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An overview of production, mechanism of action, incidences, diagnosis, prevention and mitigation of aflatoxicosis

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Abstract
Aflatoxins produced by the pathogenic fungi *Aspergillus* species in the food crops at the field before harvest or after harvest and during storage. Consumption of toxins contaminated food causes severe consequences in humans and animals. Even a low concentration is highly hazardous. The associated symptoms produced due to the intake of toxins are gene mutation leading to cancer, teratogenicity, suppression of the immune system and damage to the liver with reduction in synthesis. The other major signs include loss of appetite, reduction in growth rate, lethargic, anemia, icteric condition and mortality. Diagnosis is usually done with detection of clinical signs, lesions of the liver and by detection of aflatoxins in the contaminated feed material or milk under laboratory. Treatment protocol usually suggests for the replacement of the aflatoxin contaminated feed with a good quality diet containing appropriate level of nutrients like protein, vitamins, and trace minerals. No specific antidote is available. Though many treatments protocols are available, each method has got its own merits and demerits. Thus, it necessitates proper diagnosis and prevention of contamination in feed. As World Health organization recommends to go for alternative medicine involving plant based products to overcome the adverse effects of synthetic chemicals, numerous studies are already on pipeline investigation to mitigate the effect of aflatoxins. The current review provides insight about the fungal ecology, production of aflatoxin, mechanism of action, incidences in humans and animals, diagnosis and prevention of aflatoxicosis to ensure proper management and eventually safeguard food security.

Keywords: Aflatoxin production, effects, humans, animals, diagnosis, prevention

1. Introduction
1.1 Feed and Food safety
Feed ingredients which include rice, wheat, corn in the category of cereals along with their by-products are shared by animals and humans and the oil cakes *viz.*, de-oiled sunflower cake, soybean cake and groundnut oil cakes are being exclusively used in the livestock and poultry feed industry. In animal management systems, feed supply is the major part which assures its production. But in most of the circumstances**,** the competitive nature of raw material pricing has led to the usage of low-quality feed ingredients in feed formulation and these products in turn are maintained for longer periods of time under storage. But, the lack of proper storage conditions makes the feed and feed materials more vulnerable for contamination with the fungus. Thus the factors that affect the safety of the food grains causes a huge impact in the food chain and the presence of these toxic residues affect the food safety at large.
Thus, feed and food safety is the major issue throughout the world and this will have a direct impact on the livestock and poultry sector. As a major proportion of the cot is invested mainly in the feed, a low quality feed material will cause a huge economic impact which will be immediately visible in the animal industry. In this respect, much attention is needed to ascertain the possible contamination of feed and food by the pathogenic microbes and with special emphasis to fungi and the risk of mycotoxin production (Bullerman and Bianchini 2007) [13].

2. Fungal contamination
2.1 Fungal contamination of feed and feed ingredients

Feed spoilage by fungi exists as one of the important persistent problem inspite of the extensive research done to mitigate it (Christensen 1974) [19]. Fungi are ubiquitous and all the feedstuffs are highly prone for fungal contamination when stored. They are highly toxic as they produce secondary metabolites known as mycotoxins. So far, about 400 mycotoxins have been identified and these toxins are different from each other in terms of their structure and chemical nature. These toxins are found to be potentially harmful to humans, animals and birds (Huwig *et al.* 2001) [36].

2.2 Fungal ecology

Fungal toxins are usually produced by species belonging to five genera *viz*., *Alternaria, Aspergillus, Cladosporium, Fusarium* and *Penicillium*. Other genera including *Chaetomium, Claviceps, Diplodia, Myrothecium, Phoma, Phomopsis, Pithomyces* and *Strachybotrys* are also classified as mycotoxic fungi (Moss 1991). These fungal species liberate many different toxic compounds in the contaminating material. However, not all isolates from the same species are reported to produce toxins (Cole *et al.* 2003; Brase *et al.* 2009) [22, 9]. The major toxins synthesized from these genera include: aflatoxins (AF), ochratoxins (OT), trichothecenes, fumonisins, zearalenone (Miller 1998) [55], patulin and cyclopiazonic acid (Huwig *et al.* 2001) [36].

2.3 Conditions favouring mycotoxin production

In standing crops and stored feeds, fungi are present as a normal part of the microflora, but the physical factors like moisture, relative humidity, optimum temperature and the severity of contamination caused to the crops, chemical factors like carbon dioxide concentration, oxygen level, suitability of the substrate, application of treatment and biological factors like the crop used, growth limiting factors, insect infestation and spore load (Frisvad 1995; Wicklow 1995) [31, 98] play a major role leading to fugal contamination. Of all the factors discussed, presence of moisture and the prevalence of optimum temperature determine the mould growth and enhance mycotoxin production. Normal fungal growth occurs in the temperature range between 10 °C and 40 °C, with a pH range of 4 to 8 and with moisture levels exceeding 0.70 (Lacey 1991). Mycotoxins can enter the food and feed stuffs in several ways, but in general, it is categorized as either direct or indirect contamination (Jarvis 1990) [39]. The growth of the fungus in the feed material produces direct contamination. Sometimes, the growth of the fungus is enhanced during harvest in the field or after harvest or during storage and transportation. Indirect contamination occurs due to the toxic metabolites secreted by the fungi causing serious ill health effects in not only animals but also in humans. The effects originate from the ingestion of these fungal toxins which include AF, OT, fumonisins, citrinin, trichothecenes and zearalenone. The syndromes associated with these toxic compounds are generally referred as mycotoxicoses (Richard 2007) [80].

AF are highly carcinogenic in nature. A predisosed health condition to hepatitis B virus leads to severe fatality by causing liver damage. Ochratoxin A (OTA) is also a potent carcinogen and causes cancer in the urinary tract and damages kidney. The other dangerous mycotoxins are umonisins (which causes oesophageal cancer), trichothecenes (which are found to suppress the immune system) and zearalenone (estrogenic in nature) (Pitt 2000).

2.4 Aflatoxicosis

The toxic condition caused by AF includes acute and chronic toxicity in both animals and man with resultant damage to liver in acute phase and leading to cirrhosis of liver, tumor induction and teratogenic effects on chronic exposure (Stoloff 1977) [90]. There are four different compounds naturally produced by AF which includes B1, B2, G1, and G2. 'B' and 'G' refers to the blue and green fluorescence emitted by these compounds on exposure to an UV light under thin layer chromatography plates, while the numbers 1 and 2 mentioned as subscript indicates the major and minor compounds. AF are produced by the pathogenic fungus *Aspergillus flavus* and *Aspergillus parasiticus* which prevails both at the field conditions and during storage. They are almost prevalent in all types of soil. AF are produced by the *Aspergillus* species, *A. flavus, A. parasiticus* and *A. nomius* and other species of *Penicillium, Rhizopus, Mucor* and *Streptomyces* of which, B1, B2, G1 and G2 are the most commonly observed ones in feed materials (Smith 2002; WHO 2018) [88]. AF have become the most widely reported food-borne mycotoxin since their discovery, thus emphasizing their significance both economically and medically. Also their global occurrence has also been well documented with established monitoring methods. AF contamination has been reported to occur in a wide types of feedstuffs like corn, sorghum, barley, rye, wheat, peanuts, soya, rice, cottonseed and also in numerous other products prepared from these primary feedstuffs (Busby and Wogan 1979) [14].

2.4.1 Mechanism of action of AF toxicity

The mechanisms by which AF produce toxicity are

- AF are clearly defined as genotoxic agents and cause genetic alterations by initiating their action at the DNA level (Sugimura 2000; Sutandyo 2010) [91, 92].
- AF induces the formation of DNA adducts thereby causing genetic changes in the target cells. These changes bring about breakage of the DNA strand, causes damage to the DNA base and also produces oxidative damage (Sharma *et al*. 2018) [86].

The first mechanism of action of AFB1 will get initiated at liver, supposed to be the primary target organ of metabolism. Upon ingestion with the contaminated feed, AFB1 gets metabolized by cytochrome - P450 enzymes in the liver to the reactive genotoxic intermediates (AFB1 - 8, 9 -oxide, AFBO) or it may get hydroxylated (AFQ1 and AFM1) and also further gets demethylated (AFP1) to produce less harmful compound than AFB1. During biotransformation, it produces highly reactive intermediate chemical compound AFBO which exerts hepatocarcinogenic effect.

This intermediate compound is highly reactive and exerts genotoxicity and binds to liver cell DNA, and results in the formation of DNA adducts, *viz.*, 8, 9-dihydro-8 (N7 guanyl)-9-hydroxy-AFB1 (AFB1 N7-Gua). If the formed DNA adduct is not repaired before DNA replication, then they interact with the guanine base of the DNA resulting in the mutational effects in the tumor suppressor gene p53 (Wang and Groopman 1999; Lewis *et al.* 2005; Sutandyo 2010; Obuseh *et al.* 2011; Sharma *et al.* 2018) [37, 97, 92, 86] involved in safeguarding the system. To prevent the formation of DNA adducts, it is highly essential to detoxify the genotoxic AFBO intermediate. Hence, conjugation of AFB1 to glutathione is an important detoxification pathway that can aid in the elimination of the toxin from the system in animals (McLean and Dutton 1995).

2.4.2 Incidence of aflatoxicosis

2.4.2.1 In humans

Humans get exposed to AF by eating contaminated food. There are two pathways identified for the dietary exposure: (a) direct ingestion of AFB1 through consumption of contaminated maize, nuts and their products, (b) ingestion of milk, milk products, cheese and powdered milk contaminated with AFM1 that are produced from feed within the system of dairy cows (WHO 1979). In 1967, an outbreak was reported in 26 persons in the Taiwan rural villages. The case victims were reported to have consumed contaminated mouldy rice for up to 3 weeks and subsequent signs like edema of the legs, abdominal pain and vomiting as well as palpable liver, were observed (Ling *et al.* 1967). In 1974, a similar type of condition was observed in humans and dogs and the cases were associated with the consumption of AF contaminated mouldy corn.

The symptoms noticed were high fever, coloured urine, vomiting, oedema of feet, ascites, portal hypertension with increased mortality mainly due to gastrointestinal haemorrhage. The incidence occurred in the poor population who were forced to eat the contaminated rice due to economic reasons. (Krishnamachari *et al*. 1975a, 1975b; Keeler and Tu 1983) [42, 43].

In October 1988, mortality was reported in the Northwestern state of peak in peninsular Malaysia among Chinese children and the cause was identified to be due to acute hepatic encephalopathy (Lye *et al.* 1995) caused by AF. AFB1 has also been reported to produce human hepatic cell carcinoma (HCC) (Jackson and Groopman 1999) [37].

2.4.2.2 In animals

The sensitivity to AF varies considerably among species. Though animals are found to be generally sensitive to AF, ruminants are relatively resistant. Among the domestic livestock, the order of increasing susceptibility occurs among sheep, goat, cattle followed by pigs (Ramdell and Eaton 1990). Among the poultry industry, the outbreak is greater in ducks, followed by turkeys, geese, peasants and chickens. Within the same species, the dose of the toxin ingested plays a major role and it is mostly influenced by race, gender, age and composition of the diet (WHO 2002). In chronic aflatoxicosis, decrease in the growth rate is the most evident clinical sign noticed the animals are in the growing stage. The symptoms become prominent when the animals ingest low levels of AF for a long period of time (Leeson *et al,* 1995). Thus AF are prone to cause chronic toxic symptoms like carcinogenesis especially depending on the species, age of the animal, dose and duration of exposure (Smith 2002) [88]. Hence all animal species are highly susceptible to aflatoxicosis, but outbreak has been reported most commonly in pigs followed by sheep and cattle.

Beef and dairy cattle are highly affected by aflatoxicosis than sheep or horses. Similarly, Young animals of all the species are highly vulnerable to the effects of AF than mature animals (Cassel *et al.* 1988) [18]. Nursing animals get affected due to the exposure of AF metabolites in the milk (Jones *et al.* 1994) [44]. AF ingested by ruminants through feed forms a complex with the ingesta in the rumen, and 2 - 5 per cent of the ingested toxin reaches the intestine. If the level of AFB1 exceeds 100 µg / kg, it is highly toxic to cattle (Radostits *et al.* 1997). Among sheep and goats exposed to AF, anorexia, depression and icterus were the prominent symptoms observed. Goats are also observed to produce nasal discharge and sheep excretes dark brown urine (Hatch *et al.* 1971; Samarajeewa *et al.* 1975; Abdel-salam *et al.* 1989) [83, 1]. Green and Oehme (1976) [32] reported the first case aflatoxicosis in equine in a 15-year-old Arabian stallion which died on exposure to 54.4 ppb of AFB1 in feed. As far as swine population is concerned, young animals are highly susceptible than the old animals (Diekman *et al.* 1992) [26].

Seibold and Bailey in 1952 first reported the case of canine aflatoxicosis. In dogs fed with mould contaminated feed, they observed hepatitis "X". Thus et animals are also reported to be the victims of the condition. In dogs the LD50 value measured for AFB1 was 0.5 - 1.5 mg / kg and in cats it was 0.3 - 0.6 mg/ kg b.wt. In companion animals, outbreak of aflatoxicosis have been observed on ingestion of feed containing AFB1 at 60 ppb or greater. Thus the sensitivity to the toxic compound depends on age, hormonal status (pregnancy), and nutritional status of the animal species concerned (Rumbeiha 2001) [81].

As far as poultry industry is concerned, aflatoxicosis has been reported to cause severe economic impact in ducklings, broilers, layers, turkeys and quail (CAST 1989) [23]. However, the susceptibility varies based on the species involved, breeds and the genetic lines. Among the avian species, the comparative toxicological evaluation concluded that ducklings and turkey poults are the most sensitive species.

Goslings, quails and pheasants are reported to be in the intermediate range and the chickens considered to be resistant. Thus the susceptibility ranges are denoted in the order as ducklings > turkey poults > goslings > pheasant chicks > chickens (Muller *et al.* 1970; Leeson *et al.* 1995).

Ducklings are almost 15 times more susceptible to aflatoxicosis than the laying hens, however on comparing among the strains of the laying hens, certain strains of hens are reported to be thrice more susceptible than other species in poultry (Jones *et al.*, 1994) [44]. AF decrease immunity in the flocks and thereby enhancing susceptibility to various infections and lead to considerable economic losses in poultry industry. This occurs as a result of decrease in egg and meat production, decreased feed consumption, reduced weight gain and nutritional interactions (Pimpukdee *et al.* 2004; Jand *et al.* 2005) [38]. Increased heat stress among broiler birds with reported significant mortality (Dafalla *et al.* 1987a) [24], impaired egg production in leghorn layers (Bryden *et al.* 1980) [12], anemia, hemorrhages and liver damage (Lamont 1979), skeletal impairment leading to paralysis and lameness (Okoye *et al.* 1988), reduced production performance in broilers, (Jones *et al.* 1982) [40], high mortality rate in ducks (Bryden *et al.* 1980) [12], impaired ambulation in quail (Wilson *et al.* 1975) [99], weakened immunity in turkeys (Hegazy *et al.* 1991) and increased susceptibility to secondary bacterial infections (Bryden *et al.* 1980; Calnek *et al.* 1997) [12, 16] are the direct and indirect effects of aflatoxicosis.

3. Regulations to control mycotoxicosis

Eventhough many disease outbreaks were noticed over time, mycotoxicosis remained as the "neglected disease" until the early sixties. As time advanced there was greater impact imposed by these toxins on both human and animal population which changed the attitude and to the implementation of strict regulatory measures. It became very clear that the prevalence of these fungal metabolites in the feed materials, feed and food proves to be undesirable and should be avoided or otherwise kept as low as possible. Today the laws governing the food industry not only prohibit the introduction of these toxins but also follow strict regulatory protocols that ascertain the occurrence level of these toxins

derived either by natural contamination or by means of any industrial origin. Since mycotoxins mostly occur through natural mode of contamination, certain level of presence are unavoidable warranting their exposure must be tolerated (Pittet 1998). AF was first noted in early 1960's and caused significant health concern since then with persistent problems in food trading. To overcome these issues, certain regulations specific to mycotoxin on tamination was framed, developed and followed in several countries, initially referring only to AF but later regulations governing the safety limit for other mycotoxins such as deoxynivalenol, OTA, patulin, citrinin and zearalenone also included in the food for some countries. The choice of fixing limits for mycotoxin contamination depends on the available data on toxicology, data on the prevalence of contamination in various food and feed materials, employing different methods of sampling and final analysis of toxins in the commodities, implications for intercountry trade and the existence of sufficient food supply.

As AF cannot be completely eliminated or prevented from the feed by the existing good agricultural practices, it is always considered as an unavoidable contaminant in food. Hence, the natural way of defense tolerates some level of toxins and protect the system. However, from a regulatory perspective, it is very essential to set the maximum limit for the AF in food and feed. In some of the industrialized nations, the limit has been set ranging from 0 to 30 µg / kg for AF*B*1 in foodstuffs and 0 to 50 µg / kg for total AF contamination. This is very important for the global scenario and trade. It has been identified by Joint FAO / WHO Expert Committee on Food Additives (JECFA) and concluded that between the two different ranges of 10 µg / kg and 20 µg / kg for AFB1 in food, no significant difference in risk to health has been noticed (Boutrif 1997) [8].

The maximum permissible level for AFB1 in crops has been fixed at 2 ppb and 0.5 ppb in milk for human consumption by Food and Drug Administration (FDA) of the United States. In European countries food materials containing more than or 4 ppb of AF are not accepted and in India, the limit has been fixed at 30 ppb in food commodities (Van Egmond and Jonker 2004) [29]. Thus the limits of AFB1, AFB2, AFG1, AFG2 and AFM1 are 0 - 40 ppb for foods and in feed the limit is 0 – 1000 ppb ; for OTA, the level is 0-50 ppb in food and in feed the level is 0-1000 ppb ; for Don, the limit is 500 – 2000 ppb in food and 5-10,000 ppb in feed; for zearalenone it has been assigned at 0 - 1000 ppb in food; for patulin, the range is 0-50 ppb in foods, for diacetoxyscirpenol, the set limit is 0 - 100 ppb in feed; for chetomin, the permissible range is 0 ppb in feed; for stachybotryotoxin, the accetanace limit in feed is 0 ppb and for fumonisins, the permissible range is 0 - 1000 ppb in food and 5000-50,000 ppb in feedstuffs. Thus, the set limits promote a global harmonization for regulating the contamination with the fungal metabolites, thereby facilitating a smooth international food trade. These regulations are followed based on sound scientific principles and risk analysis which in turn helps to make recommendation on plans to be adopted for preventing mycotoxin contamination with minimized damage to the food commodity and with the final outcome of promoting a safe and wholesome supply of food and feed (Mazumder and Sasmal 2001) in the market.

4. Diagnosis of mycotoxins

Analysing mycotoxin in feed and feed materials remains as major challenge due to the co-occurrence of numerous compounds in it (Cole 1986) [21]. Detecting mycotoxin is the key step for determining the extent of contamination and thereby ascertaining the risk involved. There are three Methods involved in diagnosis *viz*., biological, physicochemical and immunochemical methods (Agriopoulou *et al*. 2020) [3].

4.1 Biological methods

Biological methods signify the changes in the normal physiology occurring in the system on exposure to a toxin. It includes, changes in the growth pattern, altered immune status, increased biochemical profile, pathological, histopathological lesions owing to organ damage and morality associated with the disease. Based on the observation of the gross, visible changes in the major organs, a rough diagnosis can be made. If the detoxifying organ liver is found to be enlarged, friable, icteric with necrosis and haemorrhage, the symptom indicates mycotoxicosis. Changes in kidney like swelling with bulging and congestion also indicates mycotoxicosis (Abidin *et al.* 2011) [2]. Though there are significant changes, these changes occur most commonly in most of the systemic disease involvement of variable cause. Hence, it may not give a definite clue for diagnosis. Mycotoxicosis often aggravates when there is a synergistic effect exhibited by the presence of more than one toxin in the feed. Interaction of two or more toxins produces a profound effect than the individual toxin concerned and under such situations, diagnosis based on biological methods may not be concluding (Schiefer 1986) [84].

4.2 Physicochemical methods

In the physicochemical method, different techniques like thin layer chromatography (TLC), high- performance liquid chromatography (HPLC), mass chromatography and gas chromatography is employed for the diagnosis. Quantification can be done easily using this methods and they have advantages of yielding a more appropriate, quicker, rapid and reproducible result even at a lower range (Egmond and Paulsch 1986) [29]. But these techniques need sophisticated laboratory. The instruments are also highly expensive (Chu 1991) [20] to arrive at a more accurate and sensitive result. The technique also employs clean up and processing of samples to increase the concentration to 10 - 1000 folds. These complications limit this method of diagnosis for the routine monitoring.

4.3 Immunochemical methods

To overcome the effects of the biological and physicochemical methods, immunochemical methods were developed by scientists to detect the toxins in the feed and food matrix. This lead to the development of a rapid, repeatable and sensitive assay to detect the mycotoxins using Enzyme-linked immunosorbent assay (ELISA). The methods use specific antibodies against the mycotoxin (Pestka 1994) which may be either monoclonal tested by Fluorescence polarization immunoassay (FPIA) (Shim *et al*. 2004; Maragos 2009) [87] and/or both monoclonal and polyclonal antibodies These methods are comparatively cheaper (Chu, 1991; Carlos, 2004) [20].

5. Prevention of mycotoxicosis

The entry of aflatoxins in the food chain or the feed can be prevented by the following three strategies.

5.1 Pre-harvest strategies: In the recent years, genetic engineering has been the main trend of combating

mycotoxicosis. The technique involves identification of the potent antifungal peptides and transfer of the same to the susceptible crops at the preharvest stage (Rajasekaran *et al*, 2009). Another strategy that can be adopted at the preharvest stage is the incorporation of antibodies into the plants. These antibodies bind with the specific toxins and help in their elimination. They can protect the plants from fungal infection (Peschen *et al.* 2004; Hu *et al.* 2008). Fungal spores remain dormant in the soil for a long duration. Hence, removing old seed heads, plant stalks and other debris from the field can eliminate the contamination. Some crops also may show increased susceptibility for specific types of fungi, so crop rotation can be followed to avoid contamination (Blaney 2001; CAC, 2003; Bricknell *et al.* 2008) [7, 10]. Poor nutrition or water deficit may impose stress to plants which in turn makes the plant more susceptible to diseases. At optimum pH in the soil, the planting has to be followed. Also enough intercrop spacing must be ensured to avoid stress during growth (Bricknell *et al.* 2006) [11].

The other important management practice is to control attack of the kernels by the insects, equipments and by following proper drying protocols. Lack of control measures before harvesting may predispose the kernels to fungal contamination (Munkvold 2003a) due to penetration of the husk. So farmers use insecticides and fungicides to counter this problem of contamination.

5.2 Harvesting strategies

It has been observed that a delay in harvesting will lead to increased contamination (Munkvold 2003b; Kaaya *et al.* 2005) [41]. When the crops are fully mature with less than 14 per cent moisture in kernels, harvesting must be done (Bricknell *et al.* 2006) [11]. The moisture content in the crops can be reduced to less than 14 per cent by means of drying the crops either before storage by natural means prior to harvest or through mechanical means post-harvest (Bricknell *et al.* 2008) [10].

5.3 Post-harvest strategies

The crops intended for future use will be stored and the period indicates the post-harvest period. The production of mycotoxin in the crop during this period depends on the suitable conditions of the substrate like ambient temperature, humidity, moisture content, insect activity and the presence of the fungus enough to initiate toxin production (Bricknell *et al.* 2008) [10]. The fungal infection of the crops occurs mostly in the field at the time of storage. The dormant fungal spores may reproduce to produce the fungal contamination or the insects or rodents may act as carrier. Fumonisins, zearalenone, deoxynivalenol (DON), and nivalenol are mostly noticed pre harvest while AF contamination is common both during re harvest and post-harvest. The level of moisture in the crop should be strictly less than 14 per cent during storage (DPI&F, 2005a). Pesticides like Phosphine fumigation and dichlorvos can be used to control pests (DPI&F, 2005b).

The control measures that were adopted at the time of storage to prevent fungal infection has to be applied even at the time of transportation. A structured and systematic potential quality framework like the Hazard analysis critical control point (HACCP) system has to be adopted to ensure monitoring right from the beginning of production till the formation of the finished product (Pineiro 2001; Brandt *et al.* 2005; Wyss 2005) [102].

5.3.1 Post-harvest Strategies - Physical methods

The seeds which are contaminated with the fungi and appear shriveled and infested with insects must be segregated from the uninfected ones (FAO 1991). Using UV light, fungi contaminated material can be segregated (Basappa and Shanta 1996) [5]. Different adsorbents can be added in the feed to control mycotoxicosis. This may reduce the carryover of mycotoxins from the feed ingredients to the finished final feed (Ramos *et al.* 1996; Huwig *et al.* 2001) [78, 36]. The common adsorbents that are used includes phyllosilicate minerals, activated charcoal, synthetic resins, zeolites or certain clays (Miazzo *et al.* 2000; Raju and Devegowda 2000) [4].

5.3.2 Post-harvest Strategies - Chemical methods

In the chemical method of control, ozone has been used and roved to be highly beneficial against AF, cyclopiazonic acid, OTA and patulin (McKenzie *et al.* 1997). Numeras fungistats.ous organic acids like ascorbic acid, benzoic acid and propionic acid are known to prevent fungal contamination and considered as fungistats (Marın *et al.* 2000). Even the method using ammoniacal treatment with heat and pressure application has proved to be highly successful against aflatoxicosis and fumonisins. But the main disadvantage faced is the generation of toxic compounds (Marth and Doyle 1979). Phosphine is an effective fumigant used to protect maize grains (Bell 2000) [6] against fungal contamination. Anti-oxidants of food grade like butylated hydroxyanisole (BHA), butylated hydroxytoluene (BHT), propylparaben (PP), L-carnitine, silymarin, water-soluble vitamins, cyaniding, licorice, lycopenes, catechins, and so on (Passone *et al*. 2009) have fungicidal and fungistatic activities.

5.3.3 Post-harvest Strategies - Biological methods

In the biological method, to counteract the fungal alterations, probiotics are used (Mombelli and Gismondo 2000; Shah, 2000) [6]. They are very essential for the biological degradation of the mycotoxins (Teniola *et al.* 2005) [94]. In recent years, biological method of detoxification is gaining momentum due to the usage of medicinal plants for the purpose. They are easily available, ecofriendly and generally more economical. The recent day research involves usage of organic extracts and essential oils from medicinal and aromatic plants to overcome the fungal contamination. They have also proved to interfere with the biosynthesis mechanism of the fungal species resulting in ultimate reduction of toxin.

6. Mitigating mycotoxicosis with plant extracts

Stoev *et al.* (2000) [89] reported that inclusion of 5 per cent level of aqueous extract of *Artichoke sp.* and *Curcuma longa* powder in chicks reduced the toxic effects of aflatoxicosis and ochratoxicosis.

Aravind *et al.* (2003) [4] conducted a study to counteract the toxic effects of mycotoxins fed at the level of AF 168 ppb, OT 8.4 ppb, zearalenone 54 ppb, and T-2 toxin 32 ppb in commercial broilers with esterified glucomannan and concluded that the toxin effect was mitigated as evidenced by alleviating the growth depression and increasing the relative weights of liver, kidney and gizzard.

The antimicrobial and antioxidant properties of *Garcinia sp* was evaluated (Tamil selvi *et al.* 2003) [93] on the growth of *A. flavus* and reported an inhibitory effect on the growth of the fungus and subsequent toxin production. They attributed the result to the presence of a major compound garcinol which

might have exhibited the anti-oxidant property and recommended it for use in neutraceuticals.
Sakhare *et al.* (2007) [82] also reported the effect of polyherbal feed supplement against mixed mycotoxicosis in broilers. Rangsaz and Ahangaran (2011) [79] concluded that addition of ethanolic extract of turmeric (*Curcuma longa*) at level of 0.05 per cent to the diet containing 3 ppm AF enhanced the overall growth performance and reduced the toxic severity of AF and suggested that turmeric extract can mitigate the condition of aflatoxicosis in chickens. Vijayanandraj *et al.* (2013) [96] evaluated the effectiveness of leaf extract of Vasaka (*Adhatoda vasica* Nees) in detoxification of AFB1 *in vitro* using thin-layer chromatography method and enzyme-linked immunosorbent assay (ELISA) and found an alkaloid in the methanolic extract of the plant which exhibited maximum effect of degradation of AFB1 after incubation for 24 h at 37°C. They observed that a time course study of AF detoxification by *A. vasica* extract revealed 69 per cent degradation within 6 h and ≥ 95 per cent degradation after 24 h of incubation. The same was also further confirmed by liquid chromatography – mass spectrometry (LC - MS) analysis

7. Conclusion
Aflatoxin contamination can be controlled by adopting good agricultural practices at the time of harvest and by ensuring proper storage facility. However, in a tropical country with optimum temperature and humidity prevalence, the feed materials are more prone for contamination. Hence periodical screening and diagnosis is highly warranted. As the toxin is extremely durable, it remains as a great challenge to eliminate the presence in the food and feed ingredients. Therefore, proper prevention and control measures can eliminate the toxin in the compounded feed. Also, mitigating the toxicosis condition at the final stage with novel methods employed using plant based materials is also gaining momentum in order to ensure food safety and security to human and animal health with the concept of bringing together under an umbrella of one health approach.

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9. Declaration of interest
The motivation to prepare this manuscript is related to the increased prevalence of mycotoxicosis in humans and animals. Since its first report, the cases related to aflatoxicosis is very common. Because mycotoxins are “natural” contaminants of foods, their formation is often unavoidable. On the whole it seems that mitigating mycotoxicosis is very crucial as the presence of toxin is ubiquitous. In such situations, numerous strategies have been adopted with new start up with plant extract based research. The recent works are gaining momentum as it proves successful in mitigating the toxins and as confirmed in the review, a lead would guide the researchers working in this field to evolve new control mechanisms. Additionally, the authors have not participated in and do not anticipate participation in any legal, regulatory or advocacy proceedings related to the concerns of the paper. The authors report no conflict of interest.

10. Disclaimer
The article was prepared by the author as she is employed in the Tamil Nadu Veterinary and Animal Sciences University. The opinion expressed in this article are the author’s own and do not reflect the view of any departments.

11. Author contributions
- Conceptualization – Sakthi Priya Muthusamy and Jagadeeswaran Appusamy
- Investigations – Jagadeeswaran Appusamy and Natarajan Amirthalingam
- Methodology - Sakthi Priya Muthusamy, Jagadeeswaran Appusamy and Natarajan Amirthalingam
- Writing, Review and editing - Sakthi Priya Muthusamy, Jagadeeswaran Appusamy and Natarajan Amirthalingam
- All authors have read and agreed to the published version of the manuscript.

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15. Consent for publication
Not applicable

16. Competing Interest
None

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