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Aflatoxin Contamination in Some Nigerian Feeds and Feedingstuffs: Highlights of Some Nutritional, Physiopathological and Economic Implications

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ABSTRACT

A number of feeds and feedingstuffs, along with the post-harvest factors predisposing them to aflatoxin contamination, are outlined. Based on field reports, the nutritional and physiopathological implications of aflatoxin as an environmental pollutant and feed contaminant in Nigeria are discussed.

Viewed from the context of the positive response of the farming population to the call for local sourcing of a substantial proportion of industrial raw materials (backward integration) by various governments, the economic implications of post-harvest deterioration of large tonnages of food crops (now being experienced nation-wide) are highlighted.

Various ameliorative measures (both short and long term) are suggested to minimize both post-harvest deterioration and the imminence of the scourge of aflatoxicosis, especially in the poultry industry.

INTRODUCTION

With the dwindling of the nation's foreign reserves, emphasis has recently been placed on 'Backward Integration'—a design by the Federal Government to encourage manufacturers to rely on local sources of industrial raw materials which had previously depended on importation. While the response to this call has been remarkable, as manifest in increased agricultural output, post-harvest deterioration arising from poor storage facilities constitutes a major drawback to the full realization of the benefits.

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One common storage problem is mouldiness and aflatoxicosis is a poisoning caused by one or more aflatoxins, toxic metabolites of certain strains of fungi, mainly *Aspergillus flavus* and *Aspergillus parasiticus.* Aflatoxins B_1 and B_2 are produced mainly by *A. flavus* while G_1 and G_2 are produced by *A. parasiticus.*

While groundnut and its products remain the most susceptible to aflatoxin contamination (Allcroft & Carnaghan, 1963), other susceptible commodities include maize, sorghum, soyabean, cotton seed and products, copra, oats, rice and wheat bran, cassava and its peels. The importance of the above ingredients in the food of man and his livestock needs little emphasis. Quite apart from animal health and the associated problems of the occurrence of aflatoxin in feeds and feddingstuffs, there is also the problem of transmission of aflatoxin through farm animals to the human foodchain.

The present account therefore examines the prevalence of aflatoxins in some Nigerian feeds and feedstufs and highlights the physiopathological and economic implication in poultry. The choice of this class of animal (poultry) for investigation is in recognition of its susceptibility to aflatoxicosis vis-a-vis its growing importance in the Nigerian economy.

PREVALENCE OF AFLATOXINS IN FEEDS AND FEEDINGSTUFFS

A survey of contemporary literature reveals an increasing wave of aflatoxin contamination of feeds and feedingstuffs and consequent poisoning of a large number of animals, expecially poultry, in several parts of the country. For example, Akinyemi *et al.* (1984) showed that aflatoxicosis was responsible for 93% mortality in a flock of 2977 3-weeks-old White Peking ducklings in Ogun State following consumption of a compounded poultry ration which, on analysis, was found to contain 3·30 μg/g (3·20 ppm) aflatoxin B_1. Asuzu and Shetty (1986) reported the death of over 1000 6-weeks-old broilers belonging to the University of Nigeria poultry farm 2 weeks after they were fed a newly purchased commercial poultry feed. Analysis of the feed for aflatoxin revealed the presence of Aflatoxin B_1 at a concentration of 265 μg/kg (2·65 ppm) of feed and day-old broilers fed the same aflatoxin contaminated diet exhibited clinical and pathological manifestations identical with naturally infected birds. In a survey of aflatoxin contamination in commercial poultry feeds in five southern states of Nigeria, Oyejide *et al.* (1986) reported that 57–62% of the chick and broiler starter, grower, broiler finishes and layer rations were contaminated with aflatoxin.

Aflatoxin B_1 levels in the 28 quantitatively analysed samples were found to vary from 0·57–2·55 μg/g (or 0·57–2·55 ppm). At the Kainji Lake Research Institute, Okaeme (1987) has recently reported cases of outbreak of aflatoxicosis in guineafowls hitherto regarded as very resistant to aflatoxin when compared with turkey, duck and chicken. During examination of the feed he found that, in some cases, the rice bran and maize used in the preparation of the feeds has visible greenish–yellow plaques due to fungal discoloration. The fungal growth isolates were mostly *Aspergillus flavus* and *Mucor* spp. which were found prevalent in processed feed, groundnut cake, rice bran and maize used in the feed preparation. On analysing the feed in the nine cases examined, he reported aflatoxin levels of 49–72·50 μg/kg with a mean of 59 μg/kg.

Although the potential contribution of animal products to aflatoxin load has not received as much attention as the plant counterpart, recent investigations (Abalaka & Eronini, 1987) have shown that cow meat samples, obtained from three locations with different sanitary qualities over a 12-month period in Zaria (Nigeria), contained aflatoxins B_1, B_2, G_1 and G_2 up to the level of 1·20–5·10 mg/kg or 1·2–5·10 ppm. This implies an accumulation of this toxin within tissues, ostensibly on account of chronic exposure of the cows to aflatoxin-contaminated feeds and/or feedingstuffs. Such a situation represents a serious health hazard to man and the more susceptible poultry when meat/bone meals derived therefrom are used as components of compound livestock feed.

Edible oils such as groundnut and palm oils which are commonly added to poultry feeds to increase the energy density and enhance feed efficiency are not immune from aflatoxin contamination. Earlier studies (Abalaka & Elegbede, 1982; Abalaka, 1984) showed that a number of the commonly available edible oils which form part of the recipe for African dishes were frequently contaminated with aflatoxin. Laboratory analysis of palm oil, palm kernel oil and groundnut oil obtained from Ibadan markets by Kuku and Agboola (1984) showed that, of the fifteen mould species isolated from such oils, six were *Aspergillus* spp. Further investigations on the individual effects of the isolates showed that *Aspergillus flavus* caused the greatest increase in the free fatty acid content of the oils, especially palm oil. Although aflatoxin was not determined in such oils, the chances of their contamination by this toxin cannot be ignored.

Recent investigations (Aletor, 1987, unpublished data) indicate that maize, brewer's grain and vitamin-mineral premixes in moulded conditions and containing hazardous levels of aflatoxin B_1 are to be found in some farms and/or feedmills located in varous parts of Ondo State. Moulded feed troughs, drinkers and poultry litters, all of which are veritable sources of aflatoxicosis, are not uncommon.

PREDISPOSING FACTORS TO AFLATOXIN CONTAMINATION

Generally, any factors which encourage the growth of *Aspergillus flavus* invariably enhance the elaboration of aflatoxins. Such conditions include bruising during and after harvest, high carbohydrate content, high temperature, high relative humidity (RH), residual fat and oxygen availability. Austwick & Ayerst (1963) first pointed out that moisture availability or RH surrounding these commodities represents the most critical of these factors.

The optimum RH for aflatoxin production is about 85–90% and minimum moisture content for the growth of *A. flavus* and aflatoxin production in groundnut is about 8% and 16%, respectively (Ashworth *et al.*, 1965). This suggests, therefore, that the risk of aflatoxin contamination, especially of groundnut and its products, can be greatly minimized if the moisture content can be kept below these critical levels during storage. The maximum growth rate of *A. flavus* occurs at 38°C although it grows well up to 42°C. However, maximal aflatoxin production occurs at 24°C. Consequently, many feed grains grown or processed in the warm humid tropics such as Nigeria would be expected to have a high risk of aflatoxin contamination.

NUTRITIONAL IMPLICATIONS OF THE GROWTH OF *A. FLAVUS* AND AFLATOXIN ELABORATION

Although information on the effects of the growth of *A. flavus* on the nutrient composition of feed ingredients is scanty, it is fairly well established

TABLE 1
Effect of Dietary Aflatoxins on Feed Conversion Efficiency (Feed/Gain)[a] in the Chicken

Aflatoxin levels (ppm)	*Aflatoxin type*				*Mean*[b]
	B_1	B_2	G_1	G_2	
0·00	2·63	2·63	2·63	2·63	$2·63 \pm 0·02^a$
0·01	2·64	2·69	2·71	2·72	$2·69 \pm 0·03^b$
0·10	2·68	2·67	2·71	2·71	$2·69 \pm 0·04^b$
1·00	2·76	2·75	2·78	2·76	$2·78 \pm 0·02^c$

[a] Feed conversion efficiency = weight of feed consumed per unit weight gained.
[b] Means followed by the same letter are not significantly ($P < 0·05$) different.
Source: Aletor *et al.* (1981*a*).

TABLE 2
Effect of Dietary Aflatoxins on Nitrogen Retention[a]

Aflatoxin levels (*ppm*)	*Aflatoxin type*				*Mean*[b]
	B_1	B_2	G_1	G_2	
0·00	0·84 ± 0·02	0·84 ± 0·02	0·84 ± 0·02	0·84 ± 0·02	0·84 ± 0·02[a]
0·01	0·80 ± 0·07	0·65 ± 0·01	0·59 ± 0·26	0·60 ± 0·01	0·66 ± 0·09[b]
0·10	0·75 ± 0·06	0·64 ± 0·10	0·58 ± 0·12	0·58 ± 0·05	0·64 ± 0·08[b]
1·00	0·59 ± 0·09	0·69 ± 0·10	0·78 ± 0·05	0·62 ± 0·04	0·64 ± 0·08[b]

[a] Nitrogen retention = the weight of food nitrogen withheld for body tissue synthesis over a given period (g/chick/day).
[b] Means followed by the same letter are not significantly ($P < 0·05$) different.
Source: Aletor *et al.* (1981*a*).

that moulds utilize the lipid present in their substrates, a situation which decreases the metabolizable energy (ME) values of moulded corn and causes depletion and/or deterioration of other nutrients (Tables 1 and 2). The vitamin E activity of blighted corn has been found to be about 40% less than the theoretical value. Thiamine has been found to be required by *A. flavus* for optimal production of Aflatoxin B_1 by Hamilton (1974). Decreased vitamin E activities associated with moulded meals, such as soya bean meal, are believed to be due to fungal destruction of the α-tocopherols.

Effect on nutrient utilization by poultry

A major concern of aflatoxicosis, from the nutritional standpoint in poultry, appears to be linked to its interaction with protein, carbohydrate, lipid and vitamin metabolism.

Aflatoxin B_1 not only affects lipid synthesis and transport but also interferes with its absorption and degradation in chickens (Smith, 1980). Aflatoxin contamination in feeds appears to increase the dietary requirements of lipotropes which are nutrients such as methionine, folic acid, choline and vitamin B_{12}, whose presence in a diet prevents fatty liver. Liver and plasma levels of vitamin A are decreased by aflatoxin in the chicken while the metabolism of the B-series of vitamins is also markedly impaired.

PATHOLOGICAL ASPECTS OF AFLATOXIN INGESTION IN POULTRY

Generally, the toxicity of aflatoxin depends on the aflatoxin in question and the species of animal. Aflatoxin B_1 seems to be most toxic followed by G_1, B_2

TABLE 3
Effect of Aflatoxin on Poultry

Effect	*Quantity of toxin (ppm)*
Acute death, hepatic necrosis and haemorrhage	1–10
Impaired immunogenesis	0·25
Reduced resistance	0·60–1·00
Decreased gain	1·5–2·50
Decreased egg production	2–8

Adapted from Smith (1980).

and G_2. Of the common species of poultry, ducks appear to be most sensitive to aflatoxin, followed by turkey, geese and pheasants while chicks appear the least sensitive.

The toxicity effects of different dietary levels of aflatoxin as well as the LD_{50} for some selected poultry species are shown in Tables 3 and 4, respectively. Various reports on a number of poultry species studied indicate that the first clinical signs of aflatoxicosis are reduced appetite, and loss of weight. Quite often there are no marked signs of disease apart from general unthriftiness until a few days before death when the animal appears dull, develops atoxia and finally becomes recumbent. The most important pathological effects which occur are liver damage and bile duct hyperplasia. Others include periportal fatty infiltration of the hepatocytes, increased SGPT and SGOT activities and the inhibition of brain ACHE activity (Aletor *et al.*, 1981*a*,*b*). Serious infiltration of the pericardium, hepatomas, enlargement of the pancreas and regression of bursa of fabricus have also been reported (Smith & Hamilton, 1969).

The development of fatty liver is regarded as a primary lesion in avian aflatoxicosis. In the chicken, the fatty liver syndrome during aflatoxicosis assumes its particular importance since about 90–95% of Total Fatty Acids are synthesized in the liver from where they are transported to other parts of the body. Accumulation of fat in the liver in aflatoxicosis therefore represents a serious disturbance in lipid metabolism.

TABLE 4
LD_{50} Levels for Aflatoxin with Some Selected Poultry Species

Animal	*LD_{50} (ppm)*
Duckling	0·36
Turkey	1·86
Chick	6·50

ECONOMIC IMPACT OF AFLATOXIN CONTAMINATION IN FEEDS AND FEEDINGSTUFFS

Perhaps one of the greatest problems of aflatoxicosis from the veterinary standpoint is the difficulty in diagnosis on account of the mild and frequently close similarities of many of the symptoms with other established diseases. Therefore, such effects as reduced growth rate, reduced feed efficiency, decreased egg production, poor carcass value and increased susceptibility to disease obviously have serious economic implications to the livestock producer. For example, dietary aflatoxin as low as 0·01–1·00 ppm can cause significant reduction in feed efficiency in the chick. Okaeme (1987) computed that each time there is an outbreak of aflatoxicosis it takes the laying guineahens 8–10 weeks to return to normal laying capacity during which period the birds must be fed with zero economic returns. Apart from outright large scale mortality which could accompany a high aflatoxin load in feed, reductions in feed efficiency by sub-lethal levels represent serious financial loss to the producer since feeding alone currently accounts for about 80% of the cost of intensive poultry production in most developing countries.

CONCLUSIONS AND RECOMMENDATIONS

It is evident that the problem of aflatoxin in poultry feed is far from simple. It would appear that acute aflatoxicosis is somewhat easier to deal with than the poor performance of birds chronically or periodically exposed to lower levels. Among the currently employed therapeutic measures against aflatoxicosis are high fat and high protein diets which are believed to ameliorate the lethal effects, especially in broilers and turkeys. Broad spectrum antibiotics are of value against secondary infection.

It must, however, be emphasized that the problem of aflatoxicosis is largely one of feed management. For example, farm bulk feed bins and the broiler house feed troughs in moist conditions are primary sites of significant amplification of aflatoxins in rations contaminated with moulded feeds. This situation may be remedied with proper attention paid to cleaning procedures of mouldy or caked feeds as well as discouraging all those conditions that favour mould growth such as wetness and high RH. This makes it imperative for the government to provide, as a matter of urgency, driers and silos in reasonable numbers, at strategic locations in all the States of the Federation. Such facilities should be made available, at subsidized rates, to local grain farmers and feed millers who currently suffer huge

financial losses on account of mould-related post-harvest deteriorative changes of their products.

The use of mould inhibitors such as copper sulphate, sodium propionate and gentian violet could also be of value. Perhaps the best precaution is the maintenance of tight controls of the quality of feed ingredients accepted in our feed mills. Proper legislative back-ups, as is done elsewhere, should be enunciated to effect such controls. Rapid mini-column screening of aflatoxin may also be used. The use of such procedures as roasting, solvent extraction, chemical inactivation or microbial degradation, have proved not only efficient but economically feasible as a means of reducing aflatoxin contamination of feed ingredients.

Aflatoxins, even at diminutive dietary levels, have been established to cause decreased growth rate and feed conversion efficiency. This situation therefore makes the above recommendations—although painstaking—worthwhile, especially when it is realised that feeding presently represents about 80% of the cost of intensive poultry production in Nigeria.

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