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*Full Length Research Paper*

# Clinical manifestation and growth performance of broiler chickens fed with T-2 toxin and co-infected with infectious bronchitis virus

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**The objective of the present study was to assess the effects of T-2 mycotoxin at dose level of 2 ppm in association with infectious bronchitis virus (IBV) infection on growth performance and clinical signs of broiler chickens. A total of 128 one-week-old chicks were classified into four groups and were treated respectively with T-2 toxin alone, IBV alone, T-2 toxin and co-infected with IBV, and untreated (control) for a period of 6 weeks. The treatment groups exhibited variable degrees of dullness, lethargy, and dehydration, intensity of which increased with duration of treatment. The T-2 toxicated birds, in addition, showed thin and haemorrhagic droppings. The birds treated with T-2+IBV exhibited severe weakness, recumbency, extending and dragging of neck on the ground and gasping (respiratory distress) at 48 h post IBV infection. Characteristic ruffled, thin shafted, uneven and fewer hairs of feathers were also noticed in almost all toxicated birds. Body weights were lower in toxin groups since 2nd week of toxin feeding. In IBV groups, however, birds did not show much difference from control, but it was higher than toxin groups. The mean percent body weight gains (BWG) and feed conversion ratios (FCR) in all treatment groups were significantly reduced from 3 weeks of toxin feeding or 3rd day post infection (DPI) onwards.**

**Key words:** Broiler chickens, growth performance, clinical signs, feather, IBV, T-2 toxin.

## INTRODUCTION

Mycotoxins are naturally occurring fungal metabolites contaminating feeds and their ingredients which are used for livestock, poultry and human consumption. Majority of the common mycotoxins are known for their cytotoxic, genotoxic, immunotoxic, carcinogenic and teratogenic effects (Binder et al., 2007, Glenn, 2007) and thus are responsible for increasing the susceptibility to several infectious agents and breakdown in immunity even in properly vaccinated flocks. In the environment they co-exist with other infectious agents like bacteria, virus or other pathogens and due to their immunosuppressive effects even the innocuous pathogens sometimes result in overwhelming diseases and cause heavy mortality in livestock and poultry. T-2 toxin is a member of trichothecene group of mycotoxins produced by *Fusarium* species and is widely prevalent in environment contaminating livestock and poultry feed. It is known for its cytotoxic and immunotoxic effects and is likely to alter pathology and pathogenesis of co-existing pathogens. The toxic effects of T-2 toxin in poultry include inhibition of protein, DNA and RNA synthesis. Cytotoxicity resulting in lesions in feathers/skin and low performance in poultry production (decrease in weight gain, poor feed conversion, egg production, and hatchability) are main features of T-2 toxicity (Shinozuka et al., 1997; Pestka et al., 2004). Infectious bronchitis virus (IBV) is a highly contagious pathogen mainly affecting farming birds with involvement of respiratory and urogenital systems.

Infectious bronchitis (IB), an acute, highly contagious and primarily respiratory infection, is characterised by tracheal rales, coughing and sneezing together with accumulation of excess mucus in bronchi; occurring at all ages and present in most poultry producing areas (Villegas, 1998). Infectious bronchitis virus (IBV) causes

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severe economic losses to the layers industry and it was also reported to reduce weight gain and feed efficiency in broiler chickens (Cavanagh and Naqi, 2003). T-2 toxin caused abnormal position of wing, reduction in feed intake and body weight gain. In laying birds it caused delayed maturation of follicles, reduced egg production, shell thickness and hatchability in poultry (Kurkure and Pande, 2008). Krishnamoorthy et al. (2008) observed that wing feathers of broiler chickens which were fed 1 ppm T-2 toxin mixed feed for 28 days were seen shortened with clear primary and secondary wing feathers on the $2^{nd}$ and $4^{th}$ week of age. This pattern of wing growth was attributed to the effect of T-2 toxin on protein synthesis as a result of liver damage and inhibition of amino acid synthesis, indirectly affecting the growth of wing feathers in broiler chickens. Mortality rate of 5% due to T-2 toxicosis was also observed by Balachandra et al. (2008) in broiler chickens. Experimental T-2 mycotoxicoses was induced by Prasath et al. (2008) in 30 turkey poults by feeding diets containing 0, 1 and 3 ppm T-2 toxin from 0 to 28 days of age. Significant ($P<0.05$) decrease in body weight gain was observed in 3 ppm T-2 toxin group from the first week onwards when compared with the control and the other treatment groups. The T-2 toxin fed birds showed inappetence, poor growth, diarrhoeic droppings, weakness and depression. Defective and abnormal formation of primary and secondary feathers in wing with short angular feathers was also observed indicating that T-2 toxin at the level of 1 and 3 ppm caused disturbance in growth performance. Similarly, IBV, in addition to the most common of clinical manifestations, resulted in depression and retarded growth in chickens (Cavanagh and Naqi, 2003) and feed consumption and weight gain are significantly reduced (Otsuki et al., 1990).

Though there is no or scant previous report on either natural or experimental interaction of IBV infection and T-2 mycotoxin in chickens, yet the possibility of their co-existence in nature could not be ruled out. Under such circumstances, T-2 toxin is expected to alter the course and pathogenic potential of IBV infection. Understanding the combined effects of T-2 mycotoxin and IBV infection on poultry health, production and productivity has been timely and demanding research work to design and apply appropriate mitigating strategies of the two. Hence, the objective of the present study was designed to make detailed investigation on clinical and growth performance disturbances in chickens fed with T-2 toxin contaminated feed alone or in association with infectious bronchitis virus (IBV) infection on clinical signs and performances of chickens.

## MATERIALS AND METHODS

### Production and analysis of T-2 mycotoxin

T-2 mycotoxin was produced on sterile maize and wheat as described by AOAC (1995). The cultures of *Fusarium sporotrichioioides var. Sporotrichioides* NRRL 3299, supplied by the National Centre for Agricultural Utilization Research (Peoria, Illinois, USA), and MTCC1894, procured from the Institute of Microbial Technology (Chandigarh, India), were used to produce T-2 mycotoxin on partially ground maize and intact wheat grains. The concentration of T-2 mycotoxin was estimated by thin layer chromatography (TLC) at Animal Feed Analytical and Quality Control Laboratory (AFAQCL), Veterinary College and Research Institute (Namakkal, Tamil Nadu, India).

### IBV Propagation and $EID_{50}$ determination

Infectious bronchitis virus (IBV) isolate (India/LKW/56/IVRI/08 serotype/pathotype) of chickens used in the study was obtained from the Avian Disease Section (Division of Pathology), IVRI, India. The isolate was propagated in embryonated chicken eggs by serial passages with its 10 fold serial dilutions. Seven groups of five eggs each were inoculated via allantoic membrane, with each dilution of $10^0$, $10^2$, $10^3$, $10^4$, $10^5$, $10^6$ and $10^7$, respectively and the presence of virus in each embryo was checked by observing the lesions (curling and dwarfing of embryos) from five to seven days after inoculation and embryo deaths were recorded. The virus titre in terms of 50% egg infective dose ($EID_{50}$) per 0.2 ml was calculated following the procedure of Reed and Muench (1938). The chickens were inoculated intranasally and intraocularly with 0.2 ml $EID_{50}$ ($10^{5.69}$/) of the IBV isolate (Indian/LKW/56/IVRIV/08) per bird.

### Experimental chicks

The experiment was conducted using a total of 128, one week-old, *Cornish-Rock* strain, both sex broiler chicks, procured from the Hatchery Unit of Central Avian Research Institute (CARI), Bareilly, India. All the experimental procedures were conducted as per the guidelines of the Institute Animal Ethics Committee (IAEC) and Committee for the Purpose of Control and Supervision of Experiments on Animals (CPCSEA). The chicks were kept under standard management conditions in the poultry shed of Avian Diseases Section (Division of Pathology), IVRI. The birds were checked for the presence of maternally derived AB (MDAB) antibodies against IBV by SVANOVIR IBV-AB ELISA kit (SVANOVIR, Sweden) to ensure their sero-negative status prior to experimental IBV infection. They were provided with fresh water *ad libitum* during the entire experimental period and anti-microbial treatment at their 1 to 2 weeks of age. The chickens were vaccinated to protect them from Newcastle disease and the experimental feeds were given as per the following.

### Experimental feed

The substrates (maize and wheat) containing the known amount of T-2 toxin were mixed to the basal feed (tested negative for mycotoxin contamination by ELISA) to make the desired concentration of T-2 toxin in the diet that is 2 ppm (2 mg/kg feed). The amount of T-2 toxin, 2 ppm, was chosen based on previous works, where 2 ppm of T-2 was found to develop pathology in chickens, whereas here its toxicity was studied in association with IBV pathogenesis and pathology. Aliquots were taken from the mixed feed and the toxin was quantified by ELISA (Romer lab, Singapore) to ensure proper mixing.

### Experimental design

One hundred and twenty eight 1-week-old broiler chicks were separately wing-tagged, weighed and randomly distributed to four groups (T-2, IBV, T-2 + IBV and control groups) of 32 birds each.

a b. c.

**Figure 1.** Clinical signs manifested by different groups at different interval. a. Toxin group, 4 WTF, Thin mucoid and haemorrhagic droppings. b. T-2+IBV group, chick at 4 (10) WTF (DPI) showed depressed, unable to move and respiratory distress. C. T-2+IBV group, chicks at 5 WTF showed weakness, depression, stretching feather and open mouth indicating respiratory distress.

To avoid IBV cross contamination, the two groups (Control and T-2 groups) were kept in the same pen with separate cages; while the other two groups (IBV and T-2-IBV groups) were kept in another pen with separate cages. The experimental diets were given to each group of chick for a period of 6 weeks starting at the age of 1 week, as per the following: T-2 Group (fed with toxicated feed from 1 to 7 weeks), IBV group (fed with toxin free basal feed from 1 to 7 weeks and infected with IBV at 3$^{rd}$ week of toxin feeding), T-2 + IBV group (fed with toxicated feed from 1 to 7 weeks and co-infected with IBV at 3$^{rd}$ week of toxin feeding), and control group (fed with toxin free control feed from 1 to 7 weeks). The birds were observed daily for clinical symptoms and mortality, weekly for body weights and feed consumption. Body weight gains (BWG) were calculated weekly and feed conversion ratios (FCR) were derived from feed intake and body weight gains by adopting the following formula:

$$FCR = \frac{\text{Average feed consumed/bird during the week (g)}}{\text{Average weight gain/bird during the week}}$$

The data generated were analysed using Graph Pad Prism (4) Software and presented as Mean±SE. One-way analysis of variance (ANOVA) was used to compare differences among groups and the means were compared by Tukey's Multiple Comparison Test and comparisons were considered significant at $P<0.05$ (Snedecor and Cochran, 1989).

## RESULTS

### Clinical signs

Birds belonging to all treatment groups exhibited variable degree of dullness, lethargy, and dehydration, with intensity of signs increasing with duration. Thin mucoid and haemorrhagic droppings (Figure 1a) in toxin groups were recorded. Severe weakness, unable to move and stand, extending and dragging of neck on the ground, stiffening of legs, torticollis and gasping (respiratory distress) followed by death of four chicks (12.5% mortality rate) at 48 h post IBV (Figure 1b and c) infection were observed in T-2+IBV group. However, no mortality was observed neither in toxin alone nor virus alone groups. In the current experiment, feathers of T-2 toxin fed chicks became ruffled, thin shaft with uneven and fewer hairs as compared to feathers of the control chicks. These changes progressed with time and were more severe in T-2 + IBV group. The feather lesions were noticed in the toxin fed birds during the entire study period starting from 3 weeks of toxin feeding, and it was more severe at the end (6 WTF) and in T-2+IBV groups (Figure 2a, b and c) as compared to control (Figure 2d).

### Growth performance

The means and standard errors for each of the parameters are given in Tables 1 and 2. The feeding of contaminated feed as well as IBV infection did not affect ($P>0.05$) feed consumption as compared with control (Table 2). Body weights in toxicated birds starting from 2(0) weeks of toxin feeding (WTF) or days of post IBV infection (DPI) and in IBV group starting 3(3) WTF (DPI) onwards, were significantly ($P<0.05$) lower as compared with that of the control group, on the other hand, the mean body weight of IBV groups was higher than toxicated groups (Table 1). The mean percent body weight gains (BWGs) of the treatment groups, were significantly ($P<0.05$) lower than that of the control group at all observation intervals (Table 2). The mean percent BWGs in T-2+IBV group were significantly ($P<0.05$) lower than the IBV groups at 5 and 6 WTF or 17 and 21 DPI (Table 2). The mean feed conversion ratios (FCR) were significantly ($P<0.05$) reduced in the toxicated groups at all intervals starting from 4 (10) WTF (DPI) onwards as compared to the control. While at 1(0) and 6(21) WTF (DPI), the mean FCR in T-2+IBV group were significantly lower as compared to T-2 group (Table 2).

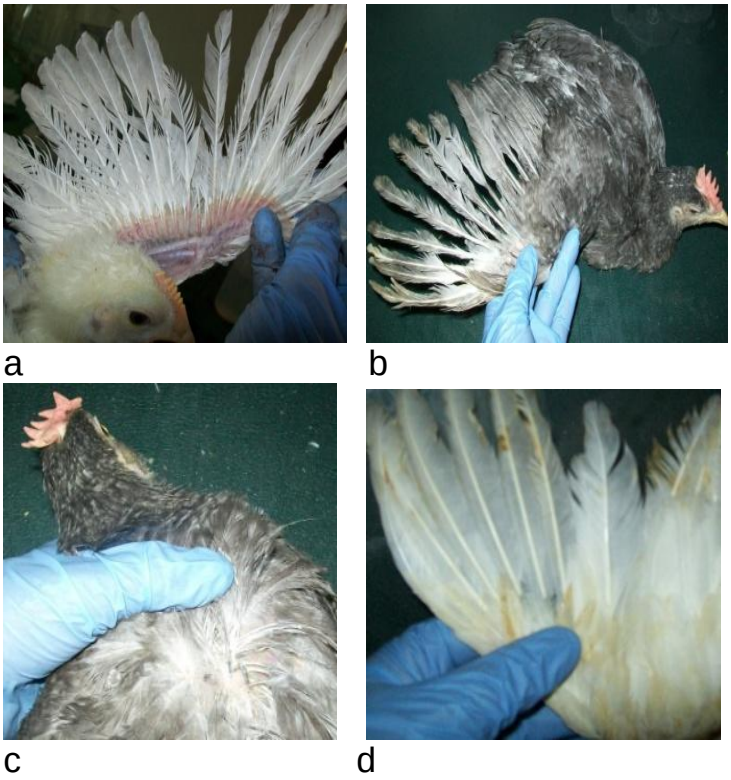


**Figure 2.** Narrow, thin and ruffled feather. a. T-2 Group, 5 WTF, Narrow and stunt feathers growth. b and c. T-2+IBV group, 5 (17) WTF (DPI), narrow feathers and loss of back hair. d. Control group, 5 week: Normal feather growth.

**Table 1.** Effect of T-2 toxin and IBV on body weights (g) in broiler chicks.

| WTF (DPI) | T-2 | IBV | T-2+IBV | Control |
|---|---|---|---|---|
| 1(0) | 153.00± 1.16$^{a}$ | 156.50± 1.29$^{a}$ | 155.00± 0.82$^{a}$ | 157.00± 0.82$^{a}$ |
| 2(0) | 221.00± 0.82$^{a}$ | 231.00± 0.82$^{b}$ | 223.00± 1.41$^{a}$ | 233.00± 0.82$^{b}$ |
| 3 (3) | 325.00± 0.82$^{a}$ | 335.60± 0.82$^{b}$ | 333.00± 0.82$^{c}$ | 340.00± 0.82$^{d}$ |
| 4(10) | 416.50± 0.41$^{a}$ | 423.30± 0.24$^{b}$ | 420.90± 0.74$^{b}$ | 444.50± 0.41$^{d}$ |
| 5(17) | 576.00± 0.82$^{a}$ | 592.50± 0.41$^{b}$ | 581.60± 0.33$^{c}$ | 647.00± 0.82$^{d}$ |
| 6(21) | 616.00± 2.16$^{a}$ | 713.00± 1.63$^{b}$ | 680.00± 1.63$^{c}$ | 770.00± 0.82$^{d}$ |

Means bearing at least one common superscript (a, b, c and d) do not differ significantly between groups ($P \leq 0.05$), WTF=Weeks of toxin feeding, DPI=days post infection.

## DISCUSSION

These clinical manifestations exhibited by the experiment birds are similar with the work done by Prasath et al. (2008) who reported diarrhoeic droppings, weakness and depression in turkey poults fed with 1 and 3 ppm T-2 toxin contaminated feed. In another study carried out by Madheswaran et al. (2005), T-2 toxin treated birds also showed brown diarrhoeic droppings, stiffening of legs and torticollis in Japanese quail. Cavanagh and Gelb (2008) reported that chicks infected with IBV exhibited characteristic respiratory signs like gasping and sneezing. However, the new thing in the present study is that it has revealed more severe clinical signs in T-2 toxicated birds and co-infected with IBV which might possibly be due to the fact that T-2 toxin might have damaged the local and systemic defence mechanisms of the chicks against the IBV infection and aggravated its pathogenicity. The effects of T-2 toxin and IBV infection on many local and systemic cellular mechanisms may be explained as the underlined reason for the development of the aforementioned clinical signs. The mortality

**Table 2.** Effect of T-2 toxin and IBV on feed intakes (g), percent body weight gains (%) and feed conversion ratios in broiler chicks.

| WTF (DPI) | Parameter | T-2 | IBV | T-2+IBV | Control |
|---|---|---|---|---|---|
| 1(0) | BWG (%) | 49.67± 1.41$^{a}$ | 49.23±1 .29$^{a}$ | 45.16±1.63$^{a}$ | 57.06±5.42$^{b}$ |
| | FI(g) | 210.00±1.41$^{a}$ | 210.00±1.41$^{a}$ | 210.00±1.41$^{a}$ | 210.00±1.41$^{a}$ |
| | FCR (%) | 35.78± 2.05$^{a}$ | 37.91±1.61$^{a}$ | 33.39±1.80$^{b}$ | 44.36±3.29$^{c}$ |
| 2(0) | BWG (%) | 27.57±0.83$^{a}$ | 26.70±1.83$^{b}$ | 29.80±1.29$^{c}$ | 30.04±1.83$^{d}$ |
| | FI(g) | 245.00±1.41$^{a}$ | 245.00±1.41$^{a}$ | 245.00±1.41$^{a}$ | 245.00±1.41$^{a}$ |
| | FCR (%) | 25.74±0.96$^{ab}$ | 24.09±0.43$^{ab}$ | 27.79±1.30$^{ac}$ | 28.59±0.85$^{bd}$ |
| 3 (3) | BWG (%) | 34.20±1.41$^{a}$ | 34.15±0.95$^{a}$ | 33.00±1.15$^{a}$ | 31.47±1.15$^{d}$ |
| | FI(g) | 280.00±1.15$^{a}$ | 280.00±1.15$^{a}$ | 280.00±1.15$^{a}$ | 280.00±1.15$^{a}$ |
| | FCR (%) | 39.67±1.43$^{a}$ | 40.96±1.31$^{a}$ | 39.31±1.21$^{a}$ | 38.24±1.18$^{a}$ |
| 4(10) | BWG (%) | 21.97±0.58$^{a}$ | 20.72±0.48$^{b}$ | 20.88±0.04$^{b}$ | 23.51±0.58$^{c}$ |
| | FI(g) | 315.00±1.83$^{a}$ | 315.00±1.83$^{a}$ | 315.00±1.83$^{a}$ | 315.00±1.83$^{a}$ |
| | FCR (%) | 29.10±1.02$^{ab}$ | 27.87±1.00$^{ac}$ | 27.05±1.13$^{bc}$ | 33.21±1.18$^{ad}$ |
| 5(17) | BWG(g) | 27.70±1.29$^{a}$ | 28.70±1.83$^{b}$ | 27.62±0.82$^{a}$ | 31.30±0.71$^{c}$ |
| | FI(g) | 315.00±1.83$^{a}$ | 315.00±1.83$^{a}$ | 315.00±1.83$^{a}$ | 315.00±1.83$^{a}$ |
| | FCR (%) | 51.71±2.47$^{a}$ | 54.02±1.77$^{a}$ | 50.06±1.97$^{a}$ | 64.37±2.66$^{b}$ |
| 6(21) | BWG (%) | 6.50±1.41$^{a}$ | 15.71±1.15$^{b}$ | 14.47±0.58$^{c}$ | 15.97±0.82$^{b}$ |
| | FI(g) | 350.00±1.63$^{a}$ | 350.00±1.63$^{a}$ | 350.00±1.63$^{a}$ | 350.00±1.63$^{a}$ |
| | FCR (%) | 30.45±0.76$^{a}$ | 32.03±1.12$^{cd}$ | 28.14±1.03$^{c}$ | 35.17±0.92$^{d}$ |

Means bearing at least one common superscript (a, b, c and d) do not differ significantly between groups ($P \leq 0.05$), WTF=Weeks of toxin feeding, DPI=days post infection, BWG=body weight gain, FI=feed intake, FCR=feed conversion Ratio.

occurred in the combined group which indicated that there was synergistic effect and T-2 toxicity might have reduced the resistance of the birds to IBV infection. Feather lesions showed by almost all toxicated birds, were important findings in this study and gained support from the earlier work by Hoerr (2003) who reported similar feather changes (thin, uneven and poorly developed feather) in birds naturally toxicated with T-2 toxin. Hoerr et al. (1982) also reported malformed feathers in T-2 toxin fed birds. Due to cytotoxic radiomimetic effects, the pattern of wing growth was attributed to the effect of T-2 toxin on protein synthesis, as a result of liver damage and inhibition of amino acid synthesis, indirectly affecting the growth of wing feathers in broiler chickens.

However, no oral lesion was observed in this study, which was reported by many previous workers (Hoerr et al., 1982; Hoerr, 2003; Madheswaran et al., 2005). The possible explanation for this may be due to difference in dosage. The present findings with regard to weekly body weights (BW), body weight gains (BWG), and feed conversion ratios (FCR) are supported by many previous workers. In a study carried out by Madheswaran et al. (2005) on the growth performances, namely (BW), (BWG), feed intake (FI) and (FCR) of chicks revealed that BW was lower in toxin groups since 2$^{nd}$ week of toxin feeding. Moreover, the preset findings are in agreement with the findings of Pande et al. (2006) who reported a significant decrease in BW of broiler chicks fed with 2 ppm T-2 toxin from the second week onwards. Diaz et al. (2005) also reported that 2 ppm level of T-2 toxin significantly reduced BW in broiler chicks at 28 days. BW in IBV groups did not show much difference from control, however, it was higher than toxin groups. The mean percent body weight gains (BWG) and feed conversion ratios (FCR) in toxin as well as virus groups (T-2, IBV and T-2+IBV) reduced from 3 weeks of toxin feeding onwards or 3 days of post IBV infection. At 5(17) and 6(21) WTF (DPI), BWGs of T-2+IBV group was significantly lower than IBV groups and the mean FCR of T-2+IBV group was significantly lower as compared to T-2 group. The reduction in BWG and FCR matched with previous findings by Diaz et al. (2005) who reported significant reduction in BWG in broiler chicks fed 2 ppm T-2 toxin for 28 days. The present observations conformed well with earlier reports (Hoerr et al., 1982; Kubena et al., 1994; Hussein et al., 2001; Ogunbo et al., 2007; Prasath et al., 2008) where T-2 toxin at 3 to 6 ppm levels reduced BWG and FCR. Moreover, Cavanagh and Gelb (2008) opined that IBV significantly reduced BWG in chicks.

However, unlike BWG and FCR, there was no change in feed intake (FI) in any of the groups during the entire experimental period and found support from Diaz et al. (2005) which contradicts the findings of Hussein et al. (2001) who observed a significant difference in FI among T-2 toxin fed broiler chickens at level of 2 ppm. Reduction in BW of T-2 toxicated chicks may be due to inflammation, contact erosion and irritation of digestive system resulting into decrease in feed digestion and absorption and consequently decrease in BWG, FCR of toxicated birds. It is concluded that T-2 toxin feeding resulted in overt clinical signs, mortality, feather lesions and lowered growth performances in broiler chicks. The severity of which being more in birds fed with T-2 toxin and co-infected with IBV. This indicated that T-2 toxin increased the susceptibility of birds to IBV infection, and it can also be concluded that T-2 toxicity may cause IBV vaccine failure. Therefore, an integrated control and prevention strategies need to be designed and implemented on both T-2 toxin contamination poultry feed and IBV infection in the poultry industry.

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