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Survey of Aflatoxicosis in Farm Animals[1]

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Over a 22-month period, 278 submissions of farm animals were made to the North Carolina Diagnostic Laboratory for suspected aflatoxicosis, and 94 cases were confirmed on the basis of finding aflatoxin in the feed and the occurrence of bile ductule proliferation. There was an annual variation in the incidence of aflatoxicosis, as well as a seasonal variation: the peak incidence occurred in the winter, and the minimum incidence occurred during the summer. The annual increase coincided with the corn harvest. All confirmed cases occurred on farms that raised and stored their own corn, and 88% were in swine. The cases were geographically localized in the eastern section of North Carolina (94% of the total cases) where 82% of the swine and 79% of the corn are produced. Mean concentration of aflatoxin in feed samples from the confirmed cases was 3,890 μg/kg, and the mean value for corn used in making the feed was 5,180 μg/kg. Only aflatoxin B_1 was found in the samples. These data were interpreted to mean that the incidence and severity of aflatoxicosis is greater than previously suspected, that poor on-farm storage of corn is a primary contributing factor, that aflatoxin formation continues during and after the milling process, and that mycotoxicoses other than aflatoxicosis may cause equal or greater problems.

Much of the justification for research and education in mycotoxicology resides in the occurrence of mycotoxicoses as an economic factor in the animal and feed industries. Despite the generally assumed widespread occurrence of mycotoxins, there have been few cases of mycotoxicoses reported in the literature. The original reports of aflatoxicosis gave brief and incomplete descriptions of the disease in turkeys (10), swine (5), cattle (6), chickens, and ducklings (1). It was not until 1970 (11) that the first documented field case of aflatoxicosis in chickens was reported that satisfied Koch's postulates as they apply to mycotoxicoses. Since then, several other field cases of aflatoxicosis have been reported that satisfied Koch's postulates and emphasized certain aspects of the disease, such as increased susceptibility to bruising (12), poor carcass pigmentation (13), high mortality (2), and interaction with vitamin deficiencies (3).

Another important aspect of mycotoxins is the concentration at which they occur under field conditions. Shotwell et al. (8), in a thorough and much-needed survey, assayed 1,311 corn samples from commercial markets in the crop years 1964 and 1965 and found minimal incidence and low concentrations. This was confirmed in later study that found 6 of 283 samples to contain 12 to 25 μg/kg (9). A survey of freshly harvested corn in the southeastern United States revealed a higher incidence (49%) and concentrations (3 to 320 μg/kg) (4). A survey of feed samples from feeding troughs in a commercial operation in which broiler chickens exhibited clinical aflatoxicosis found an even higher incidence (91%) and levels (100 to 10,000 μg/kg) of aflatoxin (10). A single sample of corn containing 101,000 μg of aflatoxin B_1 per kg, obtained during clinical aflatoxicosis in laying hens, appears to be the highest reported level of naturally occurring aflatoxin (2).

The purpose of this report was to provide additional documentation of aflatoxicosis in farm animals. The results indicated a high incidence and high concentration of aflatoxin in animal feed.

MATERIALS AND METHODS

Criteria and sources of laboratory submissions. The field submissions studied in this investigation were referred to the diagnostic laboratory (Rollins Animal Disease Diagnostic Laboratory, Animal Health Division, N.C. Department of Agriculture) by private veterinarians, county farm agents, and, in a few instances, individual farmers. Each sample was submitted with the request that the submission

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be evaluated for the possibility of aflatoxicosis. This request was usually based on illness, mortality, or unthriftiness in animals that could not be diagnosed and treated successfully as infectious diseases; on signs of hepatotoxicity; or on the occurrence of visibly moldy corn or feed. In each instance a diagnostic veterinarian from the state laboratory attempted to obtain an accurate clinical history, material for histopathological examination, and samples of corn and feed for aflatoxin analysis. For the purposes of this investigation, a diagnosis of aflatoxicosis was made when aflatoxin was found in the feed and when bile ductule proliferation in the liver had occurred. In instances when necropsy material for histopathological analysis was not available, a diagnosis of aflatoxicosis was made on the basis of the overall clinical evaluation and the occurrence of aflatoxin in the feed.

Aflatoxin assays. The suspect feed and corn samples were analyzed for aflatoxin by the chromatographic standard of aflatoxins B_1, B_2, G_1 and G_2 was obtained from the Southern Regional Research Laboratory, United States Department of Agriculture-Agricultural Research Station, New Orleans, La.

RESULTS

The total suspected submissions to the laboratory and those confirmed as aflatoxicosis over a 22-month period are presented in Fig. 1. Of 278 submissions studied, 94 were confirmed as aflatoxicosis, for an overall incidence of 25.1%. However, there was an annual variation in incidence. The period from September 1972 through May 1973 had an incidence of 19%, whereas the corresponding period from September 1973 through May 1974 had an incidence of 34%, although the total submissions for the two periods were approximately equal. Both the total suspected submissions and confirmed cases were at a maximum during the winter and at a minimum during the summer. The increase in cases occurred about the time the corn crop was harvested.

The animals involved in this survey were swine, cattle, and poultry, which accounted for 88, 7, and 5%, respectively, of the confirmed cases. There were no consistent clinical symptoms. However, gross liver damage, hemorrhage or edema of various organs, elevated body temperature, mortality (reaching as high as 40%), anorexia, and unthriftiness were commonly observed. Less commonly observed were enteritis and respiratory symptoms ranging from coughing to pneumonia. Histopathological evaluation usually revealed mild to severe centrilobular hemorrhage or necrosis, portal cirrhosis, and nodular hyperplasia of the liver. Toxic nephritis was a frequent sequence to liver damage. Peripheral leukocytosis was frequently observed.

The mean value for aflatoxin in the finished feed being consumed by the animals at the time the samples were submitted was 3,890 μg/kg and the mean value for aflatoxin in the corn ingredient of the feed was 5,180 μg/kg. The range of aflatoxin concentrations was 60 to 15,000 μg/kg. Only aflatoxin B_1 was ever found in any of the samples. That the concentration of aflatoxin in corn was higher than in the feed suggested that the corn was the primary source of aflatoxin. In all confirmed cases, the corn had been grown and stored on the farm where it was fed. Table 1 gives the geographical distribution of corn growing, hog rearing, and aflatoxicosis in North Carolina. It is apparent that there is a good correlation between them and that all three are concentrated in the eastern districts of North Carolina, with 72% of the hogs, 79% of the corn, and 94% of the confirmed cases of aflatoxicosis occurring there.

DISCUSSION

There appears to be an annual variation in the incidence of aflatoxicosis in North Carolina,

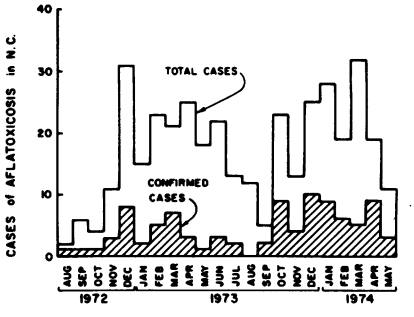

FIG. 1. *Temporal distribution of suspected and confirmed cases of aflatoxicosis in North Carolina.*

TABLE 1. *Geographical distribution of aflatoxicosis, corn culture, and swine husbandry*

North Carolina district	Aflatoxicosis[a] (no. of cases)	Corn harvested[b] (bushels × 10^6)	Hogs marketed[b] (no. × 10^4)
Northwestern	0	2.5	2.0
North central	2	4.5	12.5
Northeastern	21	22.7	39.9
Western	0	2.0	2.9
Central	0	4.3	11.9
Eastern	44	29.6	44.1
South central	3	2.5	9.4
Southeastern	24	21.2	55.2

[a] These are the cases that occurred during the time period in Fig. 1.

[b] These values were compiled by the North Carolina Department of Agriculture for the year of 1972.

because 19% of the submissions were confirmed during the first year of the survey, whereas the incidence during the second year was almost doubled, to 34%. There are two obvious explanations. Either the people submitting the cases became more discerning, or the conditions permitting aflatoxin production fluctuate on an annual basis. We suspect the latter because the incidence of confirmed cases increased when the corn crop was harvested.

An unusual observation of this survey was that all cases of aflatoxicosis occurred on farms that raised and stored their own corn. The similar geographical distribution of corn culture, swine husbandry, and incidence of aflatoxicosis supports this fact. These observations suggest that the corn had a high aflatoxin content, or at least was well seeded with *Aspergillus flavus* when it was harvested and that the corn was harvested and stored improperly. A high incidence and concentration of aflatoxin in freshly harvested corn was reported (4) in the adjacent state of South Carolina. Annual variations in aflatoxin and *A. flavus* content of corn could explain annual variations in the incidence of aflatoxicosis, but it does not explain why the cases increased to a peak during the winter and decreased to a minimum in the summer. A logical explanation for the slow increase in the fall and winter would be improper storage. The slow decrease in the spring and summer could be accounted for by assuming that by this time, the corn that will spoil has already spoiled and been discovered.

The level of aflatoxin found in the finished feed was about 75% of the level found in the corn. Since the finished feeds contained 50 to 60% corn, it is obvious that aflatoxin is coming from another ingradient or it is being formed during and after the milling process. An earlier survey (10) in North Carolina and the present study found negligible incidence and levels of aflatoxin in other ingradients, hence it seems that aflatoxin formation continues during and after feed milling.

The inconsistency between the exceptionally high concentration of aflatoxin in corn found in this survey and the low concentrations reported in other surveys of market corn (4, 8, 9) can be explained partially on the basis that our sampling was biased. Only corn that was associated with suspected aflatoxicosis was assayed in the present survey, and thus represents only a very minor portion of the total corn crop in North Carolina. Another factor that partially explains the inconsistency is that market corn usually enters commercial channels shortly after harvest where it is blended and stored under conditions that usually permit only comparatively modest formation of aflatoxin.

Yet another aspect of this survey is the lack of consistent symptoms that permit a confirmed clinical diagnosis of aflatoxicosis. This places the diagnostic veterinarian in a difficult position in which he is forced to rely on analytical and epidemiological approaches for a definitive diagnosis. Nevertheless, with adequate herd history, signs, and information about the feed, a diagnostician can reasonably include aflatoxicosis in the list of entities for differential diagnosis. The lack of consistent symptoms also poses the question of etiology of submissions that were not confirmed as aflatoxicosis. There was equal prior reason for thinking all submissions were aflatoxicosis. If it can be assumed that only half of the total submissions were truly mycotoxicoses, then mycotoxins other than aflatoxin were causing as many cases as aflatoxin in this survey.

It seems obvious that the incidence and severity of aflatoxicosis is greater than previously suspected. The magnitude of the problem will not be determined until on-the-farm surveys are conducted because the total environment of the animals must be evaluated. Once this is accomplished, rational control procedures may be developed.

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