

Altered Feathering of Chicks Caused by T-2 Toxin

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ABSTRACT Dietary T-2 toxin (0, 1, 2, 4, 8, and 16 μg./g.) was fed to 4 groups of 10 chickens at each treatment level from hatching until 3 weeks of age. Growth inhibitory levels (4, 8, and 16 μg./g.) caused abnormal feathering which appeared dose related. The chickens were sparsely covered with short feathers protruding at odd angles in comparison to controls. There were few feathers on the base of the neck, on the anterior dorsal surface of the wing, and on the side and back adjacent to the tail. Feather tips frequently were constricted and bent downward while the quill could have a reverse curve. A literature survey suggests that T-2 toxin may cause this effect through a nutritional imbalance.

INTRODUCTION

T-2 TOXIN is a mycotoxin produced by several species of Fusarium (Burmeister et al., 1972). It has been identified as 4, 15-diacetoxy-8-(3-methylbutyryloxy)-12,13-epoxy-D9-tricothecen-3-ol (Bamburg et al., 1968) and like most 12,13-epoxy-D9-tricothecen compounds it is a potent irritant and inflammatory agent (Marasas et al., 1969). In chickens it causes severe oral lesions at dietary concentrations as low as 1 μg./g. of feed (Wyatt et al., 1972b), and it has been implicated in a field outbreak of apparent fusariotoxicosis which was characterized by lesions on the feet, shanks, and heads as well as the mouth (Wyatt et al., 1972a). It also causes unique disturbances of the nervous system (Wyatt et al., 1973a) and other systemic effects which may not be directly related to its inflammatory properties (Wyatt et al., 1973b). During these studies we have consistently observed abnormal feathering in young chickens given dietary T-2 toxin. The purpose of the present communication is to provide a brief description of the abnormal feathering and to comment on some of its implications.

MATERIALS AND METHODS

T-2 toxin was produced by growing F. tricinctum NRRL 3299 on white corn grits. It was extracted and purified by the method of Burmeister (1971) to give a crystalline product melting at 150 to 152°C. One-day old male chickens of a commercial broiler strain were housed in electrically heated batteries where feed and water were available

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in the various tissues shows that in each case there was more endogenous than exogenous cholesterol (Table 6); however, these differences were not of significant magnitude to be statistically different in plasma. In view of the very rudimentary lymphatic system in the chicken, large quantities of dietary cholesterol are transported in both the free and esterified form. Since three-fourths of the cholesterol found in the aortic tissue was of endogenous origin, it is suggested that dietary cholesterol, in all probability, plays a rather small role in atherosclerosis in the chicken and that perhaps acetate or energy metabolism should be investigated as a major factor in this disease.

The portion of cholesterol and cholesterol esters in all tissues collectively, according to their source of origin is given in Table 7. It should be noted that approximately one-half of the free cholesterol in the tissues is of endogenous origin while almost three-fourths of the cholesterol esters are obtained from dietary sources. One-half of the plasma cholesterol, 64 percent esterified, is of endogenous origin. The liver data reveal 72 percent cholesterol occurs in the free form and 64 percent is of endogenous origin. Aortic tissue follows a similar pattern with 72 percent free cholesterol and three-fourths of this being endogenous.

It is possible that the results observed in Table 2 (free cholesterol) and that observed in Table 4 (total cholesterol) were markedly influenced by the fact that more exogenous cholesterol appears in the esterified form than in the free form. This might explain the observed deviations between the data presented in Tables 2 and 7.

REFERENCES


Pick, R., L. N. Katz, D. Century and P. J. Johnson, 1963. Group isolation and atherosclerosis in choles-
ad libitum under constant lighting. The feed was a commercial broiler-starter diet free of all medications. T-2 toxicosis was induced by incorporating into small portions of the diet known amounts of crystalline T-2 toxin dissolved in 50% (v./v.) aqueous ethanol. This treated feed was dried at 100° C before being mixed thoroughly into the remainder of the feed. Four replicates of 10 birds at each dose level were fed the broiler-starter diet containing 0, 1, 2, 4, 8, and 16 μg. of T-2 toxin per g. for a period of 3 weeks after hatching.

RESULTS

The feathering abnormalities occurred only at 4, 8 and 16 μg./g., which were the growth inhibitory levels, and their severity appeared to be dose related. However, not every bird was affected to the same extent and there was no readily apparent way of quantitating the feather abnormalities. Nevertheless, T-2 toxin consistently caused feather abnormalities in 3 different commercial broiler strains. Fig. 1 shows a top view of a control bird and a bird fed T-2 toxin (8 μg./g.). The bird fed T-2 toxin is sparsely covered and short

Fig. 1. Top view of a bird fed T-2 toxin (left) and a control bird (right).

Fig. 2. View of a bird fed T-2 toxin exhibiting typical shortened tail feathers.

Fig. 3. Lateral view of common feathering patterns in a bird fed T-2 toxin.

Fig. 4. Posterior view of wing feathers of a bird fed T-2 toxin.
feathers protrude from the bird at odd angles in comparison to the control bird which is fully and smoothly feathered. Fig. 2 shows another top view of a bird fed 8 μg/g. In addition to the features seen in Fig. 1, the short tail feathers are readily apparent. Fig. 3 indicates a common feather pattern. There are few feathers on the base of the neck or on the anterior dorsal surface of the wing. Less apparent is a comparative paucity of feathers on the side and back adjacent to the tail. More detail of some of the feather abnormalities are illustrated in Fig. 4. Tips of feathers frequently are constricted and bent downward. In addition, the quill may have a curve that is the reverse of a normal feather. This is particularly apparent in feathers on the anterior surface of the wing.

DISCUSSION

Abnormal feathering can be caused by genetic factors or nutritional imbalances. The extent and rapidity of feathering in chickens is controlled by specific genes (Somes, 1970). Nutrition appears to be the most important factor affecting phenotypic expression in chickens. Donaldson et al. (1955) reported poor feather condition at market age in broilers on a diet with a high energy to protein ratio. Leong et al. (1955) also reported poor feathering in New Hampshire-White Leghorn crossbred chickens on a high energy diet that was corrected by increasing the protein level of the diet. Specific amino acid deficiencies cause abnormal feathering. Arginine deficiency caused a feathering syndrome (Sanders et al., 1950) similar to that described here. Leucine deficiency (Klain et al., 1960b) and glycine deficiency (Hegsted et al., 1941) caused twisted and ruffled feathers. A lysine deficiency coupled with excess intake of other amino acids caused feather achromatosis in Barred Plymouth Rocks (Klain et al., 1960a). Magruder et al. (1952) found that the faulty feather structure caused by a "stress" ration containing 70% soybean oil meal was not corrected by vitamin therapy. Supplee (1966) found that a diet deficient in vitamin E and selenium caused shrunkened and discolored quills of the flight feather of poult and in a few instances feathers from the tail and breast were similarly affected. A similar condition has been observed in the wing feathers of chicks deficient in pyridoxine (Daghir and Balloun, 1963; Gehle and Balloun, 1965). Waibel et al. (1969) observed that biotin supplementation of a deficient diet decreased the incidence of broken feathers. Zinc deficiency causes an altered wing feathering (Supplee et al., 1961) similar to that caused by T-2 toxin but with zinc deficiency the body feathers appear normal.

The abnormal feathering caused by T-2 toxin appears unique in the type of abnormality and in the involvement of feathers over the entire body. These characteristics should be of diagnostic value in T-2 toxicosis which like other mycotoxicoses is characterized by reduced growth rate. In addition, it can add to the extensive list of abnormalities caused by mycotoxins in chickens. Presently, one may only speculate on the mechanism by which T-2 toxin causes altered feathering, but the literature strongly suggests that the metabolism of essential nutrients is impaired. A more direct effect on the follicular tissue where the feathers are formed appears also to be an attractive hypothesis. At any rate, this feather abnormality offers a new point of attack in gaining an understanding of the complex syndrome of T-2 toxicosis.

REFERENCES


Feathering and T-2 Toxin


NEWS AND NOTES

(Continued from page 1030)

HUBBARD MILLING NOTES

Dr. Richard C. Eaton has been appointed Vice President and Manager of the Feed Division of the Hubbard Milling Company, Mankato, Minnesota. He replaces Paul J. Frederick who will assume the position of President and Chief Executive Officer of Springfield Milling Corporation, a subsidiary of Hubbard Milling. For the past 2-1/2 years, Eaton has been President of a Monsanto subsidiary, Farmers Hybrid Companies, Inc., Hampton, Iowa. Prior to 1972, Eaton was associated with Ralston Purina Company.

CONRAD NOTES

Dr. Robert J. Mackin has joined Conrad’s Poultry Farm and Hatchery, Inc., Goodells, Michigan, as Director of Technical Service and Marketing. Prior to joining Concord’s, a franchise distributor of Babcock B-300, Mackin was with Babcock Poultry Farm in
ABSTRACT Laying hens consume more water on days when an egg is laid, than on non-laying days; the daily water intake is more than double—from 115 g. to 255 g. and the number of drinks taken increases proportionately. The weight of water in the egg produced is only 32 g. The requirement for this excess of water implies a considerable metabolic stress associated with egg formation. Studies have been carried out to determine why this water is consumed, and its metabolic fate.

Water ingestion started to increase about 12 h. before oviposition; it rose steadily until about 2 h. before lay, then fell sharply. Urine minute volume showed similar fluctuations. At the time when the urine output fell just before lay, plasma osmolarity was higher than usual; this might be predicted from the drop in water consumption. Alterations in the water content of the oviduct were not sufficient to explain the increased fluid intake, and total body weight remained constant in spite of the consumption of the additional 140 g. water. These findings suggest that the water has a metabolic role, as it is not retained as a net fluid surplus.

INTRODUCTION

THE formation of an egg involves the formation and transportation of considerable quantities of protein and minerals across the wall of the oviduct. In part, at least, these materials are derived from the increase in food consumption during this period (Morris and Taylor, 1967). The increased demands for raw materials require additional measures for transportation and dictate that fluid ingestion should also be increased.

Jull (1949) commented on the fact that during the first year of production a laying flock of hens consumed over 4 kg. additional water for each dozen eggs produced. Later, Lifschitz et al. (1967) reported that White Leghorn hens ingested almost twice as much water per day as cockerels. They also reported that there was a sharp reduction in fluid intake immediately before oviposition; drinking activity rose again subsequently.

These findings were confirmed by Anderson and Hill (1967), who also pointed out that in pre-lay and laying hens food and water intake were linearly related. When the supply of food was restricted, the pattern of water intake was not correspondingly altered (the converse situation was also true); it is clear that under these conditions, food and water are independent of one another.

Chapman and Black (1967), using radioisotope techniques, demonstrated that laying hens had a higher water turnover than cockerels, although this did not appear to be related to oviposition. This observation was extended by Chapman and Mihai (1972), who reported that in non-laying hens the rate of water turnover was higher than in cockerels, but that during lay the rate of turnover increased further and was associated with a rise in the size of the total body water pool from 54 to 62% of body weight. Additional evidence for the importance of water during development of the egg was presented by Ballard and Bieller (1969) and Wood-Gush and Horne (1970) who showed that in Leghorn hens both feed and water intake rose before oviposition, but fell immediately afterward.

It seems probable that important changes in water balance occur in the laying hen, and that some of these at least can be related to oviposition. The experiments presented