Aflatoxin in nuts

Not to be confused with Alpha toxin. Chemical structure of aflatoxin B1 Aflatoxins are poisonous carcinogens and mutagens that are produced by certain molds (Aspergillus flavus and Aspergillus parasiticus) which grow in soil, decaying vegetation, hay, and grains. They are regularly found in improperly stored staple commodities such as cassava, chili peppers, cottonseed, millet, peanuts, rice, sesame seeds, sorghum, sunflower seeds, sweetcorn, tree nuts, wheat, and a variety of spices. When contaminated food is processed, aflatoxins enter the general food supply where they have been found in both pet and human foods, as well as in feedstocks for agricultural animals. Animals fed contaminated food can pass aflatoxin transformation products into eggs, milk products, and meat.[1] For example, contaminated poultry feed is suspected in the findings of high percentages of samples of aflatoxin-contaminated chicken meat and eggs in Pakistan.[2] Children are particularly affected by aflatoxin exposure, which is associated with stunted growth,[3] delayed development,[4] liver damage, and liver cancer. An association between childhood stunting and aflatoxin exposure[5] has been reported in some studies[6][7] but could not be detected in all.[8][9] Furthermore, a causal relationship between childhood stunting and aflatoxin exposure has yet to be conclusively shown by epidemiological studies, though such investigations are under way.[10][11] Adults have a higher tolerance to exposure, but are also at risk. No animal species is immune. Aflatoxins are among the most carcinogenic substances known.[12] After entering the body, aflatoxins may be metabolized by the liver to a reactive epoxide intermediate or hydroxylated to become the less harmful aflatoxin M1. Aflatoxins are most commonly ingested. However the most toxic type of aflatoxin, B1, can permeate through the skin.[13] The United States Food and Drug Administration (FDA) action levels for aflatoxin present in food or feed is 20 to 300 ppb.[14] The FDA has had occasion to declare both human and pet food recalls as a precautionary measure to prevent exposure. The term "aflatoxin" is derived from the name of one of the molds that produce it, Aspergillus flavus. It was coined around 1960 after its discovery as the source of "Turkey X disease".[15] Aflatoxins form one of the major groupings of mycotoxins. Major types and their metabolites Aflatoxin B1 is considered the most toxic and is produced by both Aspergillus flavus and Aspergillus parasiticus. Aflatoxin M1 is present in the fermentation broth of Aspergillus parasiticus, but it and aflatoxin M2 are also produced when an infected liver metabolizes aflatoxin B1 and B2. Aflatoxin B1 and B2 (AFB), produced by Aspergillus flavus and A. parasiticus Aflatoxin G1 and G2 (AFG), produced by some Group II A. flavus and Aspergillus parasiticus[16] Aflatoxin M1 (AFM1), metabolite of aflatoxin B1 in humans and animals (exposure in ng levels may come from a mother's milk) Aflatoxin M2, metabolite of aflatoxin B2 in milk of cattle fed on contaminated foods[17] Aflatoxicol (AFL): metabolite produced by breaking down the lactone ring Aflatoxin Q1 (AFQ1), major metabolite of AFB1 in in vitro liver preparations of other higher vertebrates[18] AFM, AFQ, and AFL retain the possibility to become an epoxide. Nevertheless, they appear much less capable of causing mutagenesis than the unmetabolized toxin.[19] Contamination conditions Aflatoxins are produced by both Aspergillus flavus and Aspergillus parasiticus, which are common forms of 'weedy' molds widespread in nature. The presence of those molds does not always indicate that harmful levels of aflatoxin are present, but does indicate a significant risk. The molds can colonize and contaminate food before harvest or during storage, especially following prolonged exposure to a high-humidity environment, or to stressful conditions such as drought. The native habitat of Aspergillus is in soil, decaying vegetation, hay, and grains undergoing microbiological deterioration, but it invades all types of organic substrates whenever conditions are favorable for its growth. Favorable conditions for production of aflatoxins include high moisture content (at least 7%) and temperatures from 55 °F (13°C) to 104 °F (40°C) (optimum 81--86 °F).[20][21] Aflatoxins have been isolated from all major cereal crops, and from sources as diverse as peanut butter and cannabis. The staple commodities regularly contaminated with aflatoxins include cassava, chilies, corn, cotton seed, millet, peanuts, rice, sorghum, sunflower seeds, tree nuts, wheat, and a variety of spices intended for human or animal consumption. Aflatoxin transformation products are sometimes found in eggs, milk products, and meat when animals are fed contaminated grains.[1][22] A study conducted in Kenya and Mali found that the predominant practices for drying and storage of maize were inadequate in minimizing exposure to aflatoxins.[23] Organic crops, which are not treated with fungicides, may be more susceptible to contamination with aflatoxins.[24] Prevention There is very limited evidence to show that agricultural and nutritional education can reduce exposure to aflatoxin in low to middle income countries.[25] Pathology No animal species is immune to the acute toxic effects of aflatoxins. Adult humans have a high tolerance for aflatoxin exposure and rarely succumb to acute aflatoxicosis,[26] but children are particularly affected, and their exposure can lead to stunted growth and delayed development, in addition to all the symptoms mentioned below.[4] High-level aflatoxin exposure produces an acute hepatic necrosis (acute aflatoxicosis), resulting later in cirrhosis or carcinoma of the liver. Acute liver failure is made manifest by bleeding, edema, alteration in digestion, changes to the absorption and/or metabolism of nutrients, and mental changes and/or coma.[26] Chronic, subclinical exposure does not lead to symptoms so dramatic as acute aflatoxicosis. Chronic exposure increases the risk of developing liver and gallbladder cancer,[27] as aflatoxin metabolites may intercalate into DNA and alkylate the bases through epoxide moiety. This is thought to cause mutations in the p53 gene, an important gene in preventing cell cycle progression when there are DNA mutations, or signaling apoptosis (programmed cell death). These mutations seem to affect some base pair locations more than others, for example, the third base of codon 249 of the p53 gene appears to be more susceptible to aflatoxin-mediated mutations than nearby bases.[28] As with other DNA-alkylating agents, Aflatoxin B1 can cause immune suppression, and exposure to it is associated with an increased viral load in HIV positive individuals.[29][30] The expression of aflatoxin-related diseases is influenced by factors such as species, age, nutrition, sex, and the possibility of concurrent exposure to other toxins. The main target organ in mammals is the liver, so aflatoxicosis primarily is a hepatic disease. Conditions increasing the likelihood of aflatoxicosis in humans include limited availability of food, environmental conditions that favour mould growth on foodstuffs, and lack of regulatory systems for aflatoxin monitoring and control.[31] A regular diet including apiaceous vegetables, such as carrots, parsnips, celery, and parsley may reduce the carcinogenic effects of aflatoxin.[32] There is no specific antidote for aflatoxicosis. Symptomatic and supportive care tailored to the severity of the liver disease may include intravenous fluids with dextrose, active vitamin K, B vitamins, and a restricted, but high-quality protein diet with adequate carbohydrate content. In other animals In dogs, aflatoxin has potential to lead to liver disease. Low levels of aflatoxin exposure require continuous consumption for several weeks to months in order for signs of liver dysfunction to appear.[33] Some articles have suggested the toxic level in dog food is 100–300 ppb and requires continuous exposure or consumption for a few weeks to months to develop aflatoxicosis.[34] No information is available to suggest that recovered dogs will later succumb to an aflatoxin-induced disease. Turkeys are extremely susceptible to aflatoxicosis. Recent studies have revealed that this is due to the efficient cytochrome P450 mediated metabolism of aflatoxin B1 in the liver of turkeys and deficient glutathione-S-transferase mediated detoxification.[35][36] Some studies on pregnant hamsters showed a significant relationship between exposure of aflatoxin B1 (4 mg/kg, single dose) and the appearance of developmental anomalies in their offspring.[37] In 2005, Diamond Pet Foods discovered aflatoxin in a product manufactured at their facility in Gaston, South Carolina.[38][39] In 23 states, Diamond voluntarily recalled 19 products formulated with corn and manufactured in the Gaston facility. Testing of more than 2,700 finished product samples conducted by laboratories confirmed that only two date codes of two adult dog formulas had the potential to be toxic.[40] In December 2020 and January 2021, Midwestern Pet Foods recalled dog food that contained fatal levels of aflatoxin.[41] As many as 70 dogs had died from aflatoxin poisoning by January 12, 2021.[42] Detection in humans There are two principal techniques that have been used most often to detect levels of aflatoxin in humans. The first method is measuring the AFB1-guanine adduct in the urine of subjects. The presence of this breakdown product indicates exposure to aflatoxin B1 during the past 24 hours. This technique measures only recent exposure, however. Due to the half-life of this metabolite, the level of AFB1-guanine measured may vary from day to day, based on diet, it is not ideal for assessing long-term exposure. Another technique that has been used is a measurement of the AFB1-albumin adduct level in the blood serum. This approach provides a more integrated measure of exposure over several weeks or months. List of outbreaks This section needs expansion. You can help by adding to it. (December 2014) International sources of commercial peanut butter, cooking oils (e.g. olive, peanut and sesame oil), and cosmetics have been identified as contaminated with aflatoxin.[43][44][45] In some instances, liquid chromatography–tandem mass spectrometry (LC–MS/MS), and other analytical methods, revealed a range from 48% to 80% of selected product samples as containing detectable quantities of aflatoxin. In many of these contaminated food products, the aflatoxin exceeded the safe limits of the U.S. Food and Drug Administration (FDA), or other regulatory agency.[44][45][46] 2003 Kenya: acute poisoning, 120 confirmed deaths.[47][48] February–March 2013: Romania, Serbia, Croatia imported into western Europe - 2013 aflatoxin contamination. February 2013: Iowa contamination.[49] 2014 (ongoing): Nepal and Bangladesh, neonatal exposures, found in umbilical cord blood.[47] 2019 Kenya: five brands of maize flour recalled due to contamination.[50] 2021 USA: Contamination of pet food manufactured by Midwestern Pet Food, causing the deaths of at least 70 dogs.[51] 2021 (ongoing) Sri Lanka: Contaminated Coconut oil released for public consumption by local government.[52] See also Aflatoxin total synthesis 2013 aflatoxin contamination Mycotoxins in animal feed Sterigmatocystin, a related toxin Resources ^ a b Fratamico PM, Bhunia AK, Smith JL (2008). 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Detailed listing and information on all Aspergillus mycotoxins Aflatoxin, ICRISAT Aspergillusflavus.org Diamond Pet Food Recall Aflatoxin B1 Retrieved from " 2 3-MCPD Names Preferred IUPAC name 3-Chloropropane-1,2-diol Other names 3-Monochloropropane-1,2-diol; α-Chlorohydrin; Glycerol α-monochlorohydrin; Chlorodeoxyglycerol; 3-Chloro-1,2-propanediol Identifiers CAS Number 96-24-2 Y 3D model (JSmol) Interactive image ChEBI CHEBI:18721 Y ChemSpider 7018 Y ECHA InfoCard 100.002.267 EC Number 202-492-4 KEGG C18676 N PubChem CID 7290 UNII QGS78A3T6P Y CompTox Dashboard (EPA) DTXSID4020664 InChI InChI=1S/C3H7ClO2/c4-1-3(6)2-5/h3,5-6H,1-2H2 YKey: SSZWWUDQMAHNAQ-UHFFFAOYSA-N YInChI=1/C3H7ClO2/c4-1-3(6)2-5/h3,5-6H,1-2H2Key: SSZWWUDQMAHNAQ-UHFFFAOYAR SMILES ClCC(O)CO Properties Chemical formula C3H7ClO2 Molar mass 110.54 g·mol−1 Appearance Viscous, colorless liquid Density 1.32 g·cm−3 Melting point −40 °C (−40 °F; 233 K) Boiling point 213 °C (415 °F; 486 K) Hazards Safety data sheet External MSDS R-phrases (outdated) R26/27/28-R36/37/38 S-phrases (outdated) S24-S45 Except where otherwise noted, data are given for materials in their standard state (at 25 °C [77 °F], 100 kPa). N verify (what is YN ?) Infobox references Chemical compound 3-MCPD (3-monochloropropane-1,2-diol or 3-chloropropane-1,2-diol) is an organic chemical compound with the formula HOCH2CH(OH)CH2Cl. It is a colorless liquid. It is a versatile multifunctional building block.[1] The compound has attracted attention as the most common member of chemical food contaminants known as chloropropanols.[2] It is suspected to be carcinogenic in humans. It is produced in foods treated at high temperatures with hydrochloric acid to speed up protein hydrolysis. As a byproduct of this process, chloride can react with the glycerol backbone of lipids to produce 3-MCPD. 3-MCPD can also occur in foods that have been in contact with materials containing epichlorohydrin-based wet-strength resins which are used in the production of some tea bags and sausage casings.[3] In 2009, 3-MCPD was found in some East Asian and Southeast Asian sauces such as oyster sauce, Hoisin sauce, and soy sauce.[4] Using hydrochloric acid is far faster than traditional slow fermentation. A 2013 European Food Safety Authority report indicated margarine, vegetable oils (excluding walnut oil), preserved meats, bread, and fine bakery wares as major sources in Europe.[5] 3-MCPD can also be found in many paper products treated with polyamidoamine-epichlorohydrin wet-strength resins.[6] Absorption and toxicity The International Agency for Research on Cancer has classified 3-MCPD as Group 2B, "possibly carcinogenic to humans".[7] 3-MCPD is carcinogenic in rodents via a non-genotoxic mechanism.[8] It is able to cross the blood-testis barrier and blood–brain barrier.[9] The oral LD50 of 3-chloro-1,2-propanediol is 152 mg/kg bodyweight in rats.[10] 3-MCPD also has male antifertility effects [10][11] and can be used as a rat chemosterilant.[12] Legal limits The joint Food Standards Australia New Zealand (FSANZ) set a limit for 3-MCPD in soy sauce of 0.02 mg/kg, in line with European Commission standards which came into force in the EU in April 2002. History In 2000, a survey of soy sauces and similar products available in the UK was carried out by the Joint Ministry of Agriculture, Fisheries and Food/Department of Health Food Safety and Standards Group (JFSSG) and reported more than half of the samples collected from retail outlets contained various levels of 3-MCPD.[13] In 2001, the United Kingdom Food Standards Agency (FSA) found in tests of various oyster sauces and soy sauces that 22% of samples contained 3-MCPD at levels considerably higher than those deemed safe by the European Union. About two-thirds of these samples also contained a second chloropropanol called 1,3-dichloropropane-2-ol (1,3-DCP) which experts advise should not be present at any levels in food. Both chemicals have the potential to cause cancer and the Agency recommended that the affected products be withdrawn from shelves and avoided.[14][15] In 2001 the FSA and Food Standards Australia New Zealand (FSANZ) singled out brands and products imported from Thailand, China, Hong Kong, and Taiwan. Brands named in the British warning include Golden Mountain, King Imperial, Pearl River Bridge, Golden Mark, Kimlan, Golden Swan, Sinsin, Tung Chun, and Wanjasham soy sauce. Knorr soy sauce was also implicated, as well as Uni-President Enterprises Corporation creamy soy sauce from Taiwan, Silver Swan soy sauce from the Philippines, Ta Tun soy bean sauce from Taiwan, Tau Vi Yeu seasoning sauce and Soya bean sauce from Vietnam, Zu Miao Fo Shan soy superior sauce and Mushroom soy sauce from China and Golden Mountain and Lee Kum Kee chicken marinade.[16][17][18] Between 2002 and 2004, relatively high levels of 3-MCPD and other chloropropanols were found in soy sauce and other foods in China.[19] In 2007 in Vietnam, 3-MCPD was found in toxic levels. In 2004, the HCM City Institute of Hygiene and Public Health found 33 of 41 sample of soy sauce with high rates of 3-MCPD, including six samples with up to 11,000 to 18,000 times more 3-MPCD than permitted, an increase over 23 to 5,644 times in 2001,[20] The newspaper Thanh Nien Daily commented, "Health agencies have known that Vietnamese soy sauce, the country's second most popular sauce after fish sauce, has been chock full of cancer agents since at least 2001."[21] In March 2008 in Australia, "carcinogens" were found in soy sauces, and Australians were advised to avoid soy sauce.[22] In November 2008, Britain's Food Standards Agency reported a wide range of household name food products from sliced bread to crackers, beefburgers and cheese with 3-MCPD above safe limits. Relatively high levels of the chemical were found in popular brands such as Mother's Pride, Jacobs crackers, John West, Kraft Dairylea and McVitie's Krackawheat. The same study also found relatively high levels in a range of supermarket own-brands, including Tesco char-grilled beefburgers, Sainsbury's Hot 'n Spicy Chicken Drumsticks and digestive biscuits from Asda. The highest levels of 3-MCPD found in a non- soy sauce product, crackers, was 134 µg per kg. The highest level of 3-MCPD found in soy sauce was 93,000 µg per kg, 700 times higher. The legal limit for 3-MCPD coming in next year[when?] will be 20 µg per kg, but the safety guideline on daily intake is 120 µg for a 60 kg person per day.[citation needed] In 2016 the occurrence of 3-MCPD in selected paper products (coffee filters, tea bags, disposable paper hot beverage cups, milk paperboard containers, paper towels) sold on the Canadian and German market was reported and the transfer of 3-MCPD from those products to beverages was investigated.[23] Exposure to 3-MCPD from packaging material would likely constitute only a small percentage of overall dietary exposure when compared to the intake of processed oils/fats containing 3-MCPD equivalent (in form of fatty acid esters) which are often present at levels of about 0.2-2 µg/g. References ^ Fernandez-Megia, Eduardo; Correa, Juan; Rodríguez-Meizoso, Irene; Riguera, Ricardo (2006). "A Click Approach to Unprotected Glycodendrimers†". Macromolecules. 39 (6): 2113–2120. 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