INTRODUCTION

Mycotoxins are toxic compounds produced by fungi. Mycotoxicosis is the result of ingesting toxin contaminated diets by animals and humans. The term mycotoxin is derived from the Greek word "mykes" meaning fungus and the Latin word "toxicum" meaning poison.

The first outbreak of mycotoxicosis was ergotism which was discovered in 1700 and this cause and its effect were found to be associated by the consumption of ergot. The second was the outbreak of stachybotoxicosis in horses and of alimentary toxic aleukia in humans in the USSR in the 1930's. The third was the outbreak of aflatoxicosis of Turkeys in England in 1960, which was caused by feeding aflatoxin contaminated peanut meal from Brazil. Since then, aflatoxin and aflatoxicosis and other mycotoxins and mycotoxicosis have received extensive studies.

Mycotoxins associated with food and originating from *some species of Alternaria, Aspergillus, Fusarium, Penicillium* and *Claviceps spp.* are reviewed with reference to the toxicological, regulatory and economic issues arising. In 1985 the **FAO** estimated that 25% of the world's food crops are contaminated annually with mycotoxins.

Aflatoxins, are considered as a group of bis-furano isocoumarin derivatives produced by fungi from *Aspergillus* group. They have various derivatives, the most important being aflatoxin B_1 and B_2 produced by *Aspergillus flavus* and isolated often from maize and aflatoxin G_1 and G_2 produced by *Aspergillus parasiticus* commonly isolated from peanuts. However, aflatoxin B_1 is considered the most important aflatoxin and

most harmful such aflatoxins have been found to be very parent hepatocarcinogen in various species of laboratory animals tested, among which are rodents, birds, fishes and monkeys. Aflatoxins are associated with alteration of lipid metabolism, imbalance of immune system, depression of liver function, retardation of growth, and subsequent death.

Hazards of aflatoxin have been well documented. Ingestion of aflatoxin by a variety of experimental animals results in necrosis of their liver and may ultimately lead to death. Furthermore, chronic exposure of susceptible hosts to aflatoxins may cause malignant liver tumors.

E.O.S. (1990), concerning maximum limits of mycotoxin in foods recommended that, the maximum allowances of aflatoxin B_1 should be in the limits of 10 ppb, meanwhile aflatoxins B_2, G_1 and G_2 concentration in the range of 20 ppb for corn animal feeds. The maximums contents of aflatoxin B_1 in peanuts should not exceed that 5 ppb, while aflatoxins B_2, G_1 and G_2 should be in the limits of 10 ppb. Several investigators have studied various chemicals to see if they would degrade and detoxify aflatoxins. However, many chemicals which effectively detoxify aflatoxin, are not suitable for use in foods. This is true because they may reduce the nutritional quality of the food, produce off-flavors and off-odors in the food, or have toxic residues in the food. Hence, it would be desirable to find a chemical which is an acceptable additive that will also degrade aflatoxins **(Doyle and Marthe, 1978a)**.

Several procedures have been used in an attempt to detoxify aflatoxin contaminated feedstuffs, they include, ammoniation, dietary addition of non nutritive inert compounds, such as zeolites, aluminosilicates or charcoal and alteration of dietary nutrients, such as protein, selenium and vitamins.

Dietary nutritents are known to have an important interactive role in the toxicity of aflatoxins to animals. For example, increased protein, lipid, fiber, trace minerals or selective vitamins added to aflatoxins contaminated diets will decrease aflatoxin toxicity. Addition of vitamins A, E and K to a commercial diet partially protected rats from some of the adverse changes induced by aflatoxin. Vitamin E, a fat soluble vitamin antioxidant is known to reduce free radical formation in tissues, is suspected of reducing oncogenesis and can prevent certain types of hepatic necrosis. Some signs of vitamin E deficiency and aflatoxicosis are similar and one investigator proposed that aflatoxin may exert its effects by interfering with vitamin E metabolism. The liver is the target organ for aflatoxin and hepatocyctes are damaged by formation of aflatoxin epoxide during metabolism of aflatoxin by mixed functions of oxidase enzymes. Similarly, hepatotoxicitic of carbon tetrachloride results from damaging free radicals generated during the oxidation of this compound by hepatic mixed-function oxidases.

Dietary vitamin E is reported to protect rats against this carbon tetrachloride-induced lipid peroxidation presumably by maintaining intracellular concentration of glutathione peroxidase.

The aflatoxins are secondary fungal metabolites that may be produced by *Aspergillus* fungi on cereal grains during growth, harvest, storage or transportation. They can cause death in people and animals, however, the greatest economic impact comes from reduced productivity, suppressed immune function, pathological effects on organs and tissues and altered reproductive capability.

Due to the essential role of the liver in metabolism and detoxification of a wide range of toxic materials, and that of the kidney in eliminating the waste products from the body, liver and kidneys were selected as target organs.

Due to the fact that chronic ingestion of small amounts of toxins has more significance than acute exposure **(Doerr et al. 1983)**. This study was conducted to study the chronic effects of the aflatoxins on experimental animals.