Iraqi Journal of Veterinary Sciences, Vol. 21, No. 1, 2007 (75-86)

INEFFECTIVENESS OF DIFFERENT ADSORBENTS IN ALLEVIATION OF ORAL LESIONS INDUCED BY FEEDING T-2 TOXIN IN BROILER CHICKENS

A. M. SHAREEF
Department of Veterinary Public Health, College of Veterinary Medicine,University of Mosul. Mosul-Iraq.

(Received: September 19, 2006; Accepted: April 18, 2007)

ABSTRACT

One hundred and forty one-day-old-male broiler chicks (Ross), were divided into 7 groups (20 chicks/group) and fed T-2 toxin alone or with five different types of adsorbents; Montmorillonite 0.5%, Vermiculite 0.5%, Pigacine 0.5%, Gezilgure 0.5%; and the resin Perlite 0.5%. Gross and microscopic examinations reveal that all adsorbents used were unable to alleviate the oral lesions induced by T-2 toxin in broiler chickens.

عدم تاثيرأضافة أنواع مختلفة من الممتزات في تخفيف الأفات الفموية التي يحدثها سم T-2 في أفراخ فروج اللحم

عقيل محمد شريف
فرع الصحة العامة البيطرية، كلية الطب البيطري، جامعة الموصل، العراق

الخلاصة

تم تغذية 140 من افراخ فروج اللحم نوع (Ross) بعمر يوم واحد موزعة على 7 مجاميع (20 فروج لكل معاملة) على علائق ملوثة بسم T-2 بمفرده او مع اضافة خمسة انواع من الممتزات ; المونتموريلونايت؛ 0.5% الفيرميكيلولايت 0.5% والبيكاسين0.5% والكيزلكور0.5% ; وبولمير الراتنج البرلايت0.5% . لم تخفف جميع هذه الممتزات المظافه من شده الافات الفموية الناجمة عن استهلاك سم T-2 عند دراستها عيانيا ومجهريا.

INTRODUCTION

Trichothecene mycotoxins are produced by Fusarium and its perithecial stages (1). T-2 toxin, one member of group A trichothecenes, was first isolated from *fusarium tricictum* strain T-2 by Bamburg (2) and Bamburg et al., (3). Feeding T-2 toxin to broilers cause feed refusal, impaired growth and whole body pathology including caustic injury to skin and alimentary mucosa; radiomimetic injury to bone marrow, lymphoid tissues, gastrointestinal tract, and hepatitis; and thyroid alteration(4). T-2 toxin causes erosive and exudative injury to the oral mucosa of poultry fed toxin-appended diets (5, 6, and 7)).These oral lesions were characterized by the presence of focal, yellow oral plaques that progress to yellow gray, raised accumulations of exudates with underlying ulcers located near major

salivary duct openings on the palate, tongue, and buccal floor. Thick crusts accumulate along the interior margin of the beak. Oral histology confirms mucosal necrosis and ulceration, sub mucosal granulation tissue and inflammatory cells, crusts of exudates, bacterial colonies, and feed components. (5; 8; 9; 10; 11; 12; 13; 14). The most applied method for protecting against mycotoxicosis is the utilization of adsorbents mixed with the feed which are supposed to bind the mycotoxins efficiently in the gastrointestinal tract, (15). Most studies related to the alleviation of mycotoxicosis by the use of adsorbents are focused on aluminosilicates, mainly zeolites and Hydrated sodium calcium aluminosilicates(HSCAS), and aluminumonsilicate-containing clays. All consisting of aluminates, silicates and some interchangeable ions, mainly alkali metal and alkaline earth metal ions (16).Clay minerals are primarily layered silicates with the common chemical formula[Sio2$O^2$5] xy, e.g. Bentonit, vermiculite, Kaolin, Pigacin, Gezilgure. HSCAS contain calcium ions and protons which are exchanged against the naturally occurring sodium ions. They are a type of montmorillonite belonging to phyllosilicates which are composed of layers of aluminum and silicon connected in a 1:1 or 2:1 arrangements. Polymers, an ion exchange mycotoxin adsorbent resins were also tried to be used in vitro studies for adsorption of certain mycotoxins (15,17). As a general aluminosilicates are the preferred adsorbents, followed by activated charcoal and special polymers(16). The aim of the present study was to evaluate different adsorbents; Montmorillonite (HSCAS); new aluminosilicte-containig clays (Vermiculite, Pigacin, Gezilgure); and new polymer resin (Perlite), in an attempt to alleviate necrotic oral lesions induced by feeding T-2 toxin to broiler chickens.

MATERIALS AND METHODS

A total of 140, 1-d-old male meat-type chickens of the Ross line were used. The experimental design consisted of a complete randomized trial with one negative control group, one positive control group, and four feed additives : montmorillonite(HSCAS); Aluminumsilicate- containing clays Vermicullite, , pigacine, gezilgure; and polymer resin, perlite. The experimental treatments consisted of the same commercial mash, based on corn and soybean meal, according to the recommendations of the NRC (1994) (18), with T-2 toxin alone or with different additives as follows:

Group 1: no T-2 toxin or feed additive (negative control).
Group 2: 8 ppm T-2 toxin (positive control).
Group 3: 8 ppm T-2 toxin + 0.5% montmorillonite.
Group 4: 8 ppm T-2 toxin + 0.5% Vermicullite.
Group 5: 8 ppm T-2 toxin + 0.5% pigacine.
Group 6: 8 ppm T-2 toxin + 0.5% gezilgure.
Group 7: 8 ppm T-2 toxin + 0.5% perlite.

The experimental ration was checked to contain no detectable levels of aflatoxins, ochratoxins, zearalenone, and T-2 toxin by the method reported by Coker et al (1984) (19). Purified crystalline T-2 toxin was produced by culturing *F. tricinctum* NRRL 3299 according to the method reported by Burmeister et al. (20). The crystalline toxin was dissolved in acetone, added to experimental diets, and mixed to homogeneity by means of a twin- shell blender. Homogeneity was tested by analyzing the presence of T-2 toxin in each diet by thin–layer chromatography. The chemical composition of Aluminum silicate feed additives

are presented in table 1. Chickens were kept in batteries in a thermo regulated room with continuous light; feed and water were provided *ad libitum*. On 21 day of age heads were removed from all chicks and visually scored for oral lesions using a four –point scoring system ranging from 1 to 4 (5) by the same individual without knowledge the treatment groups. A lesion score of 1 indicated no visible lesions; a score of 2 was seen as one or two mouth lesions clearly visible on either the lower or upper mandible; a score of 3 was seen as more than one or two mouth lesions clearly visible on either the lower or upper mandible and back of the tongue ; a lesion score of 4 was seen as large lesions occurring at several sites within the mouth, principally on the upper and lower mandibles, the corners of the mouth, and the back of the tongue. Histopathological study of oral lesions was performed by fixing affected portions in 10% neutral buffered formalin. Fixed tissues were trimmed, embedded in paraffin, sectioned at 4 µm, and stained with hematoxylin and eosin stain. Tissue samples from treatments show oral lesions were examined microscopically. Data (pen means) for all response variables in each treatment were subjected on ANOVA using the SAS software (SAS 2000) general Linear Models procedure for factorial ANOVA. Treatment means were ranked by Duncan's multiple range test (SAS 2000). All statements of significance are based on the 0.05 level of probability (21).

RESULTS

Oral lesion scores: The oral lesion scores in all treatment groups fed purified T-2 toxin with five different aluminum silicate feed additives show no significant difference in developing oral lesions when compared with group of chicks fed T-2 toxin alone, but all were significantly ($p<0.05$) differ from control group fed no toxin or additives(Table 1).

Table 1: Effect of T-2 toxin alone and T-2 toxin with feed additives on broiler oral lesions at 21days of age[1] .

Treatments	Feed additive (%)	T-2 toxin (ppm)	Oral lesion score[2]
Control	0.00	0	0.0 b*
T-2 toxin	0.00	8	2.30 ±0.14a
T-2 toxin + montmorillonite	0.5	8	2.20±0.14 a
T-2 toxin + Vermicullite	0.5	8	2.10±0.11 a
T-2 toxin + pigacine	0.5	8	2.00±0.14 a
T-2 toxin + gezilgure	0.5	8	2.30±0.16 a
T-2 toxin + perlite	0.5	8	2.00±0.12 a

*Values within column with no common subscripts differ significantly ($p<0.05$)

[1] Values = X±SEM of two groups of 10 broilers.

[2] Lesions were scored on a scale from 0-4, with a value of 4, representing the more sever lesions.

Gross lesions: Oral lesions were reported in all groups fed T-2 toxin alone or with different feed additives and were characteristic of T-2 toxicosis which first appeared when the chicks were about 1 week old, as raised ceasous yellow-white plaques. By 2-weeks, the lesions increased in size and invaded the lingual papillae at the root of the tongue, margin of the beak, mucosa of the hard palate

and angle of the mouth. By three weeks, the size of the lesions increased to more extent that of the 2nd week. These lesions were characterized by raised accumulation of exudates with underlying ulcers located near major duct opening of the palate, tongue, and buccal floor. (Figure 1, 2, 3, 4, 5, 6)

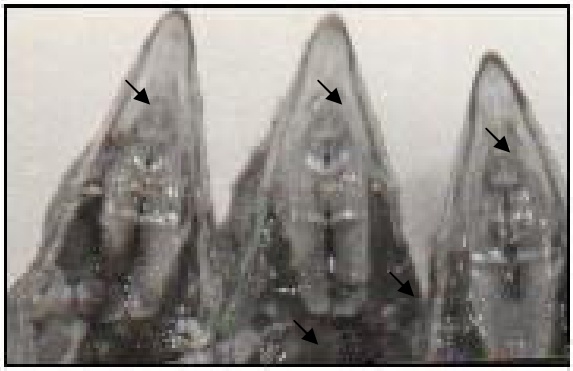

Figure 1: Beak and palate ulceration and crusting in broiler chickens following 21 days consumption T-2 toxin (8 ppm), characterized by raised accumulations of exudates with underlying ulcers located near major salivary duct openings on the palate.

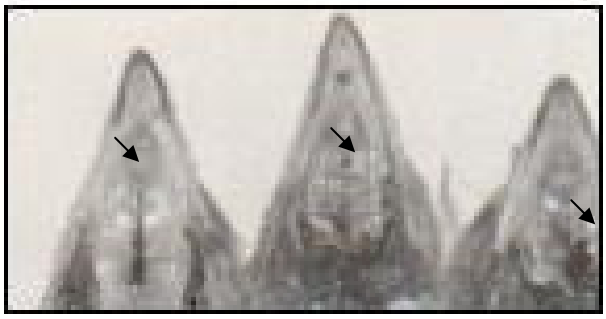

Figure 2: Beak and palate ulceration and crusting in broiler chickens following 21 days consumption 8 ppm T-2 toxin and 0.5% montmorillonite, characterized by raised accumulations of exudates with underlying ulcers located near major salivary duct openings on the palate.

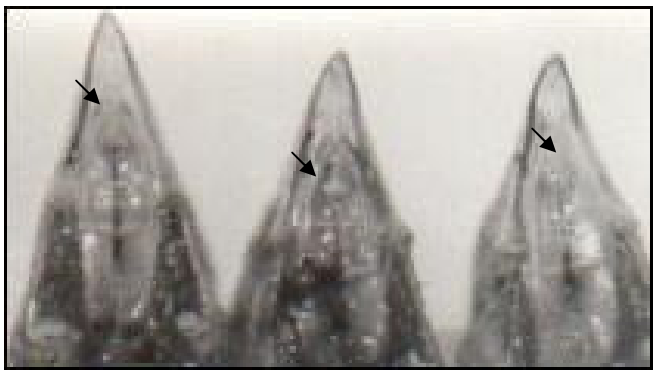

Figure 3: Beak and palate ulceration and crusting in broiler chickens following 21 days consumption 8 ppm T-2 toxin and 0.5% vermicullkite, characterized by raised accumulations of exudates with underlying ulcers located near major salivary duct openings on the palate.

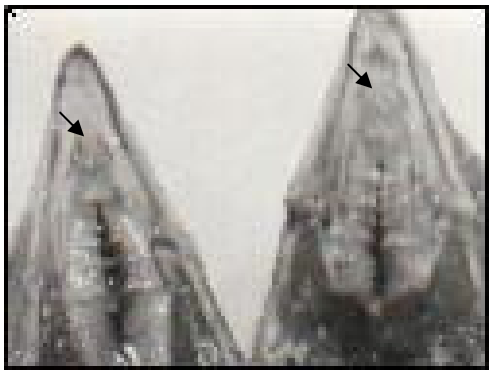

Figure 4: Beak and palate ulceration and crusting in broiler chickens following 21 days consumption 8 ppm T-2 toxin and 0.5% pegacine, characterized by raised accumulations of exudates with underlying ulcers located near major salivary duct openings on the palate.

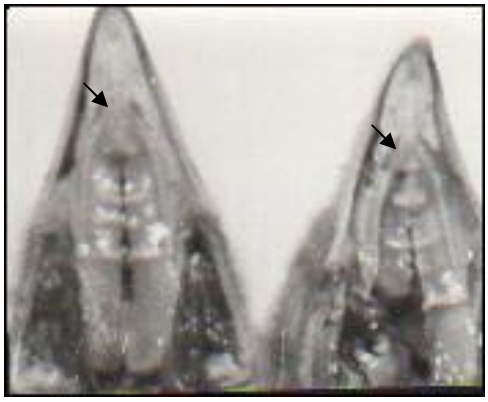

Figure 5: Beak and palate ulceration and crusting in broiler chickens following 21 days consumption 8 ppm T-2 toxin and 0.5% gezilgure, characterized by raised accumulations of exudates with underlying ulcers located near major salivary duct openings on the palate.

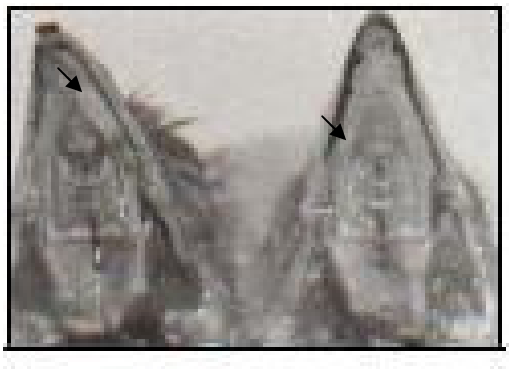

Figure 6: Beak and palate ulceration and crusting in broiler chickens following 21 days consumption 8 ppm T-2 toxin and 0.5% perlite, characterized by raised accumulations of exudates with underlying ulcers located near major salivary duct openings on the palate.

Microscopical findings: Histopathological results 0f 21-day-old experimentally fed broiler chickens 8 ppm T-2 toxin showed intense inflammation and localized necrosis in the oral cavity. The outer layer of the lesion consisted of sloughing fibrous material with bacterial colonies, while the underlying tissue was heavily infiltrated with granular leukocytes. Hyperplasia of the main salivary glands in the oral cavity was also evident (Figure 6) .

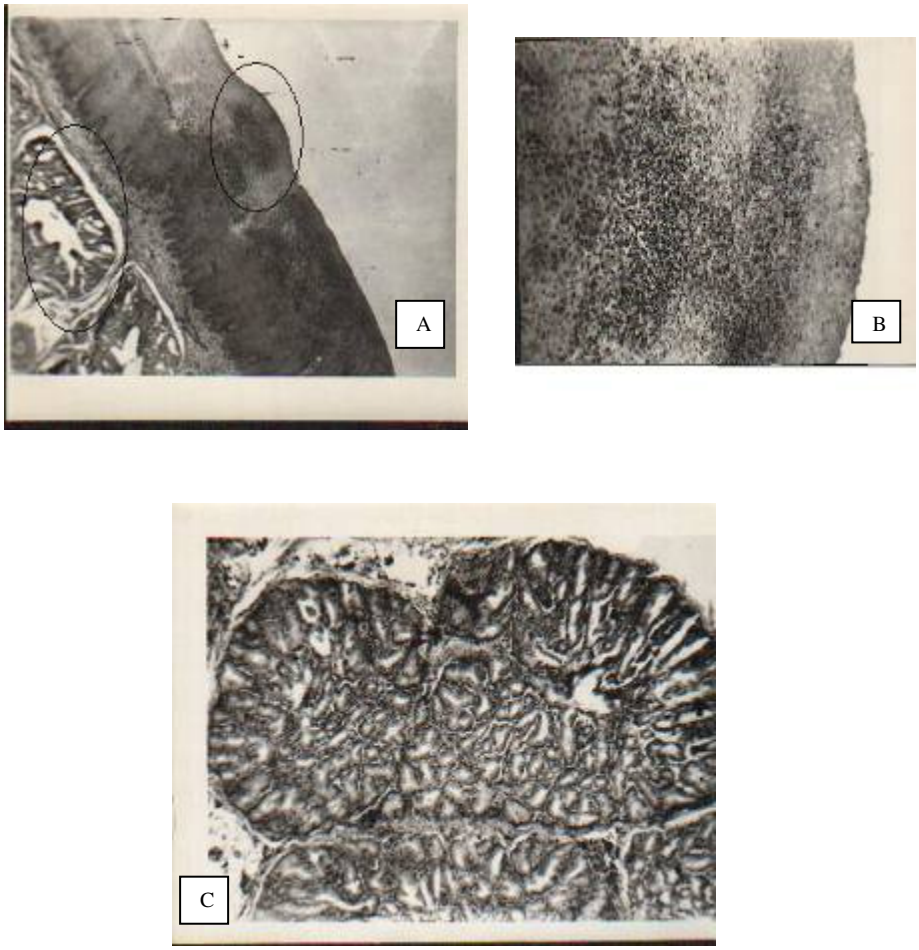

Figure 6: Photomicrograph of oral lesions of 21-day-old broiler fed purified 8 ppm T-2 toxin. (A) Oral mucous membrane show intense inflammation and localized necrosis, sloughing of upper fibrous material contained bacterial aggregations, while underlying tissue was heavily infiltrated with granular leukocytes; hyperplasia of the epithelial layer and salivary glands is also evident. H&E. X36 ;(B) Higher magnification of A, H&E. X160; (C) Hyperplasia of oral salivary gland in(A). H&E. X80.

Montmorillonite, hydrated sodium calcium aluminosilicate (HSCAS), when added at a rate of 0.5% to T-2 toxin contaminated diet was ineffective in counteracting adverse effect of T-2 toxin inducing oral lesion. Lesions were characterized by Sloughing upper fibrous material which was heavily contaminated with bacterial aggregations, while underlying tissue was infiltrated with granular leukocytes (Figure 7).

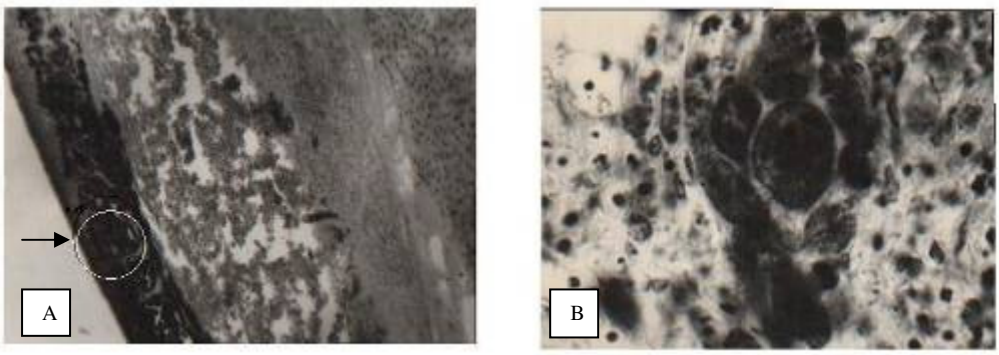

Figure 7: Photomicrograph of oral lesions of 21-day-old broiler fed purified 8 ppm T-2 toxin with 0.5%. montmorillonite. (A) Oral mucous membrane showing sloughed upper fibrous material containing bacterial aggregations, while underlying tissue was heavily infiltrated with granular leukocytes .H & E. X 160 . (B) Higher magnification of (A), showing clumps of bacteria. H & E. X 700

Addition of 0.5% vermiculite, one of the clay mineral feed additive when added to T-2 toxin contaminated diet, didn't restore the normal oral mucous membrane structure. Erosive and exudative injury still present in the broilers buccal cavity. Multiple focal necroses, interrupting the continuity of mucous membrane were noticed. fibrinosuppurative inflammation and bacterial colonies were also found in birds of this group. The underlying tissue show inflammatory response mainly of heterophils. Hyperplasia of the epithelial layer , were noted. Hypertrophy and hyperplasia of oral salivary glands were recorded (Figure 8).

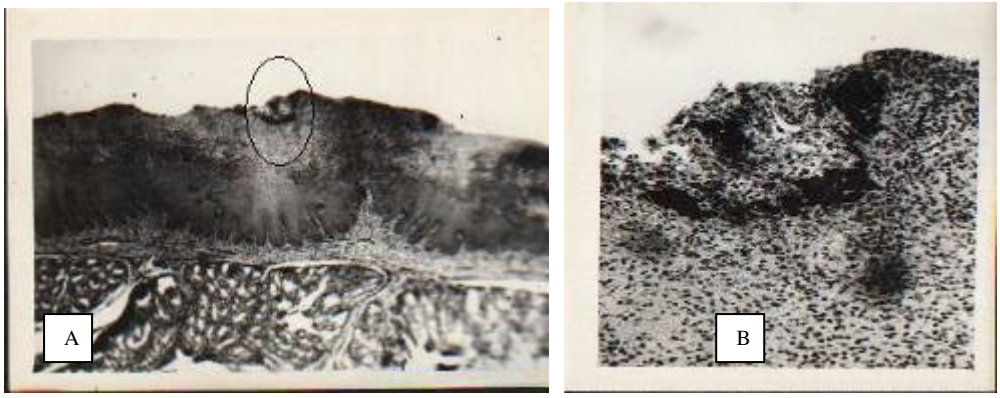

Figure 8: Photomicrograph of the mucous membrane of 21-day-old broiler fed purified 8 ppm T-2 toxin with 0.5% vermiculite,(A) showing fibrinosuppurative inflammation and bacterial colonies in the upper layer. The underlying tissue shows inflammatory response mainly of heterophils and hyperplasia of oral salivary glands. H &E X 36. (B). Higher magnification of (A), showing fibrinosuppurative inflammation and bacterial colonies in the upper layer of oral mucous membrane. H&E. X220.

Pigacin, like other mineral clays used, when added to T-2 toxin contaminated diet, did not alleviate oral lesions induced by T-2 toxin. Microscopically, lesions show hypertrophy and hyperplasia of the epithelial cells.

Increase in the thickening of the mucous membrane was noticed as a result of an increase in the amount of exudates, granulocytes and subcutaneous edema (Figure 9).

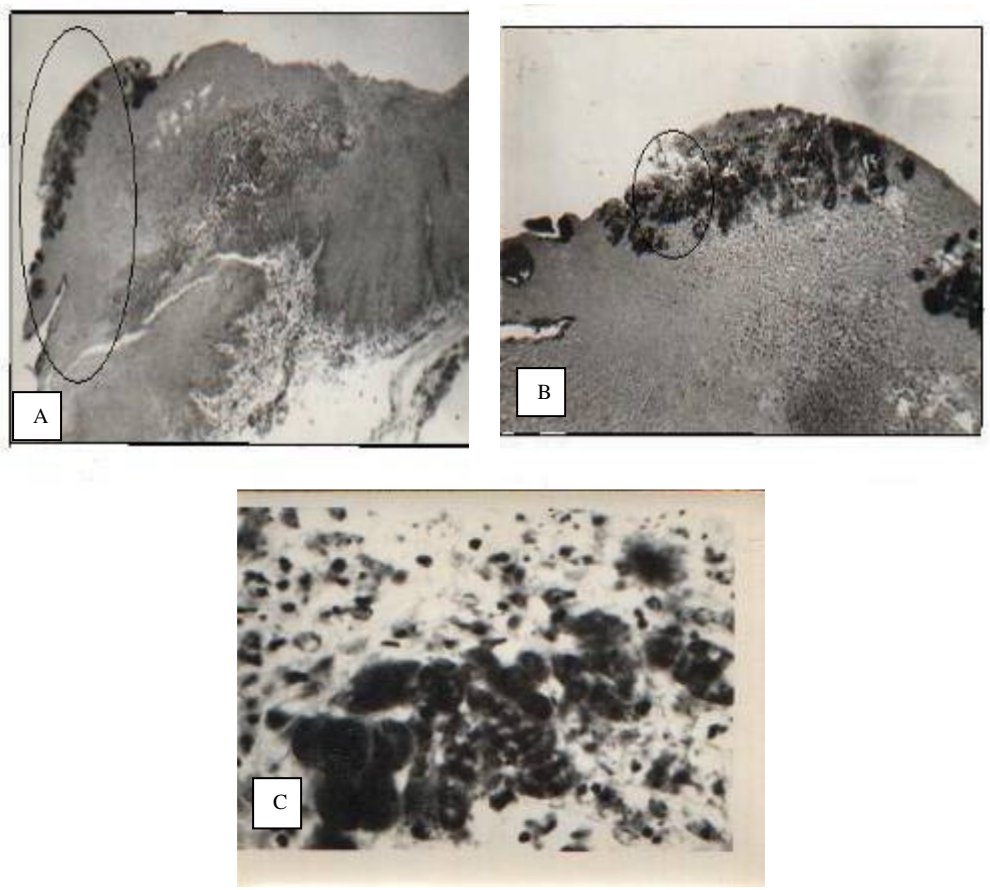

Figure 9: Photomicrograph of the mucous membrane of the buccal cavity 21-day-old broiler fed purified 8 ppm T-2 toxin with 0.5% Pigacine,(A) showing fibrinous exudates, clumps of bacteria, granulocytosis and subcutaneous edema. HY&E. X36. (B). Higher magnification of (A) H&E.X 110. (C) Higher magnification of (B), showing clumps of bacteria. H&E.X 700

The addition of 0.5% Gezilgure, the third mineral clay used, to T-2 toxin contaminated diet, was also failed to relief the degenerative effect in broiler buccal mucosa induced by the toxin. Oral histopathology confirms mucosal necrosis which varied in shape and size and had random distribution. The lesions were consisted predominantly of lower hyperplasic epithelium, layer of inflammatory cells, crusts of exudates, bacterial colonies and feed component (Figure 10).

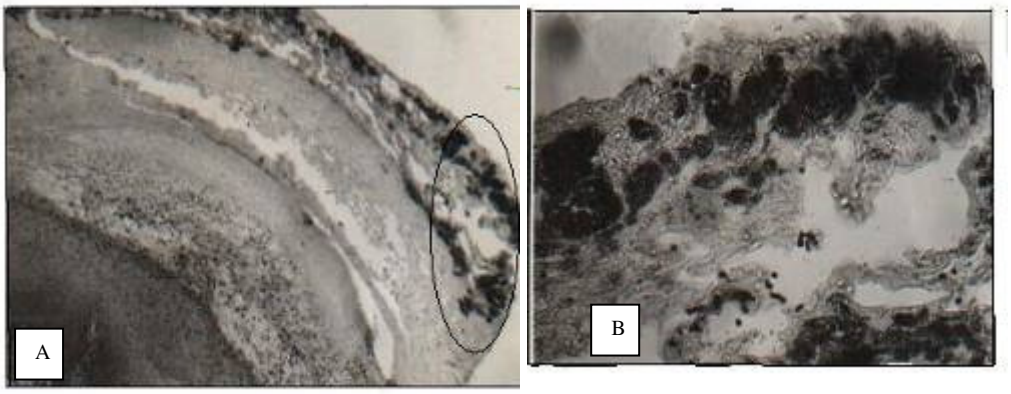

Figure 10: Photomicrograph of the mucous membrane of the buccal cavity of 21-day-old broiler fed purified 8 ppm T-2 toxin with Gezilgure,(A) showing hyperplasic epithelium, layer of inflammatory cells, crusts of exudates, bacterial colonies. H&E. X36. (B) Higher magnification of (A), show cellular detritus and clumps of bacteria and feed components. H&E. X500

Perlite, the polymer resin used in this experiment addition at a rate of 0.5% to T-2 toxin contaminated diet, was also ineffective in ameliorating oral lesions induced by feeding 8 ppm T-2 toxin. Oral lesions in birds buccal cavity of this group was characterized by focal fibrinoid necrosis, with degeneration of the epithelial cells. Microscopically there was a focal fibrinoid necrosis, with degeneration and sloughing off of the epithelial cells, edema and granulocytosis with bacterial colonies aggregations (Figure 11).

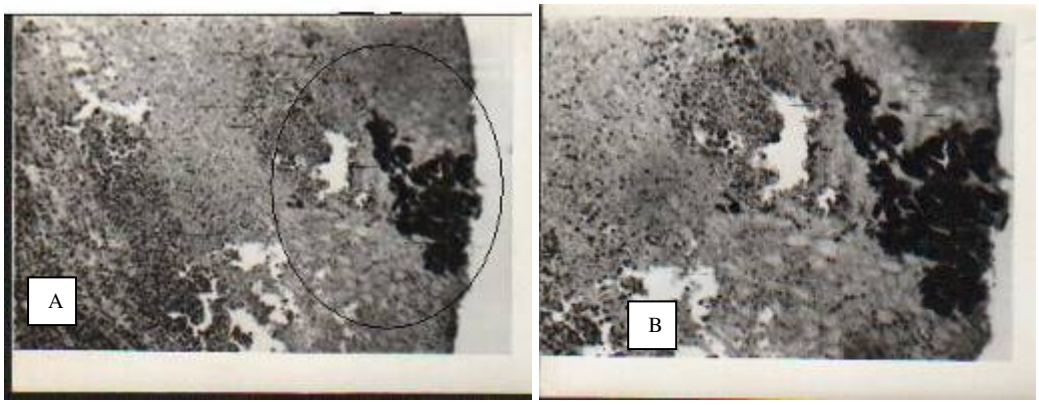

Figure 11: Photomicrograph of the mucous membrane of the buccal cavity of 21-day-old broiler fed purified 8 ppm T-2 toxin with 0.5% perlite, (A) showing focal fibrinoid necrosis, with degeneration and sloughing off of the epithelial cells, edema and granulocytosis with bacterial colonies aggregations.H&E. X75 .(B) Higher magnification of (A). H&E. X 160 .

DISCUSSION

Results of the current study indicate that broiler chicks fed diet contaminated with 8 ppm T-2 toxin develop oral lesions. Levels of this toxin reported to induce oral lesion in broilers are: 0.4 (8); I ppm (13,14); 4ppm(22); 5

ppm(23,24); 8 ppm(6,25,26,27). Gross and microscopic oral lesions induced here were resemble those reported early by Wyatt et al (12,28) in broilers fed diets containing 1ppm T-2 toxin and more. These oral lesions were also reported in broilers fed purified T-2 toxin (produced and extracted from *F. tricictum*) at a rate of 4-16 ppm for 3 weeks, and some microorganisms were isolated from these lesions (10). The erosive and exudative injury to the broiler oral mucosa, and the increase in the bacterial clumps in the sloughed outer fibrous layers appear to bear some resemblance to those described for Alimentry Toxic Aleukia in humans (12; 29). In humans, necrotic lesions were reported in gums, buccal mucosa, throat, larynx and vocal cords, contaminated with a variety of avirulent bacteria. The necrotic areas are an excellent medium for bacterial infection due to lowered résistance caused by damage to the haemopoitic and reticuloendothelial systems (30). These bacterial infections induced by T-2 causes an unpleasant odor from the mouth due to the enzymatic activity of the bacteria on proteins. In the results here, it is interesting to note that in all groups fed T-2 toxin amended with all used adsorbents, there was an increasing tendency to bacterial colonies aggregations in the necrotized, sloughed superficial layer of oral lesions. The exacerbating effect of these silica containing adsorbents (montmorillonite, 33.00%; Vermiculite, 35.12%; pigacine, 61.09%; gezilgure, 88.3%; perlite, 73.35%) in predisposing bacterial colonization may be due to the specific silica-macrophage cytotoxic interaction (31). This condition was also confirmed and reported when broiler chickens fed siliceous Ninivite (6). The increase in bacterial infection could be attributed to T-2 toxin itself in leucopenia and anemia induction (6, 7). The dramatic consequences of T-2 toxin feeding to broiler chicks could be attributed to large extent to its caustic property. The mucosal necrosis caused by Y-2 toxin is very painful to the affected birds, thus feed consumption decreases. Subsequently weight gain and feed conversion are reduced (7).

The most important feature of the adsorption is the physical structure of the adsorbent, i.e. the total charge and charge distribution, the size of the pores and the accessible surface area. On the other hand, the properties of the adsorbate molecules, the mycotoxins, like polarity, solubility, size, shape and – in case of ionized compounds- charge distribution and dissociation constants play a significant role, too. Therefore, the efficacy of every adsorption process has to be investigated in regard to the particular properties of the adsorbate (16). The applicability of aluminosilicates for the adsorption of mycotoxins has been studied for more than 20 years (32). All these studies refer to inability of aluminosilicates to counteract the negative effect of T-2 toxin and other trichothecenes (16). Of these studies which confirm the present results those used hydrated calcium sodium aluminosilicate (HSCAS) at a rate of 0.25%; 0.37%; 0.5%; and 0.8%, and showed that there was no effect against toxicity induced by 8 ppm T-2 toxin in broiler chickens (25, 26). No references are available in using clay minerals or resin polymers for counteracting T-2 toxicosis in broilers. So , it is the first time that these clay minerals and resin polymers were tested to elucidate their effectiveness in alleviating oral lesions induced by feeding purified T-2 toxin to broiler chickens. T-2 toxin has a toxic specific group on position (12, 13) responsible for the molecules negative impact on performance and health of animals. Enzymes (de-epoxydases and esterase) are at the present the only way to detoxify T-2 toxin and reduce its toxicity by converting the toxin to nontoxic product. These enzymes are contained in Mycofix® plus 3.0, and are

able to biotransformation T-2 toxin, resulting in the non toxic metabolite, de-epoxy –HT-2 toxin. This product gives a promising solution to the problem of unadsorbable trichothecenes mycotoxins in broiler feed. (7).

REFFERENCE

1- Mirocha CJ. Trichthecene toxins produced by *Fusarium*. In Shimoda W (Ed). Conference of mycotoxins in animal feeds and grains related to animal health. Food and Drug Administration Report No.FDA/BVM-79/139,1979, PP: 289-373.

2- Bamburg Jr, Riiggs NR, Strong FM. The structures of toxins from two strains of *Fusarium tricinctum*. Tetrahedron1968; 24:3329-3336.

3- Bamburg JR, and Strong FM. In Mycotoxins of the trichothecene family produced by *Fusarium tricinctum* and *trichoderma lignarum*. Phytochem 1969; 8: 2405-2410.

4- Calnek BW, Barness HJ, Beard CW, McDoougald LR, Saif YM. Disease of poultry.10th edition. Copyright. Iowa State University Press, Ames, Iowa USA 1997.

5- Chi MS, Miroch CJ. Necrotic oral lesions in chickens fed diacetoxyscirpenol, T-2 toxin, and crotocin.Poult Sci 1978; 57: 807-806.

6- Shareef AM. New methods for detoxification of poultry feeds contaminated with T-2 toxin producing fungi. Ph.D. Thesis. Sant Petersburg University. Russia 1991.

7- Jameel Z AL. The use of some adsorbents in decreasing T-2 toxin effect on broiler health and production. M Sc Thesis, Mosul University 2005.

8- Chi MC, Miroch CJ, Kurtz HJ, Weaver G, Bates F, Shimoda W, Burmeister HR. Acute toxicity of T-2 toxin in broiler chickens and laying hens. Poult Sci 1977; 56:103-116.

9- Coffin JL, Combs GF. Impaired vitamin E status of chicks fed T-2 toxin. Poult Sci 1981; 60: 385-392.

10- Hamilton PB, Wyatt RD, Burmeister H. Effect of fusariotoxin T-2 in chickens. Poult Sci 1971; 50:1583-1584.

11- Hoerr FJ, Carlton WW. T-2 mycotoxicosis in male broiler chickens. Toxicol Appl Pharmacol 1979; 48: A15 (Abstr).

12- Wyatt RD,Weeks BA, Hamilton PB, Burmeister HR. Severe oral lesions in chickens caused by ingestion of dietary fusariotoxin . Appl. Microbiol.1972; 24: 251-257.

13- Fazekas B, Hajdu ET, Tanyi J. Effect of myco-adon experimental T-2 toxicosis in broiler chickens (Hungarian).Magy. Allatory.Lapja 2000; 122: 412-416

14- Sklan D, Klipper E, Friedman A, Shelly M, Makovsky B. The effect of chronic feeding of diacetoxyscirpenol, T-2 toxin, and aflatoxin on performance, health, and antibody productionin chicks. J Appl Poult Res 2001; 10: 79-85.

15- Ramos AJ, Finkgremmels J, Hernandez E. Prevention of toxic effects of mycoxins by means of non-nutritive adsorbents compounds. J Food Prot 1996; 59: 631-641.

16- Huwig A, Freimund S, Kappeli O, Dulter H. Mycotoxin detoxication of animal feed by different adsorbents toxicology letters 2001; 122: 179-188.

17- Bauer J. Moglichkeiten zur entgiftung mykotoxin-haltiger futtermittel. Monatsh Veterinarmed 1994; 49, 175-181.
18- National Research Council. Nutrient requirements of poultry 9th reviewed. National Academic Press, Washington DC 1994.
19- Coker RD, Jones BD, Nagler MJ, Gilman GA, Walbridge AL, Panigraphi S. Mycotoxin training manual, Tropical Products Institute, London 1984.
20- Burmeister HR. T-2 toxin production by Fusarium tricinctum on solid substrate. Appl Microbiol 1971; 21: 739-742.
21- SAS Institute Users Guide: Statistics. Version 8 ed. SAS Institute Inc., Cary, NC 2000.
22- Harvey RB, Kubena LF, Rotting GE. Toxic synergism between aflatoxin and T-2 toxin in broiler chickens. Poult Sci 1988; 67: 1418-1423.
23- Huff W
24- Kubena LF, Smith EE, Gentles A, Harvey RB, Edrington TS, Phillips TD, Rottinghaus GE. Individual and combined toxicity of T-2 toxin and Cyclopiazonic acid in broiler chicks. Poult Sci 1994; 73:1390-1397.
25- Edrington TS, Kubena LF, Harvey, RB, Rottinghaus GE. Influence of a superactivated charcoal on the toxic effects of aflatoxin or T-2 toxin in growing broilers. Poult. Sci 1997; 76: 1205-1211.
26- Kubena LF, Harvey RB, Huff WE, Corrier DE, Phillips TD, Rottinghaus GE. Efficiency of hydrated sodium calcium aluminosilicate to reduce the toxicity of aflatoxin and T-2 toxin. Poult. Sci. 1990; 69: 1078-1086.
27- Kubena LF, Harvey RB, Baily RH, Buckly SA, Rottinghaus GE. Effect of hydrated sodium calcium aluminosilicate (T-Bind™) on mycotoxicosis in young broiler chickens. Poult Sci. 1998; 77: 1502-1509.
28- Baily RH, Kubena LF, Harvey RB, Buckly SA, Rottinghaus GE. Efficacy of various inorganic sorbents to reduce toxicity of aflatoxin and T-2 toxin in broiler chickens. Poult. Sci. 1998; 77: 1623-1630.
29- Wyatt RD, Hamilton PB, Burmiester HR. The effects of T-2 toxin in broiler chickens. Poult.Sci 1973; 52: 1853-1859.
30- Joffe AZ, Fusarium PF. Sporotrichoides as principle causes of alimentary toxic aleukia. In handbook of mycotoxins and mycotoxicosis (Wyllie TD and Morehouse LG Eds, Marcel Dekker, New York 1977.
31- Corrier DE. Mycotoxicosis; mechanisms of immunosuppression. Vet Immunol. Immunopathol 1991; 30: 73-87.
32- Bruin ADE. Biochemical toxicology of environmental agents. Elsevier/North-itolland biomedical press, Amsterdam 1976. pp:1286-1388.
33- Ramos AJ, Hernandez E. Prevention of aflatoxicosis in farm animals by means of hydrated sodium calcium aluminosilicate addition to feedstuffs. A review. Anim Feed Sci Technol 1997; 65: 197-206.