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Effect of Chronic V.S. Intermittent Exposure to T-2 Toxin on Reproductive Performance in Bobwhite Quail

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Abstract: Studies were conducted to determine the effect of chronic v.s. intermittent exposure to T-2 toxin on reproductive performance in adult bobwhite quail. In Experiment 1, 180 hens were orally dosed with 0% (LD_0), 20% (LD_{20}), 40% (LD_{40}) or 60% (LD_{60}) of the acute 100% lethal dose of T-2 toxin as determined in a previous experiment. One quarter of the dosage was administered each week for 3 weeks. Date of puberty was delayed 5 days as a result of the highest dose of T-2 toxin. There were no differences ($P>0.05$) in hen-day egg production or fertile hatchability of eggs as a result of intermittent exposure to T-2 toxin. Fertility and total hatchability of eggs collected from LD_{60} hens during week 2 following puberty were less ($P\leq0.05$) than from control hens. In Experiment 2, 139 hens were fed 0, 12, 16 and 20 mg T-2 toxin/kg feed for a 4 week period. Puberty was delayed 11 days among hens fed 20 mg/kg feed as compared to hens not fed T-2 toxin. Similarly, feed consumption was less ($P < 0.05$) among birds consuming any level T-2 toxin as compared to controls. No differences ($P>0.05$) in total hen-day egg production was found. However, percent fertility and total hatchability of eggs were lower ($P<0.005$) among hens receiving 20 mg T-2 toxin/kg feed as compared to control hens during the first 7 days following puberty. Results from these studies indicate that reproductive failure in wild bobwhite quail may be a consequence of mycotoxin exposure.

Key words: Bobwhite quail, T-2 toxin, reproduction

Introduction

Trichothecene mycotoxin, T-2 toxin, has long been known as an inhibitor of protein and DNA synthesis, and as a suppressor of immune cells (Rosenstein and Lafarge-Frayssinet, 1983; Mann *et al*., 1983; Holladay *et al*., 1995; Corrier and Ziprin, 1987). In addition , this toxin has been shown to have negative effects on reproduction and reproductive efficiency in a variety of laboratory and farm animals (Rousseaux and Schiefer, 1987; Weaver *et al*., 1978; Speers *et al*., 1977; Chi *et al*., 1977b; Tobias *et al*., 1992). However, little information is available on the effects of T-2 toxin in bobwhite quail. Decline of native bobwhite quail populations in the Southeastern United States has been documented since the 1960's. In Georgia alone, the decline of quail since 1966 is estimated at greater than 70% (Sisson and Stribling, 2001).

In chickens, exposure to T-2 toxin resulted in significant reductions in body weight and egg production. Speers *et al*. (1977) reported lower ($P\leq0.05$) body weights among laying hens fed 16 mg T-2 toxin/kg of feed v.s. controls (1525 g v.s. 1595 g respectively). Egg production was lower among hens fed 16 mg/kg T-2 toxin (3.46 eggs/week) v.s. those fed control diets (6.13 eggs/week). Similarly, Chi *et al*. (1977b) observed a significant ($P\leq0.05$) decrease in feed consumption, egg production, and egg shell thickness among laying hens fed 8 mg T-2 toxin/kg feed. Lower ($P\leq0.05$) fertile hatchability was observed among hens fed 2 or 8 mg T-2 toxin/kg.

Ames Plantation, (cooperator with the University of Tennessee Agricultural Experiment Station) the site of the National Championships for purity all age bird dogs, supports a bobwhite quail population that is integral to the mission of the Plantation. Over the past 25 years quail populations have been fluctuating despite management practices aimed at improving habitat for quail (Dimmick, 1992). From a peak population in 1974 (1.5 birds/acre) to a low in 1991 (0.26 birds/acre), the bobwhite population has declined nearly 83% (Dimmick, 1992). Recent analysis of soybeans left as late winter feed for quail showed the presence of T-2 toxin (Gigeous, 1999). It was hypothesized that T-2 toxin may be a contributing factor to the decreasing quail population. The objective of the following study was to determine the effect of intermittent v.s. chronic exposure to T-2 toxin on reproductive performance of adult pen-raised bobwhite quail.

Materials and Methods

Two experiments were conducted to determine the effect of intermittent (Experiment 1) v.s. chronic (Experiment 2) dietary exposure to T-2 toxin on reproductive performance in bobwhite quail. Experiments were

designed to mimic what could possibly occur in a natural situation where quail would forage from different feed plots and thus experience "intermittent" v.s. daily or "chronic" exposure to mycotoxins. Additionally, experiments were designed to limit toxin exposure only during the pre-puberal period to mimic exposure prior to the nesting period, when late winter fungal contamination of feed sources would be likely (Grizzle *et al.*, 2004). In Experiment 1, 180 bobwhite quail hens (45 hens/treatment) were randomly assigned to receive 0, 20, 40, or 60% of the acute 100% lethal dose ($LD_{100,}$ 20 mg/kg body weight [BW]) T-2 toxin (0, 12.4, 14.0, or 15.5 mg toxin/kg BW respectively) as previously determined (Grizzle *et al.*, 2004). Hens were housed in grower batteries (Petersime Company, Gettysburgh, OH; Model 515) inside a Bioshield 350 (Airo Clean Inc., Exton, PA) negative pressure biological containment unit, and provided *ad libitum* access to feed and water (Mazuri Brand Pheasant Breeder, PMI Feeds Inc., St. Louis, MO). Purified T-2 toxin (Sigma, St. Louis, MO) was dissolved in 100% ethanol, and diluted to volume with corn oil. Dosage was administered by oral gavage over a 3 week period; 1/4th dose per week (starting on day 0) so that hens received the total 0, 20, 40, or 60% LD_{100} by the end of the 3rd week. Males were maintained with hens at a rate of one male per two females, but were not gavaged with toxin, and thus not exposed. Light stimulation (16 hours/day) was initiated at the beginning of the treatment period to stimulate egg production. Data were collected on body weight change, mortality, date of puberty, 17 week hen-day egg production (HDEP), and 12 week percent fertility, fertile hatchability, and total hatchability of eggs. In Experiment 2, 139 bobwhite quail hens were assigned to 1 of 4 treatment groups; 0, 12, 16, and 20 mg T-2 toxin/kg feed. Treatments were replicated four times with 8-11 birds per replicate, for a total of 32-44 birds per treatment. T-2 toxin was dissolved into 100% ethanol, diluted to volume in corn oil, and added to commercial breeder diets (Mazuri Pheasant Breeder, PMI Feeds Inc., St. Louis, MO). Hens were housed in grower batteries inside the Bioshield 350 biocontainment unit as in Experiment 1, and were provided feed and water *ab libitum*. T-2 toxin contaminated feed was fed for a total of 28 days. Photo-stimulation, 16 hours/day, began 2 weeks (day 14) after initiation of T-2 treatment to bring hens into egg production. Males were added to the cages the day after cessation of contaminated feed treatment (2 weeks after light stimulation was initiated). Thereafter all birds were fed a non-contaminated commercial pheasant breeder feed. Data were collected on body weight change, mortality, feed consumption, date of puberty, hen-day egg production, fertility, fertile hatchability, and total hatchability of eggs for 12 weeks. Data were analyzed using the PROC FREQ and PROC MIXED procedures of SAS (1996). Means were separated by Fisher's Least Significant Difference Test. Significance was declared at $P = 0.05$.

Results

As compared to hens dosed intermittently (Experiment 1), hens consuming any level T-2 toxin in feed (Experiment 2) lost weight ($P < 0.05$) during the exposure period (Table 1). Among hens dosed once/week (Experiment 1), all birds gained weight during the 21 day period, however hens dosed with the highest amount T-2 toxin gained less weight than either controls or LD_{20} hens. In Experiment 2, all birds lost weight, and was greater among hens fed any level T-2 toxin as compared to controls (Table 1). Feed consumption declined linearly with each increase in dietary level T-2 toxin and was less among all T-2 toxin treatments as compared to controls (Table 2).

There was no mortality among hens dosed with T- 2 toxin in Experiment 1. In Experiment 2, mortality increased as dietary T-2 toxin increased. However, even at the highest level toxin, 20 mg/kg feed, only 4 birds died during the 28 day exposure period (Table 1).

Date of puberty was delayed in both experiments as a result of T-2 toxin (Table 3). In Experiment 1 date of puberty was 22 days after initiation of light stimulation for control (LD_0) and LD_{40} hens, 26 days after initiation of light stimulation for LD_{20} hens, and 27 days after initiation of light stimulation for LD_{60} hens. Likewise in Experiment 2, chronic consumption of T-2 toxin resulted in an even longer delay to puberty than Experiment 1. Date of puberty was 23, 29, 37, and 34 days after initiation of light exposure for 0, 12, 16, and 20 mg/kg feed treatments respectively.

Total experimental hen-day egg production was not affected by a pre puberal exposure to T-2 toxin in either experiment, but was numerically less for the LD_{60} hens in Experiment 1 (Table 3).

Unlike egg production, fertility and hatchability of eggs was affected by T-2 toxin treatment (Table 4). However, in both experiments, these differences were only found during the first 1-2 weeks egg production following puberty and cessation of toxin treatment when T-2 toxin would be present in the egg (Chi *et al.*, 1978a). In Experiment 1, eggs were not set during week 1 following puberty as hen-day egg production was less than 4%. During week 2, hen day egg production was > 15% and egg fertility was less among LD_{60} hens as compared to controls or LD_{20} hens. Likewise fertile hatchability and total hatchability was less among both LD_{40} and LD_{60} treated hens as compared to controls and LD_{20} hens (Table 4). In Experiment 2, 301 eggs (16.4 %) were laid during week 1 following puberty and were set for hatchability characteristics. Egg fertility and total hatchability of eggs from hens fed 20 mg/kg T-2 toxin were less than both controls and hens fed 12 mg/kg feed (Table 4). A numerical reduction in both fertility and

Table 1: Average body weights and body weight change of bobwhite quail hens intermittently or chronically treated with T-2 toxin (Experiment 1 and 2)

Treatment	Intermittent			Chronic		
	Wt. Change	% Change	Mortality	Wt. Change	% Change	Mortality
Intermittent (Chronic)	(g)	(%)	(n)	(g)	(%)	(n)
LD0 ; 0 mg/kg BW (0 mg T-2 toxin/kg)	20.88[a]	10.68[a]	0	-4.53[a]	-2.11[a]	0
LD20; 12.4 mg/kg BW (12 mg T-2 toxin/kg)	18.50[a]	8.97[a]	0	-29.90[b]	-14.01[b]	2
LD40; 14.0 mg/kg BW(16 mg T-2 toxin/kg)	18.28[ab]	8.96[ab]	0	-41.60[bc]	-19.49[bc]	3
LD60; 15.5 mg/kg BW (20 mg T-2 toxin/kg)	14.26[b]	6.87[b]	0	-47.50[c]	-22.46[c]	4

a,b,c values in the same column with different letters are significantly different (P£.05)

Table 2: Average daily feed consumption (grams of feed/bird) of bobwhite quail hens chronically treated with T-2 toxin for a 4 week period (Experiment 2)

Treatment	Week 1	Week 2	Week 3	Week 4	Total
0 mg T-2 toxin/kg	14.90[a]	15.91[a]	16.68[a,b]	22.14[a]	17.40[a]
12 mg T-2 toxin/kg	13.21[a]	11.84[b]	17.68[a]	17.20[b]	14.98[b]
16 mg T-2 toxin/kg	12.23[a]	11.29[b]	16.25[b]	15.97[b]	13.93[b]
20 mg T-2 toxin/kg	12.01[a]	10.56[b]	15.02[b]	16.93[b]	13.63[b]

a,b Values in the same column with different letters are significantly different (P£.05)

total hatchability was seen among hens fed 16 mg T-2/kg feed but was not different than control fed hens. During the second week following puberty no differences in fertility, fertile hatchability or total hatchability were observed. However as compared to controls, numerically these traits were less among hens fed either 16 or 20 mg/kg feed T-2 toxin (data not shown). Hens fed the 16 or 20 mg T-2 diets during the pre-puberal period laid eggs that were 7-10% lower in fertility (84.98% and 86.59% for 16 and 20 mg T-2 toxin diets respectively) than control hens (95.41%). The difference between 16 mg and control birds approached significance (P≤0.086). Likewise, total hatchability was numerically lower among 16 and 20 mg/kg fed birds (76.85% and 76.07% respectively) v.s. controls (83.74%).

Discussion

This study was designed to compare the effects of intermittent v.s. chronic exposure to T-2 toxin on reproductive performance in adult bobwhite quail. The intent was to mimic the type of mycotoxin exposure wild quail might experience if ingesting mycotoxin infested soybeans, a common feed grain left over winter for wild birds. If wild quail were continually ingesting mycotoxin infested soybeans, the chronic study would closely duplicate this situation; whereas if they were ingesting mycotoxin infested soybeans on an irregular basis, the intermittent study would mirror this situation.

The negative effect of T-2 toxin on body weight gain has been well documented in many poultry species. Ruff *et al.* (1992) observed a significant (P≤.05) reduction in body weight over a three week period in bobwhite and Japanese quail fed diets containing 4, 8 and 16 mg T-2/kg feed from day 1 of age. Kubena *et al.* (1995) observed a 26% decrease in weight gain in turkey poults fed 5 mg T-2 toxin/kg feed from hatch to day 21 of age as compared to controls. Chi *et al.* (1977a) observed a 43.7% decrease in weight gains of day old broiler chicks dosed with 6.5 mg T-2/kg BW during a 30 day trial. Later, Kubena *et al.* (1989) confirmed these results and reported significant (P≤0.05) reductions in body weight gain of broiler chicks fed T-2 toxin from day 1 to 3 weeks of age as compared to controls. Our data agree with these reports in that hens chronically fed T-2 toxin (Experiment 2) lost weight during the pre puberal period. Hens dosed intermittently on a weekly basis did not lose weight during the pre puberal period as a result of T-2 toxin exposure, and actually gained weight as would be expected. However, those dosed with the highest dose T-2 toxin gained the least amount, and this was significantly less than controls (Table 1). As a result of the continual exposure to T-2 toxin, onset of puberty in Experiment 2 hens was delayed an average 8 days longer than those intermittently exposed in Experiment 1. Thus late winter exposure to mycotoxin contaminated feed sources may delay nesting in wild quail.

In both experiments, overall (12 week) fertility, fertile hatchability, and total hatchability of eggs was not affected by pre puberal exposure to T-2 toxin. However, in agreement with Chi *et al.*, (1977b, 1978b) and Tobias *et al.* (1992), fertility and hatchability characteristics were impacted during those time periods when T-2 toxin would be present in egg yolk. In both experiments fertility and total hatchability of eggs was less among hens dosed with the LD_{60} or fed 20 mg/kg feed T-2 toxin as compared to controls during the first 2 weeks following puberty (Table 4). As reported by Chi *et al.* (1977b),

Table 3: Total experimental hen-day egg production (HDEP) and days to puberty after initiation of light stimulation among bobwhite quail hens intermittently or chronically treated with T-2 toxin (Experiment 1 and 2)

Treatment	Intermittent		Chronic	
	Puberty	HDEP (17 wk)	Puberty	HDEP (12 wk)
Intermittent (Chronic)	d	%	d	%
LD0 ; 0 mg/kg BW (0 mg T-2 toxin/kg)	22	67.2	23	51.9
LD20; 12.4 mg/kg BW (12 mg T-2 toxin/kg)	26	65.4	29	54.5
LD40; 14.0 mg/kg BW (16 mg T-2 toxin/kg)	22	69.5	37	53.6
LD60; 15.5 mg/kg BW (20 mg T-2 toxin/kg)	27	59.9	34	55.5

Table 4: Percent fertility (F), fertile hatchability (FH), and total hatchability (TH) of eggs laid by bobwhite quail hens exposed to T-2 toxin during week 2 (intermittent, Experiment 1) or week 1 (chronic, Experiment 2) following puberty.

	Intermittent			Chronic		
Treatment	F	FH	TH	F	FH	TH
Intermittent (Chronic)	%	%	%	%	%	%
LD0 ;0 mg/kg BW (0.0 mg T-2/kg feed)	100.00^{a}	89.47^{ab}	89.47^{ab}	100.00^{a}	82.20	82.20^{a}
LD20; 12.4 mg/kg BW (12.0 mg T-2/kg BW)	96.60^{a}	92.00^{a}	89.61^{a}	96.75^{a}	84.86	82.08^{a}
LD40; 14.0 mg/kg BW (16.0 mg T-2/kg BW)	89.84^{ab}	78.75^{b}	71.59^{b}	89.44^{ab}	86.61	77.84^{ab}
LD60; 15.5 mg/kg BW (20.0 mg T-2/kg BW)	85.71^{b}	75.64^{b}	64.86^{c}	81.23^{b}	79.19	65.82^{b}

a.b.c Values in the same column with different letters are significantly different (P£.05)

consumption of diets containing 2 and 8 mg T-2 toxin/kg feed by laying hens had no effect on the number of fertile eggs, but hatchability of fertile eggs from these hens was less than that of control hens ($P \leq 0.05$). Likewise, Tobias *et al.* (1992) reported poor hatchability accompanied by reduced feed intake among laying hens fed diets containing 5 and 10 mg T-2 toxin/kg feed, and concluded that total hatchability was a result of T-2 toxin induced infertility. Chi *et al.* (1978a) and Tobias *et al.* (1992) reported that T-2 toxin and its metabolites are deposited into egg yolk for 11 days following cessation T-2 exposure. Therefore, the early decline in percent fertility as observed in our experiments may be the result of yolk T-2 content. As observed by (Chi *et al.*, 1978b), T-2 toxin was cleared from the body after the first 2 weeks egg production and no further experimental changes were observed; which is the same as our results. No differences in hatchability characteristics were observed after the 2nd week egg production in either experiment. Based upon our results, we conclude that T-2 toxin may have similar effects on adult Bobwhite quail as have been reported in chickens. However, it appears that slightly higher levels of T-2 may be needed to elicit the same effects in pen-raised quail. Levels of 1, 3, 5, 8, and 10 mg T-2 toxin/kg feed resulted in body weight depression, growth retardation, reduced egg shell thickness and hatchability of fertile eggs in chickens (Wyatt *et al.*, 1975; Kubena *et al.*, 1989; Tobias *et al.*, 1992). The levels of T-2 toxin used in our study were nearly twice these values. It has been documented that there are large differences in toxin metabolism within avian species (Pan and Fouts, 1979; Dalvi *et al.*, 1987). Biotransformation is the sum of processes by which a foreign chemical or xenobiotic is subjected to chemical change by living organisms. Hydrolysis of ester linkages seem to be the major pathway in the metabolism of T-2 toxin with hydrolysis of the ester at carbon 4 being the primary site of attack (Leeson *et al.*, 1995). Species differences in drug and toxin metabolism have been attributed to different levels and forms of liver enzymes (Dalvi *et al.*, 1987). The hepatic microsomal enzymes thought to participate in detoxifying reactions include cytochrome, benzphetamine N-demethylase, aniline hydroxylase, and glutathione S-transferase (Dalvi *et al.*, 1987). Since it takes more T-2 toxin in quail to obtain similar effects, one would assume that bobwhite quail have higher levels of detoxifying enzymes than chickens. However, available information indicates that quail have lower levels of at least some hepatic microsomal enzymes (cytochrome P-450, aniline hydroxylase, glutathione S-transferase) than chickens (Dalvi *et al.*, 1987; Enkvetchakul *et al.*, 1995). Reduction of the 12,13 epoxide ring, necessary for toxicity (Uneo, 1986) by anaerobic microflora present in the gastrointestinal tract however, is an additional, and important component in the detoxification of T-2 toxin (Leeson *et al.*, 1995), and may explain differences between chickens and quail. To date, we have no reasonable explanation as to why bobwhite can withstand higher levels than chickens. Exposure to T-2 toxin during the pre-puberal period may be a contributing factor to nest failure of wild quail particularly when combined with immune suppression and increased susceptibility to predation after consumption of mycotoxins.

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