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PATHOLOGY OF FUMONISIN TOXICITY IN THE ORGANS OF GASTROINTESTINAL SYSTEM IN BROILER CHICKEN*

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Fumonisin B₁ (FB₁) has now emerged as one of the most important mycotoxins which contaminates food and poultry feed (Scott, 1993; Shetty and Bhat, 1997). Various biochemical and pathological effects of FB₁ in turkeys and chicken have been described (Espada *et al.*, 1994; Ledoux *et al.*, 1996; Bermudez *et al.*, 1997). In this paper pathological changes induced by fumonisin toxin in the digestive tract of broiler chicken dosed with FB₁ are reported.

The freeze-dried culture of *Fusarium moniliforme* MRC826 was a kind gift from Professor W.F.O. Marasas of South African Medical Research Council. It was sub-cultured in autoclaved glass fruit jars containing 400 grams of maize and incubated at room temperature in the dark in an open room for a period of 7 weeks. After incubation, the cultures were autoclaved and dried in hot air oven at 40°C for 48 hours. The dried cultures were ground into powder and stored at 4°C until required. Two different batches of pooled dried maize cultures yielded 1600 and 4500 ppm of FB₁ toxin, respectively on estimation by thin layer chromatography (Rottinghaus *et al.*, 1992). A total of 120 day-old commercial broiler chicks were divided into four groups of 30 chicks each and fed with FB₁ at 0 (group 1), 80 (group 2), 160 (group 3) and 320 ppm (group 4) from day one to four weeks. The percentage of *Fusarium moniliforme* culture material incorporated in the diets were 0, 1.8, 3.6 and 7.2 respectively for the four groups. Six birds from each group were sacrificed at 1st, 2nd and 3rd

week and the remaining birds at 4th week. Detailed post-mortem was conducted on the sacrificed birds and tissue pieces from oesophagus, crop, proventriculus, gizzard and intestine were collected in 10% formal saline for histopathological study.

Grossly the mucosa of crop was found to be thickened predominantly in the birds of group 4 (320 ppm) after the 3rd week and 4th week of toxin exposure. Hypovitaminosis A caused by fumonisin toxicity (Hall *et al.*, 1995) might have caused this condition with increased keratinization. Proventriculus of the birds of groups 3 & 4 (160 and 320 ppm) during the fourth week revealed tenacious mucus, which might be due to the direct irritating effect of fumonisin and not due to the culture material since the culture material added were less than eight per cent of the diet (Brown *et al.*, 1992). Intestines of all the toxin treated birds showed catarrh with copious mucus secretion from second to fourth week. More or less similar findings were reported by Marijanovic *et al.* (1991).

Microscopically the mucosa of crop was thrown into folds with papillomatous projections and also showed hyperkeratosis. Focal areas of mononuclear cell infiltration was observed in the sub mucosa. These changes were consistently seen in all the treated groups (groups 2, 3 and 4) from second week onwards till the end of experiment. In the oesophagus hyperplasia of lining epithelial

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cells, infiltration of mononuclear cells in the lamina propria and dilatation of oesophageal glands (Fig. 1) were consistently seen from second week to fourth week in all the treated groups (groups 2, 3 and 4). Proventriculus showed lengthening of villi and infiltration of cells in the lamina propria, which were encountered in all the treated groups throughout the experiment. In addition, mucosal epithelial villi appeared shortened and broadened with infiltration of cells in the lamina propria in chicks of group 4 (320 ppm) from third week onwards. The koilin layer of gizzard in the birds of group 4 (320 ppm) was very much thickened from second week onwards and the sub mucosal glands were also found to be dilated. Intestines of all the treated groups (groups 2, 3 and 4) showed catarrhal changes characterized by increased mucus cell activity and mononuclear cell infiltration after third and fourth week of toxin exposure. Most of the lesions observed in this study in crop, oesophagus, proventriculus and gizzard mimicked symptoms of vitamin A deficiency (Calnek *et al.*, 1991). This is in conformity with earlier report wherein decreased serum vitamin A level was noticed in broiler chicken treated with FB₁ (Hall *et al.*, 1995).

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