Sources, Transformation, and Hazards of Mycotoxins in Milk: Postprint

Authors: Gao Yanan, Wang Jiaqi, Zheng Nan

Date: 2017-10-11T00:00:00+00:00

Abstract

Mycotoxin contamination represents one of the primary risks to milk quality and safety, with major types including aflatoxins (AFs), ochratoxins (OT), zearalenone (ZEA), fumonisins (FUM), deoxynivalenol (DON), T-2 toxin (T-2), among others. Mycotoxins in milk primarily originate from animal feed. Based on existing domestic and international literature reports, this paper provides a comprehensive review of the sources, transformation, hazards, and maximum limits of mycotoxins in milk.

Full Text

Mycotoxins in Cow' s Milk: Origin, Transformation, and Hazards

GAO Yanan[1],[2],[3], WANG Jiaqi[1],[2],[3], ZHENG Nan[1],[2],[3]*

[1]Ministry of Agriculture-Milk Risk Assessment Laboratory, Beijing 100193, China
[2]Ministry of Agriculture-Milk and Dairy Product Inspection Center, Beijing 100193, China
[3]State Key Laboratory of Animal Nutrition, Institute of Animal Science, Chinese Academy of Agricultural Sciences, Beijing 100193, China

Abstract: Mycotoxin contamination represents a major risk factor for milk quality and safety. The primary mycotoxins detected in milk include aflatoxins (AFs), ochratoxins (OT), zearalenone (ZEA), fumonisins (FUM), deoxynivalenol (DON), and T-2 toxin. These mycotoxins predominantly originate from contaminated animal feed. This review synthesizes existing literature to examine the sources, metabolic transformation, hazards, and regulatory limits of mycotoxins in cow' s milk.

Keywords: mycotoxin; milk; origin; hazard

According to the Food and Agriculture Organization (FAO), approximately 25% of global grain production is contaminated with varying levels of mycotoxins. In China, this problem is particularly severe, with contamination rates exceeding 90% [1-2]. The major mycotoxins include aflatoxins (AFs), ochratoxins (OTs), zearalenone (ZEA, also known as F-2 toxin), deoxynivalenol (DON), and T-2 toxin. Huang et al. [5] detected multiple mycotoxins in milk, including aflatoxin M1 (AFM1), ochratoxin A (OTA), ZEA, and α-zearalenol (α-ZEL), with 15% of samples containing two toxins, 45% containing three, and 22% containing four, demonstrating widespread co-occurrence. Feed contamination serves as the primary source of mycotoxins in milk, with the type and concentration of feed mycotoxins directly determining those found in milk [6]. Ruminants are generally considered more tolerant to mycotoxins than monogastric animals because rumen protozoa can detoxify and sequester certain mycotoxins such as OTA, ZEA, T-2, and DON, providing some protection to dairy cows [7]. However, some mycotoxins undergo opposite transformations, being metabolized by rumen microorganisms into more toxic compounds rather than being degraded. For example, ZEA is converted to the more potent α-ZEL [8]. Following metabolic processing, feed mycotoxins can be transferred into milk, posing potential threats to human health.

1.1 Aflatoxins (AFs)

AFs are produced by storage fungi, primarily *Aspergillus* species, with optimal growth conditions of 25–30°C and 80–90% relative humidity [9]. Consequently, *Aspergillus* proliferation and AF secretion increase under hot, humid conditions. AFs predominantly contaminate raw meal, corn, cottonseed meal, and silage [10-11]. Contamination patterns show geographic variation: in Mianyang, China, the detection rate for aflatoxin B1 (AFB1) in feed reached 100%, with an overall exceedance rate of 3.9% [12], whereas in Shanghai' s Pudong district, both detection rates and average concentrations were relatively low [13].

Research indicates that when dairy cows ingest AFB1 at concentrations of 1–10 g/mL, rumen microorganisms metabolize less than 10% of the toxin [14]. The remaining 90% passes to the liver, where hydroxylation converts it to the less toxic AFM1 [15]. The resulting AFM1 can bind with glucuronic acid or be transferred to urine and milk through systemic circulation [16]. Valenta et al. [17] reported a conversion rate of 1–2% from dietary AFB1 to milk AFM1, with high-yielding cows showing rates up to 6.2% [18]. Therefore, AFM1 transfer to milk is generally considered to range from 0.1% to 6.0%, with an accepted average of 1.7% [19]. Based on this 1.7% conversion rate, milk AFM1 would exceed safety limits in the United States (0.5 g/kg) when dietary AFB1 exceeds 30 g/kg dry matter, and would exceed EU limits (0.05 g/kg) when dietary AFB1 exceeds 3 g/kg dry matter. Strict control of feed AFB1 content is therefore essential to prevent milk AFM1 contamination and protect human health.

1.2 Ochratoxin A (OTA)

OTA is a toxic secondary metabolite produced by *Aspergillus ochraceus* and *Penicillium verrucosum*, prevalent in temperate regions. *Aspergillus ochraceus* can grow at 8–37°C, with optimal growth at 24–31°C and 95–99% humidity, and thrives at pH 3–10. OTA primarily contaminates wheat, barley, corn, oats, and dried beans [20]. EU and Chinese surveys indicate relatively low OTA contamination in grains and feed, with levels between 5.2–80.0 g/kg [21-22]. However, investigations in Shanghai' s Pudong district revealed that feed and feed ingredients were predominantly contaminated with DON, OTA, and ZEA, with OTA detection rates of 46.81% [13]. These findings demonstrate uneven geographic distribution of OTA contamination.

In ruminants, ingested OTA is converted to the less toxic ochratoxin α (OTα) by rumen microorganisms, affecting only calves with underdeveloped rumens [23]. Healthy dairy cows metabolize OTA at approximately 0.01‰, equivalent to 12 mg OTA per kg of feed [24]. Studies show that OTA and its metabolite OTα appear in milk only when intake reaches 1.66 mg/kg body weight [25], suggesting that feed may not be the primary source of milk OTA. Recent reports indicate that milk and dairy products can be contaminated with OTA during storage and transportation [19,26-27]. Therefore, both feed content and post-production handling must be considered when addressing milk OTA contamination.

1.3 Zearalenone (ZEA)

ZEA is an estrogenic mycotoxin produced by field fungi *Fusarium* species, thriving in hot, low-humidity conditions. It primarily contaminates corn, wheat, rice, barley, millet, and oats [28]. Analysis of feed samples from the US, Europe, and Asia showed ZEA detection rates of 45% with an average concentration of 233 g/kg [29]. A global survey of 17,316 feed and feed ingredient samples found a 36% detection rate with an average of 101 g/kg [30], indicating severe ZEA contamination requiring enhanced monitoring.

Rumen microbial degradation of ZEA produces at least five metabolites: zearalanone (ZAN), α-zearalanol (α-ZAL), β-zearalanol (β-ZAL), α-zearalenol (α-ZEL), and β-zearalenol (β-ZEL). Kiessling et al. [31] found α-ZEL concentrations approximately double those of β-ZEL. After 21 days of ZEA intake at 544.5 mg/d, dairy cows showed detectable ZEA and α-ZEL in milk with a conversion rate of 0.06% [14]. Research demonstrates dose-dependent conversion, with rates ranging from 0.008% to 0.016% when cows ingested 1.8–6.0 g ZEA [25]. These results indicate that ZEA rarely accumulates in tissues and transfers to milk at very low efficiency.

1.4 Fumonisin B1 (FB1)

Fumonisins are water-soluble secondary metabolites produced by *Fusarium moniliforme*, with optimal growth around 25°C. To date, 28 fumonisins and analogs have been identified, with FB1 being the most toxic. Fumonisin

contamination is globally prevalent, primarily affecting corn and wheat feed ingredients. Silva et al. [32] reported that 22% of Portuguese corn samples contained fumonisins, some exceeding EU limits. Global surveys show contamination trends by continent: Oceania > Africa > Latin America > Asia > North America > Europe [33].

Few studies have reported FB1 transfer from feed to milk. Oral administration of FB1 at 5 mg/kg body weight yielded no detectable FB1 in milk [21,34], and in vitro studies showed low rumen conversion rates [49]. However, Hammer et al. [35] detected FB1 in milk after intravenous injection of 0.046–0.067 mg/kg body weight. The European Food Safety Authority (EFSA) concluded that only trace amounts transfer to milk, posing minimal human health risk [8].

1.5 Deoxynivalenol (DON)

DON is produced by field fungus *Fusarium* species, with optimal growth at 5–25°C. Crops are typically contaminated during growth, and the fungus can persist post-harvest through asexual reproduction. DON concentrations are generally high in barley, wheat, and corn, but low in rye, sorghum, and rice. Contamination shows geographic patterns: analysis of 481 feed samples from East China revealed 67% exceedance in wheat and bran; in South China, 48% of 185 samples exceeded limits; and in North China, 33% of 96 samples exceeded limits [36].

Ruminants generally possess strong DON degradation capacity, preventing adverse effects unless intake exceeds metabolic capacity. In healthy ruminants, ingested DON is rapidly converted to de-epoxy-deoxynivalenol (DOM-1) by rumen microorganisms, a form with only 1/54th the toxicity of DON. Studies show that when cows receive 1.9 mg/kg body weight DON, less than 1% is absorbed [37]. At higher doses of 2,933–5,867 g/kg body weight, only 27 ng/mL DOM-1 was detected in milk [38]. These findings demonstrate that DON is metabolized and degraded in both ruminants and non-ruminants without bioaccumulation, making it a low-priority public health concern in animal products.

1.6 T-2 Toxin

T-2 toxin is widely distributed in nature, with production favored by low temperatures, temperature fluctuations, high moisture, and neutral to acidic conditions. It commonly contaminates corn, wheat, barley, and oats, causing various toxic symptoms when ingested by animals. Chen [39] detected T-2 in 100% of 176 feed samples from 18 Chinese provinces. Shan et al. [40] similarly found 100% detection in 116 feed ingredients from Northeast China, though no samples exceeded limits. While contamination is widespread, severity appears relatively low, though high detection rates warrant continued vigilance.

As a major mycotoxin contaminating Chinese feed, T-2 primarily damages hematopoietic and immune tissues. All species are sensitive, with pigs being most susceptible. Ruminants show greater tolerance due to rumen microbial

degradation. Reported transfer rates from feed to milk range from 0.05% to 2.00% [4,14].

2 Hazards and Regulatory Limits of Milk Mycotoxins

Mycotoxins pose significant health risks through immunotoxicity, nephrotoxicity, and hepatotoxicity, making them critical hazards in milk safety. AFM1 and OTA are particularly concerning due to their carcinogenic, mutagenic, and teratogenic effects, with OTA potentially posing greater risks to infants. Currently, only AFM1 has established maximum limits in milk globally, while other mycotoxins are regulated through provisional tolerable weekly intake (PTWI) values. More comprehensive limits are needed to better protect human health.

2.1 AFM1 Hazards and Limits

AFM1 was classified as a Group 1 carcinogen by the International Agency for Research on Cancer (IARC) in 2002. Its target organ is the liver, causing severe vascular permeability disruption and central nervous system damage. AFs exert toxicity through two primary pathways: (1) interfering with RNA and DNA synthesis, thereby disrupting protein synthesis and cellular metabolism, causing systemic damage [41]; and (2) binding to DNA, inhibiting methylation, altering gene expression and cell differentiation, and activating oncogenes while reducing disease resistance [42]. Regulatory limits for AFM1 vary by country .

Analysis of 22,189 global milk samples revealed that 1,709 Asian samples (7.7%) exceeded EU limits, followed by Africa (1.1%), Europe, and the United States (0.5%) [43]. Lower AFM1 levels in European milk likely reflect lower AF contamination in feed. Sadia et al. [44] reported average AFM1 levels of 0.252 g/L in Pakistani milk, while Indian milk contained 0.1–3.8 g/L [45], posing serious health threats. Conversely, Iranian milk showed lower levels of 0.013–0.250 g/L [46], and UHT milk contained 0.021–0.087 g/L [47]. Regional variations likely reflect differences in climate, geography, farming practices, and analytical methods [48].

2.2 OTA Hazards and Limits

OTA ranks second only to AFs in importance and hazard, classified as a Group 2B human carcinogen by IARC. Its primary target organ is the kidney, causing tubular degeneration and functional impairment. OTA exhibits potent nephrotoxicity, hepatotoxicity, neurotoxicity, and immunotoxicity, with teratogenic, carcinogenic, and mutagenic effects. Its toxicity manifests through three mechanisms: (1) inhibiting mitochondrial respiration, causing ATP depletion; (2) suppressing DNA/RNA synthesis and phenylalanine-tRNA ligase activity, thereby inhibiting protein synthesis; and (3) inducing oxidative damage and increasing lipid peroxidation [50].

The Joint FAO/WHO Expert Committee on Food Additives (JECFA) estab-

lished a PTWI of 100 ng/kg body weight for OTA. Analysis of milk from Italy, Norway, France, Sweden, and China showed OTA levels of 5.0–84.1 ng/L [5,27,51-53], insufficient to exceed PTWI for adults but potentially hazardous for infants with a tolerable daily intake (TDI) of 5 ng/(kg body weight · d) due to their high milk consumption. Sudanese milk containing 2,730 ng/L OTA posed adult health risks [26], possibly due to sudden dietary changes or high protein feed ratios reducing rumen degradation capacity. Despite these risks, no countries have established maximum limits for OTA in milk.

2.3 ZEA Hazards and Limits

IARC (1993) classified ZEA as a Group 3 possible carcinogen with estrogenic effects targeting the reproductive system. Structurally similar to endogenous estrogen, ZEA competes for estrogen receptor (ER) binding, activating estrogen response elements and causing estrogenic syndrome [54]. Consumption during pregnancy may cause abortion, stillbirth, and teratogenesis. Since ZEA is not completely metabolized, residues can accumulate, making feed monitoring crucial [28].

JECFA recommended a provisional maximum tolerable daily intake (PMTDI) of 0.5 g/kg body weight for ZEA and its metabolites. Analysis of 400 milk samples from Egypt, the UK, and China detected maximum ZEA levels of 12.5 g/kg [5,55-57]. An adult (50–70 kg) would need to consume 2.0–2.8 L of milk daily at this concentration to exceed PMTDI, suggesting minimal risk from milk exposure. However, metabolites warrant attention: α-ZEL is three times more toxic than ZEA and has been detected in Chinese milk at 73.5 ng/kg [5].

2.4 FB1 Hazards and Limits

FB1 is classified as a Group 2B human carcinogen. Its mechanism remains unclear, but structural similarity to sphingosine suggests neurotoxicity targeting the brain. The EU Commission set a PMTDI of 2 g/kg body weight for individual and combined FB1, FB2, and FB3. Maragos et al. [58] detected FB1 in 1 of 155 milk samples at 1,290 ng/L, while Gazzotti et al. [59] found FB1 in 8 of 10 samples, with a maximum of 430 ng/kg. Even the highest reported level (1,290 ng/L) is unlikely to exceed PMTDI for adults, though limited monitoring data suggest need for enhanced surveillance.

2.5 DON and T-2 Hazards and Limits

DON and T-2 are trichothecene mycotoxins. Among approximately 170 trichothecenes, Type A (including HT-2 and T-2) and Type B (including DON, 3-ADON, and 15-ADON) are distinguished by functional groups. DON damages gastrointestinal mucosa through absorption, while T-2 enters immune organs (thymus, bone marrow, liver, spleen) via blood, inhibiting DNA/RNA transcription and translation through its sesquiterpene structure, thereby suppressing protein synthesis and impairing immunity and reproduction [60]. T-2

also causes DNA single-strand breaks in lymphocytes and inhibits mitochondrial respiration, causing energy deficiency [61].

The EU Commission set a PMTDI of 60 ng/kg body weight for HT-2 and T-2, and 1 g/kg for DON. DON and T-2 are rarely detected in milk; only 5 of 20 Danish milk samples contained DOM-1 (DON metabolite) at 0.3 ng/mL [62]. Both ruminants and non-ruminants effectively degrade DON to less toxic forms without bioaccumulation. Therefore, DON should be considered a low-priority hazard.

Conclusion

Mycotoxins in milk pose serious threats to human and animal health. Consumption of contaminated feed can reduce milk yield and alter composition, while transferring toxins into milk. Current research focuses primarily on AFM1, which is globally detected in milk samples. However, milk also contains OTA, ZEA, FB1, α-ZEL, and DOM-1, requiring comprehensive monitoring. Prevention strategies include: avoiding moldy feed, maintaining dry and hygienic storage conditions, limiting feed inventory duration, and using mycotoxin binders. Risk monitoring programs and multi-mycotoxin detection technologies are essential for ensuring milk safety. Current detection methods include thin-layer chromatography, HPLC, LC-MS/MS, and ELISA. Future research should develop more efficient, simplified methods for simultaneous multi-mycotoxin detection and establish appropriate limits based on actual consumption patterns and contamination levels to better protect human health.

References

[1] IHESHIULOR O O M, ESONU B O, CHUWUKA O K, et al. Effects of mycotoxins in animal nutrition: a review[J]. Asian Journal of Animal Sciences, 2011, 5(1): 19–33.

[2] YIN Qinggang, WANG Feng, ZHAO Guohua, et al. Research progress on control techniques for zearalenone in grains and feed[J]. Feed Research, 2009(6): 32–35.

[3] HUSSEIN S H, BRASEL J M. Toxicity, metabolism, and impact of mycotoxins on humans and animals[J]. Toxicology, 2001, 167(2): 101–134.

[4] CAVRET S, LECOEUR S. Fusariotoxin transfer in animal[J]. Food and Chemical Toxicology, 2006, 44(3): 444–453.

[5] HUANG L C, ZHENG N, ZHENG B Q, et al. Simultaneous determination of aflatoxin M1, ochratoxin A, zearalenone and α-zearalenol in milk by UHPLC-MS/MS[J]. Food Chemistry, 2014, 146: 242–249.

[6] ZHENG Nan, WANG Jiaqi, HAN Rongwei, et al. Analysis of major risk factors for milk quality and safety II. Mycotoxins[J]. China Animal Husbandry & Veterinary Medicine, 2012, 39(3): 1–9.

[7] LIU Dan, YI Hongqin, XU Guozhong, et al. Toxic effects of feed mycotoxins on dairy cows[J]. Shanghai Journal of Animal Husbandry and Veterinary Medicine, 2009(4): 65-66.

[8] The European Food Safety Authority. Opinion of the scientific panel on contaminants in the food chain [CONTAM] related to fumonisins as undesirable substances in animal feed[R]. Parma: The European Food Safety Authority, 2005, 235: 1-32.

[9] YANG Limei, SHEN Guangrong. Hazards and prevention of mycotoxins in feed[J]. Feed Industry, 2003, 24(12): 53-55.

[10] DING X X, LI P W, BAI Y Z, et al. Aflatoxin B1 in post-harvest peanuts and dietary risk in China[J]. Food Control, 2012, 23(1): 143-148.

[11] KELLER L A M, GONZÁLEZ PEREYRA M L, KELLER K M, et al. Fungal and mycotoxins contamination in corn silage: monitoring risk before and after fermentation[J]. Journal of Stored Products Research, 2013, 52: 42-47.

[12] GOU Shuang. Investigation on aflatoxin B1 contamination in feed in Mianyang City[J]. Feed Wide Angle, 2013(12): 30-32.

[13] WANG Zheng, YAN Minming, NI Weizhong, et al. Investigation on mycotoxin contamination in feed and feed ingredients from large-scale farms in Pudong District, Shanghai[J]. Animal Husbandry and Veterinary Medicine, 2013, 45(10): 85-87.

[14] YIANNIKOURIS A, JOUANY J P. Mycotoxins in feeds and their fate in animals: a review[J]. Animal Research, 2002, 51(2): 81-99.

[15] KUILMAN M E M, MAAS R F M, FINK-GREMMELS J. Cytochrome P450-mediated metabolism and cytotoxicity of aflatoxin B1 in bovine hepatocytes[J]. Toxicology in Vitro, 2000, 14(4): 321-327.

[16] FINK-GREMMELS J. Mycotoxins in cattle feeds and carry-over to dairy milk: a review[J]. Food Additives & Contaminants Part A, 2008, 25(2): 172-180.

[17] VALENTA H, GOLL M. Determination of ochratoxin A in regional samples of cow' s milk from Germany[J]. Food Additives and Contaminants, 1996, 13(6): 669-676.

[18] VELDMAN A, MEIJS J A C, BORGGREVE G J, et al. Carry-over of aflatoxin from cows' food to milk[J]. Animal Science, 1992, 55(2): 163-168.

[19] COFFEY R, CUMMINS E, WARD S. Exposure assessment of mycotoxins in dairy milk[J]. Food Control, 2009, 20(3): 239-249.

[20] WANG Shoujing, HU Peng, RU Yi, et al. Fungal toxin contamination in grains and its control technology[J]. Food and Nutrition in China, 2012, 18(3): 13-16.

[21] RICHARD J L. Some major mycotoxins and their mycotoxicoses—an overview[J]. International Journal of Food Microbiology, 2007, 119(1/2): 3-10.

[22] BINDER E M, TAN L M, CHIN L J, et al. Worldwide occurrence of mycotoxins in commodities, feeds and feed ingredients[J]. Animal Feed Science and Technology, 2007, 137(3/4): 265-282.

[23] Whitlow L W, Hagler W M. Mycotoxins: a review of dairy concerns[C]//Mid-South Ruminant Nutrition Conference. Raleigh, NC: North Carolina State University, 2005: 47-58.

[24] HULT K, TEILING A, GATENBECK S. Degradation of ochratoxin A by a ruminant[J]. Applied and Environmental Microbiology, 1976, 32(3): 443-444.

[25] PRELUSKY D B, VEIRA D M, TRENHOLM H L, et al. Metabolic fate and elimination in milk, urine and bile of deoxynivalenol following administration to lactating sheep[J]. Journal of Environmental Science and Health, Part B, 1987, 22(2): 125-148.

[26] ELZUPIR A O, MAKAWI S Z A, ELHUSSEIN A M. Determination of aflatoxins and ochratoxin A in dairy cattle feed and milk in Wad Medani, Sudan[J]. Journal of Animal and Veterinary Advances, 2009, 8(12): 2508-2511.

[27] PATTONO D, GALLO P F, CIVERA T. Detection and quantification of ochratoxin A in milk produced in organic farms[J]. Food Chemistry, 2011, 127(1): 374-377.

[28] YU Miao, WANG Qiuxia. Research progress on mycotoxins in feed[J]. Feed Wide Angle, 2013(12): 21-24.

[29] RODRIGUES I, NAEHRER K. A three-year survey on the worldwide occurrence of mycotoxins in feedstuffs and feed[J]. Toxins, 2012, 4(12): 663-675.

[30] STREIT E, NAEHRER K, RODRIGUES I, et al. Mycotoxin occurrence in feed and feed raw materials worldwide: long-term analysis with special focus on Europe and Asia[J]. Journal of the Science of Food and Agriculture, 2013, 93(12): 2892-2899.

[31] KIESSLING K H, PETTERSSON H, SANDHOLM K, et al. Metabolism of aflatoxin, ochratoxin, zearalenone, and three trichothecenes by intact rumen fluid, rumen protozoa, and rumen bacteria[J]. Applied and Environmental Microbiology, 1984, 47(5): 1070-1073.

[32] SILVA L J G, LINO C M, PENA A, et al. Occurrence of fumonisins B1 and B2 in Portuguese maize and maize-based foods intended for human consumption[J]. Food Additives and Contaminants, 2007, 24(4): 381-390.

[33] ZHANG Yibing, BAO Lei, CHU Qinghua. Detection and analysis of mycotoxins in agricultural products[M]. Beijing: Chemical Industry Press, 2006: 51-78.

[34] SCOTT P M, DELGADO T, PRELUSKY D B, et al. Determination of fumonisins in milk[J]. Journal of Environmental Science and Health, Part B, 1994, 29(5): 989-998.

[35] HAMMER P, BLUETHGEN A, WALTE H G. Carry-over of fumonisin B1 into the milk of lactating cows[J]. Milk Science International, 1996, 51(12): 691-695.

[36] HUANG Junheng, HUANG Guangming, LI Wanhua. Analysis of mycotoxin contamination in feed and feed ingredients from 19 provinces in 2015[J]. Swine Production, 2016(2): 14-16.

[37] PESTKA J J. Deoxynivalenol: toxicity, mechanisms and animal health risks[J]. Animal Feed Science and Technology, 2007, 137(3/4): 283-298.

[38] CÔTÉ L M, DAHLEM A M, YOSHIZAWA T, et al. Excretion of deoxynivalenol and its metabolite in milk, urine, and feces of lactating dairy cows[J]. Journal of Dairy Science, 1986, 69(9): 2416-2423.

[39] CHEN Xinyi. Overview of mycotoxin contamination in feed raw materials and compound feed in some provinces and cities of China from 2009-2010[J]. Zhejiang Journal of Animal Science and Veterinary Medicine, 2011(2): 7-10.

[40] SHAN Anshan, ZHOU Changlu, ZHANG Yuanyuan, et al. Determination of mycotoxin content in different feed ingredients in Northeast China[J]. Journal of Northeast Agricultural University, 2013, 44(5): 96-100.

[41] WANG Xiaoxiao, WANG Baowei, WANG Xin, et al. Hazards, detection, and detoxification methods of aflatoxins in livestock and poultry[J]. China Feed, 2011(13): 33-36.

[42] XIE Guanghong, CHEN Cheng, XU Chuang, et al. Research on detection methods for aflatoxins[J]. Feed Industry, 2007, 28(6): 53-56.

[43] FLORES-FLORES M E, LIZARRAGA E, DE CERAIN A L, et al. Presence of mycotoxins in animal milk: a review[J]. Food Control, 2015, 53: 163-176.

[44] SADIA A, JABBAR M A, DENG Y J, et al. A survey of aflatoxin M1 in milk and sweets of Punjab, Pakistan[J]. Food Control, 2012, 26(2): 235-240.

[45] SIDDAPPA V, NANJEGOWDA D K, VISWANATH P. Occurrence of aflatoxin M1 in some samples of UHT, raw & pasteurized milk from Indian states of Karnataka and Tamilnadu[J]. Food and Chemical Toxicology, 2012, 50(11): 4158-4162.

[46] FALLAH A A, RAHNAMA M, JAFARI T, et al. Seasonal variation of aflatoxin M1 contamination in traditional Iranian dairy products[J]. Food Control, 2011, 22(10): 1653-1656.

[47] HESHMATI A, MILANI J M. Contamination of UHT milk by aflatoxin M1 in Iran[J]. Food Control, 2010, 21(1): 19-22.

[48] ASI M R, IQBAL S Z, ARIÑO A, et al. Effect of seasonal variations and lactation times on aflatoxin M1 contamination in milk of different species from Punjab, Pakistan[J]. Food Control, 2012, 25(1): 34-38.

[49] IQBAL S Z, JINAP S, PIROUZ A A, et al. Aflatoxin M1 in milk and dairy products, occurrence and recent challenges: a review[J]. Trends in Food Science & Technology, 2015, 46(1): 110-119.

[50] HÖHLER D. Ochratoxin A in food and feed: occurrence, legislation and mode of action[J]. Zeitschrift für Ernährungswissenschaft, 1998, 37(1): 2-12.

[51] BREITHOLTZ-EMANUELESSON A, PALMINGER-HALLÉN I, WOHLIN P O, et al. Transfer of ochratoxin A from lactating rats to their offspring: a short-term study[J]. Natural Toxins, 1993, 1(6): 347-352.

[52] SKAUG M A. Analysis of Norwegian milk and infant formulas for ochratoxin A[J]. Food Additives and Contaminants, 1999, 16(2): 75-78.

[53] BOUDRA H, BARNOUIN J, DRAGACCI S, et al. Aflatoxin M1 and ochratoxin A in raw bulk milk from French dairy herds[J]. Journal of Dairy Science, 2007, 90(7): 3197-3201.

[54] DENG Youtian, YUAN Hui. Research progress on toxicity mechanism of zearalenone[J]. Progress in Veterinary Medicine, 2007, 28(2): 89-92.

[55] XIA X, LI X W, DING S Y, et al. Ultra-high-pressure liquid chromatography-tandem mass spectrometry for the analysis of six resorcylic acid lactones in bovine milk[J]. Journal of Chromatography A, 2009, 1216(12): 2587-2591.

[56] EL-HOSHY S M. Occurrence of zearalenone in milk, meat and their products with emphasis on influence of milk heat treatments[J]. Archiv für Lebensmittelhygiene, 1999, 50(6): 140-143.

[57] Final SCOOP Task 3.2.10. Collection of occurrence data of Fusarium toxins in food and assessment of dietary intake by the population of EU Member States. Subtask II: Zearalenone. European Commission, Directorate-General Health and Consumer Protection[G]. [S.l.] Scientific Cooperation on Questions Relating to Food, 2003: 239-482.

[58] MARAGOS C M, RICHARD J L. Quantitation and stability of fumonisins B1 and B2 in milk[J]. Journal of the Association of Official Analytical Chemists, 1994, 77(5): 1162-1167.

[59] GAZZOTTI T, LUGOBONI B, ZIRONI E, et al. Determination of fumonisin B1 in bovine milk by LC-MS/MS[J]. Food Control, 2009, 20(12): 1171-1174.

[60] JIN Lu, DONG Guozhong. Effects of deoxynivalenol on animal immunity and reproductive performance[J]. Feed Research, 2012(3): 18-21.

[61] ZOU Guangxun, ZHANG Hongxia, HUA Rima. Research progress on toxic effects and mechanisms of T-2 toxin[J]. Asian Journal of Ecotoxicology, 2011, 6(2): 121-128.

[62] SØRENSEN L K, ELBÆK T H. Determination of mycotoxins in bovine milk by liquid chromatography tandem mass spectrometry[J]. Journal of Chromatography B, 2005, 820(2): 183-196.

Note: Figure translations are in progress. See original paper for figures.