**THE ADVERSE HEALTH EFFECTS ASSOCIATED WITH AFLATOXIN HAZARD**

Name

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Subject

Instructor

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Aflatoxins are secondary metabolites produced mainly by fungal species *Aspergillus flavus*, A. *parasiticus* and A. *nominus*, which are mostly found in areas with hot and humid climates (Elliot, 2023). Aflatoxins can occur on foods such as groundnuts, treenuts, maize, rice, figs and other dried foods, spices, crude vegetable oils and cocoa beans, as a result of fungal contamination before and after harvest. When such contaminated food is processed or consumed it may cause both chronic and acute hepatocellular damage. The most common types of aflatoxins are B1(AFB1), B2(AFB2), G1 (AFG1) and G2(AFG2).Aflatoxin B1 is the most common in food and among the most potent genotoxic and carcinogenic aflatoxins followed by G1, B2 and G2. Aflatoxins cannot be destroyed boiling nor do they confer, colour, flavour or smell and therefore can go unnoticed.

Aflatoxins have negative effects on the anatomical status of humans by causing DNA damage, cancer and development abnormalities in embryos under long-term exposure (Peles., et al 2021). After consumption of contaminated food, the alternating groups of carbonyl and methylene called polyketides, are absorbed, modified and transferred to different parts of the body (Pandey., et al 2021). Long-term consumption of Aflatoxin contaminated food can result in aflatoxicosis, a severe poisoning that can be life-threatening and mainly causes liver damage. In a recent research done by WHO in 2022, it shows that children receive a higher exposure to all aflatoxin contaminated food types, followed by teenagers and adults who are least exposed group.

The existing research shows that infants and young children have a higher possibility of experiencing the adverse effects of mycotoxins due to their undeveloped metabolic system, higher in take to body weight ratio, higher metabolic rates, and lesser detoxification capability relative to adults (Jankovic., et al 2020). A growing body of scientific evidences has highlighted a relationship between aflatoxin exposure and childhood stunting. pre-natal and neonatal exposures of aflatoxins in early life and child linear growth have been demonstrated through trans-placental transfer (Hifza., et al 2021), human breast milk, weaning and post-weaning foods which contributes to exposure to aflatoxin and severe child growth impairment and long-term health implications. According to a research carried out by Milievic et al (2021), it was established that pasteurized and UHT milk have the highest level of contamination (79%) and the greatest mean concentration of AFM1 (22.34 ± 0.02 ng kg−1), while cheese had the lowest mean concentration (1.36 ± 0.01 ng kg−1) [[20](https://www.mdpi.com/2072-6651/15/4/246#B20-toxins-15-00246)]. The main contributor to the risk of hepatocellular carcinoma (HCC) resulting from AFM1 exposure was the consumption of milk products, in the form of pasteurized and UHT milk, with estimated cases of 0.00038 and 0.00039 per 100,000 individuals per year for the lower bound and upper bound scenarios, respectively. Interestingly, the age group of 1–3 years was associated with the highest risk of HCC (0.00034), indicating no health risk for the groups assessed. Toddlers were estimated to have a higher daily exposure to AFM1 in milk compared to children aged 3–9 years, with an estimated daily intake of 0.164 and 0.193 ng kg−1 bw day−1 for the lower and upper bound exposure scenarios, respectively.

In adults, repeated exposure to low doses of aflatoxins over a lifetime causes chronic diseases, the most frequent and severe of which is cancer. Although dietary intake of aflatoxins has been classically associated with primary liver cancer, i.e., HCC and bile duct hyperplasia (Ruan et al.,2019) other organs, such as the kidney, the pancreas, the bladder, bone and viscera have also been reported to develop cancer upon exposure to these mycotoxins. According to Marchese et al (2019), aflatoxins have been reported to cause lung cancer via inhalation and skin cancer via direct contact.

Liver cancer is one of the most common and deadly type of cancer diseases whose occurrence has been strongly correlated with dietary exposure to aflatoxins. Liver injury and hepatocellular carcinoma (HCC), one of the major types of liver cancer, are considered the main toxic impact of AFB1 (Fan & Xie, 2021). Worldwide, approximately 5–28% of HCC occurrences are attributed to AF exposure (Benkeroum, 2020). Globally, a total of 905,677 new cases (corresponding to a crude rate of 11.6 cases per 100,000 people) and 830,180 deaths (corresponding to a crude rate of 10.7 cases per 100,000 people) due to liver cancer in both sexes and all ages were estimated in 2020. Based on the total number of cases, liver cancer is ranked the 6th and 3rd cancer type in incidence and mortality, respectively, worldwide (IARC, 2021). More than 80% of HCC cases come from developing countries (Resham, 2019). HCC has become a serious health problem, especially in sub-Saharan African countries and countries of southeast Asia, and it is also increasing in Europe and the United States (McCullough, 2019. Recently, Hatipoglu et al (2022), demonstrated that AFB1 can induce oxidative stress by generating reactive oxygen species (ROS) and causing lipid peroxidation. This leads to a significantly increased level of malondialdehyde (MDA) and decreased activities of glutathione (GSH) and superoxide dismutase (SOD). Additionally, AFB1 triggers the release of pro-inflammatory cytokines, including tumor necrosis factor-a (TNF-a), interleukin-1b (IL-1b), and interleukin-6 (IL-6). Abnormal liver function tests that included high levels of AST and ALT further explained the loss of hepatocyte structural integrity. AFB1 can disrupt cell membrane permeability and the mitochondrial membrane in hepatocytes, leading to liver damage.

In addition to the classically known adverse health effects of aflatoxins, there is increasing body of evidence that chronic exposure to aflatoxins can also be responsible for neurodegenerative disorders. The AFBO and ROS generated by CYP450 enzymes and aflatoxin-induced oxidative stress, respectively, react with functional macromolecules in neuronal brain cells where they inhibit lipid and protein synthesis to induce their degeneration (Wild et al.,2021). Aflatoxins have also been reported to disrupt the structure and function of mitochondria of brain cells, which impedes oxidative phosphorylation and leads to their apoptosis. In addition, the detection of aflatoxins in brain tissues of kwashiorkor-deceased children and their association with Rey’s disease (cerebral edema and neuronal degeneration) is a strong indication that aflatoxins can cross the brain-blood barrier and infiltrate the nervous system that they degenerate (Alsayyah et al.,2021).

Two major methods are used for detecting aflatoxin level in human. The first technique is by determining AFB1-guanine adduct in the patient urine. Presence of aflatoxin B1-guanine adduct indicates aflatoxin B1 exposure within last 24 hours. The method measures current exposures alone. The second method is the measurement of the level of AFB1-albumin adduct in serum of blood. This method offers additional cohesive measures of exposures to aflatoxins over many weeks, and sometimes months. Identification of aflatoxicosis in humans is challenging due to variations of the clinical symptoms as well as presence of some conditions including immune system suppression resulting from infectious diseases.

In 2020, it was 60 years since the discovery of aflatoxins, which are among all mycotoxins, considered to be the most agriculturally important and harmful. Particular attention should be taken to improve the situation of crops prone to aflatoxin contamination such as peanut, maize, sorghum, and sunflower that are grown in agro climatic zones (hot and humid) favorable to aflatoxin production.

Reference

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