Effects of T-2 toxin on growth performance, feather quality, tibia development and blood parameters in Yangzhou goslings

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ABSTRACT T-2 toxin is a dangerous natural pollutant and widely exists in animal feed, often causing toxic damage to poultry, such as slow growth and development, immunosuppression, and death. Although geese are considered the most sensitive poultry to T-2 toxin, the exact damage caused by T-2 toxin to geese is elusive. In the present study, a total of forty two 1-day-old healthy Yangzhou male goslings were randomly allotted seven diets contaminated with 0, 0.2, 0.4, 0.6, 0.8, 1.0, or 2.0 mg/kg T-2 toxin for 21 d, and the effects of T-2 toxin exposure on growth performance, feather quality, tibia development, and blood parameters were investigated. The results showed that T-2 toxin exposure significantly inhibited feed intake, body weight gain, shank length growth, and organ development (e.g., ileum, cecum, liver, spleen, bursa, and tibia) in a dose-dependent manner. In addition, the more serious feathering abnormalities and feather damage were observed in goslings exposed to a high dose of T-2 toxin (0.8, 1.0, and 2.0 mg/kg), which were mainly sparsely covered with short, dry, rough, curly, and gloss-free feathers on the back. We also found that hypertrophic chondrocytes of the tibial growth plate exhibited abnormal morphology and nuclear consolidation or loss, accompanied by necrosis and excessive apoptosis under 2.0 mg/kg T-2 toxin exposure. Moreover, 2.0 mg/kg T-2 toxin exposure triggered erythropenia, thrombocytosis, alanine aminotransferase, and aspartate aminotransferase activity, as well as high blood urea nitrogen, uric acid, and lactic dehydrogenase levels. Collectively, these data indicate that T-2 toxin had an adverse effect on the growth performance, feather quality, and tibia development, and caused liver and kidney damage and abnormal blood parameters in Yangzhou goslings, providing crucial information toward the prevention and control of T-2 toxin contamination in poultry feed.

Key words: T-2 toxin, goslings, growth performance, feather quality, tibia development

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INTRODUCTION

T-2 toxin is a virulent type A trichothecene secreted by *Fusarium spp.*, and is widely present in maize, barley, wheat, oats, and other cereal crops, which are common animal feed ingredients (Escrivá et al., 2015). Of 420 Chinese feedstuff samples, T-2 toxin incidence was 79.5%, and levels ranged 10 to 735 μg/kg, posing a potential threat to animals (Wang et al., 2013). T-2 toxin ingestion by animals can produce acute or chronic effects, with serious toxic effects on feed intake (**FI**), growth and development, reproductive capacity, and health (Binder et al., 2007; Schuhmacher-Wolz et al., 2010). Lower FI, reduced weight gain, growth retardation (Wei et al., 2019), lower egg production and quality, decreased hatchability (Dazuk et al., 2020), coagulopathy (Singh et al., 2020), and feather abnormalities (Wyatt et al., 1975; Manafi et al., 2015) are visible signs of T-2 toxin poisoning in poultry (Sokolović et al., 2008). The toxic effects of T-2 toxin include inhibition of protein, DNA and RNA synthesis, and immunoglobulin production (Szabó et al., 2019). In addition, T-2 toxin accelerates the generation of reactive oxygen species (**ROS**) that induces oxidative stress, which further leads to inflammatory responses and apoptosis (Mackei et al., 2018; Huang et al., 2021; Zhang et al., 2021). The hepatic apoptosis rate and pathology of chickens were

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aggravated with the increase of T-2 concentration that ranged 0.5 to 2.0 mg/kg, which induced the mitochondria-mediated apoptosis by producing ROS and promoting cytochrome c translocation and apoptosomes formation (Yin et al., 2020). It has been reported that 4.0 mg/kg of T-2 toxin triggers oxidative stress and inflammatory responses to renal damage in mice (Huang et al., 2021). In addition, 0.5 mg/kg T-2 toxin disturbed various endogenous metabolic changes and pathways and oxidative stress, causing the accumulation of amino acids and nucleotides in the liver, kidney, and spleen of broiler chickens (Wan et al., 2016). T-2 toxin targets tissues in an actively and rapidly dividing state through several toxic mechanisms that cause severe damage (Sokolović et al, 2008), such as the intestines, liver, kidneys, spleen, bursa, feathers, and bones. Thus, as T-2 toxin is widely present in animal feed, it often causes a decrease in animal performance and tissue damage.

Some studies have concluded that T-2 toxin is extremely hazardous to poultry. Among poultry, geese are more sensitive to T-2 toxin than chickens, ducks (Mézes et al., 1999; Fernye et al., 2018), and other mammals such as rodents (Li et al., 2011) and ruminants (Gholampour Azizi et al., 2014). Considering the effects of T-2 toxin on lipid peroxidation and the glutathione redox system, geese are the most sensitive poultry species, followed by ducks and chickens, and the liver is the most sensitive tissue, followed by plasma and red blood cells (Mézes et al., 1999). Geese showed upper alimentary distress and died after foraging on moldy barley, which was associated with T-2 mycotoxicosis; however, experimental mice exhibited only slight symptoms under long-term exposure (Puls and Greenway, 1976). Ruminants are more resistant to the T-2 toxin than non-ruminants because of microbial degradation within rumen microorganisms and the lack of phosphatidylcholine in the membrane (DeLoach et al., 1989; Kuca et al., 2008). Different doses of toxins affect the degree of toxicity in animals. For chickens, the Lethal Dose 50% (**LD50**) of T-2 toxin for broilers was 4.97 mg/(kg·b.w) and the LD50 for laying hens was 6.27 mg/(kg·b.w) (Chi et al., 1977b; Li et al., 2022). T-2 toxin causes impaired performance, DNA fragmentation in spleen leukocytes, and elevated serum immune globulin A (**IgA**) levels in a dose-dependent manner in broilers, especially with 13.5 mg/kg contamination (Rezar et al., 2007). For geese, 0.1, 0.2, and 0.3 mg/kg/day T-2 toxin reduced hatching rates and was dose-dependent, with a significant increase in mortality at levels above 0.8 mg/kg/day (Vanyi et al., 1994).

As the effects of T-2 toxin on geese production performance and tissue damage have been rarely reported, and studies have mainly focused on European geese and reproduction performance, it was unclear whether Chinese geese have similar or different responses at different T-2 toxin concentrations. Therefore, the objective of this study was to investigate the effect of T-2 toxin with different concentrations on growth performance, feather quality, tibia development, and blood parameters in Yangzhou goslings.

MATERIALS AND METHODS

Ethics Statement

All animal protocols were approved by the Animal Care and Use Committee of the Yangzhou University.

Animals and Sample Collection

Forty-two healthy 1-day-old Yangzhou male goslings were randomly divided and raised in stainless-steel cages (90 cm × 60 cm × 45 cm) in an animal housing facility at Yangzhou University (Yangzhou, Jiangsu, China) with insulation lamps, and the temperature was controlled at 25°C to 30°C. Goslings had free access to the growing diet (Table 1) supplemented with 0, 0.2, 0.4, 0.6, 0.8, 1.0, and 2.0 mg/kg T-2 toxin and water during the growing period (1−21 d of age). T-2 toxin (CAS NO: 21259-20-1, 50 mg, purity >98%, Wuhan Grint Biological Technology Co., Ltd., Wuhan, China) was dissolved in 10 mL of non-aqueous ethanol to obtain a primary solubility of 5 mg/mL of mother liquor, which was divided into ten tubes and stored at −20°C. Before usage, enzyme-free water was added to achieve the target concentration and then sprayed onto the feed, stirred well, evaporated naturally for 30 min and stored at room temperature for later use. The real T-2 toxin concentration in each diet (0.009, 0.208, 0.412, 0.621, 0.814, 1.020, and 2.033 mg/kg) was determined using enzyme-linked immunosorbent assay (**ELISA**; CAT NO: EKT-060, Qingdao Pribolab Biotech Co., Ltd., Qingdao, China) to ensure the accuracy of T-2 toxin supplementation. After 21 d of daily feeding management and recording, goslings were fasted overnight for 6 h before sample collection. On the 21st day, all goslings from each group were sacrificed. After capturing images and scoring feathering, live body weight (**BW**) and shank length (**SL**) were measured, followed by blood collection. After cleaning with 75% alcohol, 4 mL of blood was collected from the right wing vein. Half (2 mL) was mixed with K_2EDTA in a plasma collection tube, placed on ice for 10 min, and

Table 1. Ingredient composition and nutritional level of the growing diet for 1−21 day-old Yangzhou goslings.

Ingredient composition (%)		Nutritional level (%)	
Corn	66.60	ME (MJ/kg)	11.64
Soybean meal	26.50	Crude protein	17.04
Rice husk	0.50	Crude fiber	3.66
Stone powder	0.50	Crude fat	2.48
Calcium hydrogen phosphate	1.25	Ca	0.68
Methionine	0.35	Total phosphorus	0.66
Salt	0.50	Available phosphorus	0.41
Premix	3.80	Lysine	0.86
Total	100.00	Methionine	0.60

Note: The premix per kilogram contains 1,200, 000 IU VA; 400,000 IU VD; 1,800 IU VE; 150 mg VK; 60 mg VB1; 600 mg VB2; 200 mg VB6; 1 mg VB12; 3,000 mg niacin; 900 mg D-pantothenic acid; 50 mg folic acid; 4 mg biotin; 35 g choline; 6 g Fe; 1 g Cu; 9.5 g Mn; 9 g Zn; 30 mg Se; and 50 mg I. Nutrient levels were calculated values.

sent to the Yangzhou Centers for Disease Control and Prevention (**CDC**) for routine blood tests. Another 2 mL sample was centrifuged and sent to the Yangzhou University Animal Hospital for serum biochemical tests. The weights of the heart, liver, spleen, lungs, kidneys, gizzard, proventriculus, bursa, and pancreas were measured. The weight and length of intestines (duodenum, jejunum, ileum, cecum, and rectum) were also measured after cleaning to remove intestinal contents and adherent fat. After removal of adhesions, the left tibial phenotypes were observed, and the length and weight were measured. For histological analysis, tibial growth plate (**TGP**) samples (approximately 1.5 cm^3) were fixed in 10% formalin overnight, followed by storage in fresh 10% formalin, hematoxylin and eosin (**HE**) staining, and histomorphological analysis.

Growth Performance and Organ Indexes

The average FI was recorded daily on a pen basis during the 21-d exposure to the T-2 toxin. After 6 h of fasting, live individual BW and SL were measured, and the goslings were sacrificed on the 21st day. Different organs were collected sequentially, and the relevant contents and adhesive substances were removed for weighing. Organ indices were calculated as organ weight (g)/live weight (g) × 100%.

Feather Scoring and Injury Degree

On the 21st day, the feather scoring of the neck, back, thorax-abdomen, wing, and tail for all goslings was counted, and back-feather damage scoring of individuals was also calculated. The feather scoring system for evaluating cleanliness and damage was adapted from previous studies (Morrissey et al., 2014; Mahmoud et al., 2015; Liu et al., 2021). Briefly, a score of 0 indicates that the feather was completely clean and covered. A score of 1 indicates less clean or damaged feathers, with no bare skin. A score of 2 indicates unclean feathers or less than 3 × 3 cm injured regions. A score of 3 indicates less dirty feathers or a denuded area of more than 3 × 3 cm. A score of 4 represents dirty feathers or visible skin. The total score for various parts of the body indicates the total father cleanliness. The lower the feather scoring, the less feathers were contaminated or damaged, and the better the welfare condition.

HE Staining and Histomorphological Analysis of the Tibial Growth Plate

HE staining was performed following the guidelines of the HE dye solution kit sets (CAT NO: GP1012, GP1001, GP1010, and GP1031, Wuhan Servicebio Biotechnology Co., Ltd., Wuhan, China). Briefly, TGP samples were fixed in 10% formalin, decalcified for 1 month, dehydrated until transparent, and embedded in paraffin. Subsequently, sections were prepared, placed in water for extension, and dried. The sections were then placed in xylene, ethanol, and 75% ethanol, and washed with water. Next, the sections were stained with hematoxylin, followed by staining with eosin. Finally, the sections were placed in ethanol and xylene and sealed with neutral gum. TGP histological images were captured using a micro-image analysis system (CaseViewer 2.4, 3DHISTECH Ltd., Budapest, Hungary).

Hematological Parameters Tests

After blood collection, half the blood was transferred to a K_2EDTA-tube for routine blood parameter analysis performed using an automatic blood cell counter (MEK-8222K, NIHON KOHDEN CORP., Tokyo, Japan), including white blood cells (**WBC**), red blood cells (**RBC**), hematocrit (**HCT**), mean corpuscular volume (**MCV**), red blood cell distribution width (**RDW**), hemoglobin (**HGB**), mean corpuscular hemoglobin (**MCH**), mean corpuscular hemoglobin concentration (**MCHC**), platelet (**PLT**), platelet (**PCT**), mean platelet volume (**MPV**), platelet distribution width (**PDW**), and platelet-larger cell ratio (**P-LCR**). The remaining non-anticoagulant blood samples were processed through centrifugation at 3,000 rpm for 10 min at 4°C to determine serum biochemical indicators using a fully automatic biochemical analyzer (AU480, Beckman Coulter K.K, Tokyo, Japan), containing alanine aminotransferase (**ALT**), aspartate aminotransferase (**AST**), **AST/ALT**, blood urea nitrogen (**BUN**), uric acid (**UA**), total protein (**TP**), albumin (**ALB**), globulin (**GLOB**), **ALB/GLOB**, calcium (**Ca**), phosphorus (**P**), **Ca/P**, alkaline phosphatase (**ALP**), and lactate dehydrogenase (**LDH**).

Statistical Analysis

SPSS software (version 25.0, IBM Corp, Armonk, NY) was used to analyze the statistical significance of the difference between the T-2 toxin contamination and control groups. All data were tested using the Shapiro-Wilk (N < 50) and homogeneity tests to ensure the normality and homogeneity of variances among the various groups. The data of average daily FI were analyzed using one-way repeated measures analysis of variance (**ANOVA**). After the Mauchly's test of sphericity, a simple effect analysis was executed and the significant comparison of different T-2 toxin dosages at the same age was presented. Other data were expressed as the mean ± standard error (**SEM**) and subjected to one-way ANOVA followed by Tukey's honestly significant difference (**HSD**) multiple range test. If the data (e.g., feather scoring, RBC, PLT, and Ca/P) were not normal and homogeneous, it was subjected to the non-parametric test (Kruskal-Wallis tests) and results were expressed as medians (interquartile range) and presented in figures with box plots (median, first, and third quartiles). Statistical significance was set at $P < 0.05$.

RESULTS

Effect of T-2 Toxin on Growth Performance and Organ Development

To determine the effect of T-2 toxin on the growth performance of goslings, the relevant parameters were measured. Goslings in the control group (0 mg/kg) appeared active with tidy plumage and normal feed intake, droppings, and body weight. Compared with the control, the average daily FI ($P < 0.05$; Figure 1A; Supplementary table 1) and 21-d total FI ($P < 0.05$; Figure 1B) changed in a dose-dependent manner during T-2 toxin exposure. This was consistent with the fact that BWG and final BW significantly decreased in all T-2 toxin groups during the 21-d exposure period ($P < 0.05$; Figure 1C). SL is closely related to BW, meat production, and slaughter performance. In terms of shank growth, SLG and the final SL significantly decreased in the 2.0 mg/kg T-2 toxin-contaminated groups ($P < 0.05$; Figure 1D).

Goslings were in a state of delayed organ development under 21-d T-2 toxin exposure. Compared with the control group, the weights of the liver, spleen, gizzard, and bursa decreased in all T-2 toxin groups, in contrast to the increased weight indexes of the heart, lungs, kidneys, proventriculus, and pancreas in some groups ($P < 0.05$; Table 2). In addition, the weights of the jejunum, ileum, cecum, and rectum; the lengths of the ileum, cecum, and rectum; and the indices of the ileum and cecum in some T-2 toxin groups were significantly lower than those in the control, especially with higher T-2 toxin doses ($P < 0.05$; Table 3).

Effect of T-2 Toxin on Feather Quality

Overall, it was clear that feather quality varied with T-2 toxin exposure in a dose-dependent manner in goslings. Based on appearance, T-2 toxin caused feathering abnormalities, especially through short, dry, rough, curly, and gloss-free feathers sparsely covering the back (Figure 2A). Interestingly, the feather cleanliness scoring (Figure 2B) and back-feather damage scoring (Figure 2C) also increased in a dose-dependent manner ($P < 0.05$), and the most serious damage appeared in the 2.0 mg/kg T-2 toxin group.

Effect of T-2 Toxin on Tibia Growth and Development

Furthermore, the tibial growth plate and morphological structure can reflect longitudinal bone growth and health to a certain extent, and tibial development is also a major contributor to support BW. T-2 toxin clearly slowed tibial growth (length and weight) in a dose-dependent manner ($P < 0.05$), especially in the 2.0 mg/kg T-2 toxin group (Figure 3A, B). As shown through HE staining and histomorphological analysis (Figure 3C), the TGP was subdivided into resting, proliferative, hypertrophic, and calcified zones, with clear cell morphology. Compared with the control group, the TGP exhibited abnormal morphological structure in the 2.0 mg/kg T-2 toxin group. Chondrocytes were arranged in a crowded and stagnant state with increasing density. Importantly, some chondrocytes in the hypertrophic zones manifested abnormal morphology and nuclear

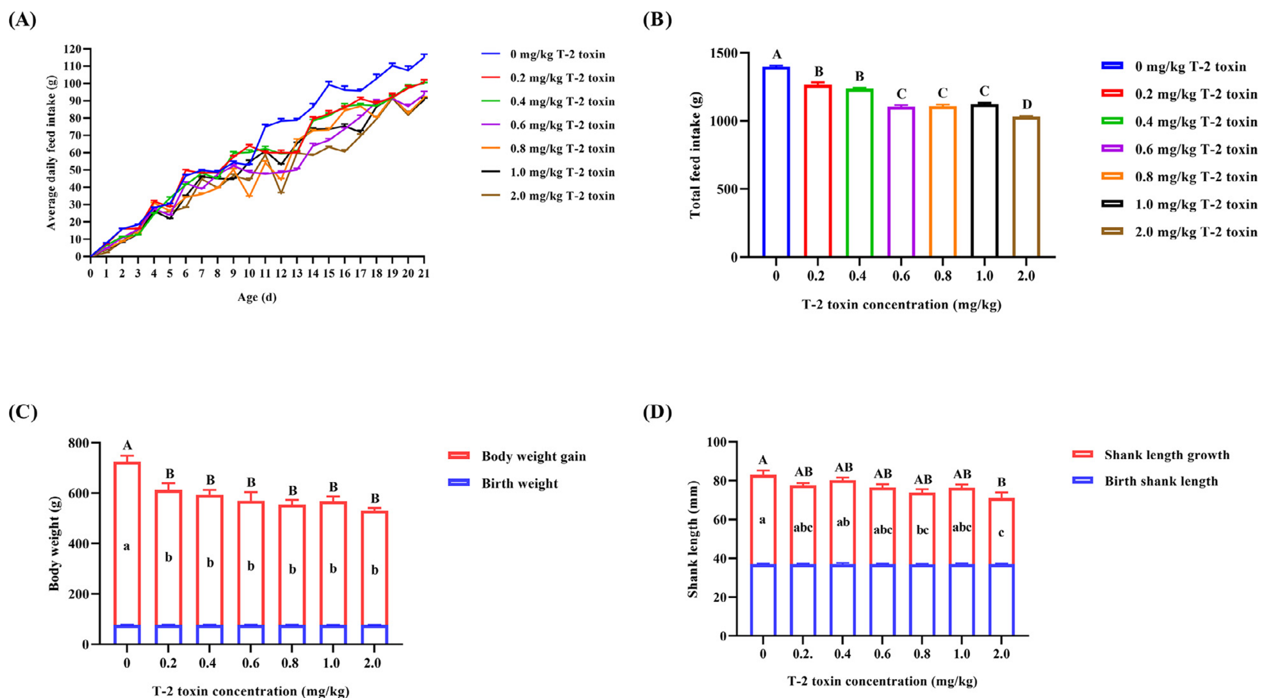

Figure 1. Average daily feed intake (A), total feed intake (B), body weight, body weight gain (C), shank length and shank length growth (D) of Yangzhou goslings exposed to 0, 0.2, 0.4, 0.6, 0.8, 1.0, and 2.0 mg/kg T-2 toxin from birth to 21-day-old. Note: The values were presented as the means ± SEM. N = 6. The significance analysis of Figure 1A was shown in Supplementary table 1. In others, no superscripts or the same superscripts in the shoulder or heading mark of peer data indicated that the differences were not significant ($P > 0.05$) and different superscripts indicated significant differences ($P < 0.05$). Lowercase letters represented partial significance, and uppercase letters represented overall significance. It was the same in the following figures.

Table 2. Organ weight and index of 21-day-old Yangzhou goslings under T-2 toxin exposure from birth.

Organ	Item	0 mg/kg T-2	0.2 mg/kg T-2	0.4 mg/kg T-2	0.6 mg/kg T-2	0.8 mg/kg T-2	1.0 mg/kg T-2	2.0 mg/kg T-2
Heart	Weight (g)	5.56 ± 0.21	5.46 ± 0.25	5.35 ± 0.05	5.27 ± 0.23	5.20 ± 0.36	5.29 ± 0.17	5.05 ± 0.10
	Index (%)	0.77 ± 0.01^{b}	0.90 ± 0.04^{ab}	0.91 ± 0.03^{ab}	0.93 ± 0.03^{a}	0.94 ± 0.06^{a}	0.93 ± 0.02^{a}	0.95 ± 0.02^{a}
Liver	Weight (g)	24.05 ± 0.23^{a}	17.61 ± 0.12^{bc}	18.00 ± 0.71^{b}	18.22 ± 0.82^{b}	17.30 ± 0.29^{bc}	18.32 ± 0.16^{b}	15.86 ± 0.22^{c}
	Index (%)	3.34 ± 0.13	2.90 ± 0.14	3.05 ± 0.15	3.26 ± 0.25	3.13 ± 0.07	3.24 ± 0.10	3.00 ± 0.05
Spleen	Weight (g)	1.22 ± 0.06^{a}	0.74 ± 0.02^{b}	0.74 ± 0.08^{b}	0.82 ± 0.12^{b}	0.66 ± 0.07^{b}	0.67 ± 0.10^{b}	0.84 ± 0.01^{b}
	Index (%)	0.17 ± 0.01	0.12 ± 0.01	0.13 ± 0.01	0.14 ± 0.02	0.12 ± 0.01	0.12 ± 0.02	0.16 ± 0.01
Lung	Weight (g)	4.03 ± 0.09^{bc}	3.94 ± 0.25^{c}	3.93 ± 0.32^{c}	5.45 ± 0.06^{a}	5.19 ± 0.18^{a}	5.04 ± 0.10^{ab}	4.78 ± 0.39^{abc}
	Index (%)	0.56 ± 0.01^{b}	0.65 ± 0.06^{b}	0.67 ± 0.07^{b}	0.97 ± 0.06^{a}	0.94 ± 0.02^{a}	0.89 ± 0.03^{a}	0.90 ± 0.06^{a}
Kidney	Weight (g)	7.11 ± 0.14	7.16 ± 0.06	7.54 ± 0.49	7.20 ± 0.29	7.07 ± 0.39	6.92 ± 0.47	6.85 ± 0.38
	Index (%)	0.98 ± 0.03^{b}	1.18 ± 0.04^{ab}	1.27 ± 0.08^{a}	1.29 ± 0.09^{a}	1.27 ± 0.04^{a}	1.21 ± 0.05^{ab}	1.29 ± 0.06^{a}
Gizzard	Weight (g)	40.93 ± 3.14^{a}	30.32 ± 0.64^{bc}	30.36 ± 0.57^{bc}	32.61 ± 1.55^{b}	23.90 ± 1.70^{c}	30.85 ± 0.12^{bc}	26.45 ± 1.43^{bc}
	Index (%)	5.62 ± 0.31^{a}	4.99 ± 0.22^{ab}	5.15 ± 0.21^{ab}	5.78 ± 0.29^{a}	4.31 ± 0.24^{b}	5.46 ± 0.17^{a}	4.99 ± 0.23^{ab}
Proventriculus	Weight (g)	5.22 ± 0.04^{ab}	5.00 ± 0.10^{ab}	5.74 ± 0.27^{a}	5.56 ± 0.01^{ab}	4.84 ± 0.35^{b}	5.31 ± 0.07^{ab}	5.71 ± 0.24^{a}
	Index (%)	0.72 ± 0.03^{c}	0.82 ± 0.04^{bc}	0.98 ± 0.07^{ab}	0.99 ± 0.06^{ab}	0.87 ± 0.04^{bc}	0.94 ± 0.03^{ab}	1.08 ± 0.03^{a}
Bursa	Weight (g)	1.00 ± 0.08^{a}	0.65 ± 0.03^{b}	0.69 ± 0.08^{b}	0.57 ± 0.06^{b}	0.71 ± 0.02^{b}	0.74 ± 0.02^{b}	0.76 ± 0.04^{b}
	Index (%)	0.14 ± 0.01^{ab}	0.11 ± 0.01^{b}	0.12 ± 0.01^{ab}	0.10 ± 0.01^{b}	0.13 ± 0.00^{ab}	0.13 ± 0.01^{ab}	0.14 ± 0.01^{a}
Pancreas	Weight (g)	2.62 ± 0.08	3.04 ± 0.04	2.76 ± 0.18	2.72 ± 0.16	2.69 ± 0.06	2.81 ± 0.06	2.60 ± 0.16
	Index (%)	0.36 ± 0.01^{b}	0.50 ± 0.02^{a}	0.47 ± 0.04^{a}	0.48 ± 0.02^{a}	0.49 ± 0.01^{a}	0.50 ± 0.02^{a}	0.49 ± 0.02^{a}

Note: The values used for ANOVA analysis are presented as the means ± SEM and the values used for the non-parametric test are presented as medians (interquartile range). N = 6.
[a,b,c]Means within a row with no common superscript differ significantly ($P < 0.05$).

consolidation or loss, accompanied by necrosis and excessive apoptosis.

Effect of T-2 toxin on Blood Parameters

Blood is the main and most important component of the internal environment of the body, and changes in blood composition reflect the metabolic status and health status of the animal body. In routine blood tests (Table 4), compared with the control group, RBC and RDW decreased in the 2.0 mg/kg T-2 toxin group, along with an increase in MCH ($P < 0.05$). In addition, PLT rose in the 2.0 mg/kg T-2 toxin group, accompanied by a reduction in MPV and P-LCR ($P < 0.05$).

Serum biochemical tests (Table 5) showed that, compared with the control, the liver function-related parameters (ALT, AST, and AST/ALT) increased significantly to varying degrees in most T-2 toxin groups ($P < 0.05$). Furthermore, the renal function relative parameter BUN increased in most T-2 toxin groups ($P < 0.05$), whereas UA increased only in the 2.0 mg/kg T-2 toxin group ($P < 0.05$). In addition, TP increased significantly only in the 2.0 mg/kg T-2 toxin group ($P < 0.05$), whereas there were no significant changes in the ALB, GLOB, and ALB/GLOB. Moreover, 0.4 to 0.8 mg/kg of T-2 toxin accelerated the rise of phosphorus and caused the Ca/P ratio to decrease ($P < 0.05$). Similarly, Ca decreased only in the 0.8 mg/kg T-2 toxin group ($P < 0.05$). However, ALP increased in 1.0 and 2.0 mg/kg T-2 toxin groups ($P < 0.05$). Finally, LDH showed a distinct improvement in most T-2 toxin groups ($P < 0.05$).

DISCUSSION

The T-2 toxin is the most toxic secondary metabolite of all trichothecenes produced by diverse *Fusarium spp.* (for example, *F. acuminatum, F. poae*, and *F. sporotrichioides*), which widely occurs in cereal crops for animal feed production (Janik et al., 2021). Many agricultural technical and environmental factors induce T-2 toxin pollution in crop growth, harvest and processing, feed production, food processing, and animal production,

Table 3. Intestinal weight, index, and length of 21-day-old Yangzhou goslings under T-2 toxin exposure from birth.

Index	Item	0 mg/kg T-2	0.2 mg/kg T-2	0.4 mg/kg T-2	0.6 mg/kg T-2	0.8 mg/kg T-2	1.0 mg/kg T-2	2.0 mg/kg T-2
Duodenum	Weight (g)	4.55 ± 0.26^{a}	4.47 ± 0.10^{ab}	4.32 ± 0.40^{ab}	3.54 ± 0.19^{b}	3.97 ± 0.15^{ab}	3.80 ± 0.22^{ab}	3.70 ± 0.15^{ab}
	Index (%)	0.63 ± 0.02^{b}	0.73 ± 0.03^{ab}	0.72 ± 0.04^{ab}	0.81 ± 0.06^{a}	0.71 ± 0.01^{ab}	0.67 ± 0.04^{ab}	0.70 ± 0.01^{ab}
	Length (cm)	23.00 ± 0.22	23.13 ± 1.48	21.67 ± 0.88	20.00 ± 0.96	20.33 ± 0.67	19.50 ± 0.48	20.50 ± 1.20
Jejunum	Weight (g)	15.64 ± 0.34^{a}	13.63 ± 0.52^{ab}	14.00 ± 0.79^{ab}	13.61 ± 0.52^{ab}	11.73 ± 0.41^{bc}	12.31 ± 0.61^{b}	9.69 ± 0.36^{c}
	Index (%)	2.16 ± 0.03^{ab}	2.23 ± 0.04^{ab}	2.39 ± 0.19^{a}	2.42 ± 0.14^{a}	2.12 ± 0.09^{ab}	2.16 ± 0.05^{ab}	1.83 ± 0.06^{b}
	Length (cm)	58.58 ± 3.10	56.75 ± 1.97	55.08 ± 1.07	56.00 ± 2.92	51.17 ± 0.46	57.50 ± 1.14	52.00 ± 1.46
Ileum	Weight (g)	15.13 ± 0.42^{a}	12.68 ± 0.94^{ab}	12.21 ± 0.72^{b}	11.93 ± 0.46^{b}	11.78 ± 0.65^{b}	10.05 ± 0.41^{bc}	8.45 ± 0.39^{c}
	Index (%)	2.09 ± 0.01^{ab}	2.07 ± 0.11^{ab}	2.05 ± 0.08^{ab}	2.12 ± 0.10^{a}	2.13 ± 0.11^{a}	1.77 ± 0.01^{bc}	1.59 ± 0.04^{c}
	Length (cm)	55.67 ± 3.13^{a}	52.00 ± 1.10^{ab}	53.25 ± 1.09^{ab}	49.17 ± 1.70^{ab}	49.67 ± 2.07^{ab}	50.33 ± 0.42^{ab}	46.67 ± 1.31^{b}
Cecum	Weight (g)	3.41 ± 0.13^{a}	2.79 ± 0.04^{ab}	2.34 ± 0.30^{bc}	1.92 ± 0.21^{c}	2.05 ± 0.16^{bc}	1.91 ± 0.13^{c}	1.99 ± 0.17^{c}
	Index (%)	0.47 ± 0.01^{a}	0.46 ± 0.02^{a}	0.39 ± 0.05^{ab}	0.33 ± 0.03^{c}	0.37 ± 0.03^{bc}	0.33 ± 0.02^{c}	0.37 ± 0.02^{bc}
	Length (cm)	33.33 ± 1.12^{a}	30.00 ± 0.37^{ab}	29.50 ± 0.80^{ab}	28.42 ± 0.70^{b}	28.50 ± 1.28^{ab}	29.83 ± 1.60^{ab}	26.50 ± 1.38^{b}
Rectum	Weight (g)	3.34 ± 0.18^{a}	2.80 ± 0.09^{ab}	2.84 ± 0.09^{ab}	2.87 ± 0.18^{ab}	2.90 ± 0.10^{ab}	2.39 ± 0.09^{bc}	2.01 ± 0.05^{c}
	Index (%)	0.46 ± 0.03^{abc}	0.46 ± 0.01^{abc}	0.48 ± 0.01^{ab}	0.51 ± 0.04^{ab}	0.52 ± 0.02^{a}	0.42 ± 0.02^{bc}	0.38 ± 0.01^{c}
	Length (cm)	10.67 ± 0.53^{a}	9.00 ± 0.41^{b}	8.00 ± 0.52^{bc}	6.33 ± 0.31^{d}	6.50 ± 0.26^{cd}	7.17 ± 0.21^{cd}	6.33 ± 0.17^{d}

Note: The values used for ANOVA analysis are presented as the means ± SEM and the values used for the non-parametric test are presented as medians (interquartile range). N = 6.
[a,b,c,d]Means within a row with no common superscript differ significantly ($P < 0.05$).

Figure 2. Lateral view of feathering patterns (A), feather cleanliness scoring (B) and back-feather damage scoring (C) of 21-day-old goslings exposed to 0 (a.), 0.2 (b.), 0.4 (c.), 0.6 (d.), 0.8 (e.), 1.0 (f.), and 2.0 (g.) mg/kg T-2 toxin from birth.

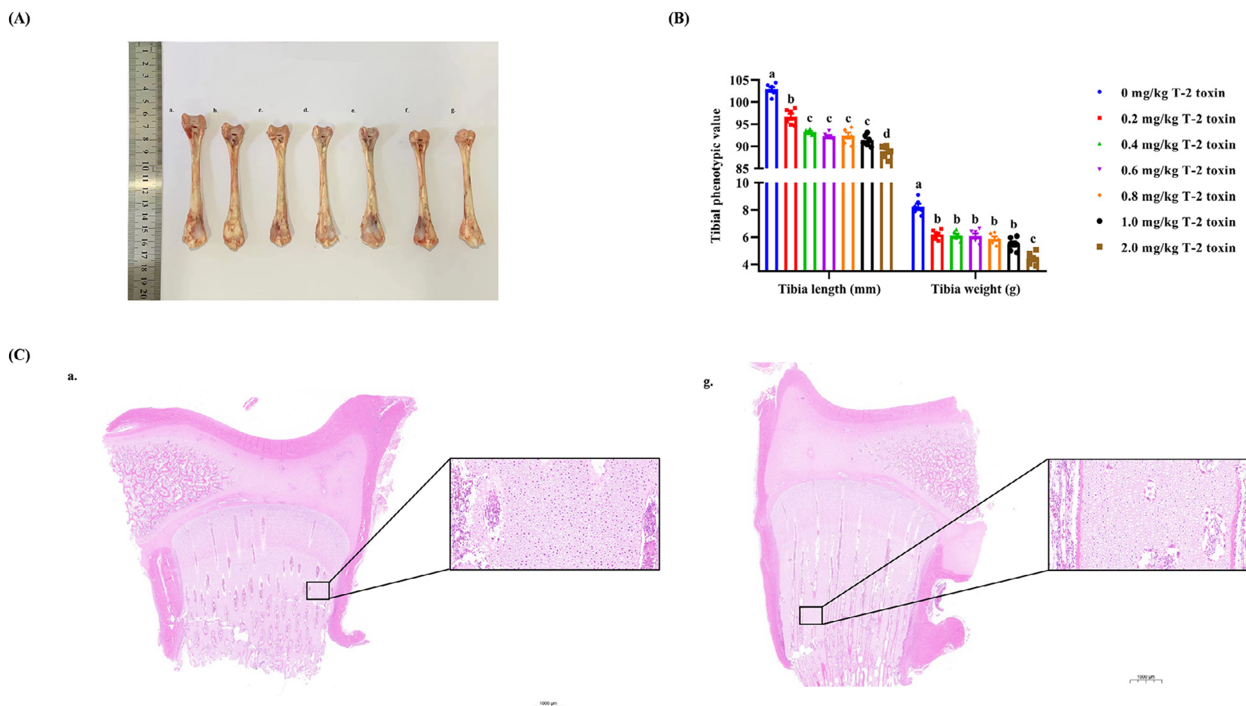

Figure 3. The tibia phenotype (A), length and weight (B) and tibial growth plate morphological structure (C) of 21-day-old goslings exposed to 0 (a.), 0.2 (b.), 0.4 (c.), 0.6 (d.), 0.8 (e.), 1.0 (f.), and 2.0 (g.) mg/kg T-2 toxin from birth.

Table 4. Routine blood parameters of 21-day-old Yangzhou goslings under T-2 toxin exposure from birth.

Index	0 mg/kg T-2	0.2 mg/kg T-2	0.4 mg/kg T-2	0.6 mg/kg T-2	0.8 mg/kg T-2	1.0 mg/kg T-2	2.0 mg/kg T-2
WBC (10^9/L)	334.78 ± 8.66	326.06 ± 11.41	334.46 ± 8.75	328.78 ± 7.84	334.17 ± 11.49	312.75 ± 19.73	356.77 ± 17.84
RBC (10^{12}/L)	1.52 (1.46−1.57)^{a}	1.37 (1.32−1.52)^{a}	1.39 (1.19−1.44)^{a}	1.23 (1.18−1.23)^{a}	1.29 (1.17−1.41)^{a}	1.12 (1.12−1.15)^{a}	1.16 (1.11−1.24)^{b}
HCT (%)	20.55 ± 2.03	21.67 ± 1.20	20.22 ± 1.36	20.66 ± 1.11	21.62 ± 1.45	18.63 ± 0.81	18.41 ± 0.47
MCV (fL)	153.68 ± 4.91	150.61 ± 3.04	154.40 ± 3.87	164.57 ± 1.76	167.22 ± 5.10	153.90 ± 2.64	160.18 ± 7.69
RDW (%)	29.33 ± 1.38^{a}	28.22 ± 0.78^{ab}	26.32 ± 0.50^{abc}	27.62 ± 0.72^{ab}	23.97 ± 0.40^{bc}	27.40 ± 0.84^{ab}	22.62 ± 1.57^{c}
HGB (g/L)	110.40 ± 2.89^{ab}	110.20 ± 2.28^{ab}	95.13 ± 2.93^{c}	104.52 ± 2.95^{abc}	114.90 ± 1.73^{a}	102.65 ± 2.73^{bc}	112.62 ± 2.80^{ab}
MCH (pg)	73.67 ± 1.05^{b}	77.65 ± 3.39^{b}	80.02 ± 5.05^{b}	84.73 ± 5.29^{ab}	90.50 ± 3.17^{ab}	85.53 ± 4.55^{ab}	97.64±2.95^{a}
MCHC (g/L)	476.00 ± 18.46	516.17 ± 24.51	545.33 ± 45.83	513.33 ± 27.16	555.17 ± 40.76	561.50 ± 36.06	614.83±27.49
PLT (10^9/L)	132.00 (115.75 −178.00)^{ac}	188.00 (155.25 −206.25)^{c}	121.50 (119.00 −125.25)^{abc}	113.00 (105.00 −130.50)^{abc}	147.00 (138.00 −148.50)^{abc}	147.00 (128.75 −175.25)^{ab}	177.00 (176.25 −181.00)^{b}
PCT	0.14 ± 0.01^{ab}	0.18 ± 0.02^{a}	0.12 ± 0.01^{bc}	0.10 ± 0.01^{c}	0.15 ± 0.00^{ab}	0.13 ± 0.01^{bc}	0.18± 0.00^{a}
MPV (fL)	10.80 ± 0.16^{a}	10.62 ± 0.07^{ab}	10.47 ± 0.13^{abc}	9.45 ± 0.08^{d}	10.23 ± 0.05^{abc}	10.12 ± 0.22^{bc}	9.88±0.13^{cd}
PDW (%)	9.80 ± 0.62^{a}	9.40 ± 0.24^{ab}	9.48 ± 0.09^{ab}	8.18 ± 0.19^{b}	9.55 ± 0.07^{a}	9.60 ± 0.22^{a}	9.82±0.29^{a}
P−LCR (%)	27.66 ± 0.25^{a}	26.82 ± 0.34^{a}	26.10 ± 0.81^{ab}	20.25 ± 0.67^{c}	24.88 ± 0.41^{ab}	24.43 ± 1.39^{ab}	22.82±0.76^{bc}

Abbreviations: HCT, hematocrit; HGB, Hemoglobin; MCH (HGB/RBC), mean corpuscular hemoglobin; MCV (HCT/RBC), mean corpuscular volume; MCHC (HGB/HCT), mean corpuscular hemoglobin concentration; MPV, mean platelet volume; PDW, platelet distribution width; P-LCR, platelet-larger cell ratio; PLT, Platelets; PCT, plateletocrit; RDW, red blood cell distribution width; RBC, red blood cells; WBC, white blood cells.

Note: The values used for ANOVA analysis are presented as the means ± SEM and the values used for the non-parametric test are presented as medians (interquartile range). N = 6.

^{a,b,c,d}Means within a row with no common superscript differ significantly ($P < 0.05$).

which threatens animal health, production, and human food safety, resulting in significant economic losses and health issues (Magnoli et al., 2019; Janik et al., 2021). Geese, especially goslings, are the most to T-2 toxin damage among poultry, and relevant studies are limited to increasing the difficulties of prevention and control management in production.

Effect of T-2 Toxin on Growth Performance and Organ Development

In this study, the growth performance of goslings was significantly reduced by 21-d T-2 toxin consumption, including FI, BW, BWG, SL, and SLG, as well as by the delay of organ development. The lower growth performance under T-2 toxin toxicity in goslings was consistent with the changes observed in male broiler chicks (Wei et al., 2019), White Roman geese (Lin et al., 2018) and Brown Tsaiya and Kaiya ducklings (Tso et al., 2021). The effect of T-2 toxin on growth performance and health clearly depends on animal age, dosage, and exposure time (Li et al., 2011; Janik et al., 2021). T-2 toxin causes contact irritation and metabolic toxic damage to the rostrum, oral cavity, and gastrointestinal tract, thus negatively affecting FI and causing further injuries to the blood system, liver, kidneys, and other organs (Yohannes et al., 2012). Moreover, the decline in FI caused by the T-2 toxin is an iconic effect that is well explained by the previously observed food refusal phenomenon and strange feeding behaviors regulated by various appetitive central and peripheral modulators, gut satiety hormones, and proinflammatory cytokines (Gaigé et al., 2014; Sheng et al., 2019), as well as growth hormone deficiency (Liu et al., 2017; Zhang et al., 2020). In addition, T-2 toxin inhibits protein, DNA, and RNA synthesis, and subsequently affects physiological functions by influencing the synthesis and secretion of key molecular precursors and raw materials (Sokolović et al., 2008). In addition to affecting morphological structures, T-2 toxin also affects digestion, absorption, and metabolic

Table 5. Serum biochemical parameters of 21-day-old Yangzhou goslings under T-2 toxin exposure from birth.

Index	0 mg/kg T-2	0.2 mg/kg T-2	0.4 mg/kg T-2	0.6 mg/kg T-2	0.8 mg/kg T-2	1.0 mg/kg T-2	2.0 mg/kg T-2
ALT (U/L)	24.63 ± 0.53^{d}	25.93 ± 0.88^{cd}	28.25 ± 0.37^{bcd}	33.62 ± 1.39^{ab}	30.83 ± 1.90^{abc}	30.13 ± 1.75^{abcd}	35.22 ± 1.90^{a}
AST (U/L)	14.44 ± 0.41^{d}	18.83 ± 0.72^{d}	33.82 ± 0.98^{ab}	36.45 ± 0.74^{a}	25.95 ± 2.39^{c}	28.12 ± 2.16^{bc}	37.10 ± 0.63^{a}
AST/ALT	0.59 ± 0.01^{e}	0.73 ± 0.02^{de}	1.20 ± 0.03^{a}	1.09 ± 0.04^{ab}	0.83 ± 0.04^{cd}	0.93 ± 0.03^{bc}	1.07 ± 0.06^{ab}
BUN (mmol/L)	0.54 ± 0.02^{d}	0.65 ± 0.04^{cd}	0.84 ± 0.03^{abc}	0.72 ± 0.05^{cd}	0.98 ± 0.07^{ab}	0.78 ± 0.02^{bc}	1.03 ± 0.10^{a}
UA (μmol/L)	242.57 ± 8.82^{b}	237.37 ± 12.81^{b}	239.33 ± 38.54^{b}	239.03 ± 25.17^{b}	222.18 ± 13.21^{b}	194.93 ± 14.19^{b}	354.20 ± 20.85^{a}
TP (g/L)	36.80 ± 0.06^{b}	37.60 ± 0.88^{ab}	41.13 ± 0.84^{ab}	41.67 ± 2.07^{a}	41.20 ± 0.58^{ab}	40.23 ± 1.12^{ab}	41.57 ± 0.50^{a}
ALB (g/L)	14.57 ± 0.10	13.48 ± 0.22	15.13 ± 0.88	15.20 ± 0.59	15.47 ± 0.40	14.67 ± 0.37	14.95 ± 0.13
GLOB (g/L)	22.23 ± 0.07	24.12 ± 0.71	26.00 ± 1.60	26.47 ± 2.00	25.73 ± 0.19	25.57 ± 1.28	26.62 ± 0.44
A/G	0.66 ± 0.01	0.56 ± 0.01	0.61 ± 0.08	0.59 ± 0.06	0.60 ± 0.01	0.58 ± 0.04	0.56 ± 0.01
Ca (mmol/L)	2.49 ± 0.05^{ab}	2.49 ± 0.06^{ab}	2.62 ± 0.05^{ab}	2.35 ± 0.14^{b}	1.97 ± 0.05^{c}	2.75 ± 0.11^{a}	2.74 ± 0.04^{a}
P (mmol/L)	2.45 ± 0.02^{b}	2.40 ± 0.04^{b}	3.17 ± 0.04^{a}	3.45 ± 0.09^{a}	3.37 ± 0.21^{a}	2.55 ± 0.23^{b}	2.52 ± 0.08^{b}
Ca/P	1.01 (0.99−1.01)^{ab}	1.01 (1.01−1.03)^{ab}	0.85 (0.83−0.85)^{abc}	0.66 (0.65−0.66)^{bc}	0.59 (0.54−0.94)^{c}	1.22 (0.99−1.22)^{a}	1.05 (1.03−1.13)^{a}
ALP (U/L)	432.00 ± 40.07^{bc}	442.00 ± 28.04^{bc}	449.67 ± 10.36^{bc}	496.50 ± 11.28^{abc}	408.17 ± 40.29^{c}	570.33 ± 42.99^{ab}	629.67 ± 51.27^{a}
LDH (U/L)	496.00 ± 8.43^{d}	601.67 ± 19.16^{cd}	923.50 ± 11.07^{ab}	1125.67 ± 41.39^{a}	810.67 ± 87.04^{bc}	739.00 ± 26.62^{bcd}	802.00 ± 105.30^{bc}

Abbreviations: A/G, ALB/GLOB; ALB, albumin; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; Ca, calcium; GLOB, globulin; LDH, lactate dehydrogenase; P, phosphorus; TP, total protein; UA, uric acid.

Note: The values used for ANOVA analysis are presented as the means ± SEM and the values used for the non-parametric test are presented as medians (interquartile range). N = 6.

^{a,b,c,d,e}Means within a row with no common superscript differ significantly ($P < 0.05$).

utilization, thus causing the nutritional needs of the body for the development of organs and induction of immunotoxicity to be unmet (Rafai et al., 2000; Manafi et al., 2015). This also results in oxidative stress (Rezar et al., 2007; Chen et al., 2019; Yin et al., 2020), inflammation (Sun et al., 2022), apoptosis (Chen et al., 2019; Yin et al., 2020), and autophagy (Yin et al., 2020; Sun et al., 2022). Consequently, the organ weights and indices measured in our study also changed significantly in a malnourished and stunted environment owing to the decreased FI and weakened digestion, absorption, and metabolism through T-2 toxin exposure. Atrophy of the liver, spleen, gizzard, and bursa and the elevated organ indexes of the heart, lungs, kidneys, proventriculus, and pancreas, as well as the weight loss of the jejunum, ileum, cecum, and rectum, and the descended organ indexes of the ileum, cecum, and the shortened lengths of the ileum, cecum, and rectum were observed. Interestingly, these results are similar to those of previous studies, especially in chicks (Rezar et al., 2007; Manafi et al., 2015) and ducklings (Rafai et al., 2000; Tso et al., 2021).

Effect of T-2 Toxin on Feather Quality

Feathers play a role in regulating fowl body temperature, conserving heat, and resisting external damage, thus affecting appearance evaluation, health, productivity, and animal welfare. We discovered that T-2 toxin induced a significant decrease in feather quality in a dose-dependent manner compared to the control, particularly on the back, which is consistent with the delayed development of plumage in young ducks (Hayes and Wobeser, 1983; Rafai et al., 2000) and chickens sparsely covered in short protruding feathers (Wyatt et al., 1975; Yohannes et al., 2012). Similar to beak/oral lesions in other studies, T-2 toxin causes contact corrosion and irritation and generates malformed feathers in birds that are thin, uneven, shedding, and dirty (Yohannes et al., 2012). Additionally, T-2 toxin has adverse effects on nutritional imbalances (arginine, pyridoxine, and zinc deficiency), causing abnormal feathering; a more direct influence (necrosis) is specifically observed on the follicular tissue (Wyatt et al., 1975; Leeson and Walsh, 2004), the layer of regenerative cells in the feather base, and the basilar layer of the ramus (Hoerr et al., 1981; Yohannes et al., 2012). Moreover, T-2 toxin inhibits keratin synthesis and causes feather abnormalities (Nguansangiam et al., 2003). In practice, feather lesions and low growth performance are the main characteristics of T-2 toxin toxicity, as an indication of poisoning in poultry production (Yohannes et al., 2012).

Effect of T-2 Toxin on Bone Growth and Development

Leg (femur, tibia, and metatarsus) growth, development, and health are key concerns for growth performance, health, animal welfare, and profits in the poultry industry (Guo et al., 2019). SL, the indicator of metatarsus development, is a monitoring body size parameter of growth and development because of its close correlation with BW (Gao et al., 2010; Ukwu et al., 2014). In addition to BW, the inhibitory effect of T-2 toxin on growth was directly reflected in the slow growth of SL. Interestingly, there was a significant SL shortening as well as a significant drop in SLG after 21-d 2.0 mg/kg T-2 toxin exposure. Leg bones grow similarly and synchronously with BW to support BWG and life activities, thus directly influencing meat quality and skeletal abnormalities in meat geese (Yu et al., 2022). It is well known that the T-2 toxin is an environmental risk factor related to bone malformation, especially chondrocyte damage in the TGP, which are central sites regulating endochondral growth of chondrocyte formation, maturation, and turnover for bone elongation and repair (Farquharson and Jefferies, 2000; He et al., 2012). For instance, T-2 toxin is implicated in tibial dyschondroplasia (**TD**), which is characterized by the accumulation of immature, poorly differentiated, unmineralized, and avascular cartilage owing to intoxication and growth retardation of long bones in chickens (Nascimento et al., 2001; He et al., 2012). Furthermore, T-2 toxin participates in the pathological process of human Kashin-Beck disease (**KBD**) with chondrocyte damage to bone formation and morphogenesis (proliferation, differentiation, migration, and apoptosis) and imbalanced extracellular matrix (**ECM**) homeostasis (inhibition of synthesis and increase in degradation) (Li et al., 2016; Chang et al., 2017; Ning et al., 2021). Similarly, compared to the control, 2.0 mg/kg of T-2 toxin significantly inhibited the growth and development of tibia in goslings, reducing their length and weight. Breakage, necrosis, and excessive apoptosis of TGP chondrocytes in the hypertrophic zones were also observed. Our limited study could not confirm these TGP chondrocyte injuries as a TD or KBD model, which requires further investigation. However, we focused on these similar histopathological features under T-2 toxin exposure. Nutrient intake, hormone balance, and blood flow are key factors for normal bone growth and development (Waldenstedt, 2006; Marenzana and Arnett, 2013), and are toxicological targets of T-2 toxin. In this study, we speculated that T-2 toxin as a neurotoxin may lead to anorexia nervosa, reduce nutrient delivery, and cause hematological disorders such as anemia and variations in serum enzyme activities (e.g., Ca, P, and ALP), which may cause bone marrow necrosis and hematopoietic dysfunction, further aggravating blood burden, abnormal long bone (tibia and metatarsus) growth and development, and forming a recurring cycle, which was partly shown in chickens (Hoerr et al., 1982a,b; Janssens et al., 2000). T-2 toxin also reduces DNA synthesis in chondrocytes because of a basic depression of protein, DNA, and RNA (Wright Jr et al., 1987; Sokolović et al., 2008; He et al., 2012). Furthermore, T-2 toxin facilitates growth retardation and anomalous structural lesions of the TGP, with some unusual apoptotic and necrotic areas interfering with normal proliferation, differentiation, migration, mineralization of chondrocytes and synthesis, degradation of ECM, and the specific molecular

expression mechanism (Ning et al., 2021). In addition, inflammatory cytokines (mainly interleukin 1β) potently induce matrix degrading enzyme MMP expression in chondrocytes, inhibit the expression of matrix components, and evoke cell apoptosis, affecting chondrocyte growth under T-2 toxin exposure (Chang et al., 2017). Moreover, the T-2 toxin enhances catabolic activity and inhibits anabolic reactivity in hypertrophic chondrocytes through the ROS-NF-κB-HIF-2α pathway (Tian et al., 2012). Finally, leg bone development abnormalities (defective tibia and metatarsus) may affect walking and balance, cause paralysis (clearly observed in the 2.0 mg/kg T-2 toxin group in the final week), and increase mutual contact and the occurrence of feather pecking.

Effect of T-2 Toxin on Blood Parameters

The blood system is an important factor in maintaining homeostasis of the internal environment and is essential for the metabolism of tissues and cells throughout the body and for the sustainability of life, as it contains various nutrients, cellular metabolites, hormones, enzymes, and antibodies. In the present study, 2.0 mg/kg of T-2 toxin diminished blood RBC, RDW, MPV, and P-LCR, and elevated MCH and PLT, promoting the occurrence of hemorrhagic anemia syndrome, platelet dysfunction, and coagulopathy (Doerr et al., 1981; Parent-Massin, 2004). Notable changes in serum enzyme activity can reflect vital organ damage under toxic effects. Consistent with previous studies (Pande et al., 2006), T-2 toxin also triggered serum ALT, AST, and AST/ALT dependent on dosage, exhibiting abnormal hepatic dysfunction and damage owing to hepatic degeneration and subsequent leakage of enzymes into circulation (Wang et al., 2009). It has been reported that T-2 toxin causes liver glycolipid metabolism disruption (Kang et al., 2020), induces hepatic pathological symptoms (Garcia et al., 2003), provokes oxidative stress (Yang et al., 2016), and leads to apoptosis as well as CYP450 drug metabolism disorders (Osselaere et al., 2013) in animals. Furthermore, in line with previous reports (Pande et al., 2006), the increased serum BUN and UA levels, which is catabolized waste, indicated potential kidney injury caused by T-2 toxin (Gowda et al., 2010). BUN is the main end-product of protein metabolism and can be used as a diagnostic and screening indicator of glomerular filtration (Gowda et al., 2010). Under normal renal function conditions, small molecules are filtered out of the glomerulus. When glomerular filtration is reduced, particularly with renal insufficiency, BUN levels increase because of retention. UA is a waste product of purine catabolism, and high UA can readily lead to gout in the severe condition of long-term purine metabolism disorder and renal insufficiency, triggering immune responses (Rock et al., 2013). Few studies have reported the clinical symptoms of visceral gout in geese fed with moldy food containing T-2 toxin, but subsequent attempts at the experimental production of gout have failed (Chi et al., 1977b; Siller, 1981). However, another explanation is that this increase was dependent on the poor utilization of protein for the inhibition of protein synthesis by T-2 toxin rather than on kidney dysfunction (Chi et al., 1977c; Coffin and Combs Jr, 1981; Singh et al., 2020). Therefore, the causes and relationships between BUN, UA, and renal injury of T-2 toxin require further research in the future. In addition, serum ALB, GLOB, and A/G remained unchanged, whereas TP increased with T-2 toxin contamination in diets, which is inconsistent with earlier findings (Pande et al., 2006; Manafi et al., 2015; Tso et al., 2021), which were accompanied by decreased serum TP, ALB, and GLOB under hepatic injury and immunosuppression. Here, it appeared not to be a direct result of protein synthesis inhibition or immunosuppression, but a slight stimulatory effect. In addition, the Ca level was halved under 0.8 mg/kg T-2 toxin contamination and a stimulation of serum P and a decreased Ca/P ratio of 0.8 mg/kg T-2 toxin and a promotion of serum ALP in the presence of 1.0 and 2.0 mg/kg T-2 toxin were observed, which was consistent with the previous studies (Chi et al., 1977a; Kubena et al., 1989). The aberrant alterations of Ca, P, Ca/P, and ALP indicate that T-2 toxin had a significant negative effect on normal Ca absorption and ALP activation, thus disturbing the calcification and development of bones (Chi et al., 1977a; Sergeev et al., 1990; Yadav et al., 2011). Previous studies have shown that excessive phosphorus enhances bone fragility in laying fowls and the imbalance of calcium and phosphorus possibly triggers bone disease in growing poultry (Chi et al., 1977a; Waldenstedt, 2006; Elwinger et al., 2016). With multiple organ damage (for example, liver, kidney, and bones), serum LDH increased, similar to previous research because of the cytotoxicity of T-2 toxin on cell viability to induce the release of intracellular LDH into the circulatory system (Pande et al., 2006; Yang et al., 2016; Yang et al., 2019).

CONCLUSIONS

Collectively, T-2 toxin exhibits a negative influence on growth performance, feather quality, tibia development, and blood parameters in Yangzhou goslings, and is therefore hazardous to animal health and welfare and reduces economic efficiency.

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DISCLOSURES

The authors declare no conflicts of interest.

SUPPLEMENTARY MATERIALS

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.psj.2022.102382.

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