Subacute Toxic Effects of Dietary T-2 Toxin in Young Mallard Ducks

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ABSTRACT

Young Mallard ducks (*Anas platyrhynchos*) were fed diets containing purified T-2 toxin at levels of 20 or 30 ppm for two or three weeks. Ingestion of T-2 toxin was associated with reduced weight gain and delayed development of adult plumage. Affected ducks developed caseonecrotic plaques throughout the upper alimentary tract, especially in oropharynx and ventriculus. Several ducks also developed severe ulcerative, proliferative esophagitis and proventriculitis. Generalized atrophy of all lymphoid tissues consistently occurred. The manifestations of T-2 mycotoxicosis in Mallard ducks were mostly attributable to irritant toxicity to the alimentary mucosa. The T-2 toxin caused neither hematopoietic suppression nor a hemorrhagic syndrome in ducks. These alimentary lesions of T-2 mycotoxicosis in ducks do not resemble diseases of native waterfowl presently being recognized in routine surveillance of waterfowl mortality in Saskatchewan.

Key Words: Mycotoxins, trichothecenes, T-2 toxin, *Fusarium*, *Anas platyrhynchos*.

RÉSUMÉ

Cette expérience consistait à donner à des jeunes canards malards, durant deux ou trois semaines, une diète qui contenait 20 ou 30 ppm de toxine T-2 purifiée. L'ingestion de cette toxine s'accompagna d'une baisse du gain de poids et d'un retard du développement du plumage adulte. Les canards affectés développèrent des plaques caséeuses et nécrotiques, tout le long du tube digestif supérieur, particulièrement dans l'oropharynx et le gésier. Plusieurs développèrent en plus une inflammation ulcéreuse et proliférative de l'oesophage et du proventricule. Une atrophie de tous les tissus lymphoïdes se produisit régulièrement. Les manifestations de la mycotoxicose provoquée par la toxine T-2, chez ces canards, se révélèrent surtout imputables à son action irritante sur la muqueuse du tube digestif. Cette toxine ne causa toutefois pas d'inhibition hémopoïétique ou de syndrome hémorragique. Les lésions du tube digestif des canards attribuables à la toxine T-2, ne ressemblent pas à celles des maladies de la sauvagine qu'on identifie actuellement, à l'occasion d'une surveillance routinière de la mortalité de la sauvagine, en Saskatchewan.

Mots clés: mycotoxines, trichothécènes, toxine T-2, *Fusarium*, *Anas platyrhynchos*.

INTRODUCTION

Several fungi of various genera, particularly *Fusarium*, *Stachybotrys* and *Myrothecium*, may produce mycotoxins on cereal plants or grain under cold moist conditions (1, 2). The most important of these mycotoxins are the estrogenic zearalenone group and the trichothecene group (1-3). Although *Fusarium* contamination of cereals is frequent (4), natural occurrence of trichothecenes, mainly T-2 toxin and deoxynivalenol (vomitoxin) has been infrequently detected in animal feeds (3-5, 6). Some reported outbreaks of fusariotoxicosis in poultry in Canada (7, 8), U.S.A. (3, 9) and Europe (10) have been attributed to T-2 toxin produced by *Fusarium* spp., particularly in grain left in the field over winter.

Although T-2 toxin is clearly capable of causing mycotoxicoses in livestock, the disease syndromes in which it has been implicated are poorly characterized. There is strong evidence that it causes inflammatory lesions in the upper alimentary tract of chickens (11-15), turkeys (16) and geese (7, 17). It has also been implicated as a cause of hemorrhagic syndromes (1, 18) and pancytopenic diseases (1, 10) of livestock but neither of these mycotoxic diseases has been reproduced in experimental studies in poultry (11-13, 14, 16), cattle (19) or swine (19, 20) fed purified T-2 toxin in the feed. In

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limited surveys of mycotoxins in feed, trichothecenes have infrequently been detected (3, 5, 6). However, the extent of natural occurrence of T-2 toxin is largely unknown, in part due to the lack of suitable routine analytical methods for detecting it and other trichothecenes in moldy feed (3).

In the prairie region of Canada, various waterfowl feed in cereal stubble fields during the spring migration. Birds feed on grain which was spilled in the field during the fall harvest, and could potentially be exposed to *Fusarium*-infected, trichothecene-contaminated grain. Many livestock species, including chickens (11-14), turkeys (16), geese (7) and pigs (19, 20) are reluctant to consume feed contaminated with T-2 toxin, but will sometimes consume enough to develop fusariotoxicosis. We considered that routine surveillance of naturally occurring diseases of native waterfowl might give some indication of the natural prevalence of T-2 toxin in overwintered grain. Accordingly, we conducted the present study to establish the effects of feed-borne T-2 toxin in young Mallard ducks, so that signs of T-2 toxicosis in native waterfowl might be recognized.

MATERIALS AND METHODS

Twenty young Mallard ducklings (*Anas platyrhynchos*) were acquired as hatchlings (from Dr. J.L. Shapiro, Avian Behaviour Laboratory, University of Manitoba, Winnipeg, Manitoba, Canada). These ducks were originally derived from the native population but had been bred in captivity and hatched in an incubator. They were reared together in a brooder unit on coccidiostat-free duck and goose starter (Federated Cooperative, Saskatoon, Saskatchewan, Canada) until approximately 400-500 g. At this stage they were placed in weight-matched pairs (one male, one female) in wire battery cages on open mesh floors and given pelleted ration and water *ad libitum*.

The experimental rations were prepared by mixing appropriate quantities of a stock solution of crystalline T-2 toxin (Makor Chemicals, Jerusalem, Israel) in absolute ethanol (10 mg/mL) in 700 mL of tapwater, to which 1000 g of ground starter ration were added. The resulting wet mash was extruded into pellets that were air dried for 24 hours and then frozen at -20°C. The control diet was similarly prepared using 3 mL of ethanol per 700 mL water and 1000 g of dry feed. Diets were thawed and supplied fresh each day.

The experimental diets were first introduced when the ducks were 780 ± 200 g. Groups were randomly selected according to the design in Table I. Group A were controls, group B were on 20 ppm and group C were on 30 ppm of T-2 toxin.

Ducks were observed each day when fed and were physically examined and weighed twice weekly. At the beginning of the experiment, three ducks (two in group B, one in group C) became lame, inappetant and lethargic. They were killed and were found to have fibrinous osteomyelitis, pericarditis and necrotizing hepatosplenitis from which *Staphylococcus aureus* was isolated. These three ducks were replaced on the experiment on day 1 with similarly reared healthy ducks and all ducks were prophylactically treated with intramuscular injections of trimethoprim/sulfadiazine solution (Trivetrin, Burroughs Wellcome, Kirkland, Quebec, Canada) at 0.05 mL per duck twice daily on days 2, 3 and 4. One duck in group C (30 ppm) became lame during the experiment but was physically bright and eating well. This duck and all others displayed no evidence of staphylococcal osteomyelitis or septicemia at subsequent necropsy examination.

At termination, according to the schedule in Table I, each duck was weighed and blood was drawn from the brachial vein. Hemoglobin concentration, microhematocrit, blood smears stained with Wright's-Giemsa were obtained and blood was collected in EDTA antocoagulant. Serum concentrations of protein and cholesterol and serum activities of glutamate-pyruvate transaminase (GPT), glutamate-oxaloacetate transaminase (GOT) and alkaline phosphatase (AP) were determined on a multianalyser (ABA-100, Abbott Laboratories, Dallas, Texas). After collection of blood, the ducks were killed with intravenous sodium pentobarbital and subjected to complete necropsy examination. Weights of liver, spleen, thymus and bursa were obtained. Samples of these tissues and of bone marrow, myocardium, lung, kidney, gonad, adrenal, thyroid, pancreas, brain, oral mucosa (3 areas), esophagus (3 levels), proventriculus, ventriculus, duodenum, ileum, ceca and colon were fixed in neutral buffered formalin for light microscopic examination. Smears of bone marrow stained with Wright's-Giemsa were also examined. Tissues, other than those above, with macroscopic abnormalities were also examined by light microscopy.

TABLE I. Experimental Design for Examination of the Subacute Toxic Effects of Different Levels of Dietary T-2 Toxin in Young Mallard Ducks

	Group A	Group B	Group C
Level of T-2 toxin (ppm)	0	20	30
For two weeks	4	4	4
For three weeks	4	4	—

Groups contained four ducks (two male, two female) examined at each time

The data obtained for each time point were analysed by one-way analysis of variance. Means of groups on different levels of toxin were compared using Student-Neuman-Keuls' multiple range tests using a commercially available computer program (21). Means different from one another at $p \leq 0.05$ were considered significant.

RESULTS

Ducks fed T-2 toxin in the diet were reluctant to eat their feed and frequently spilled it. Some ducks

TABLE II. Growth Rates of Young Mallard Ducks Fed Dietary T-2 Toxin at Levels of 20 and 30 ppm for up to Three Weeks

Group	Level of T-2 toxin (ppm)	Day 0	Day 7	Day 14	Day 21
A	0	840 ± 90	940 ± 80[a]	1030 ± 70[a]	—
B	20	810 ± 30	710 ± 10[b]	510 ± 40[b]	—
C	30	860 ± 40	700 ± 60[b]	720 ± 100[b]	—
A	0	730 ± 80[a]	860 ± 80[a]	910 ± 30[a]	1012 ± 60[a]
B	20	860 ± 30[b]	780 ± 20[b]	710 ± 40[b]	720 ± 60[b]

Groups for day 14 and day 21 termination grouped and analysed separately
[a,b]Weights expressed as group means ± SEM (N = 4, except for Group C on day 14 where N = 3). Means compared at each time point and followed by different letters are significantly different at $p \leq 0.05$

mixed their feed with large amounts of their drinking water and turned leftover feed into a wet slurry. By comparison, ducks on the control diets ate their feed readily with little spillage or spoilage. Ingestion of T-2 toxin was associated with progressive weight loss during the experiment (Table II) whereas controls gained weight at the expected rates.

Within a few days on the diets, ducks fed T-2 toxin appeared unthrifty with ruffled feathers and had reduced subcutaneous tissue hydration. They progressively became emaciated, with retarded development of adult plumage. Some ducks had bilateral paresis or drooping of the wings. By day 7, several ducks on T-2 toxin displayed small, yellow plaque-like inflammatory lesions in the oral cavity, especially on the posterolateral aspects of the tongue. Less frequently, similar plaques were also observed on the palate. Some ducks on the toxic diets did not exhibit oral lesions but those that did also had mild symmetrical perioral dermatitis with hyperkeratosis at the commissures of the mouth (Fig. 1).

Of the 12 ducks fed toxic diets, one fed 30 ppm died on day 8 and another from this group died on day 14. Another duck fed 20 ppm died on day 13. Death in each of these three ducks was preceded by constant decline of body weight during the experiment, and all three were markedly dehydrated at the time of death.

At necropsy examination, ducks fed T-2 toxin were in poor condition with very little subcutaneous or abdominal body fat. The severity of this effect was not dose-dependent as two of the four ducks on 30 ppm were bigger than all ducks on 20 ppm after 14 days (Table II). The T-2 toxin produced no significant specific changes in hepatic or splenic weights except for a reduction in relative splenic weight at 21 days (Table III). However, there was significant and marked atrophy of thymus and bursa of Fabricius in both treated groups (Fig. 2). The thymus was reduced to approximately 30% of control values, and the bursa was approximately 50% of control values, when corrected for weight reductions (Table III).

Histologically, changes in the thymus were due to an extreme to total depletion of all cortical thymocytes with collapse of the stroma

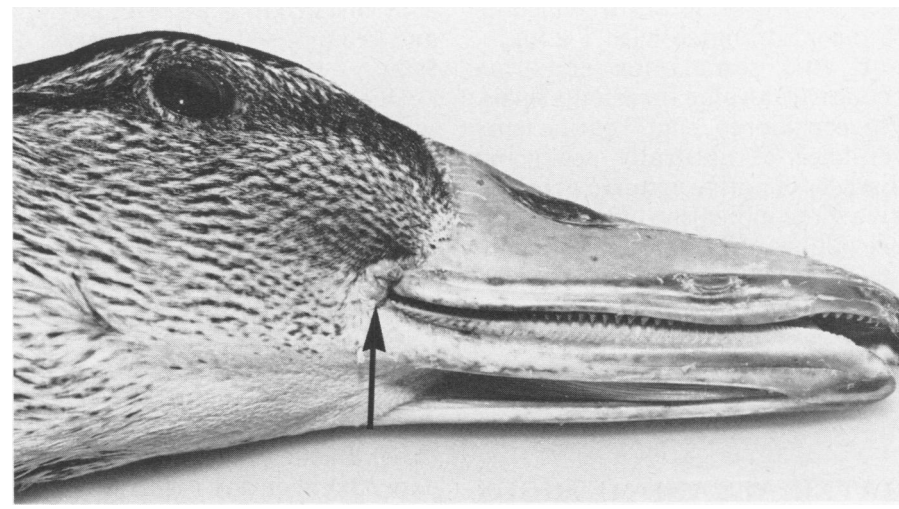

Fig. 1. Typical mild dermatitis and hyperkeratosis at the commissure of the mouth (arrow) of a duck fed dietary T-2 toxin (30 ppm) for 14 days.

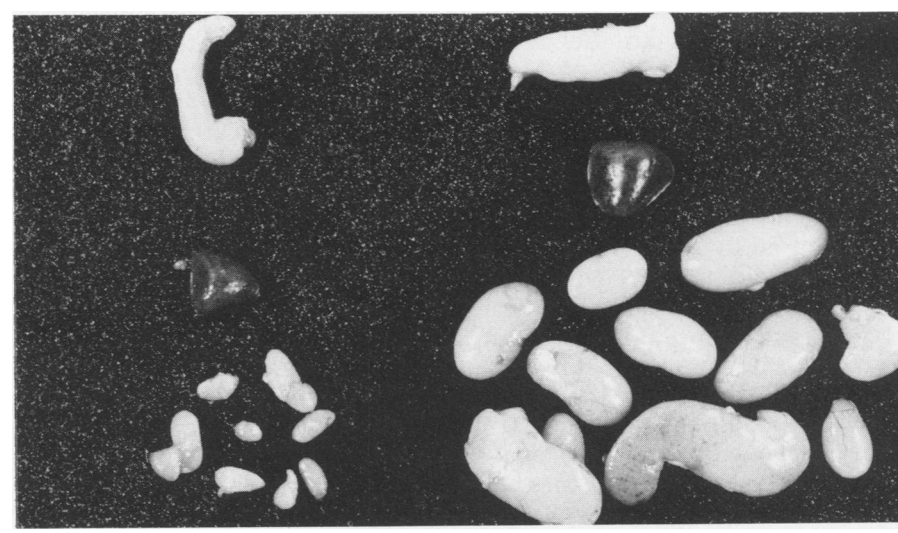

Fig. 2. Marked atrophy of bursa (top), spleen (middle) and thymic lobes (below) of a duck fed T-2 toxin (20 ppm) for 21 days. Normal tissues from a control duck are shown on the right.

TABLE III. Body and Organ Weights of Young Mallard Ducks Fed Dietary T-2 Toxin for Two or Three Weeks

Variable	Day	Level of Dietary T-2 Toxin 0 ppm	20 ppm	30 ppm	P
Body weight	14	1025 ± 71[a]	514 ± 44[b]	725 ± 100[b]	0.002
(body wt) g	21	1012 ± 59	721 ± 59	—	0.01
Hepatic weight g	14	29.3 ± 2.3[a]	15.1 ± 1.9[b]	24.6 ± 6.5[ab]	0.05
	21	30.6 ± 1.4	26.4 ± 3.8	—	0.34
Relative hepatic weight	14	2.93 ± 0.38	2.98 ± 0.32	3.30 ± 0.47	0.79
(% of body wt)	21	3.04 ± 0.16	3.60 ± 0.38	—	0.22
Splenic weight g	14	0.66 ± 0.07[a]	0.27 ± 0.05[b]	0.40 ± 0.08[b]	0.007
	21	0.61 ± 0.07	0.26 ± 0.02	—	0.003
Relative splenic weight	14	0.06 ± 0.002	0.05 ± 0.01	0.06 ± 0.01	0.66
(% of body wt)	21	0.06 ± 0.004	0.04 ± 0.004	—	0.006
Thymic weight g	14	5.12 ± 1.34[a]	0.19 ± 0.03[b]	0.36 ± 0.08[b]	0.005
	21	6.45 ± 0.89	0.45 ± 0.24	—	0.001
Relative thymic weight	14	0.49 ± 0.13[a]	0.04 ± 0.003[b]	0.05 ± 0.01[b]	0.006
(% of body wt)	21	0.63 ± 0.05	0.06 ± 0.03	—	0.0001
Bursal weight g	14	1.20 ± 0.22[a]	0.35 ± 0.02[b]	0.35 ± 0.08[b]	0.0009
	21	0.93 ± 0.10	0.31 ± 0.06	—	0.002
Relative bursal weight	14	0.12 ± 0.02[a]	0.06 ± 0.01[b]	0.05 ± 0.01[b]	0.03
(% of body wt)	21	0.09 ± 0.01	0.04 ± 0.01	—	0.002

[a,b]Values are means (± SEM) of separate groups of four ducks
In any horizontal row, means followed by the same letter or by no letter do not differ at p = 0.05 by SNK multiple range tests

into a residuum of epithelial cells. Similarly, the atrophic bursae exhibited marked depletion of follicular lymphoblasts and mature lymphocytes. Lymphocytes populations in splenic white and red pulp were also depleted in all ducks fed T-2 toxin.

Ducks fed T-2 toxin displayed various inflammatory lesions of the upper alimentary tract (Table IV). In addition to the oral lesions observed clinically, there were similar focal yellow caseous necrotic plaques in the posterior pharynx, on the posterolateral aspects of the tongue, and on the pharyngeal aspect of the larynx (epiglottis). Such oral lesions were observed only in ducks fed 30 ppm for 14 days, or 20 ppm for 21 days although not in all ducks in these two groups (Table IV). Some ducks in both groups on T-2 toxin had extensive necrotizing esophagitis (Fig. 3) of the caudal third of the esophagus. This lesion varied in severity from mild hyperkeratosis and catarrhal inflammation (Fig. 3) through to severe fibrinous exudation overlying extensive mucosal ulceration. In this latter lesion there was sometimes a large lumenal cast of exudate containing feed particles and some feathers (Fig. 5). In general, severe low esophageal exudative lesions were observed in ducks without oral lesions and were associated with a more precipitous drop in weight and physical condition. Histologically, the necrotizing lesions of the oral cavity and esophagus were similar, being characterized by focal mucosal ulceration or erosion with laminated deposits of fibrin, mucus, leukocytes especially heterophils, and necrotic debris (Fig. 6). An additional finding in ducks fed 20 ppm for 21 days was moderate hyperplasia of submucosal mucous glands in the tongue, pharynx and esophagus.

In the proventriculus, necrotizing or erosive lesions were infrequent, being seen only in the two ducks that died while on the 30 ppm level (Table IV). One of these (No. 888) had recent acute hemorrhage into the proventriculus. Generally, mild changes due to excessive mucus secretion with few leukocytes were observed in treated ducks (Fig. 3). In the ventriculus, consistent changes were an irregular thickening of the lining, usually associated with focal deep ulceration (Figs. 3 and 4). Histologically, there was focal irregular but marked hyperplasia of the mucosa. Few significant alterations were observed in the rest of the lower alimentary tract. Mild depletion of lymphocytes in the lamina propria and submucosa of the small intestine and the cecal tonsils was evident in some ducks on T-2 toxin.

Few significant effects of dietary T-2 toxin were observed in the hematopoietic system. Treated ducks exhibited only mild reductions of hemoglobin and packed

TABLE IV. Summary of the Distribution and Severity of Ulcerative and Exudative Lesions in the Upper Alimentary Tracts of Mallard Ducks Fed Dietary T-2 Toxin (20 ppm or 30 ppm) for Fourteen or Twenty-One Days

Duck	Dietary level of T-2 toxin (ppm)	Duration of T-2 toxin (days)	Perioral skin	Tongue	Pharynx and epiglottis	Esophagus — upper	Esophagus — middle and lower	Esophageal impaction	Proventriculus	Ventriculus
888	30	8[a]	0[b]	1	0	1	4	1	3	4
891	30	14[a]	0	1	1	2	5	5	3	3
892	30	14	4	4	0	0	1	0	0	4
893	30	14	4	4	4	0	2	0	0	2
890	20	13[a]	0	0	0	0	4	0	0	3
895	20	14	0	0	0	3	5	0	0	3
898	20	14	0	0	0	2	5	5	0	4
900	20	14	0	0	0	0	3	0	0	3
913	20	21	3	4	5	0	2	0	2	2
915	20	21	0	0	0	1	5	5	0	2
917	20	21	2	3	0	1	1	0	1	4
918	20	21	4	4	2	0	2	0	1	4

[a]Died or killed *in extremis*
[b]Lesion severity ratings: 0 — No significant change; 1 — Microscopic lesions only; 2 — Mild gross lesions; 3 — Moderate gross lesions; 4 — Severe lesions; 5 — Extreme, extensive lesions

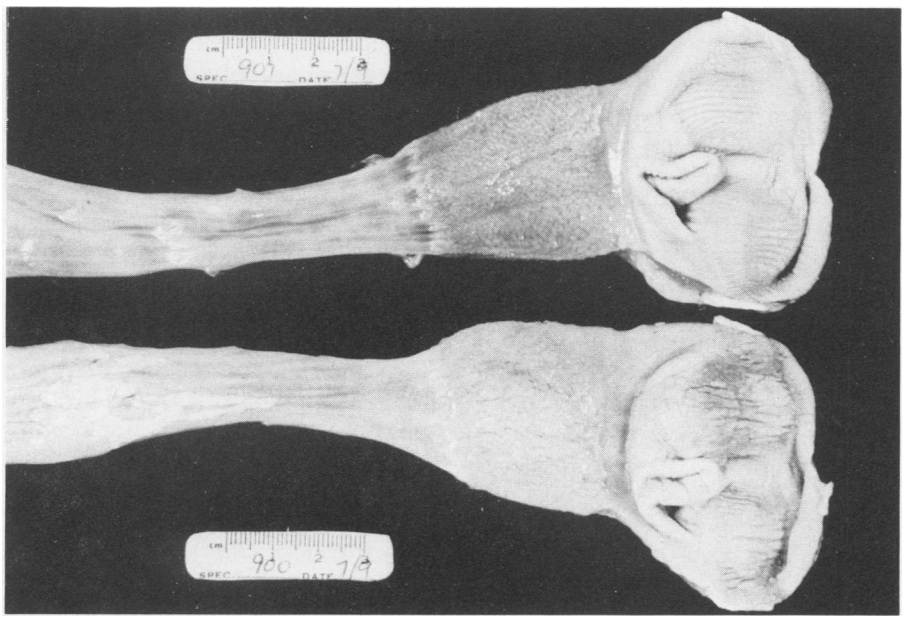

Fig. 3. Mild fibrinous esophagitis, catarrhal proventriculitis and ulcerative ventriculitis of a duck fed dietary T-2 toxin (20 ppm) for 21 days (below). A normal digestive tract of a control duck is shown for comparison (above.)

cell volume (Table V). Normal hematopoietic activity was evident in bone marrow sections. Numerous immature erythrocytes with slightly basophilic cytoplasm were present in peripheral blood of both control and treated ducks.

No physiologically significant alterations were detected in plasma protein, serum protein, cholesterol, GPT, GOT, and AP (Table VI). With the exception of one duck with acute proventricular hemorrhage associated with deep mucosal ulceration, no hemorrhagic tendency was evident in these ducks.

DISCUSSION

In the present study, young Mallard ducks fed relatively high levels of dietary T-2 toxin for two or three weeks developed a disease pattern characterized by retarded growth, necrotizing upper alimentary tract lesions and severe generalized lymphoid depletion. This pattern of T-2 mycotoxicosis resembled those described for other avian species fed dietary T-2 toxin, including young chickens (2,

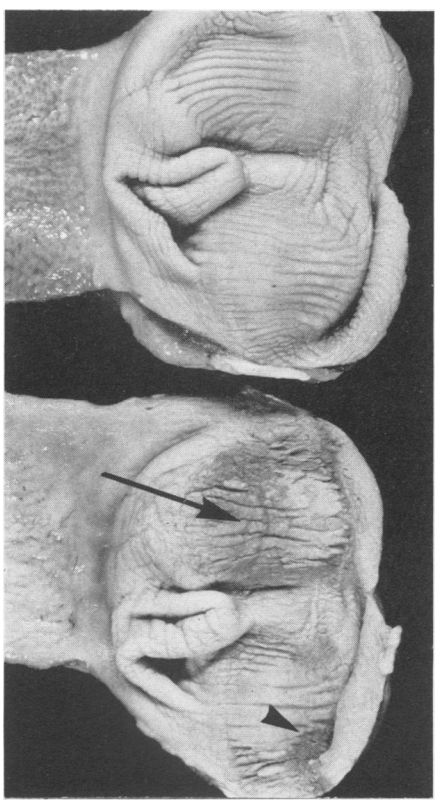

Fig. 4. Close-up view (from Fig. 3) of ventricular lesions induced by T-2 toxin. Note the thickened encrusted surface (arrow) and the focal ulcerations (arrowhead).

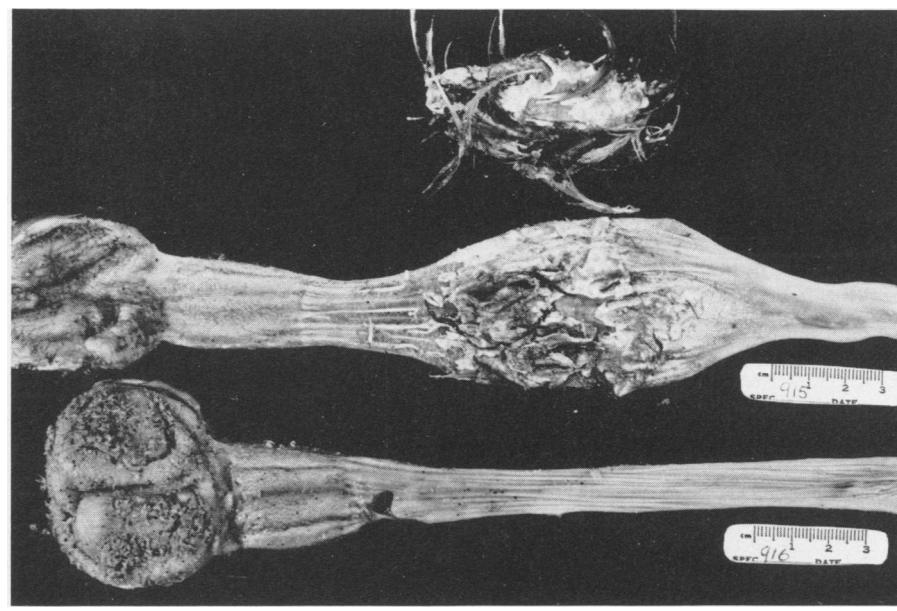

Fig. 5. Severe esophagitis with a cast of exudate, mucus, feed and feathers in the midesophageal region of a duck fed T-2 toxin (20 ppm) for 21 days. A normal control digestive track is also shown (below).

TABLE V. Hemoglobin Concentration and Packed Cell Volume of Young Mallard Ducks Fed Dietary T-2 Toxin for Two or Three Weeks

Variable	Day	Level of Dietary T-2 Toxin 0 ppm	20 ppm	30 ppm	P
Hemoglobin g/100 mL	14	13.5 ± 0.5	11.8 ± 0.7	12.5 ± 1.0	0.41
	21	12.9 ± 0.2	11.5 ± 0.2	—	0.005
Hematocrit percent	14	41.4 ± 2.0	35.2 ± 3.0	37.6 ± 3.3	0.35
	21	40.9 ± 0.7	35.6 ± 0.6	—	0.001

Values are means (± SEM) of separate groups of four ducks

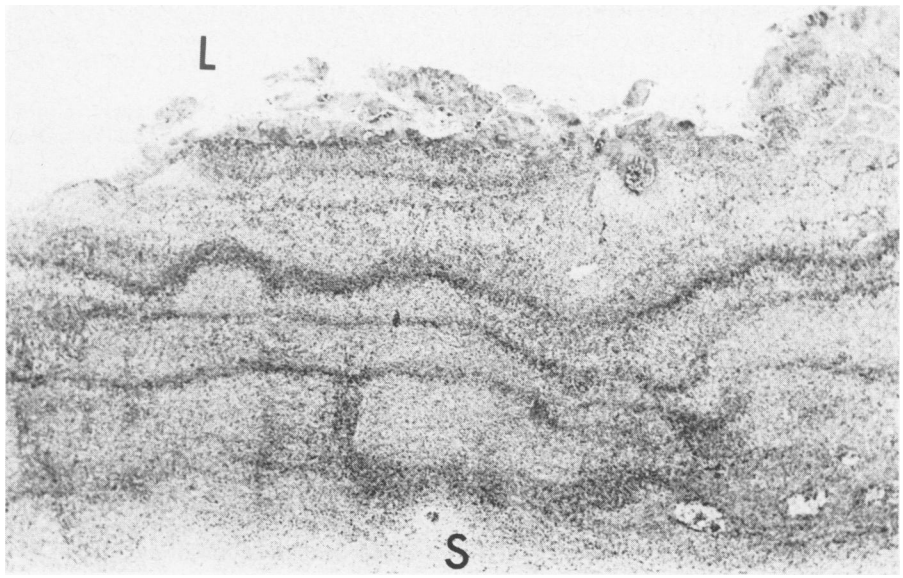

Fig. 6. Microscopic appearance of layers of fibrin and heterophils in exudate overlying ulcerated esophageal submucosa of a duck fed dietary T-2 toxin. L is the lumen; S is the submucosa. Hematoxylin and eosin. X50.

16) broiler chickens (12), laying hens (13), turkey poults (16) and geese (17), except that the lesions observed in Mallard ducks were much more severe and extensive than recognized in these other species. In experimental T-2 toxicosis in these other avian species, lower levels of T-2 toxin (up to 16 ppm) have been employed, so Mallard ducks are not necessarily more susceptible. However, due to the potent food refusal activity of T-2 toxin and similar trichothecenes (22), the domestic avian species may be reluctant to eat feed containing the levels employed in this study. The results clearly demonstrated that Mallard ducks will voluntarily ingest diets containing levels of T-2 toxin sufficient to cause a severe disease due to the irritant toxicity of T-2 toxin.

A major objective of the present study was the characterization of severe T-2 mycotoxicosis in Mallard ducks to see which patterns of toxicity were exhibited. The T-2 toxin and similar trichothecene mycotoxins have been implicated in various mycotoxicoses of livestock, including:

1) food refusal, emesis and weight loss (3, 7),
2) dermal and mucosal irritation (7, 9),
3) a poorly characterized hemorrhagic syndrome involving alimentary hemorrhage or generalized tissue hemorrhages (1, 18) and
4) hematopoietic suppression, especially anemia, thrombocytopenia and leukopenia (1, 10).

Of these syndromes, the first two have been clearly demonstrated as consistent patterns of T-2 mycotoxicosis in domestic poultry (10-16), pigs (19, 20), cattle (19) and laboratory rodents (23, 24). The latter two are largely nonreproducible with purified dietary T-2 toxin, although nonlethal gastrointestinal hemorrhage has been observed in swine (20). Hematopoietic suppression, the basis of pancytopenic mycotoxicoses such as alimentary toxic aleukia of man and livestock (1, 10) and stachybotryotoxicosis of livestock (1) has not been reproduced by dietary T-2 toxin in poultry (10-15), swine (19, 20) or cattle (19) but has been demonstrated only in mice (23). Results of the present study in Mallard ducks fed high levels of T-2 toxin indicate that this species resembles other avian and mammalian livestock in that neither a hemorrhagic syndrome, nor hematopoietic suppression occur in response to dietary T-2 toxin alone. One duck fed the 30 ppm level did die with acute hemorrhage in the upper alimentary tract but this was secondary to deep ulcerative lesions in the proventricular mucosa and thus was not attributable to either a hemostatic or a thrombocytopenic disease. No evidence of hypoplasia of bone marrow was detected in any of the ducks fed T-2 toxin. Although red cell values were lower in T-2 toxin fed ducks, this change was physiologically of little significance.

Severe depletion of lymphoid tissues, characterized by thymic, bursal and splenic atrophy consistently occurred in response to dietary T-2 toxin. Similar but less severe effects have been described in young chickens and turkey poults fed T-2 toxin (16). Lymphoid depletion in animals fed T-2 toxin

TABLE VI. Serum Biochemical Values from Young Mallard Ducks Fed Deitary T-2 Toxin for Two or Three Weeks

Variable	Day	Level of Dietary T-2 Toxin 0 ppm	20 ppm	30 ppm	P
Plasma protein g/dL	14	4.2 ± 0.2	4.4 ± 0.5	4.7 ± 0.7	0.75
	21	4.1 ± 0.2	4.3 ± 0.1	—	0.59
Serum protein g/dL	14	3.8 ± 0.1	4.1 ± 0.5	4.4 ± 0.7	0.58
	21	3.9 ± 0.2	4.1 ± 0.1	—	0.37
Cholesterol mg/dL	14	198 ± 11[a]	264 ± 16[b]	263 ± 22[b]	0.03
	21	225 ± 12	248 ± 15	—	0.29
Glutamate pyruvate transaminase activity (GPT)IU/L	14	29 ± 6	29 ± 9	138 ± 112	0.37
	21	17 ± 1	20 ± 4	—	0.51
Glutamate oxaloacetate transaminase activity (GOT)IU/L	14	20 ± 4	12 ± 2	151 ± 130	0.33
	21	24 ± 2	30 ± 4	—	0.22
Alkaline phosphatase activity (AP)IU/L	14	597 ± 19[a]	241 ± 27[b]	506 ± 139[a]	0.03
	21	622 ± 153	399 ± 32	—	0.21

[a,b]Values are means (± SEM) of separate groups of four ducks
In any horizontal row, means followed by the same letter or by no letter do not differ at p = 0.05 by SNK multiple range tests

is largely attributable to inanition effects of the unpalatable diet (16-24). However, T-2 toxin is also extremely toxic directly to germinal populations of all lymphoid tissues (23, 25) and is directly immunosuppressive (26). Presently, insufficient information is available to define the functional significance of such depletion of immune tissues.

Determinations of serum protein and cholesterol concentrations and GPT, GOT and AP activities did not indicate biologically significant hepatotoxicity of T-2 toxin in Mallard ducks. This data is in agreement with observations in chickens (13, 14) and other species in which T-2 toxin is not hepatotoxic.

Although T-2 toxin consistently caused inflammatory and ulcerative lesions in the upper alimentary tract, the distribution of the most severe lesions was variable (Table IV). Ducks fed 20 ppm for two weeks had no lesions in the oral cavity or pharynx whereas ducks fed 30 ppm for two weeks consistently had oral lesions, some of which were extensive and severe. By three weeks, three of four ducks on 20 ppm exhibited oral lesions. Of more interest was the observation of severe esophagitis in ducks with few or mild oral lesions, and mild esophageal lesions in ducks with severe oral lesions. This alternative pattern was evident in ducks fed either level of T-2 toxin for two or three weeks. Diet consumption rates were not recorded in this study so it is unknown if these patterns relate to the feeding habits of the ducks. Ducks that more readily ate the feed may have developed more severe esophageal lesions. Three of 12 ducks that died during the feeding trial all had severe esophageal lesions and mild or no oral lesions. Two of these ducks also had ulcerative proventriculitis, a lesion that was absent or very mild in all others on the diet. This is limited evidence for considering that increased toxic feed consumption was responsible for fewer oral and more severe esophageal lesions.

The characteristic lesions of dietary T-2 mycotoxicosis of Mallard ducks bore little resemblance to naturally occurring diseases recognized in native waterfowl in Saskatchewan. Considering that most migratory waterfowl have access to unharvested or spilled grain left on the ground over winter, necropsy submissions from ducks dying in spring and early summer might be an indicator of the natural levels of occurrence of T-2 toxin in a region considered climatically favourable for trichothecene production. In our experience, the alimentary lesions produced in Mallard ducks do not resemble any naturally occurring diseases so far recognized. This tentatively suggests that either there is minimal occurrence of T-2 toxin, or that the waterfowl avoid eating it. It is possible that the less specific effects of T-2 toxin, namely illthrift and lymphoid depletion could occur in ducks consuming small quantities of T-2 toxin in the absence of alimentary mucosal lesions. However, these changes are common components of many lethal diseases and thus have little value in the recognition of T-2 mycotoxicosis.

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