studies on the behaviour of AFM1 during cheese ripening seem to represent changes in the recovery of toxin by the method during the different phases of the study rather than real changes in the level of AFM1 in cheese (Brackett and Marth 1982a).

Several investigations on the partitioning of AFM1 during cheese production staring with different milk contamination levels reported a wide range of distribution of AFM1 between whey and curd. Some authors observed that half or more of the AFM1 was in the way: 50%, 50%, 66%, 100%, 60%, and 53-58% according to Grant and Carlson (1971), Stubblefield and Shannon (1974), Blanco et al. (1988a), Purchase et al. (1972), Lopez. et al. (2003), and Huseyin Oruc et al. (2007), respectively. In contrast, others reported that most of AFM1 was with curd: ranging from 66% to 72%, from 73% to 77%, 80%, 100%, and 59.1% according to Marshaley et al. (1986), El Deeb et al. (1992), Mckinney et al. (1973), Allcroft and Carnaghan (1963), and Deveci (2007), respectively. Kaniou-Grigoriadou et al. (2005) observed that enrichment factor in the production of Feta cheese made from naturally contaminated milk ranged between 4.3 and 5.6. Kamakar et al. (2008) showed that the mean concentration of toxin in curd and cheese was 3.12 and 3.65-fold more than that in whey and 1.68 and 1.80 fold more than that in cheese milk, respectively.

It is thought that since AFM1 is a semi-polar component, it has less affinity to serum protein (Applebaum et al., 1982). Regarding the affinity of AFM1 with proteins, Recently, Barbiroli et al. (2007) indicated that there is no simple physical method to remove AFM1 from ovine and caprine milk. Neither ultrafiltration, nor acidic or enzymatic treatments were able to influence the toxin’s interaction with casein or whey proteins. Only the combined action of heat and low pH (as used in ricotta cheese production) was able to denature whey proteins to a point where they lost their AFM1-binding capacity.

According to Blanco et al. (1988a) these contrasting results can be attributed to different factors such as ex-

Table 2 Occurrence and content of AFM1in Cheese Samples.

Year	Region	Samples	+ samples	Range(ng/kg)	Refernce
1990	Syria	*	0	NA	Haydar et al.
1991	Kazakhestan	*	*	*	Nikov et al.
1993	Japan	37	0	NA	Tabata et al.
1995	Japan	41	0	NA	Taguchi et al.
1995	Spain	35	16	20-200	Jose Barios et al.
2001-2002	Turkey	600	30	100-800	Yaroglu et al.
2005-2008	Iran	80	66	150-2410	Kamkar
2004	Italy	41	4	79.5-389	Virdis et al.
2005	Turkey	100	99	0-4100	Tekinsen and Ucar
2005-2007	Kuwait	40	32	23.8-452	Dashti et al.
2008	Spain	72	0	-	Cano-Sancho et al
2010	Iran	75	49	30-313	Fallah
2011	Iran	72	59	30-1200	Fallah et al.

NA: Not Applicable *: Not Reported

Table 3. Occurrence of AFM1 reported in some regions .

Year	Region	type	Samples	+ samples	Range(ng/kg)	Refernce
2005	Turkey	Butter	92	92	10-7000	Tekinsen and Ucar
2005	Turkey	Butter	27	25	0-100	Aycicek et al.
	Iran	Infant formula	120	116	1-14	
2005		Milk-based cereal weaning food	80	72	3-35	Oveisi et al.
2005	Egypt	Beast milk	443	248	4.2- 889	Polychronaki et al.
2005-2007	Kuwait	Breast milk	12	5	8.83-15.2	Dashti et al.
		Kashk	125	53	28-291	
2010		Doogh	136	25	13-53	Fallah
	Iran	Butter	31	8	13-26	
2011		Ice Cream	36	25	15-132	Fallah et al.

traction techniques, methodology, type and degree of milk contamination, differences in milk quality, expression of the results, the presence of a small portion of curd in whey which could influence AFM1 concentration, and the cheese manufacture process.

The incidence of positive cheese samples for AFM1 (Table 2) seem to be widely variable. Taguchi et al. (1995) found no positive samples in imported cheese in Japan. Virdis et al. (2008) detected few positive samples, whereas Tekinsen and Ucar (2008) observed a high incidence of positive samples. As regards the contamination level, several authors (Kamkar, 2006; Fallah, 2010) found a maximum contamination level over 1000 ng of AFM1 per kg. This latter contamination level could be hazardous.

AFM1 in yogurt

Several studies have been conducted regarding the effect of yogurt manufacturing on AFM1 content. Some authors reported no influence on aflatoxin M1 content (Van Egmond and Paulsch, 1986). In contrast, Bakirci (2001) detected variable increases of AFM1 content in yogurt related to the milk. The effect of fermentation was assessed by Govaris et al. (2002). They reported that AFM1 levels in all yoghurt samples showed a significant decrease from those initially present in milk. This decrease in AFM1 was attributed to factors such as low pH, formation of organic acids or other fermentation by-products, or even to the presence of lactic acid bacteria. The low PH during fermentation alters the structure of milk

Table 4. Maximum acceptable level of AFM1 milk and milk products in some regions.

Country	Maximum Acceptabe Level (ng/l)	Type
European Union	50	Milk
Austria	50	Milk
Argentina	50	Milk
Bulgaria	500	Milk
Germany	50	Milk
Australia	20	Children's milk
Sweden	50	Liquid milk products
Netherlands	20	Butter
Switzerland	50	Milk and milk products
	250	Cheese
Belgium	50	Milk
USA	50	Milk
Czech Republic	100	Children's milk
	500	Adult's milk
Serbia	500	Milk
Iran	50	Raw, Pasteurized, and UHT milk
	200	Cheese
	20	Butter
France	30	Children's milk< 3 years
	50	Adult's milk
Turkey	50	Milk and milk products
	250	Cheese
Brazil	500	Milk

Source: **Hamid, 2011**

proteins such as the caseins leading to formation of yoghurt coagulum. The change in casein structure during yoghurt production may affect the association of AFM1 with this protein (Brackett and Marth 1982) causing adsorption or occlusion of the toxin in the precipitate.

As to AFM1 stability over storage of yogurt, Van Egmond et al. (1986) observed no reduction of AFM1 in yogurt stored for 7 days at 7 ºC. Megalla and Hafez (1982) observed complete transformation AFB1 in its hydroxy derivative AFB2A caused by the acids present in yogurt. Whereas, Rasic et al. (1991) revealed a high reduction (up to 97%) of AFM1 in yogurt and acidified milk. El Deeb et al. (1992) observed that enzymatic, microbial, and particularly acid coagulation caused degradation of AFM1 in buffalo milk. Maryamma et al. (1990) reported a high reduction of AFM1 in fermented goat milk. As a result of Study by Govaris et al. (2002), during refrigerated storage, AFM1 was rather more stable in the yoghurts with pH 4.6 than with pH 4.0. The percentage loss of the initial amount of AFM1 in milk was estimated at about 13 and 22% by the end of the fermentation, and 16 and 34% by the end of storage for yoghurts with pHs 4.6 and 4.0, respectively.

Since it is known that exposure of the aflatoxin molecule to strong acid, such as trifluoracetic acid, can cause its acid-catalyzed hydration, leading, for example, from AFB1 to AFB2A (Cohen and Lapointe, 1981), but not its degradation or neutralization, the effect of the weak acidity of yogurt on aflatoxin should be more investigated. Some investigations have been conducted related to the effect of aflatoxin on nutritive properties of yogurt. El Deeb et al. (1992) observed some negative effects of AFM1 on Lactobacillus bulgaricus (cell wall thickening and shortening of cell chain lenghth) and Staphylococcus thermophilus (cell wall thickening and cell shape changing from coccoid to oval). Rasic et al. (1991) found that S. thermophilus was affected by the presence of in milk during fermentation of yoghurt, exhibiting longer cell chains in the contaminated than in the uncontaminated yoghurt samples. Similarly, Govaris et al. (2002) observed that the growth rate of S. Thermophilus and curdling time were affected by the higher level and not by the lower level of AFM1. Unlike cheese and milk samples, the presence of AFM1 in yogurt has not frequently been studied. Thus, more investigations are needed because:

- currently, human consumption of yogurt has greatly increased
- there are contradictory data on AFM1 stability over manufacture and storage in the literature
- The presence of aflatoxin in yogurt could reduce the nutritional values of its consumption.

AFM1 in Other Milk Products

Many other milk products such as cream, butter, ice cream may contain AFM1. The presence of AFM1 in these

products has rarely been investigated and could be of interesting aspects for study. Some surveys conducted on the occurrence of AFM1 in milk products are reported in (Table. 4). In a study by Bakirci (2001), the levels of AFM1 in the products made from contaminated milk namely butter, butter milk, cream, skim milk was investigated. The mean
AFM1 level found in cream samples was 64.4% of AFM1 concentration of bulk-tank milk.

Whereas, mean AFM1 level of skim milks was 3% higher than those of bulk-tank milk. These values were close to the results given by Van Egmond et al. (1986), and lower than the values given by Wiseman et al. (1983). Levels of AFM1 in butter samples in the study were less, and they were as 33.80% of AFM1 amounts of bulk-tank milk. Mean AFM1 levels obtained from buttermilk samples were similar to those of bulk-tank milk (mean 83% of it).
The same results were reported by Grant and Carlson (1971). During butter processing, protein membrane around fat globules is broken down and serum phase is separated. Due to the chemical structure of AFM1 and its affinity to casein, it adsorbs on this fraction of protein (Yousef & Marth, 1989), therefore, cream contained less AFM1 than milk, and butter contained less amount of AFM1 than cream. As a result of the associate effects of these factors, AFM1 concentration occurs in lipid phase (like butter and cream) less than serum phase and protein fraction (Grant & Carlson, 1971).

AFM1 is frequently observed in the aflatoxin exposed individuals and in the breast milk. AFM1 toxicity in this respect is important as it is known that within aflatoxin exposed nursing mothers it can provide a source of aflatoxin exposure to the infant (El-Nezami, 1995). The occurrence of AFM1 in breast milk has been investigated in some regions. There is increased awareness of the link between growth and health of the fetus and infant, and disease risk in later life. Long term pre and postnatal exposure to aflatoxins could be one of the factors contributing to growth faltering and/or the early onset of hepatocellular carcinoma (HCC) in countries with a high incidence of the disease. Additionally, the presence of other aflatoxins, B1, B2, G1, G2 and M2, has also been reported in breast milk (IARC, 2002). The identification and understanding of factors determining the presence of toxicants in human milk is important and may provide a strong basis for controlling the transfer of chemicals to the infants through breast milk.

Losses Due To Aflatoxin Infection

In addition to financial losses and economic damage to agricultural and animal husbandry industries, losses due to aflatoxin contamination of foods include major pharmaceutical and health costs to treat food poisoning. Based on Food and Agriculture Organization (FAO,2011) reports, annually, about 20% of the foods produced in the world are contaminated by mycotoxins; in which aflatoxins have a greater share than the others. Prevalence of cancer and livestock disease in farms, weakening of livestock immune system, reduction in milk production and productivity are a few exam ples of dam ages to food and livestock industry. Considering huge economic losses and public health protection, prevention and neutralization of the toxins in livestock feed and food products of animal origin such as milk is essential (Mili evi et al., 2010).

Permitted Levels of Aflatoxin in Milk and Milk Products

Some countries have set permitted levels of aflatoxins in food in order to control and reduce detrimental effects of these toxins. These levels are variable and depend on economic and developing status of the countries (Galvano et al., 1996). In US, Food and Drug Administration (FDA) has permitted a total amount of 20 ng/g in livestock feed and 0 .5 g/kg or 50 ng/l in milk (Ellis et al., 1 995). In European countries, permitted levels of aflatoxin M 1 in milk, milk products and baby food are 0.005 mg/kg (Creppy, 2002). Also, different countries have set different regulations for permitted levels of aflatoxin in livestock feed. For instance, European Union (EU) has set permitted levels of aflatoxin from 0.05 to 0.5 g/kg. Factors such as weather conditions are also effective in determining permitted levels of aflatoxin. However, according to US regulations the level of AFM1 in milk should not be higher than 500 ng/kg (Stoloff et al., 1991). There are thus differences in maximum permissible limit of AFM1 in various countries.

Currently the limits of AFM1 are highly variable depending upon the degree of development and economic standing of the countries. According to Hamid (2011) the maximum limits for aflatoxineM1 in milk and milk products in various countries is illustrated in table 4.

Mariko (2012) reported the limits of aflatoxin M_1 for milk and milk products and in some cases for infant and products for infants, worldwide, as illustrated in Table 5

What to Do if Milk Is Detected With Action Levels of Aflatoxin

If aflatoxin is detected in milk, it is critical that records be maintained of all feeds, feeding practices, milk quantities and contamination levels, plus animal health and performance. If the grain or related feed is fed to other animals, these records should be maintained also. After milk has been detected with greater than 0.5 ppb of aflatoxin in one load, all grain products fed to animals should be removed from the ration immediately and new

Table 5. Aflatoxin M_1 limits in different food stuffs .

Country	Foodstuffs	Aflatoxin M_1 (µg/kg)
EU Bosnia and Herzegovina	Raw milk, heat-treated milk and milk for the manufacture of milk-based products	0.050
Turkey	Infant formulae and follow-on formulae, including infant milk and follow-on milk	0.025 (products ready to use)
	Dietary foods for special medical purposes intended specifically for infants	0.025 (products ready to use)
China	Milk and milk products (for milk powder, calculated on a fresh milk basis)	0.5
	Formulated foods for infants (milk or milk protein based)	0.5 (calculated on a dry powder basis)
	Formulated foods for older infants and young children (milk or milk protein based)	0.5 (calculated on a dry powder basis)
	Formulated foods for special medical purposes intended for infants	0.5 (calculated on a dry powder basis)
Codex, GCC, India, Kenya, USA	Milk	0.5
Argentina	Milk, liquid including milk used in the manufacture of milk and milk products and reconstituted milk	0.5 (1)
	Milk, powder	5.0
	Milk formula	ND
Mexico	Pasteurised, ultrapasteurised, sterilised and dehydrated milk, milk formula and combined milk products	0.5 (1)
South Africa	Milk	0.05

ND: Not Detectable, (1) Given in µg/l.

grain and/or related items replaced in the diet. As cottonseed and corn are the most likely sources of aflatoxin contamination, these grains should be tested to determine their level of aflatoxin. The exception to the preceding statement is when an additional milking has occurred on the dairy and the milk then tests below 0.5 ppb aflatoxin. In this case, the feed should be tested before it is removed from the feed supply (Jodie, 2012).

It is illegal to sell grain with levels greater than 20 ppb aflatoxin for lactating dairy cows, and the seller of the grain is responsible for damage resulting from the sale of grain. However, in most cases, perhaps 60 percent of the time, the exact source of feed contamination is not determined. In cases of severe contamination and over a prolonged period of time, some data indicate that animals may experience adverse health effects which can affect both present and long-term performance. Calves also are more susceptible to aflatoxin and have died as a result of aflatoxin contamination in feed (Jodie, 2012).

Immediately after aflatoxin is detected in milk, the ration should be reformulated with ingredients that contain less than 20 ppb aflatoxin. If the level of milk contamination exceeds 0.5 ppb on a second test, a special dietary chemisorbent should be added to the diet at recommended levels. These compounds include clays (bentonites) at 1 percent of the diet, activated carbon at 1 percent of the diet and glucomannan (Mycosorb®) at 0.05 percent of the diet on a dry matter basis. Some empirical observations indicate that the animal may consume levels of bentonite up to one pound a day. Limited data is available on the numerous compounds that are available to absorb the aflatoxin in the digestive system. However, in one study, about 1/4 pound of hydrated sodium calcium aluminino silicate (HACA – a compound approved for feed as an anti-caking agent) was shown to reduce aflatoxinM1 in milk about 50 percent when cattle consumed feed containing 200ppb aflatoxin. Silky clay loan soil and bentonite have a similar effect but have not been well studied. Many commercially avail- able products also theoretically will bind aflatoxinM1 and should result in lower aflatoxin in milk. Generally, the cost of using the commercially available products is greater than the cost of using bentonite to bind aflatoxin (Jodie, 2012).

Afalatoxin Reduction in Milk and Milk Products

Indirect Methods of Aflatoxin Reduction in Livestock Feed

Milk contaminated with aflatoxins is produced mostly from use of infected feed. Therefore, reducing aflatoxin contamination indirectly via control of livestock feed hy-

giene is possible. To achieve the aim, principles and health considerations during farming and crop production in farms and livestock feed factories, storage of livestock feed in traditional and industrial warehouses is necessary (Cleveland et al., 2003). Livestock feed is mostly include corn, cotton seed and canola, dietary supplements, wheat bran, dried bread, fat powder and alfalfa. Given that a significant percentage of the feed composition is derived from crops, health consideration during planting, harvesting and storage of crops are factors affecting grain quality. Drought, rainfall, infection by insects and high moisture during flowering can be referred as major causes of aflatoxin occurrence in farms. Adherence to appropriate irrigation programs, planting varieties resistant to moisture and molds, weed control, insecticides application, harvesting at appropriate time, crop rotation in order to reduce the risk of pathogen transfer from current farming year to the next year and fertilizing oil are useful ways of preventing pre-harvest aflatoxin infection. In addition, appropriate storage of crops which includes placing crops on clean and dry surfaces, protecting crops from moisture, heat, insects and use of fungicides are effective ways of reducing the infection (Wu et al., 2008). Adherence to hygienic conditions in factories and livestock feed warehouses both in traditional and industrial levels is another important way of controlling and reducing aflatoxin infection.

Controlling mold growth and aflatoxin formation in traditional farms and warehouses is highly important. In this regard, several studies have been carried out on quality of livestock feed and the amount of aflatoxin in produced milk (Creppy, 2002). For example, it has been shown that the amount of aflatoxin in milk produced in autumn and winter is higher compared to spring and summer (Panariti, 2001). This is because in cold seasons, feeding livestock on fresh forages is not possible due to unfavorable weather conditions and farmers have to use stored forages. Regarding that warehouse improper temperature and moisture conditions favor mold growth; therefore, it is necessary to improve storage conditions of livestock feed (Panariti, 2001).

Results in some countries have shown that meeting safety conditions of livestock feed has led to decrease aflatoxin infection (Akande, 2006).

In addition, protecting feed from infection sources, inhibition of microorganisms' propagation in feed, alleviating infection and inhibition of reinfection are regarded as principles of controlling infection in industrial livestock feed factories. To obtain these ends, continuous and careful monitoring of different procedures of storing raw materials and production, controlling moisture content and temperature by ventilation systems, use of clean and hygienic facilities such as mills and mixers and avoiding from broken or damaged grains when preparing feed are necessary (Degirmencio lu et al., 2005).

Moreover, absorbents, chemicals, microorganisms and ionizing rays can be used to prevent mold growth and development of the molds when initial infection has been occurred (Sinha, 1998). Several researches have been conducted on using absorbents in infected livestock feed (Alexander et al., 2001; Dakovic´ et al., 2008). Use of aflatoxin absorbents in infected feed is a promising way of reducing this infection in livestock feed. Through binding to absorbents, aflatoxins present in feed inhibits from toxic reactions in livestock body as well as from absorption into digestive tract. Properties of the absorbents when choosing them to hinder the aflatoxin infection should include capability of toxicity inhibition in livestock feed, not having detrimental effects on livestock health and being economic (Dakovic´ et al., 2008). Studies on application of aflatoxin absorbents have been conducted for years. Some aflatoxin absorbents in infected feed include active carbon, alumino (clay, bentonite, montmorillonite, zeolite and phyllosilicates), complex carbohydrates (cellulose and polysaccharides present at cellular wall of yeasts and bacteria such as glucomannans, peptidoglycans), synthetic polymers such as cholestyramine and polyvinyl pyrrolidone and its derivatives. Although this method leads to good results in the laboratory conditions, the use of these substances in livestock body is different and requires time-consuming and various experiments. Livestock species, age and genus influence results of the experiments (Alexander et al., 2001; Dakovic´ et al., 2008).

Furthermore, chemical inhibitors include one or a series of organic acids such as propionic, sorbic, benzoic and acetic acids, organic acid slats such as calcium propionate, potassium sorbate and solid or liquid copper sulfate (Sinha, 1998). Also, with respect to FDA standards, use of ammonia for neutralizing aflatoxin in livestock feed has been permitted in US. 95% of aflatoxin in feed has been alleviated with the aid of gaseous or liquid ammonia (Prudente and King, 2005). When applying ammonia, if conditions such as time of use, temperature and concentration are met, reduction of aflatoxin infection is carried out more effectively. Ammonia alleviates toxicity of aflatoxin B1 and converts it to non-toxic compound of aflatoxin D1 through hydrolyzation of aflatoxin B1 and its decarboxylation (Prudente and King, 2005).

Use of different microorganisms such as lactobacillus pentosus and lactobacillus brevis is another way of reducing aflatoxin in livestock feed. Mechanism of Aflatoxin B1 removal by lactic acid bacteria is not through metabolic degradation, but through binding this toxin to cellular wall of the bacteria. However, some researchers have attributed removal property of aflatoxin by the aforesaid bacteria to their ability in producing acid and lowering pH (Haskard et al., 2001). Application of ionizing rays such as gamma ray in humid environment is also a way of aflatoxin B1 reduction in livestock feed. Aflatoxin B1 decomposes water against gamma ray in moist environ-

ment and releases radicals, thus creating conditions required for decomposition of aflatoxins. In dry conditions, aflatoxin B1 shows high resistance to detrimental effects of gamma ray (Dakovic´ et al., 2008).

Direct Methods of Aflatoxin Reduction in Milk

Toxin absorbents, chemical and biological methods are also used directly for reducing aflatoxin in milk (Bovo et al., 2012). Use of toxin absorbents is one of the main methods to reduce aflatoxin amount in milk. Absorbent soils such as bentonite, vermiculite, hydrated sodium calcium aluminosilicate (HSCAS) and active carbon have been known as absorbent compounds for absorbing various toxins in aqueous environments (Applebaum and Marth, 1982a). For instance, bentonite has been known as an effective reducer of aflatoxin M1 in infected milk (Applebaum and Marth, 1982a). Binding capacity and stability of compounds formed between absorbent and toxin are highly variable and influenced by temperature and PH. Information about the effect of absorbents on milk constituents is scarce; however, it has been shown that these substances have slight effect on nutritional quality of milk (Ellis et al., 1995).

In a study, effect of bentonite on milk protein content was not considerable and maximum reduction in protein content was only 5% (Applebaum and Marth, 1982a). It should be mentioned that with respect to acceptability of absorbents as healthy additives by international authorities and ability to separate them from milk after absorption of aflatoxin, further investigations are in progress. In addition to absorbents, chemicals such as hydrogen peroxide used for storage of some food products have reduced aflatoxin in milk at different conditions of time and concentration (Applebaum and Marth, 1982b). Use of hydrogen peroxide combined with other additives like riboflavin and lactoperoxidase as well as hydrogen peroxide application along with heat treatment have led to more satisfactory results in reducing aflatoxin in milk. In addition, potassium sulfite has been used for neutralizing aflatoxin M1 in milk (Applebaum and Marth, 1982b).

Biological methods as inexpensive and easy techniques of reducing aflatoxin have also been of interest. Therefore, widespread studies on identifying effective microorganisms are being conducted (Bovo et al., 2012).

For instance, in a study, Flavobacterium aurantiacum which is gram-negative, aerobic, bacilli like and non-sporogenous bacterium has been used for reducing aflatoxin in milk (Line et al., 1994). The bacterium can use aflatoxin present in milk as a substrate and convert it to non-toxic substances. However, formation of proteolytic and lipolytic enzymes by this microorganism leads to undesirable changes in milk flavor (Line et al., 1994).

CONCLUSIONS

Aflatoxins are produced on livestock feed in appropriate moisture and temperature conditions for mold growth. Consumption of feed infected by aflatoxins leads to different problems in reproductive, digestive and respiratory tracts of livestock causing infected milk production. Consumption of infected milk by human incurs major hygienic and pharmaceutical costs to society. Therefore, in order to prevent from introduction of aflatoxin M1 into food industry cycle, its precursor namely aflatoxin B1 should be controlled. To obtain this, meeting hygienic conditions, appropriate storage and control of livestock feed at all stages of planting, growing, harvesting, producing and storing are necessary. For this reason, milk and milk products have to be controlled continuously by accurate and reliable analytical techniques for presence of AFM1 contamination. It is also extremely important to maintain low levels of AFM1 in the feeds of dairy animals. In order to achieve this, dairy cow feds should be kept away from contamination as much as possible. Therefore, animal feeds should be checked regularly for Aflatoxin and, particularly important, storage conditions of feeds must be strictly controlled. The regulatory limits are widely variable and there has been little scientific basis in their setting. Efforts should be made in attempting to provide further and extensive scientific information on human health hazards related to low-level long term Aflatoxins exposure and to standardize the already existing regulatory limits for Aflatoxins. . The occurrence of AFM1 in cow milk and milk products is widespread and the occurrence of Aflatoxin and their metabolites in human breast milk is of great concern. Since serious health hazards to the mother, fetus, and infant could occur. Therefore extensive and periodic surveys should be performed in the incidence and occurrence of AFM1 in milk and milk products.

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