Vet.Med.J.,Giza. Vol.43,No.1.(1995):25-30.

EFFECT OF DEXAMETHASONE ON IMMUNITY AGAINST CRYPTOSPORIDIA INFECTION IN CHICKENS

By

SALWA F. AWADALLA, EMILY F. NAGUIB, BOTHINA A. BADAWY and SAFFAA H. HEGAZI

Poultry Dept. Animal Health Research Institute , Dokki, Egypt.

SUMMARY

The present study was planned to elucidate the influence of dexamethasone administration of Cryptosporidium oocysts output, body-weight gain, organs weight, total and electrophoretic patterns of serum proteins in chickens infected with Crytosporidia. The results revealed the following findings and conclusions:

1- The maximal body weight gain was observed in case of non-infected control chickens. Severe retardation of weight gain was recorded in infected group and infected chickens treated with DEX.

2- The minimum Cryptosporidium oocysts output was recorded in control chickens, while there was a significantly higher total oocysts output in infected chickens treated with DEX.

3- There was a significant decrease in weight of spleen, bursa of Fibricius and thymus of chickens in both groups of infected and infected treated with DEX. The lowest weight of these organs was noticed in infected chickens treated with DEX.

Total proteins, albumin, alpha, beta and gamma globulins decreased significantly in infected chickens treated with DEX, while there was a significant increase in albumin and gamma globulin in the group of infected chickens. The A/G ratio of infected and DEX treated groups was less than in control.

INTRODUCTION

McDonald (1977) reported that glucocorticoids reduce lymphocytes and eosinophils, decrease growth.

The ability of glucocorticoids to affect immune responses is well established. Corticosteroids have been known to cause immunosupression. Chronic administration of glucocorticoids has been demonstrated to decrease the weight of lymphoid organs such as thymus, bursa of Fabricius and spleen (Donker and Beuving 1989). Corticosteroids are also known to reduce circulating numbers of lymphocytes (Onsrud, 1981; Davison et al., 1988) and monocytes (Thompson and Van Furth, 1970); these effects are though to be mediated by cell destruction and/or altering leukocyte traffic to and from lymphoid tissues (Fauci, 1975).

Redig et al., (1984) found that, in vitro addition of corticosterone has been shown to suppress proliferation of chicken and turkey lymphocytes.

Gillis et al., (1979a) found that dexamethosone has been known to suppress both lymphocyte pro-

liferation and 11-2 production in response to both Con-A and PHA.

Cryptosporidium spp. are coccidian parasites that inhabit the microvillous border of epithelial surfaces of vertebrates (Anderson, 1982). Among avians, Cryptosporidium has been recovered from chickens, turkeys, quail, peacock chicks, black throated finches, red-lored parrots, and domestic goose (Whittington and Wilson, 1985 and Lindsay et al., 1986).

Current et al., (1986) have given the name Cryptosporidium baileyi to the species found in the domestic chicken.

Cryptosporidium baileyi parasitizes the caecum, bursa of Fabricius, cloaca, trachea, nasal cavities, infraorbital sinuses, larynx and bronchi of broiler chickens but clinical cryptosporidiosis occurs only when the respiratory tract is infected (Itakura et al., 1984 and Lindsay and Blagburn, 1986).

Rehg et al. (1988) reported that rat models of persistent Cryptosporidiosis have been reported, but the rats must be exogenously immunosuppressed by the administraion of hydrocortisone acetate, cyclophosphamide, or dexamethasone.

Blagburn et al., (1978) found that weight gain of infected birds with cryptosporidia may be reduced 1 to 2 weeks postinoculation.Parasite-induced lesions are generally confined to the bursa of Fabricius and cloaca. These lesions consists of epithelial hyperplasia and hypertrophy with underlying inflammatory response. Bursal follicle atrophy may also be observed.

The aim of this work is to study the effect of dexamethasone administration on immunity against cryptosporidia infection in chickens

MATERIAL AND METHODS

Thirty, one day-old Coccidia-free chicks w used for this investigation. The chicks were k under hygienic condition in cages. They were vided into three equal groups. Group 1 was k as non-infected negative control. Group 2 w orally infected with 1 X 10^6 oocysts Cryptosp idium per chick by a stomach tube and is consi ered as infected group. The 3rd group received 1 10^6 oocysts of Cryotosporidium and was injecte intramuscularly with 0.1 mg dexamethasor (SIGMA) per chick every other day, seven inje tions in total and is considered as ifected treate with dexamethasone (DEX) group. Chickens o all experimental groups were challenged with 2 10^6 oocysts of Cryptosporidium per chicken at 2 days of age.

Feces were collected on days 8 through 11 afte challenge in separated containers, weighed an mixed thoroughly with a mixer. Oocysts output o Cryptosporidium was counted according to the method of Blagburn et al., (1987). All feces were processed within 48 hours after collection.

All experimental chickens were weighed before experimental infection and at the time of the final fecal collection. Overall weight gain during the experimental period was calculated

Serum samples were collected into clean dry vials after 10 days post-challenge and kept in at -20°C until use for the determination of total proteins according to the method of Gornall et al., (1949) and protein electrophoretic pattern according to the method of Bicrer (1964).

Killing of the experimental chickens was done at the end of the experiment. Spleen, bursa and thymus were separated and weighed.

RESULTS

Table (1) showed that infected chickens treated with DEX produced significantly higher total Cryptosporidium oocysts (13.80±0.06) output than control (4.00 ± 0.01) and infected (8.20 ± 0.03) chickens. The lowest Cryptosporidium oocysts output was noticed in control chickens.

It is apparent from Table (1) that there was a significant decrease ($P < 0.01$) in weight gain of chickens in both groups of infected and infected treated with DEX in comparison with that of control. The maximal weight gain was recorded in control chickens, while the minimal weight gain was noticed in infected chickens treated with DEX.

Table (2) indicated that there was a significant decrease in weight of the spleen, bursa Fabricius and thymus of chickens in both groups of infected and infected treated with DEX in comparison with that of control one. The lowest weight of these organs was found in the infected chickens treated with DEX.

Table (3) revealed that there was a significant decrease in serum total protein, albumin, alpha, beta and gamma globulins in infected chickens treated with DEX in comparison with that of control. There was a significant decrease of albumin and gamma globulin in infected chickens than control. The A/G ratio of both groups of infected and infected treated with DEX was less than control one.

Table (1): Effect of dexamethasone (DEX) on weight gain and oocysts output ($X 10^5$) per gram of feces from chickens infected with Cryptosporidia.

Group	Oocysts count ($X 10^5$)	% weight gain
Control	$4.00^a \pm 0.01$	$85.51^a \pm 2.40$
Infected	$8.20^b \pm 0.03$	$75.50^b \pm 1.50$
Infected treated DEX	$13.80^c \pm 0.06$	$61.30^c \pm 2.29$

Different letters of mean in the same column show significant differences at $P < 0.01$.

Table (2): Effect of dexamethasone and Cryptosporidia infection on spleen, bursa and thymus weight in chickens.

Group	Organs Weight (g)		
	Spleen	Bursa	Thymus
Control	0.17 [a] ± 0.01	0.41 ± 0.02	0.44 [a] ± 0.04
Infected	0.11 [b] ± 0.01	0.26 [b] ± 0.02	0.29 [b] ± 0.02
Infected treated DEX	0.08 [c] ± 0.02	0.14 [c] ± 0.01	0.20 [c] ± 0.01

Different letters of mean in the same column show significant differences at P< 0.01.

Table (3): Total and electrophoretic patterns of serum proteins in chickens infected with Cryptosporidia and treated with dexamethasone (DEX).

Group	Total protein (g%)	albumin (g %)	Globulin (g %)				A/G ratio
			Alpha	Beta	Gamma	Total	
Control	3.62 ±0.10	1.92 ±0.02	0.46 ± 0.04	0.50 ±0.05	0.65 ± 0.05	1.70 ± 0.12	1.13
Infected	3.75 ±0.09	1.78* ±0.03	0.35 ± 0.04	0.38 ±0.09	1.24* ± 0.10	1.97 ±0.09	0.90
Infected treated DEX	2.98* ±0.09	1.40* ±0.05	0.26* ± 0.03	0.15* ±0.02	1.17* ± 0.03	1.58 ±0.10	0.89

Mean ± standard error

* Significantly different from control at P < 0.01.

DISCUSSION

The results obtained in the present study revealed that, the maximl body weight gain was observed in case of non-infected control chickens. Severe retardation of weight gain was recorded in both groups of infected and infected chickens treated with DEX. These results are in agreement with those recorded by McDonald (1977), Blagburn et al.., (1987) and Awadalla et al. (1994).

The minimum Cryptosporidium oocysts output was recorded in non infected control chickens, while there was a significantly higher total oocysts output in the group of infected chickens treated with DEX. This might be due to immunosupression effects of dexamethason in chickens.

Concerning the effect of dexamethasone and Cryptosporidia infection of weight of lymphoid

organs such as thymus, spleen and bursa of Fabricius in chickens, the data showed clearly that there was a significant decrease in weight of these organs in both groups of infected and infected treated with DEX. The minimal weight of those lymphoid organs was recorded in infected chickens treated with DEX. Such effect might be attributed to the immunosuppression action of dexamethasone which caused atrophy of lymphoid organs such as spleen, bursa and thymus in chickens. These resullts are in agreement with those obtained by Donker and Beuving (1989).

Total proteins, albumin, alpha, beta and gamma globulins were significantly decreased in infected chickens treated with DEX, while there was a significant decrease in albumin and gamma globlin in the group of infected chickens.

The A/G ratio of infected and DEX treated groups was less than control one. These effects are due to the immunosuppression of immune system in chicke\ns. These results are in agreement with those obtained by Gillis et al., (1979a), Radig et al., (1984) and Davison et al., (1988).

REFERENCES

Anderson, B.C., (1982): Cryptosporidiosis: A review. J. Am. Vet. Med. Assoc. 180: 1455: 1457.

Awadalla, S.; Naguib, E. Hegazi, S. and Badawy, B. (1994): Effect of concurrent infections with cryptosporidia and Eimeria tenella in chickens. Accepted in Vet. Med. Journal, Giza.

Bicrer, B.W. (1964): Electrophoretic analysis of blood serum and plasma proteins of normal horses. Am. J. Vet. Res., 30: 2237-2241.

Blagburn, D.L.; Lindsay, D.S.; Giambrone, J.J.; Sundermann, C.A. and Hoerr, F.J. (1987): Experimental Cryptosporidiosis in broiler chickens. Poult. Sci. 66: 442-444.

Current, W.L. Upton, S.J. and Haynes, T.B. (1986): The life cycle of Cryptosporidium baileyi (Apicomplexa, Cryptosporidudae) infecting chickens. J. Protozol, 33: 289-296.

Davison, T.F.; Mission, B.H.; Williams, R.A. and Rea, J. (1988): Effect of increased circulating corticosterone in the immature fowl on the blastogenic response of peripheral blood lymphocytes. Dev. Comp. Immunol. 12: 131-144.

Donker, R.A. and Beuving, C. (1989): Effect of corticosterone infusion on plasma corticosterone concentration, antibody production, circulating leukocytes and growth in chicken lines selected for humoral immune responsiveness. Br. Poultry Sci. 30: 361-369.

Fauci, A.S. (1975): Mechanisms of corticosteroid action on lymphocytes subpopulations. I. Redistribution of circulating T. and B. lymphocytes to the bone marrow. Immunology, 28: 660-668.

Gillis, S.; Crabtree, G.R. and Smith, K.A. (1979a): Glucocorticoid induced inhibition of T. cell growth factor production. Immunology, 123: 1624-1631.

Gornall, A. Bardawill, L. and David, G. (1949): Determination of total protein. J. Biol. Chem., 177:751.

Itakura, C.; Goryo, M. and Umemura, T. (1984). Cryptosporididial infection in chickens. Avian pathol. 13: 487-499.

Lindsay, D.S.; Blagbura, B.L; Sundermann, C A.; Hoerr, F.J. and Ernest, J.A. (1986): Experimental Cryptosporidium infections i chickens; Oocysts structure and tissue specificity. Am. J. Vet. Res. 47: 876-879.

McDonald., L.E. (1977): Veterinary endocrinology and reproduction, 2nd ed., Lea and Febiger, Philadelphia.

Onsrud, M. (1981): Influence of in vivo hydrocortisone on some human blood leucocyte sub-populations. II. Effects on T.cell-monocyte cooperation. Acta. Pathol. Microbiol. 89: 321-327.

Redig, P.T.; Dunnette, J.L.; Mauro, L. Sivanadan, V. and Markham, F. (1984): The in vitro respose of turkey lymphocytes to steroid hormones. Avian Dis. 29: 373-383.

Rehg, J.E.; Hancock, M.L. and Woodmansee, D.B. (1988): Characterization of a dexamethasone-treated rat model of Cryptosporidial infection J. Infect. Dis 158: 1406-1407.

Siegel, H. S. (1971): Adrenals, stress and the environment. Poultry Sci. 27: 327-349.

Thompson, J. and Van Furth , R. (1970): The effect of glucocorticosteroids on the proliferation and kinatics of promonocytes and monocytes of the bone marrow. Exp. Sci. 137: 10-21.

Whittington, R.J. and Wilson, J.M. (1985): Cryptosporic sis of the respiratory tract in a pheasant. Aust. Vet. J. 284-288.