Chapter 4

Avian Mycotoxicosis in Developing Countries

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Additional information is available at the end of the chapter

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1. Introduction

Avian mycotoxicosis refers to all the diseases caused by the effect of mycotoxin in birds. These diseases may not be pathognomonic and sometimes subclinical and difficult to diagnose. The problem is worldwide but effort will be made to localise the effect of these diseases in developing countries. Developing countries have more than 50% of total meat and egg production in global poultry market: [1]. The global poultry meat market is 86.8 million tonnes, consisting of chicken: 85.6%, turkey: 6.8%; duck: 4.6%; goose and guinea fowl: 2.6%.

Years	world	Developed countries	Developing countries	Share (%) of developing countries
1970	15	11	4	26
1975	19	13	5	31
1980	26	18	8	31
1985	31	21	10	33
1990	41	26	15	37
1995	55	28	26	48
2000	69	33	36	53
2005	81	37	44	55
(Increase %)	437	227	1,043	-

Table 1. Development of poultry meat production in developed and developing countries (million tonnes).

Years	world	Developed countries	Developing countries	Share (%) of developing countries
1970	20	15	5	24
1975	22	16	6	27
1980	26	18	8	32
1985	31	19	12	39
1990	35	19	16	46
1995	43	17	25	59
2000	51	18	33	64
2005	59	19	40	68
(Increase %)	195	29	758	-

Source: [1]

Table 2. Development of poultry hen egg production in developed and developing countries (million tonnes)

The world market poultry meat and egg market is been influenced by the production and managemental style from the developing countries. Avian mycotoxicosis is a great constraint in poultry industry, because the disease is characterized by immunosuppresion, hepatotoxicity, nephrotoxicity, loss of egg production, mutagenicity and tetratogenicity.

Mycotoxin are antinutritive factor present in feed ingredients and in concentrated feed, they are a group of secondary fungal metabolites of low molecular weight, diverse and ambiguous in nature, which are specifically implicated in causing toxic effect in animals and man [2,3]. Mycotoxicosis has been a major but unrecognized food safety issue for several centuries. They are naturally occurring contaminants that causes health related problems when it gets into the body through natural route of ingestion, inhalation or may be absorbed through the skin [4]. They are endogenously generated in foods as a result of secondary metabolism [5]. These metabolites are synthesized in or on food surfaces and transported through the food chain[6]. Mycotoxin production takes place in the mycelium after active fungal growth, but may accumulate in specialized structures such as sclerotia, conidia or in surrounding area [7]. Animal studies have shown that, besides acute effects, mycotoxins can cause carcinogenic, mutagenic and teratogenic effects. Mycotoxins-contaminated poultry feed can lead to the transfer of toxins through meat and egg to human beings.

The Food and Agriculture Organization [8] estimating that as much as 25% of the world's Agricultural commodities are contaminated with mycotoxins, leading to significant economic losses. Mycotoxigenic fungi genera include; *Aspergillus, Penicillium* and *Fusarium.* The important mycotoxin in the developing countries include aflatoxins, ochratoxins, citrinin, T-2 toxin, deoxynivalenol (DON), fumonisins and zearalenone.

Fungi genera	Associated mycotoxin
Aspergillus	Aflatoxin, Ochratoxins, cyclopiazonic acid, patulin, sterigmatocystin, gliotoxin, citrinin.
Peniccillium	Ochratoxin, citrinin, patulin, penicillic acid, cyclopiazonic acid, penitrem A, griseofulvin.
Fusarium	Fumonisins, moniliformin, zearalenone, zearalenol, deoxynivalenol, nivalenol, 15- acetyldeoxynivalenol, 3-acetydeoxynivalenol, T-2 toxin, iso T-2 toxin, acetyl T-2 toxin, t-2 triol, T-2 tetraol, fusarenon- X, diacetoxyscripentriol, neosalaniol, fusaric acid.
Claviceps	Ergot alkaloids.

[9]

Table 3. Showing fungi genera and the associated mycotoxin.

2. Avian aflatoxicosis

2.1. Aetiology

Avian aflatoxicosis is a disease of poultry caused by aflatoxin. Aflatoxins (AF) are widely distributed toxins produced by *Aspergillus*. Of the over 180 species of Aspergillus, only a few are aflatoxigenic. After the discovery of AF in the 1960s, A. flavus and *A. parasiticus* of the section *Flavi* were the only known AF producers producing the B and B/G types of AF, respectively [10]. Other aflatoxigenic species that subsequently emerged are *A. nomius* (B and G types), *A. bombycis* (B and G AF), *A. ochraceoroseus*, and *A. pseudotamarii* (B type), but they occur less frequently [11,12]. *A. tamarii, A. parvisclerotigenus* (B types*), A. rambellii* and certain members of *Aspergillus subgenus Nidulantes* namely: *Emericella venezualensis* [13] and *E. astellata* [14] have now been included in the growing list of aflatoxigenic species. *A. arachidicola* sp. Nov. and *A. minisclerotigenes sp*. Nov that produce both forms of the toxin, are the latest emerging aflatoxigenic species. The unexpected new comer is *A. niger*, an ochratoxin (OT) producer which was discovered over four decades ago but was never associated with AF synthesis. However, in a search for aflatoxigenic fungi in Romanian medicinal herbs, [15] showed the capacity of some strains of *A. niger* to produce AFB1 [16].

AFBI is a member of aflatoxin group is one of the most carcinogenic natural product formed in nature [17]. AF has been detected in most countries of the world. Four toxins soon identified: Aflatoxin B1, B2, G1, G2-blue or green florescence under UV-light.•AflatoxinB1most important -highly carcinogenic and widespread occurrence in foods •(B1> M1> G1> B2> M2~ G2). Aflatoxin M1: hydroxylated product of B1appears in milk, urine, and feces as metabolic product

2.2. Factor enhancing AF prevalence in feed ingredients and poultry feed

There are several factors enhancing the prevalence of aflatoxigenic fungi and aflatoxin production in developing countries. The factor include the following:

The food materials must be infected by aflatoxigenic fungi which deposit the toxins on feed ingredients and concentrated feeds.

The substrate which may be feed ingredients and concentrated feeds posseses a source of energy in the form of carbohydrates and organic and inorganic source of nitrogen, trace elememts and moisture for growth of mould and toxin production [8].

Among cereal, the size and integrity of the seed coat also affects the susceptibility of fungal infection and mycotoxin formation [18]

The environmental condition favouring mould growth and AF production are hot and humid conditions, the optimal temperature varies between 24^0Cand 28^0C [19] and seed moisture content of at least 17.5% [20]. These conditions that favours mould growth are present in most developing countries.

Soil type also affect the level of AF contamination of crops for example, light sandy soil support rapid growth of fungi [21]

Presence of other microorganism either bacteria for example presence of *Streptococcus lactis* and *Lactobacillius casei* causes reduction in AF production by *A. parasiticus* [20]. Meanwhile fungal metabolites like rubratoxin and cerulenin enhances AF production [16].

Agricultural practices also affect AF contamination of feed ingredient. Off season harvesting and harvesting system that enhances seed breakage would also increase the degree of AF production [22].

A well aerated storage condition used in most developing country to store feed ingredients increases metabolism and subsequent AF production [23].

2.3. Occurrence of AF

AF has been found as contaminants in animal feed ingredient worldwide. The occurrence in developing countries is more because there is no strict food and feed quality control programmes to reduce the burden of AF. Also their environmental condition presented as hot and humid climate makes most developing country vulnerable to AF in poultry feed. Among the four AF that are of significance in poultry include; AFB1, B2, G1and G2. AFB1 was detected mostly from animal and feed ingredients from developing countries. Among the feeds ingredients, sorghum, wheat, maize were the most investigated, data on groundnut cake, cotton seed meal and fish meal showed high level of AF contamination. The highest level of AFB1 contamination of feed ingredient were reported in corn from Pakistan 25μg/kg [24], Nigeria wheat was found to be contaminated with 17.10-20.53 μg /kg [25].

Higher level of contamination of AF were found in the animal feed than the feed ingredient possibly because of the storage condition which allowed growth and proliferation of mycotoxigenic fungi. In Nigeria AF was detected in poultry feed at 0.0-67.9 μg/kg [26], wheat 17.10-20.53 μg/kg [25], millet 1370-3475 μg/kg [23].

AF contamination of feed and feed ingredients has been a major concern in many developing country like Pakistan where concentration ranging from 24-37.62 μg/kg were found in poultry feed and poultry feed ingredient [24].

Commodity	Country	Type of AF	Incidence	Range (µg/kg)	Mean level ±SD (µg/kg)	References
Poultry/livestock	Nigeria	AFB1	6/13	0.0-67.9	15.5	[26]
Animal feed	Kenya	AFB1	703/830	0.9-595	8.9-46.0	[27]
Animal feed	South Africa	AF	17/23	0.8-156	39.8	[28]
Poultry feed	Morocco	AFB1	14/21	0.3-58	8.4	[29]
Sorghum	Malawi	AFB1	2/15	1.7-3.0	2.35 ±0.65	[30]
Wheat	Kenya	AFB1	23/50	0-7	1.93	[31]
Wheat	Tunisia	AFs	15/51	4.0-12.9	6.7±2.4	[32]
Wheat	Nigeria	AFB1	-	17.10-20.53	19.00±1.67	[25]
Wheat	Algeria	AFB1	30/53	0.13-37.42	>5	[33]
Wheat	South Africa	AFB1	13/238	0.5-2.0	>2	[34]
Maize	Ghana	AF	30/30	6.20-29.50	13-596	[35]
Maize	Uganda	AF	22/49	1.00-1000	-	[22]
Millet	Nigeria	AFB1	12/49	1370.28-3495	2587.47±78.23	[23]
Mouldy Sorghum	Nigeria	AFB1	93/168		199.51±26	[36]
Poultry feed	Pakistan	AFB1	60%		37.62	[24]
Corn	Pakistan	AFB1	8/13		25	[24]
Rice broken	Pakistan	AFB1	3/5		21	[24]
Wheat	Pakistan	AFB1	3/5		19	[24]
Cotton seed meal	Pakistan	AFB1	5/5		22	[24]
Fish meal	Pakistan	AFB1	3/5		24	[24]
Broiler starter (Crumbs)	Pakistan	AFB1	6/11		20	[24]
Broiler starter (Mash)	Pakistan	AFB1	8/14		21	[24]
Layer Starter (Crumbs)	Pakistan	AFB1	16/30		26	[24]
Layer Grower (Crumbs)	Pakistan	AFB1	3/7		23	[24]
Layer Grower (Mash)	Pakistan	AFB1	3/5		32.5	[24]
Layers (Crumbs)	Pakistan	AFB1	23/60		21	[24]
Layers (Mash)	Pakistan	AFB1	25/58		23.5	[24]
Poultry Feed	China	AFB1	38.2		29.7	[37]
Poultry Feed	South Africa	Aflatoxin	22/62		0.7±0.7	[38]
Maize	Morroco	AFB1	16/20		1.57±0.78	[29]

[16]

Table 4. Showing occurrence of AF in animal feed and feed ingredients in developing countries

An unacceptably high level of concentration of AF that ranges from 1-1000 μg/kg was found in maize from Uganda[22] About 70% of wheat samples investigated in Algeria were contamination at a range of 0.13-3742mg/kg [33]

Animal species	Clinical signs Performance effect	Gross pathology	Histopathology	References
Duckling	inappetance, reduced growth. abnormal vacuolation, feather. picking, discoloration of leg/feet. lameness, ataxia, convulsion.	Liver enlarged, pale and shrunken. Kidney enlarged and pale Hydropericardium, ascitis.		[40]
Turkey	inappetance, unsteady gait, Recumbency, anaemia.	body in good condition, generalized and edema. Liver and kidney Congested enlarged and firm. Gallbladder was full.		[41, 42]
Chicken	Same in turkey and duckling Increase mortality Loss of production Increased susceptibility to infectious disease and vaccination failure.	Same in turkey and duckling	Fatty vacuolation of the liver. Karyomegaly and prominent nucleoli in hepatocytes, Bile duct proliferation.	[40-44]
Chicken (broiler)	impared performance lower testis weight and Semen volume, Spermatocrit and Testosterone value.		Abnormal spermatozoa, cessation of spermatogenesis in seminiferous tubules.	[45-46]

Table 5. Main clinical signs, performance and pathological features in food producing animals exposed to AF in selected studies.

2.4. Pharmacological interaction

Aflatoxicosis has effect on plasma half-life, thus it affects drug effect in the body. [47] observed that chlortetracycline plasma concentrations were lowered due to decreased drug binding to plasma protein [48]. Though opinion differed considerably on sparing or aggravating effect caused by the addition of chlortetracycline to feed contaminated with aflatoxin [49,50].

2.5. Metabolism and residues

In broilers, metabolites of aflatoxins BI and B2 concentrated in kidney and liver but cleared within 4 days. Then metabolism of Aflatoxin B1 into conjugated aflatoxins B2a

and Ml occurred in the liver, which will be metabolized to aflatoxicol [51-54]. Aflatoxin BI was excreted in the bile, urine, and feces as 6 major metabolites [55]. The half-life of aflatoxin BI in laying hens is about 67 hours [56], though feed: egg transmission is about 5000:1 [57]. Most aflatoxin excreted through the bile and intestine, but aflatoxin BI and aflatoxicol were detected in ova and eggs for 7 days or longer [58-59]. Aflatoxin BI accumulated in reproductive organs and its subsequent transmission to eggs and hatched progeny (yolk sac and liver) in poultry[60]. It is well established that AFB_1 is both carcinogenic and cytotoxic. For example, synthesis of both RNA and DNA was inhibited when AF (5mg/kg of feed) was given to rats over a 6-week-period. The activated AFB_1 metabolite (i.e. AFB_1-8, 9-epoxide) forms a covalent bond with the N_7 of guanine [61] and forms AFB_1-N_7-guanine adducts in the target cells. The results are G_T transversions, DNA repair, lesions, mutations and subsequently tumor formation [57]. The reactive epoxide can also be hydrolyzed to AFB_1-8, 9-dihydrodiol which ionizes to form a Schiff's base with primary amine groups in the proteins [58]. The short-lived epoxide AFB_1 has also been associated with coagulopathy due to reduced synthesis of vitamin K and other clotting factors as a result of sub-lethal intoxication of animals [62]. With regard to the cytotoxic effects, AFB_1 has been shown to induce lipid peroxidation in rat livers leading to oxidative damage to hepatocytes [63]. A more recent study [64], has demonstrated that AFB_1 can inhibit cyclic nucleotide phosphodiesterase activity in the brain, liver, heart, and kidney tissues.

3. Avian ochratoxicosis

3.1. Aetiology

This is a disease of bird caused by Ochratoxins. Ochratoxins are among the most toxic mycotoxins to poultry. They are nephrotoxins found in grains and feeds worldwide [65- 66]. Ochratoxins are isocoumarin compounds linked to L-b-phenylalanine and are designated A, B, C, and D, because of their methyl and ethyl esters. Ochratoxin A (OTA) is the most common and most toxic, and is relatively stable. OTA production is dependent on different factors such as temperature, water activity (*a*w) and medium composition, which affect the physiology of fungal producers. In cool and temperate regions, OTA is mainly produced by *Penicillium verrucosum* [67, 68] or *P. Nordicum* [69, 70]. *P. verrucosum* mainly contaminates plants such as cereal crops, whereas *P. nordicum* has been mainly detected in meat products and cheese [69]. In tropical and semitropical regions, OTA is mainly produced by *Aspergillus ochraceus* [71-73]. *A. ochraceus* is also referred to as *A. allutaceus* var *allutaceus* [71]. *A. ochraceus* have been reported in a large variety of matters like nuts, dried peanuts, beans, spices green coffee beans and dried fruits, but also in processed meat and smoked and salted fish [71]. Two other species of *Aspergillus section nigri*, *A. niger* var *niger* [74-75] and *A. carbonarius*[76,77] have been reported as OTA producers. The OTA contamination of substrate such as cereals, oilseeds and mixed feeds in warm zones is thought to be due to *A.niger* var *niger* in addition to *A. ochraceus* species [78], whereas *A. carbonarius* seems to be more common in grapes, raisins and coffee [79-80].

Recently, [81] isolated two new OTA producing *Aspergillus* species from coffee beans. These species, *A. lacticoffeatus* and *A. sclerotioniger*, need further investigations and are provisionally accepted in section *Nigri.* In addition, another *Aspergillus* species, *A. alliaceus* also named *Petromyces alliaceus* isolated from onions [82] has been previously reported as OTA producer under laboratory conditions [83]. This species has been suspected to be responsible for the occasional OTA contamination in Californian figs [84, 85] an Argentinean medicinal herbs [67]. The biosynthetic pathway for OTA has not yet been completely established. However, labeling experiments using both 14C- and 13C-labelled precursors showed that the phenylalanine moiety originates from the schikimate pathway and the dihydroisocoumarin moiety from the pentaketide pathway. The first step in the synthesis of the isocoumarin polyketide consists in the condensation of one acetate unit (acetyl-CoA) to four malonate units. Recent data showed that this step requires the activity of a polyketide synthase [86]. Moreover, the gene encoding polyketide synthase appears to be very different between *Penicillium* and *Aspergillus* species [86]. In *A. ochraceus,* the gene of polyketide synthase is expressed only under OTA permissive conditions and only during the early stages of the mycotoxin synthesis [86]. No such data are presently available on *penicillium*. In *Penicillium* species, ' [86]' observed that *P.nordicum* and *P. verrucosum* use two different polyketide synthases for OTA synthesis. This difference is probably related to the *P. verrucosum* ability to produce citrinin, also a polyketide-based mycotoxin, in addition to OTA.

Destined Specie	Country	Type of samples	Type of Grain composition	LOD (LOQ)	Incidence (%)	Average	Range	References
Poultry	Kuwait	Raw ingredients	Wheat bran	5	10/14(71.4%)	4.6	n.d. -12.1	[87]
			soybean meal		18/21(85.8%)	7.9	n.d – 40	
			Yellow maize		31/32 (96.8%)	6.38	n.d. –14.5	
		Feed	Layer mash	-	20/20 (100%)	9.6	5-16.7	
			broiler starter		13/14 (93%)	8.0	nd-9.1	
			Broiler finisher	-	17/19 (90%)	6.1	nd-14.3	
Poultry	Venezuela	Concentrated feed	-	-	47 /50 (94%)	-	2.56 -31.98	[88]
Poultry	Argentina	Feed	Corn (60%)	10	38%	27		[89]
Poultry	China	Feed			61.5%	7.0		[37]
Poultry	India	Raw ingredients	Wheat		4/12 (33.3%)	60.75	n.d. -98	[90]
			Maize		5/12 (41.7%)	104.4	n.d. -140	
			Rice		3/12 (25%)	20	n.d. -25	
			Sorghum		2/12 (16.7%)	34	n.d. -38	
			Barley		3/12 (25%)	26	n.d. -38	
			Grams		2/12 (16.7%)	12.5	n.d - 15	
			Ground nut		2/12 (16.7%)	39	n.d.48	
			Millet		3/12 (25%)	2.5	n.d. -3	
			Cotton seed meal		2/10 (20%)	31	n.d. -42	
			Soybean meal		2/10 (20%)	31	n.d. -42	
			Rapeseed meal		2/10 (20%)	19	n.d. -28	
			Sunflower meal		3/10 (30%)	49	n.d. -68	
			Guar meal		2/10 (20%)	21	n.d. -22	
			Corn gluten meal		2/10 (20%)	21	n.d. -22	

Source: [91]

Table 6. Showing the occurrence of Ochratoxin in developing countries in a selected surveys.

3.2. Occurrence of ochratoxin

OTA was reported by [90] in raw ingredient for making poultry feed were found to be contaminated at range of 00-140 μg/kg in maize 00-98mg/kg in wheat followed by sunflower meal at 00-68 μg/kg. The incidence in India ranges from 16-41% [90]. Also high level of contamination was reported in soya meal from Kuwait n-d-40 μg/kg [87]. [88] reported a contamination level of 2.56-31.98 μg/kg in Venezuela concentrated poultry feed investigated.

Animal specie	Production Phase (age)	Experimental (dosage)	Clinical signs and (duration)performance effects	Gross pathology	References
Laying hens) (*Isa brown*	Laying (28 week-old)	Artificially contaminated feed (100-2000) ug/kg (30 days)	Decrease of egg mean weight Decrease of number of eggs placed Shell decalcification/thinning Egg altered conformation	Kidney Congestion and hemorrhages Increased size Liver - Yellowish	[92]
Broiler chickens	Fattening (6 day-old)	Artificially contaminated feed (100-2000) μg/kg (28 days)	Decreased growth rate Reduced feed consumption Decreased feed conversion Reduced serum protein and albumin.	Not evaluated	[93]
Laying hen (*Isa brown)*	Laying (11month old)	Naturally contaminated diet (160-332) ug/kg	Debility Reduced egg production	Kidneys Congestion Hemorrhage Renomegalia Liver yellowish color Diffuse Hepatomegalia Scattered necrotic foci Congestion Hemorrhage	[94]
Broiler chickens	Fattening (1 day-old)	Artificially contaminated Feed (0-800) ug/kg (5 weeks)	Significantly decreased of -Body weight -Feed consumption - Feed conversion ratio - Increased mortality	Not assessed	[95]

Source: [91]

Table 7. Showing the main clinical signs, performance and pathological features in poultry exposed to OTA in selected studies

4. Funmonism

4.1. Aetiology

The fumonism, are food borne carcinogenic mycotoxin that affect animals and man. They are evaluated as group 2B component carcinogen [96]. It has various analog. Twenty eight

fumonisin analogs separated into four main groups, as fumonisin A, B, C, and P series has been identified. The fumonisin B (FB) analogs, comprising toxicologically important FB_1, FB_2, and FB_3, are the most abundant naturally occurring fumonisins, with FB_1 predominating and usually being found at the highest levels [96] in feed ingredients and poultry feed in world wide. FB_1 accounts for about 80% of the total fumonisins produced inthis substrates, while FB_2 usually makes around 20% and FB_3 usually makes up from about 5% when cultured on corn or rice or in liquid medium [97-100].

Different *Fusarium* species have been reported to produce fumonisins

4.2. Occurrence of fumonisin

FB1 a potent carcinogen was found in maize investigated from developing countries like Argentina, Benin, Egypt, Nepal, Honduras, Malawi, Zambia Botswana, and Tanzania at a range between 35- 65,000 μg/kg. [101-109]. Poultry feed investigated in china was contaminated with 1854.3mg/kg(37).

No doubt the climatic condition to the agricultural practices in these country allow the growth of fungi and subsequent elaboration of toxin in their substrate.

Country	Nature of samples	Number of samples Positive/total	Highest level (μg/kg)	References
Argentina	maize	16/17	2000	[101]
Benin	maize	20/21	2310	[102]
Egypt	maize	2/2	2380	[103]
Nepal	maize	12/24	4600	[104]
Honduras	Maize	24/24	6555	[105]
Zambia	maize	20/20	1710	[102]
China	Poultry Feed	82%	1854.3	[37]
Botswana	Maize		35-255	[106]
Mozambique	Maize		240-296	[106]
Malawi	Maize		ND-115	[106]
Tanzania	Maize		ND-160	[106]
Honduras	Maize		68-6555	[107]
Uruguay	Maize		ND-3688	[108]
Coastarica	Maize		1700-4780	[109]

Table 8. Occurrence of FB1 In Feed Ingredient In Developing Countries

Animal Species	Type of toxin	Clinical signs	Gross pathology	Histopathology	References
Laying hen	FB1	Black sticky diarrhea Reduction in food intake, egg production and body weight. Lameness, increased mortality and impaired immunity.	Kidney, pancreas and liver enlargement. Enlargement of proventiculus. Atrophy of lymphoid Organ. Rickets.	Liver has multifocal necrosis of hepatocytes hyperplasia of hepatocytes and bile ductules. Intestine had villous atrophy Globlet cell hyperplasia lymphoid cell depletion from thymus.	[110] [111]
	FB1+ FB2 + moliniformin.			Decrease in blood cell counts Haemoglobin, PCV and white Blood cell count. Abnormal erythrocyte Formation	[112]
Chicken Embryo	FB1	100% mortality	pathological changes in liver, heart, lung, Musculoskeletal system, Intestine, testis and brain		[113]
Duckling	FBs		slightly swollen and Reddened liver, low body fat And loss of weight		[113, 114]
Turkey And poult	FB1	loss of weight	cerebral encephalomalasia	increased sphingamine to Sphingosine ratio in the Serum by distrupting Biosynthesis of ceramide and sphingolipid metabolism Hepatocellular and biliary hyperplasia	[115, 116]

Table 9. Showing the main clinical signs, performance and pathological features in poultry exposed to fumonism in selected studies

5. Fusariotoxin poisoning

Synonyms Fusariomycotoxicosis, trichothecene mycotoxicosis, T-2 toxicosis, vomitoxicosis, zearalenone toxicosis. They are responsible for various diseases of birds in developing countries.

5.1. Aetiology

The trichothecenes include deoxynivalenol (DON), 3, monoacetyldeoxynivalenol (3-AcDON), 15, mono-acetyldeoxynivalenol (15-AcDON), nivalenol (NIV), HT-2 toxin (HT-2), neosolaniol (NEO), T-2 toxin (T-2), T-2 tetraol and T-2 triol, diacetoxyscirpenol (DAS), MAS-monoacetoxyscirpenol (MAS) and fusarenone-X.

Different fungi species of the general *Fusarium* are responsible for the production of this group of mycotoxins. Major producers of trichothecenes are F. *graminearum, F. culmorum, F. cerealis, F. poae*.

5.2. Occurrence of trichothecenes

Occurrence of trichothecenes in feed ingredients and poultry feed in developing countries is as a result of ubiquitous nature of the fungi which are generally found when certain cereal crops like maize, wheat, corn, millet where grown under stressful condition such as drought. These mould occur in soil, hay and especially grains undergoing microbial and possibly enzymatic degradation [20]. Direct contamination or indirect contamination of feed may occur. Direct contamination occur when the poultry feed were infected with mycotoxigenic fungi.

Indirect contamination may be as a result of fungi elaborating its toxin into the substrate, the incriminating fungi may be removed but the toxin remained in the poultry feed made from such feed ingredients.

Since moulded feed are part of the diet of animal in developing counties, thus all poultry feed are suspect and may contain different level of Trichotheceus toxin.

DON has been reported in maize from Nigeria, South Africa, Argentina, Brazil Pakistan at different concentration that ranges from 0.05-2650 mg/kg [116-120].

6. Diagnosis

Diagnosis is made through observing the appropriate field signs, finding gross as well as microscopic tissue lesions, and detecting the suspected toxin in grains, forages, or the ingesta of affected animals. However, the tests required to detect these toxins are complex and few diagnostic laboratories offer tests for multiple trichothecenes in developing countries. The samples of choice include both refrigerated and frozen carcasses for necropsy examination and a representative sample of the suspected contaminated grain source. Because the toxin is produced under cold conditions, the grain sample should be frozen rather than refrigerated for shipment to the diagnostic laboratory.

Country	Feed samples	Type of toxin	Range (μg/kg)	References
Nigeria	Maize	DON	9.5-745.1	[116]
	Maize	3-acDON	0.7-72.4	
	Maize	DAS	1.0-51.0	
Poland	Wheat	DON	2-40	[121]
	Wheat	NIV	0.01	
	Wheat	ZEN	0.01-2	
Bulgaria	Wheat	DON	up to 1.8	[122]
	Wheat	ZEN	up to 0.12	
Finland	Feed and grains	DON	0.007-0.3	[123]
	Feed and grain	ZEN	0.022-0.095	
Norway	Wheat	DON	0.45-4.3	[124]
Netherland	Wheat	DON	0.020-0.231	[125]
	Wheat	NIV	0.007-0.203	
	Wheat	ZEN	0.002-0.174	
South Africa	Maize	DON	up to 1.83	[126]
	Maize	NIV	up to 0.37	
South Africa	Cereals and Animal feed	ZEN	0.05-8.0	[117]
Philippine	Maize	NIV	0.018-0.102	[127]
	Maize	ZEN	0.059-0.505	
Thailand	Maize	ZEN	0.923	[127]
Korea	Maize	DON	Mean-0.145	[128]
	Maize	NIV	Mean-168	
Vietnam	Maize powder	DON	1.53-6.51	[129]
	Maize powder	NIV	0.78-1.95	
China	Maize	DON	0.49-3.10	[129]
	Maize	NIV	0.6	
Japan	Wheat	DON	0.029-11.7	[130]
	Wheat	NIV	0.01-4.4	
	Wheat	ZEN	0.053-0.51	
New Zealand	Maize	DON	Max 3.4-8.5	[131]
	Maize	NIV	Max 4.4-7.0	
	Maize	ZEN	Max 2.7-10.5	
U.S.A	Wheat	DON	up to 9.3	[132]
Canada	Wheat and barley	DON	up to 0.5	[133]
	Wheat and barley	ZEN	up to 0.3	
	Maize	DON	0.02-4.09	[134]
Argentina	Wheat	DON	0.10-9.25	[118]
Brazil	Wheat	DON	0.47-0.59	[119]
	Wheat	NIV	0.16-0.40	
	Wheat	ZEN	0.04-0.21	
Pakistan	Maize	Nivalenol	500-2650	[120]
	Maize	DON	136-2656	
	Maize	3-ac DON	100-850	
	Maize	ZON	1250	
	Maize	15ac DON	100	
	Maize	DAS	364-750	
	Maize	HT-2	100-500	
	Maize	T-2	143-1125	

Table 10. Occurrence of Trichothecenes in Feed Ingredients In Developing Countries

Domestic Animal	Type of toxin	clinical signs	Gross lesions	Histopathology	References
Domestic poultry		Gastrointestinal bleeding Neurological abnormality Flaccid paralysis, weakness Of the neck and wing muscles Characteristic drooping head And wings. Immune suppression which May predispose bird to Secondary bacteria infection	Skin and mucosal surface inflammation which affected subcutaneous fluid over head and neck. Multiple haemorrhage and pale area in skeletal muscle.		[136]
Chicken	DAS T-2 toxin	necrotic lesion on the tongue reduces growth rate and efficiency	Lesion in palatine, sublingual, internal angle of the tongue.		[137]
	DAS	feed refusal Haemorrhagic disorder Prolong clothing time Mild diarrhea, fatigue, ataxia Poor feathering, poor feather Quality and soft bones, Subcutaneous edema, Degeneration of neck muscle.	Enlargement of liver, and gallbladder. perivascular edema of brain, depletion of lymphocyte in thymus and bursa of fabriciuos	it affects Coagulation factors VII, prothrombin fibrinogen	[138]
	T-2 And Aflatoxin	Decrease egg production increase egg shell breakage			[139]
Duck	DON	100% mortality and high Concentration	dehydration and heamorrhage along the intestinal epithelium		[140]
	T-2 toxin	reduced body weight		altered serum And plasma Level	[141]
	DON	reduced egg production			[142]
Goose And turkey	T-2 toxin	decrease egg production	degenerative changes in ovaries Interruption of maturation of Follicle. Peritonitis, bleeding, Wrapped oviducts, necrosis and Spleen amilodiosis. Catarrhal Enteritis.		

Table 11. Showing the main clinical signs, gross and histopathological changes in some poultry species.

7. Management of avian mycotoxicosis

This involves various practises that reduces fungal contamination of feedstuff and possible use of mycotoxin binders in feed. The clinical signs seen in avian mycotoxicosis are not pathognomonic so the presence of toxin in feed with the associated clinical signs may help clinician to make effective diagnosis and withdrawal of contaminated feed can help to ameliorated the disease condition. Preventive and control of mould in feed is key to achieving control of avian mycotoxicosis.

Following good agricultural practices during both pre-harvest and post-harvest conditions would, minimize the problem of contamination by mycotoxins such as aflatoxins, ochratoxin and trichothecene mycotoxins. These include appropriate drying techniques, maintaining proper storage facilities and taking care not to expose grains.

7.1. Prevention and control of mycotoxins in stored grains and seeds

7.1.1. Dry the feed ingredients

Fungi cannot grow or mycotoxins be produced in properly dried foods, so efficient drying of commodities and maintenance of the dry state is an effective control measure against fungal growth and mycotoxin production.

To reduce or prevent production of most mycotoxins, drying should take place as soon after harvest and as rapidly as feasible. The critical water content for safe storage corresponds to a water activity (a_w) of about 0.7. Maintenance of feeds below 0.7 a_w is an effective technique used throughout the world for controlling fungal spoilage and mycotoxin production in foods.

Problems in maintaining an adequately low a_w often occur in the tropics, where high ambient humidity make control of commodity moisture difficult. Where grain is held in bags, systems that employ careful drying and subsequent storage in moisture-proof plastic sheeting may overcome this problem.

While it is possible to control fungal growth in stored commodities by controlled atmospheres or use of preservatives or natural inhibitors, such techniques are almost always more expensive than effective drying, and are thus rarely feasible in developing countries.

7.1.2. Avoid grain damage

Damaged grain is more prone to fungal invasion and therefore mycotoxin contamination. It is thus important to avoid damage before and during drying, and in storage. Drying of maize on the cob, before shelling, is a very good practice.

Insects are a major cause of damage. Field insect pests and some storage species damage grain on the head and promote fungal growth in the moist environment of the ripening grain. In storage, many insect species attack grain, and the moisture that can accumulate from their activities provides ideal conditions for the fungi. To avoid moisture and mould problems, it is essential that numbers of insects in stored grain be kept to a minimum. Such

problems are compounded if the grain lacks adequate ventilation, particularly if metal containers are used.

7.1.3. Ensure proper storage conditions

While keeping commodities dry during storage in tropical areas can be difficult, the importance of dry storage cannot be overemphasized. On a small scale, polyethylene bags are effective; on a large scale, safe storage requires well-designed structures with floors and walls impermeable to moisture. Maintenance of the water activity of the stored commodity below 0.7 is crucial.

In tropical areas, outdoor humidities usually fall well below 70% on sunny days. Appropriately timed ventilation, fan-forced if necessary, will greatly assist the maintenance of the commodity at below 0.7 aw. Ideally, all large-scale storage areas should be equipped with instruments for measuring humidity, so that air appropriate for ventilation can be selected.

Sealed storage under modified atmospheres for insect control is also very effective for controlling fungal growth, provided the grain is adequately dried before storage, and provided diurnal temperature fluctuations within the storage are minimised.

If commodities must be stored before adequate drying this should be for only short periods of no more than, say, three days. Use of sealed storage or modified atmospheres will prolong this safe period, but such procedures are relatively expensive and gaslight conditions are essential.

A proven system of storage management is needed, with mycotoxin considerations an integral part of it. A range of decision-support systems is becoming available covering the varying levels of sophistication and scale involved.

7.2. Control

Control of mycotoxin in poultry feed is important and it should be hinged on eliminating mycotoxin from the food chain. Mycotoxygenic fungi are naturally found in soil and air, which makes it difficult to prevent their contamination of agriculture commodities. Nevertheless attempts should be made to control factors that affects the growth of mycotoxigenic fungi and the subsequent toxin production. Factors which include warm temperature between (20^{o}c to 30^{o}c), high moisture content (20-25%), water activity (a_w) of about 0.7aw and relative humidity of 70% and above. These factors enhance fungi growth and mycotoxin production (143,145).

Before harvesting of crops damage to grains as a result of field insect pest and some storage species damage grains and promote fungal growth in the environment of ripening grain. Strategies used in various preventive measure in poultry feed involves good agronomic practices, detoxification of mycotoxin in grains use of mould inhibitors, genetic approach through improved breeds of plants.

7.2.1. Physical decontamination

Decontamination of mycotoxin from cereal crops used in the production of poultry feed can be classified as physical decontamination, biological decontamination and chemical decontamination [146]. [145] suggest the following method of elimination of mycotoxin in grains which include, Density segregation and floatation, cleaning and washing, seiving, dehulling, hand picking, irradiation, milling, thermal degeneration.

[147] observed that washing using distill water resulted in 65%-95% reduction of DON (16-24mg/kg) and 2% to 61% of ZEN (0.9-1.6mg/kg) in contaminated barley and corn.

Density segregation in certain liquid or fractionation by specific gravity help to segregate fungi infected and mycotoxin contamination grains used in the production of poultry feed.[148]. It was also observed that fumonisms present in broken corn kernels is about 10 fold higher than that in intact corn therefore the separation based on the size has been suggested. [147, 146]

Irradiation is also a useful tool in inactivation of some mycotoxin reported ultrasonication been used in contaminated corn without affecting the grain composition. Several workers reported the use of Gamma irradiation to reduce Zearelenone DON and T-2 toxin in corn, wheat. [148-150]

7.2.2. Biological decontamination

This involved systemic degradation of toxins leading to a less toxin product. [151] reports a fungus yeast *Expoliata spinnifera* was able to grow on fumonisin B1as a sole of carbon source. The hydrolysis of fumonisin B1 yields free Tricarboxylic acid and aminopental, the intermediate aminopentol undergo oxidative deamination. *Sacchromyces cerevisiae* ferment zearalenone converting it into beta-zearelenol, which has less activity compared to the parent compound.[152]

Feed additive like mycotoxin inactivates trichothecenes by enzymatic decontamination of the 12-13 epoxy ring, and zealenone by the enzymatic opening of the lactone ring [148-152]

7.2.3. Detoxification of mycotoxin feed

Moist ozone and dry ozone were able to reduce DON concentration in contaminated corn up to 90% and 70% respectively [153]. [154] reports 79% reduction in fumonism level in corn.

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