

AFLATOXIN : FOOD TOXICANT

* Prafulla U. Shah

Aflatoxins were initially isolated and identified as the causative toxins in turkey X disease (necrosis of the liver) in 1960 when over 1,00,000 turkeys died in England.¹ There are four generally recognized Aflatoxins designated B₁, B₂, G₁ and G₂. (Figure 1 & 2)^{13,6}. The metabolites M₁ and M₂, which are found in milk²⁶. The order of toxicity is B₁ greater than G₁, greater than G₂, greater than B₂. However aflatoxin B₁ is the major mycotoxin produced by most species under culture conditions⁶. Because of this and its toxicity, B₁ is the most frequently studied of the four. Aflatoxins, have shown to be Carcinogenic, teratogenic and mutagenic in many avian and animal species including non-human primates and there is an epidemiological evidence showing that it is involved in the induction of human primary liver cancer⁶. There are a group of complex formed by some strains of the mould *Aspergillus flavus* which may grow on groundnut and other foods when stored in damp, warm conditions after harvesting. The aflatoxins particularly, aflatoxin B₁ (AFB₁) is known toxic bifuranocoumarin compounds in animal system^{14,19}. The action of aflatoxins forms two groups of phenomena.^{13,19,27} 1. A series of the rapid phenomena associated with toxicity. 2. The slow phenomenon of carcinogenesis.

Aflatoxins in Foods- In some parts of the world human foods have been found to be contaminated with small amounts of aflatoxins and may contribute to a high incidence of hepatoma in some areas.¹⁹ In India aflatoxins have been found in the commodities like groundnuts (1000-5000 ug/kg). Cottonseed (1100 to 25000 ug/kg). Sorghum, rice, wheat, peas, beans, almonds, walnuts, potato, sweet potato, figs, plums and samples of cow's milk have been found to contain aflatoxins in amounts up to 4mg/lit.^{20,24}

Health Aspect :- Epidemiological, clinical and experimental studies reveal that exposure to large doses (76000 mg) of aflatoxin may cause acute toxicity with lethal effect whereas exposure to small doses for prolonged periods is carcinogenic¹¹. The adverse effects of aflatoxins on animal can be categorized into acute and chronic toxicity.

Acute Toxicity: Acute toxicity is caused when large doses of aflatoxin are ingested. This is common in livestock. The principal target organ for aflatoxin is the liver. Acute toxicity causes jaundice, and a decrease in essential serum proteins synthesized by the liver. Other general signs of Aflatoxicosis are edema

of the lower extremities, abdominal pain and vomiting².

Chronic Toxicity : This is due to long-term exposure of moderate to low aflatoxin concentration. The symptoms include decrease in growth rate, lowered milk or egg production and immunosuppression. There is some observed carcinogenicity, mainly related to aflatoxin B₁. Liver damage is apparent due to the yellow color that is characteristic of jaundice and the gall bladder becomes swollen. Immunosuppression is due to the relativity of aflatoxin^{22,18}.

The effects of aflatoxin B₁ (AFB₁) in Animals : In animals a single dose of aflatoxin causes acute poisoning with severe liver damage and AFB₁ is the potent hepatic carcinogen.²⁹ Only 0.2 ug/day in the diet for 470 days induces liver tumors in 100% of the rats, study showed that aflatoxin in ducks developed hepatic tumors by the 180 day and had neoplastic nodules on the 90th day. They were also hepatocellular carcinoma. Cholangiocellular carcinoma and chronic hepatitis.¹⁶ Domestic animals i.e. pets and agricultural, monkeys and laboratory rats and mice have been adversely affected by aflatoxin (AFB₁). These effects include liver cancer, mutation, immunosuppression, lung injury and birth defects. Aflatoxins are making animals highly susceptible to infection by variety of various microorganisms, includes sheep, cattle, mice, rats, rabbits, pigs, poultry among others. Author observed that laboratory rats were highly affected by AFB₁ toxicity, it leads to highest liver damage and also other body organs like kidney, lungs and heart damaged.^{25,27}

Other pathological features in cattle are blood coagulation defects. A single dose of aflatoxin causes increases in plasma enzymes (Aspartate aminotransferase, lactate dehydrogenase, glutamate dehydrogenase, gamma glutamyl transferase and alkaline phosphatase) and in bilirubin probably reflect liver damage.²¹ Other abnormal clinical findings are proteinuria, ketonuria, glycosuria and hematoma.¹ An experiment was conducted by the author to observe the role of antioxidant vitamin like E, A and C on aflatoxicosis in adult albino rats. Thirty adult male albino rats weighing 92.5 to 168.0 gm were divided into three groups. The rats in each group were matched for age and body weight and were caged individually in ordinary galvanized iron cages. The rats fed a 10% casein protein diet with varying levels of vitamin E for

* Smt. A.S.C. Mahila Arts & Home Science College, Mahesana.

period of 28 days. Body weight was recorded once in a week and on 29th day animals of control group were injected DMSO (DiMethy Sultoxide) equivalent to their body weight and experimental animals from each group were injected AFB₁ (2 mg / kg body weight). Animals were sacrificed exactly 24 hours after injection and blood was collected for different analysis. The liver, kidney, lung and heart were collected and washed out with saline & were used for estimation of different parameters. Author observed that the effect of aflatoxin on liver, lung, kidney and heart & the values were highly significant in liver vit E content (p<0.001), lung (P<0.01) and kidney (P<0.05). There fore, the aflatoxin affected the various body organs & the liver is primary target for aflatoxicosis.²⁷

The effect of Aflatoxins in Humans The effect of Aflatoxin in Humans B₁ has been linked to hepatocellular carcinoma. Cancer in 67 men who had involved particles contaminated with aflatoxin was replaced in 11-year follow up study. They worked in a mill crushing peanuts and other oilseeds. Two of the men developed fatal liver disease, while eleven developed cancer of various organs. The 13 men had inhaled does estimated to between 160-395 ug/cubic meter/man/wk. In 55 matched control men. 4 developed cancer and none died from liver disease.¹⁹ Over 200 villages in western India experienced an outbreak of disease affecting humans and dogs. The illness was characterized by jaundice, rapidly developing ascites, portal hypertension and a high mortality rate. Death usually occurred from massive gastrointestinal bleeding. These diseases were confirmed to they very poor. Who are badly molded corn containing aflatoxin a concentration of 6.25 to 15.5 ppm. The average daily intake was 1.6 mg of aftatoxin.^{7,9} Multiple symptoms and clinical finding that included characterized the Rye-like syndrome reported in various places around the world. Disturb consciousness, fever, convulsions, vomiting disturbed respiratory rhythm, altered muscle tone and altered reflexes. The onsets of illness include coughing, rhinorrhea, sore throat, and earache. Slightly enlarged firm yellow liver and a pale slightly widened rehal cortex. A high rate of mortality occurred 81% of the diagnosed cases.^{18,12}

Aflatoxicoses in humans was reported in many countries like India, China, Thiland and several African countries. In African and Asian countries where environmental conditions favour the aflatoxin contamination, threat to human health from aflataxin is quiet high. Studies on aflatoxin is exposure and incidence of liver cancer in places like china and west Africa showed that the situation was alarming.⁴ In Various parts of the world it was observed that children are also affected by AFB₁ toxicity because of their fetal, childhood environment including status of the pregnment mother and infants are considered critical for growth and risk of disease in earlier life. Malnutrition is the common problem in developing countries among the children. High exposure of aflatoxin leads to various malnutrition disease like Kwashiorkor, marasmas, vitamin deficiencies etc. A study in West Africa showed significant co-correlations among the aflatoxin exposure and student growth in children who are exposed to aflatoxin right for neonatal stages.⁸ Apart from that due to the capacity of aflatoxins to cross the placental barrier can cause genetic defects at fetal stages itself.¹⁶

Conclusion-Aflatoxin toxicity in humans or animals is characterized by liver damage. Aflatoxin is present in the food chain. They have been found in human cord blood and apparently can enter the developing fetus in humans and animals.^{12,6,7,27} Aflatoxins are present in many foods like milk, peanuts, maize, sorghum, coconut, cottonseed, edible oil, rice, wheat, sweet potato, potato.²⁷ Aflatoxins have been found in human breast milk.⁶ To minimize (overcome) the aflatoxin toxicity we must have to aware the community around us by Nutrition education, various health program. The rates of death due to cancer are very high in all over the world and the situation is alarming. Aflatoxin toxicity can be overcome by daily intake of antioxidant vitamins specially A, E & C and some minerals i.e. copper, selenium, iron, magnesium in our daily diet.²⁹ Author observed in animals that varios levels of vitamin E and A in the animal (rats) diet leads to lower the liver damage^{27,12} Now it is high time to protect the world from natural and artificial toxicants.

REFERENCE

1. Aflatoxins National library of Medicine Hazardous substance Database. Toxnet (National Data Network). 2002.
2. Ananth SB & Tand W. Importance of Aflatoxins in human and liver stock health. www.afalotoxin.info/health.asp#top3. Asao et.al (1963) Aflatoxins B and G. J. Amer. Chem. Soc. 85:1706.
4. (Aspen cancer conference-2001).
5. Appelgren LE, Arora RG (1983) Distribution studies of ¹⁴C-labelled aflatoxin B₁ and ochratoxin A in pregnant mice. Vet Res Comm 7:141.
6. Ciegler AM Bennet JW (1980) Mycotoxins and mycotoxicoses. Bioscience 30:512.
7. Denning DW et al (1990) Transplacental transfer of aflatoxin in humans. Carcinogenesis 11:1033.
8. El-Nesami H.S. et al (1995) Aflatoxin M₁ in human breast milk samples in Victoria, Australia and Thailand. Food Chem. Toxicol 33:173.
9. Gong YY, Cardwell K, Hounsa A, Egal S, Turner PC, Hall AJ and Wild CP. 2002. Dietary aflatoxin exposure and impaired growth in young children from Benin and Togo: cross sectional study; BMJ 2002; 325:20-21.
10. Groopman J.D. Cain, L.G. & Kensler, T.W. (1986) Aflatoxin exposure in human population : Measurements and relationship to cancer : CRC crit Rev 19, 113-145.
11. Groopman, J.D. and Kensler W.(1996). Temporal patterns of aflatoxin. Albumin adducts in hepatitis B surface antigen-positive and antigen-negative residents of Daxin Qidong county people's Republic of china. Cancer Epidemiology 1. Asao T, et al (1963) Aflatoxins B and G. J Amer Chem. Soc 85:1706.
12. Groopmann JD and Wild (1994-2001). CRC Critical Reviews in Toxicology 1999 Chapter 19 113-124.