Chapter 2
Aflatoxins in Indian Food Products

Abstract Hepatitis cases have been reported in India in the past due to consumption of food contaminated by some aflatoxin, a mycotoxin produced by *Aspergillus flavus* and generally developed in food articles grown and/or stored in hot and moist environment. The main target organ of hepatic disease, caused by regular consumption of aflatoxin-contaminated foods, is liver which may suffer from jaundice and cancer in later stages. Milk is an ideal food for such patients provided it is free from aflatoxins. The Indian Food Safety and Standards Regulation, 2011 enlists aflatoxins among crop contaminants and naturally occurring toxins. In the European Union, food regulation ascertains much lower values for maximum aflatoxin contents in food articles than that the Indian food low does. Indian food business operators seldom have to face consequences due to high aflatoxin contents, particularly in samples of exported goods, despite the fact that detoxification (removal of aflatoxins from foods) to some extent is possible.

Keywords Aflatoxin · Aspergillus · Crop contaminant · Detoxification · Food rejection · Moist storage · Mycotoxin

Abbreviations

AAA	Aromatic amino acid
BCAA	Branched-chain amino acid
DNA	Deoxyribonucleic acid
EU	European Union
FBO	Food business operator
FSSAI	Food Safety and Standards Authority of India
ICRISAT	International Crop Research Institute for the Semi-Arid Tropics
IUPAC	International Union of Pure and Applied Chemistry
mRNA	Messenger ribonucleic acid
ppb	Part per billion
ppm	Part per million
U.S. FDA	United States Food and Drug Administration

R.K. Sharma and S. Parisi, *Toxins and Contaminants in Indian Food Products*,
DOI 10.1007/978-3-319-48049-7_2

2.1 Introduction

In 1974, the people of India came across to recognise hepatitis as a result of the consumption of maize contaminated with *Aspergillus flavus*. The outbreak of disease, lasted for 2 months, was confined to the Western Indian tribal population belonging to Banswada district of Rajasthan and Panchmahals district of Gujarat. The people suddenly began to show the symptoms of ascites and oedema of lower limbs and portal hypertensions. Hepatitis was reported in 200 villages confirming 106 deaths. The analysis of contaminated maize samples showed that the diet of affected people contained the fungus *A. flavus* in the range of 6.25–15.6 parts per million (ppm). This result means affected people might had consumed 2000–6000 μg/kg or parts per billion (ppb) of aflatoxins, daily over a period of 1 month (Krishnamachari et al. 1975). Tandon, Krishnamurthy and coworkers presumed that an epidemic of jaundice in north-Western India (1974) was also due to toxic hepatitis which affected both humans and dogs (Tandon et al. 1977). However, the word 'aflatoxicosis' had appeared in public news domain of India in the 1960s with reference to the sudden death of 2219 chicks in poultry farms of Mysore and other parts of Karnataka state (Gopal et al. 1969). In October, 1985, the egg production dropped from 85 to 40 % in and around Warangal in Andhra Pradesh, as impact of severe aflatoxicosis in poultry; this outbreak gradually increased when bird mortality rate decreased sharply after the feed—maize and groundnut cake, contaminated by aflatoxin—was changed (Sastry et al. 1965). The *post-mortem* examination of dead birds revealed liver lesions while aflatoxin content in feed samples was detected to be 600 ppb (Choudary 1986). In 1994, more than 0.2 million broiler chicken died in Ranga Reddy district of Andhra Pradesh after eating aflatoxin-contaminated groundnut cake feed. The International Crop Research Institute for the Semi-Arid Tropics (ICRISAT) revealed this fact (ICRISAT 2002). That conclusion means that the establishment of aflatoxin-disease correlation is till to date a challenging task, despite innovative laboratory testing procedures. In fact, the diagnosis of aflatoxicosis—like other mycotoxicoses—is difficult due to its symptoms being similar to those of diseases with other causes as well as presence of several mycotoxins along with aflatoxins in foods or feeds which synergise effects.

It may be remembered that the name 'aflatoxin' was first created around 1960 when it was discovered that the source of Turkey X—an avian disease spread in Great Britain—was an *A. flavus* toxin (Wannop 1961). The toxic material was extracted from it and chromatographically separated into four distinct compounds based on fluorescent colour—B for blue and G for green—with scripts relating to relative mobility in early 1960s (Nesbitt et al. 1962; Sargeant et al. 1963); two forms B_1 and G_1 were synthesised in late 1960s and early 1970s (Buechi and Weinreb 1971). The first reliable correlation between aflatoxin contamination and hepatomegaly among the children of Canara district of Karnataka in India was reported in mid-1970s (Sreenivasmurthy 1977). Despite several limitations of symptom distinction and co-mycotoxins' synergy effects, Sreenivasmurthy's studies of the correlation aflatoxin–hepatomegaly are till to date considered worthy

when a hepatitis, is likely to be named 'aflatoxicosis.' On the other hand, the scientific knowledge of aflatoxin development in farm produce is extremely clear. Reddy and Raghavender report that adequate food monitoring programs are needed with relation to the possible occurrence of mycotoxins in notable amounts, because related outbreaks continue to be signalled in India (Reddy and Raghavender 2007).

It is worth mentioning that the prevention of mycotoxin contamination in farm yields is progressively becoming tougher worldwide due to global warming and flash floods. India is called the land of climatic contrasts with temperatures shot up to 50 °C in certain parts of Rajasthan and south-west Punjab in hot summer afternoon as well as dipped up to −40 °C in cold arid region of Cargill in severe winter night. Therefore, global warming and flash floods in India can determine the fast development of aflatoxins in food articles. Fast deforestation gives rise to adverse environmental conditions that affect farm produce in both pre-harvest and postharvest stages; therefore, Food business operators (FBO) in India have to face consequences as export consignment rejections and credibility loss.

2.2 Chemistry of Aflatoxins

2.2.1 The Aflatoxin/Foodborne Diseases Correlation

With relation to intoxications and infections, there are basically two types of food-borne diseases. Intoxications are food-borne diseases caused by the consumption of intoxicants like:

(a) Synthetic insecticides (chemicals) sprayed in farms, or
(b) Naturally poisonous plant or animal tissues, or
(c) Metabolic toxic products formed by bacteria, algae and fungi.

On the other hand, infections are caused by the enterotoxigenic or invasive penetration of pathogenic microorganisms into the body, via foods, and the consequent production of mycotoxins. Aflatoxins are mycotoxins produced by *A. flavus* and *A. parasiticus* species of fungi which colonise and contaminate crops before harvest or during storage in generally hot and moist environments. Host crops for these fungi species include maize, sorghum, groundnut, rice, wheat, cassava, pistachio, cashew, almond, cottonseed, turmeric, chilli, peppers and even the cattle fodder. Aflatoxin can enter in form of feed in cattle farms or dairies too if made from affected fodder and oil seed cakes (ground nut, cottonseed, etc.). Should animals graze aflatoxin-contaminated feed, obviously they would produce milk containing a different form of aflatoxin as the result of the metabolisation of the original molecule in the consumed feed. Eggs and other poultry products can be contaminated in the same way with aflatoxins when birds consume such kind of affected grains.

The disease can be observed in humans and animals, including birds, due to the consumption of aflatoxin-contaminated foods or feeds: this disease is called aflatoxicosis. It is worth mentioning that the presence of *Aspergillus* fungi does not

always indicate harmful levels of aflatoxins. Actually, this contamination might be safe to some extent if present in minor amount, but the consumption of *Aspergillus*-contaminated food or feed is always risky (Hudler 1998). Aflatoxins are heterocyclic compounds and exert toxic effects after consumption in the body within several ways. High-level aflatoxin consumption can give rise to hepatic necrosis, resulting later in cirrhosis or carcinoma of the liver. Generally, the patient might be in very serious condition in absence of cures at early stages: consequences might lead the subject to coma and even death. It has been observed that adult humans can tolerate these mycotoxins with low consequences, while children may suffer serious damages and animals are not so resistant (Abbas 2005; Williams et al. 2004). Aflatoxins are among the most carcinogenic substances known (Hudler 1998).

2.2.2 Types of Aflatoxins

20 fungal metabolites are known: at least 14 of these compounds are mostly studied as typical aflatoxins. Only six of these molecules—aflatoxins B_1, B_2, G_1, G_2, M_1 and M_2 are normally found in foods.

The most toxic among all types, aflatoxin B_1, is produced by both *A. flavus* and *A. parasiticus*. The same thing can be affirmed for aflatoxin B_2, the dihydro-derivative of aflatoxin B_1. Aflatoxins G_1 and G_2 (the last compound is the dihydro-derivative of the G_1 type) are produced by *A. parasiticus*. Aflatoxin M_1 is the metabolite of aflatoxin B_1 in human and animal milk; the type M_2—the metabolite of aflatoxin B_2—is also found in human and animal milk.

2.2.3 Chemical Structure of Aflatoxins

The chemical structures of three of the six major aflatoxins are shown in Figs. 2.1, 2.2 and 2.3 with relation to aflatoxins B_1, G_1 and M_1, respectively. The following list shows basic properties of above-mentioned six aflatoxins:

(a) Aflatoxin B_1 (Fig. 2.1). This compound[1] exhibits blue fluorescence; crystals have melting points between 268 and 269 °C (letter B)
(b) Aflatoxin B_2. Its crystals have melting points between 286 and 289 °C. The compound[2] exhibits blue fluorescence

[1]International Union of Pure and Applied Chemistry (IUPAC) name: (6aR-*cis*)-2, 3, 6a, 9a-tetrahydro-4-methoxycyclopanta(c)furo[3′, 2′; 4, 5] furo[2, 3-h] [1] benzopyran-1,11-dione.

[2]IUPAC designation: (6aR-*cis*)-2, 3, 6a, 8, 9, 9a-hexahydro-4-methoxycylopenta (c) furo [3′, 2′; 4, 5] furo [2, 3-h] [1] benzopyran-1,11-dione.

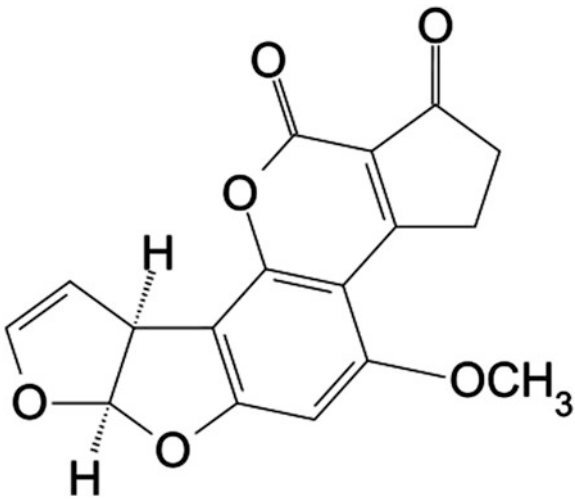

Fig. 2.1 The chemical structure of aflatoxin B_1. BKchem version 0.13.0, 2009 (http://bkchem.zirael.org/index.html) has been used for drawing this structure

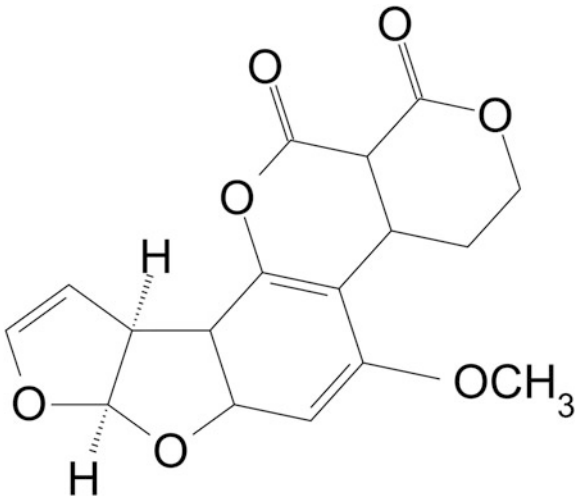

Fig. 2.2 The chemical structure of aflatoxin G_1. BKchem version 0.13.0, 2009 (http://bkchem.zirael.org/index.html) has been used for drawing this structure

(c) Aflatoxin G_1 (Fig. 2.2). Crystals have melting points between 244 and 246 °C. The compound[3] exhibits green fluorescence (letter G)
(d) Aflatoxin G_2. The compound[4] exhibits green-blue fluorescence. Its crystals have melting points between 237 and 240 °C

[3]IUPAC name: 3, 4, 7aα, 10aα-tetrahydro-5-methoxy-1*H*,12*H*-furo[3′, 2′; 4, 5]furo[2, 3-h]pyrano [3, 4-c] (1) benzopyran-1,12 dione.

[4]IUPAC designation: 3, 4, 7aα9, 10, 10aα-hexahydro-5–methoxy-1H, 12*H*–furo[3′, 2′: 4, 5]furo [2, 3-h] pyrano [3, 4-c] [1] benzopyran–1, 12-dione.

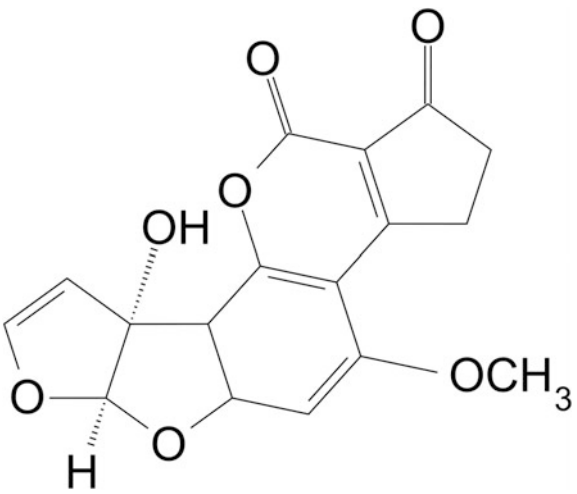

Fig. 2.3 The chemical structure of aflatoxin M_1. BKchem version 0.13.0, 2009 (http://bkchem.zirael.org/index.html) has been used for drawing this structure

(e) Aflatoxin M_1 (Fig. 2.3). The compound[5] exhibits blue–violet fluorescence. It is found in milk; consequently, the affix 'M' exists in its name. Crystals of aflatoxin M_1 have melting point: 299 °C

(f) Aflatoxin M_2. Its crystals have melting point: 293 °C. This compound[6] exhibits violet fluorescence.

2.2.4 *Physiological Actions of Aflatoxins*

Aflatoxicosis is primarily a hepatic disease: as a result, its main target organ in humans and other mammals is liver. In other words, aflatoxins are capable of developing liver diseases in mammals including humans. The liver, found in front of the stomach at the top of the abdominal cavity, is the vital organ that tends to protect body from several poisons, insecticides and environmental pollutants. The main function of liver is the transformation of these harmful chemicals into harmless or less harmful products that can be removed from the body in bile or urine. It also removes food particles and microbial products from blood coming from intestines. A well-functioning liver is characterized by a normal pattern of amino acids circulation with the balanced concentrations of branched-chain amino acids (leucine, isoleucine and valine) and aromatic amino acids (phenylalanine, tyrosine and tryptophan) leading to the production of true neurotransmitters. When

[5]IUPAC name: 2, 3, 6a 9a–tetrahydro—9a hydroxy–4–methoxycyclopenta[c] furo [3′, 2′ :4, 5] furo [2,3-h] [1] benzopyran–1,11–dione.

[6]IUPAC designation: 2, 3, 6a, 8, 9, 9a hexahydro–9a–hydroxy–4- methoxycyclopenta[c] furo [3,′ 2′: 4, 5] furo [2, 3—h] [1] benzopyran–1, 12–dione.

food-containing toxic substances are consumed beyond tolerance limits, the person might suffer from chronic liver disease, called hepatic disease or hepatitis. The patients with chronic liver disease are likely to be intolerant with relation to enteral proteins. Therefore, the balance between branched-chain amino acids (BCAA) and aromatic amino acids (AAA) is disturbed, with decrease in BCAA concentration and increase of AAA amount, in the case of hepatic disease. In other words, BCAA/AAA ratio falls in patients with hepatic encephalopathy with the production of false neurotransmitters. Commercial enteral nutrition products formulated for patients with chronic liver disease have low levels of total proteins, high BCAA concentrations and low AAA amounts (Alpers et al. 2002).

The high-level aflatoxin intake initially produces an acute hepatic necrosis which later on results in cirrhosis or carcinoma of the liver. Bleeding, oedema and mental changes are the common symptoms of acute liver failure. In the later stage, the patient might suffer from jaundice and subsequently liver cancer if BCAA/AAA ratio is not timely maintained along with reduction of protein contents. The prolonged exposure of aflatoxin may lead to the increased degradation of heme (the part of hemoglobin of blood) into bilirubin (the pigment of bile) and cause jaundice. It is worth mentioning that bilirubin—the major end-product of biological breakdown of heme—is the chromophore responsible for colouration in various forms of jaundice; should it exceed limit exposure, high risk of developing liver cancer should be expected, as aflatoxin metabolites may intercalate into deoxyribonucleic acid (DNA) and alkylate bases through epoxide moiety. Probably, this is the cause for mutations in the p–53 gene (Aguilar et al. 1993).

With concern to foods for liver-disease patients, these products should necessarily be rich in BCAA, but the amount of total proteins should be relatively low. Furthermore, taken meals should release less carbon dioxide per calorie. This reflection means fat/carbohydrate ratio should relatively be higher, with high water contents. Such a naturally available wonder food is milk.

Cow milk normally contains 86.5 % of water, 3.4 % of proteins, 0.7 % of ash, 3.0–4.6 % of fat matter, and the remaining 4.8–6.4 % of lactose. However, needless to say that milk given to patient should be almost free from or quite well within prescribed limits for M-type aflatoxins, and the patient should not suffer from lactose intolerance. At this point, it is essential to know toxicity of aflatoxins and its prescribed limits in Indian commodities.

2.3 Toxicity of Aflatoxins

The toxicity of aflatoxins B_1, B_2, G_1 and G_2 is mostly measured as oral LD_{50} (dosage of the toxin at which 50 % of test animals are killed) in μg per 50 grams of body weight (1-day old duckling), while for toxic aflatoxins like M_1 and M_2 it is measured as oral LD_{50} in μg per duckling. Carnaghan, Buchi and Holzapfel have extensively measured LD_{50} measurements with relation to aflatoxins. In general, the

most toxic aflatoxins appear to be types B_1 and M_1: LD_{50} are ≤ 18 and 12–16 mg/kg, respectively), while aflatoxin G_2 seems to show very low values if compared with other aflatoxins (Budavari and O'Neil 1989; D'Mello 2003; Westendorf 1999).

2.4 Placement of Aflatoxins in Food Safety and Standards Regulations 2011

2.4.1 Limits of Aflatoxins in Indian Food Commodities

The FSSR 2011 enlists aflatoxins among crop contaminants like patulin and ochratoxin—antibiotics from metabolites of a number of fungi, such as *A. clavatus, A claviforme, A. gigateus, A. sulphureus, Penicillium patulum*—and naturally occurring toxic substances like agaric acid, hydrocyanic acid, hypericin, and safrole (FSSR 2011, The interested Reader is invited to consult the FSSR 2011, Chap 2, Sect. 2). For example purposes, it can be noted here that patulin is allowed in apple juice and apple juice ingredients in other beverages up to 5.0 mg/kg, while aflatoxin M_1 is permitted in milk up to 0.5 mg/kg only.

2.4.2 Chemical Structures of Crop Contaminants and Naturally Occurring Toxins Other Than Aflatoxins

Patulin[7] and ochratoxin A,[8] like aflatoxins, are crop contaminants related to groups of fungal metabolites (Budavari and O'Neil 1989). The former compound (Fig. 2.4) possesses a furo-pyran structure and the latter has a typical benzo-pyran structure. Agaric (or agaricic) acid,[9] hydrocyanic acid, hypericine[10] and safrole[11] (Fig. 2.5) are not crop contaminants, but those exist in flora as naturally occurring constituents with recognised toxicity in nature.

---

[7]IUPAC name: 4-hydroxy-44-furo [3, 2-c] pyran -2 (6*H*)-one.

[8]IUPAC designation: (*R*)-*N* [(5-chloro -3, 4-dihydro -8-hydroxy-3-methyl-1-oxo-1*H*-2-benzopyran-7-yl) carbonyl]-*L*-phenylalanine.

[9]IUPAC name: 2-hydroxy-1, 2, 3-nonadecane-tricarboxylic acid.

[10]IUPAC designation: hexahydroxy dimethyl phenanthro perylene dione-1, 3, 4, 6, 8, 13, - hexahydroxy—10, 11- dimethyl phenan-thro [1, 10, 9, 8—opqra] perylene—7, 14-dione.

[11]IUPAC name: 4-allyl -1, 2 methylene dioxy benzene.

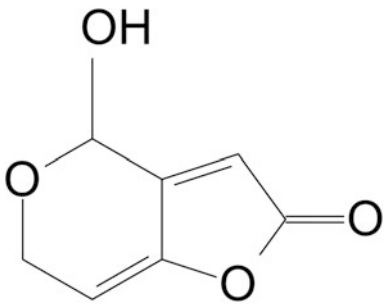

Fig. 2.4 The chemical structure of patulin. BKchem version 0.13.0, 2009 (http://bkchem.zirael.org/index.html) has been used for drawing this structure

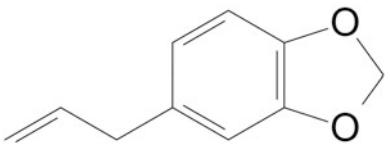

Fig. 2.5 The chemical structure of safrole. BKchem version 0.13.0, 2009 (http://bkchem.zirael.org/index.html) has been used for drawing this structure

2.5 Comparison of Aflatoxin Limits in Indian Food Law with EU Standards

The maximum permissible limit for aflatoxins (B_1, B_2, G_1, G_2, etc.) contents in all food commodities for sale in Indian market is 30 μg/kg or ppb, while the tolerance value for aflatoxins M in milk is 0.5 μg/kg. If these aflatoxins limits in Indian food commodities, as per FSSR 2011, are compared with European Union (EU) standards, the EU regulation looks much more stringent. In detail, EU regulations are considered among the stringiest food laws if compared with Indian regulations and those of other countries, around 100 in number, which have regulations governing aflatoxins in food and feed.

The EU sets limits for aflatoxin B_1 and for total aflatoxins in nuts, dried fruits, cereals and spices. Limits vary according to commodity and range from 2 to 12 μg/kg for B_1 and from 4 to 15 μg/kg for total aflatoxins. There is also a limit of 0.050 μg/kg for aflatoxin B_1 and M_1 in milk and milk products in EU regulations. Maximum aflatoxin B_1 and M_1 limits for infant foods in EU regulations are 0.10 and 0.025 μg/kg, respectively (Lawley 2013).

The EU regulation permits the maximum total aflatoxin contamination limit varying from 4 to 15 μg/kg (general commodities), while total aflatoxin limit for all commodities is 30 μg/kg in India. Therefore, food commodities (with the exclusion of milk), in India may legally have aflatoxin contamination as high as 2–7.5 times that similar food articles in Europe. Indian milk may legally be 10 times more contaminated than EU milk when speaking of aflatoxin M_1 (0.5 versus 0.05 ppb).

2.6 Indian Market Surveys for Aflatoxin Contents in Commodities

Fungi are capable to produce aflatoxins in food commodities in favourable conditions (higher moisture, temperature and adequate substratum). Aflatoxin contamination in food articles in India is observed to be highest when humidity is above 13 % and temperature is between 24 and 37 °C.

The warm and wet sea-shore regions of southern part of India possess the favourable environment for fungal synthesis of aflatoxins in foods. A valuable study was conducted on market samples of food grains, such as Bengal gram, *bajra/cumbu*, maize and *jowar*/sorghum and grain flour procured from Chennai, Tamil Nadu, in the first decade of the twenty-first century with the aim of analysing aflatoxin B_1: recovery percentage for this aflatoxin was reported to be 90 % (Ramesh et al. 2013). In detail, the contamination of aflatoxin B_1 was found to be 68.18 % in food grains whereas 100 % in grain flour. This result might be due to improper postharvest technology and storage conditions; consequently, the assessment of contamination should be improved. Ramesh and co-workers have also reported, on the basis of their own study, that analysed food grains for aflatoxins were within safe limit of Indian and United States Food and Drug Administration (U.S. FDA) standards, except maize, but higher than EU standards (Ramesh et al. 2013).

The surveys of aflatoxin M_1 in commercial milk samples and infant formula milk samples in Goa (in early 2010s) and different parts of India (in mid-2000s) are mostly found to exceed not only EU recommended limits but also Codex Alimentarius, Food Safety and Standards Authority of India (FSSAI) and U.S. FDA recommended limits. Similar results have been published by the ICRISAT with reference to '*Aspergillus* and aflatoxins in groundnuts' and consequent high values in milks. In detail, the ICRISAT has recently revealed that aflatoxin levels in peanuts (area: southern India) may be 40-times higher than allowed limits with relation to Indian permitted levels (Gandhi 2016).

2.7 Consequences for Food Business Operators

The issue of aflatoxins contamination in crops, finished grains and processed foods is often concerned with environmental conditions from pre-harvest to storage steps. The warm humid environment during harvesting in semi-arid tropical zones (and sea shores also) cannot be defined nowadays as a natural event because of the Earth global warming since decades; most probable causes are generally considered deforestation and pollution increase in air and water. The storage step is surely correlated with anthropic activities; therefore, highly elevated levels of aflatoxins in food articles detected in market surveys in India are mainly ascribed to the responsibility of human beings. Although aflatoxin contamination begins to

develop in the pre-harvest stage (abnormally hot moist environment), it should be noted that farmers are not responsible for the current weather conditions. The horizontal urbanisation on the original (or natural) forest-pasture tract, particularly on mountains, seems to be the most accountable phenomenon for vast deforestation. Consequently, remarkable temperature augments and a considerable contribution to global warming on a local scale are observed.

FBO have to face the consequences for high aflatoxins contents in samples of both indigenous and exported goods. India has been exporting spices, nuts and grains with difficulty and sometimes facing rejections due to stringent requirements with close limit of aflatoxin contents in food articles. ICRISAT has recently observed the situation in context of both the public health and the business scenario in India (Gandhi 2016):

The dense mountain forestation is the first requirement, in India, to maintain unfavourable conditions for aflatoxin formation in crops. Predictable consequences should obviously lead to agribusiness promotion with reduced export consignment rejections. Second, detoxification of aflatoxin containing foods can perhaps work to some extent in Indian agribusiness improvement. Roasting treatments on certain food articles (example: roasting coffee at 180 °C), γ-radiation treatments of grains, and fermentation processes on milk (for curd or yoghurt productions) are currently employed to partly detoxify *Aspergillus*-contaminated foods with the possible reduction of aflatoxin levels in food articles to 30 % at least (Herzallah et al. 2008).

The solvent extraction for aflatoxins removal is somewhat more effective; however, this process may produce toxic by-products due to the use of polar solvents like alcohols and ketones. Biological decontamination is nowadays considered quite safe to remove aflatoxins. For example, *Flavobacterium aurantiacum* is reported to reduce aflatoxin B_1 amounts in contaminated corns (Khanafari et al. 2007).

References

Abbas HK, editor. Aflatoxin and food safety. Boca Raton: CRC Press; 2005.

Aguilar F, Hussain SP, Cerutti P. Aflatoxin B1 induces the transversion of G– > T in codon 249 of the p53 tumor suppressor gene in human hepatocytes. Proc Natl Acad Sci USA. 1993;90 (18):8586–90.

Alpers DH, Stenson WF, Bier DM. Manual of nutritional therapeutics. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2002.

Budavari S, O'Neil MJ. The Merck index: an encyclopedia of chemicals, drugs, and biological. 11th ed. Inc, Rahway: Merck & Co.; 1989.

Buechi G, Weinreb SM. Total syntheses of aflatoxins M_1 and G_1 and an improved synthesis of aflatoxin B_1. J Am Chem Soc. 1971;93(3):746–52. doi:10.1021/ja00732a032.

Choudary C. An outbreak of 'fatty liver syndrome' in commercial layer farms. Poult Advis. 1986;19:59–60.

D'Mello JPF (ed). Food safety: contaminants and toxins. Oxon: CABI Publishing; 2003.

Gandhi MS. 'Carcinogens now even in samples considered safest': Maneka Gandhi advises against drinking milk. Firstpost.com; 2016. http://www.firstpost.com/india/carcinogens-now-

even-in-samples-considered-safest-maneka-gandhi-advises-against-drinking-milk-2571542.html. Accessed 04 July 2016.
Gopal T, Zaki S, Narayanaswami M, Premlata S. Aflatoxicosis in fowls'. Indian Vet J. 1969;46:348–9.
Herzallah S, Alshawabkeh K, Fataftah AA. Aflatoxin decontamination of artificially contaminated feeds by sunlight, γ-radiation, and microwave heating. J Appl Poult Res. 2008;17(4):515–21. doi:10.3382/japr.2007-00107.
Hudler GW. Magical mushrooms, mischievous molds: the remarkable story of the fungus kingdom and its impact on human affairs. Princeton: Princeton University Press; 1998.
ICRISAT. Aflatoxin: a deadly hazard, 1 May, 2002. International Crop Research Institute for the Semi-Arid Tropics (ICRISAT). http://www.icrisat.org/newsroom/news-releases/icrisat-pr-2002-media9.htm. Accessed 05 July 2016.
Khanafari A, Soudi H, Miraboulfathi M. Biocontrol of Aspergillus flavus and aflatoxin B_1 production in corn. J Environ Health Sci Eng. 2007;4(3):163–8.
Krishnamachari KAVR, Bhat RV, Nagarajan V, Tilak TBG. Investigations into an outbreak of hepatitis in parts of Western India. Indian J Med Res. 1975;63(7):1036–48.
Lawley R. Aflatoxins. Food safety watch: the science of safe food. 2013. www.foodsafetywatch.org/factsheets/aflatoxins/. Accessed 05 July 2016.
Nesbitt BF, O'Kelly J, Sargeant K, Sheridan A. Aspergillus Flavus and Turkey X disease: toxic metabolites of aspergillus flavus. Nature. 1962;195:1062–3. doi:10.1038/1951062a0.
Ramesh J, Sarathchandra GV, Sureshkumar V. Survey of market samples of food grains and grain flour for Aflatoxin B1 contamination. Int J Curr Microbiol Appl Sci. 2013;2(5):184–8.
Reddy BN, Raghavender CR. Outbreaks of aflatoxicoses in India. Afr J Food Agric Nutr Dev. 2007;7:5.
Sargeant K, Carnaghan RBA, Allcroft R. Toxic products in groundnuts. Chem Origin Chem Ind (London). 1963; 53.
Sastry GA, Narayana JV, Rama Rao P, Christopher KJ, Hill KR. A report on the groundnut toxicity in Murrah buffaloes in Andhra Pradesh (India). Indian Vet J. 1965;42:79–82.
Sreenivasmurthy V. Mycotoxins in foods—a public health problem. Arogya J Health Sci. 1977;3:4–13.
Tandon BN, Krishnamurthy L, Koshy A, Tandon HD, Ramalingaswami V, Bhandari JR, Mathur MM, Mathur PD. Study of an epidemic of jaundice presumably due to toxic hepatitis in North West India. Gastroenterology. 1977;72(3):488–94.
Wannop CC. The histopathology of turkey "X" disease in Great Britain. Avian Dis. 1961;5(4):371–81. doi:10.2307/1587768.
Westendorf J. Natural compounds. In: Marquardt H, Schäfer SG, McClellan RO, Welsch F, editors. Toxicology. San Diego and London: Academic Press; 1999.
Williams JH, Phillips TD, Jolly PE, Stiles JK, Jolly CM, Aggarwal D. Human aflatoxicosis in developing countries: a review of toxicology, exposure, potential health consequences, and interventions. Am J Clin Nutr. 2004;80(5):1106–22.

http://www.springer.com/978-3-319-48047-3

Toxins and Contaminants in Indian Food Products

Sharma, R.K.; Parisi, S.

2017, VII, 65 p. 16 illus., 1 illus. in color., Hardcover
ISBN: 978-3-319-48047-3