

Studies on susceptibility of native and white Lohmann layer chickens breeds to infectious bursal disease virus isolate FY.97

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This study was done to evaluate susceptibility, protective titer level of maternal derived antibodies (MDAbs) of different chicken breed against virulent Infectious bursal disease virus (IBDV) local isolate Fy97 and prediction the optimal time for vaccination. All breeds were experimentally infected orally with IBDV isolate Fy97 every 5 days following detection of MDABs by ELISA. Clinical signs, mortality, lesions and Bursal Histopathology and lesion score were taken as criteria for comparison. Morbidity rates were observed as $\geq 30\%$ in Fayoumi and Dandrawi infected at 15 days of age and in Senawi and Baladi and Lohmann at 20 days of age. All breeds showed clinical signs of infection at 30-35 days of age where Senawi breed showed the highest values (65 and 70%) followed by Fayoumi (55 and 55%), Dandrawi (50%), Baladi (55-45%) and Lohmann (50-45%). Mortality rates due to IBD infection varied from 0 to 35% in respective to age, in Fayoumi and Lohmann breeds where maximum 35 and 40% occurred at 30 day of age; respectively. Mortality in Dandrawi and Senawi varied from 5 to 40% and pass in close manner at all intervals with the highest value at 30 days of age while Baladi chicks showed same values but lower only at 20 and 25 days. Mean lesion scores in Fayoumi were the lowest at all intervals followed by Lohmann, Senawi, Baladi and Dandrawi. Results of ELISA titers at time of infection showed that Senawi chicks having the highest titers followed by Lohmann, Baladi, Dandrawi and Fayoumi at most intervals. So it necessitates more clarification of the causes of these phenomena and the role of genetics in protection against IBDV infection.

The clinical signs and mortality of infectious bursal disease (IBD) usually persist 3-5 days with high morbidity and the mortality rate may reach 10-20% (Cosegrove, 1962), while in some cases mortality have exceeded 30% (Bygrave and Fraghar, 1970). The highly virulent strains of the standard serotype I IBDV showed natural mortality reached over 25-30% in broilers and 90% in experimental infection (Chettle *et al.*, 1989).

In Egypt the disease was first recorded in commercial broiler chickens on the basis of histopathological examination (El Sergany *et al.*, 1974). The first isolation and identification of IBDV was performed by (Ayoub and Malek, 1976). The outbreaks of IBD were reported by (Bastami, 1980; Mousa *et al.*, 1983 and 1986; Amer *et al.*, 1984 and 1986; El Batrawi and El Kady, 1990). The very virulent IBDV strain was reported in Egypt by (El Batrawi, 1990). Breed variation in disease susceptibility has already been shown for IBDV and many other diseases of poultry (Bumstead *et al.*, 1991; Hassan *et al.*, 2004) and it is clear that a range of different genes affect susceptibility to different diseases.

Recently, it was suggested that over all immunocompetence can be improved by line selection for high antibody response of young chicks to controlled immunization with a single antigen (Yunis *et al.*, 2002). This study was carried out to investigate the possible variation in breed susceptibility to experimental infection with very virulent IBD virus

Materials and methods

Embryonated chicken eggs (ECE). Specific pathogen free (SPF) ECE were obtained from Ministry of Agriculture and cultivation of lands, production of SPF embryonated eggs project Kom-Oshim, Fayoum. ECE were used for virus propagation and titration of the virulent IBDV strain through chorioallantoic membrane (CAM) route of inoculation.

Experimental chicks. A total number of 240 chicks were used during this study including 4 native breeds (Fayoumi, Dandrawi, Sinawi, and Baladi) were obtained from (Al-Azab project for poultry production, Fayoum) and one foreign breed (Lohmann white) obtained from (Al-Wadi commercial company for poultry production). Dam hens of native breeds were aged 22 weeks

and received oil IBD vaccine at 14 and 18 weeks of age; while the foreign breed was aged 26 weeks and vaccinated at 23 and 33 weeks. From each breed 60 and 240 one-day old chicks were used for studying decaying of maternal antibody and studying breed susceptibility to tested IBDV virulent strains; respectively. These chicks were floor reared under natural day light and feed on balanced commercial ration.

IBDV Strains. Highly virulent IBDVs (VvIBDV) Isolate (Bursal homogenate) isolated from El-fayoum governorate, Egypt (1997) was kindly supplied by Newcastle diseases department, Veterinary Serum and Vaccine research Institute, Abbasia, Cairo, Egypt, this virus was propagated in SPF-ECE with end titers $10^{7.8}$ then propagated in 3 weeks old chicks for testing its pathogenicity before use (Ibrahim, 2001).

IBD ELISA Kits. IBDV-ELISA Kits were obtained from Kikegaard and Perry laboratories (Kpl), U.S.A.

Serum samples. Blood samples were collected in clean dry, sterile Wassermann tubes. The tubes containing blood samples were left in horizontal position for an hour at room temperature and then left for another hour at 4°C then centrifuged at 3000 rpm. for 15 minutes. Serum samples were carefully separated in a small Eppendorf vials, labeled and kept at -20°C till used.

IBDV titration. IBDV titration was performed according to (Thangavelu *et al.*, 1998) as SPF embryos were inoculated according to (Hitchner, 1970). The embryo infective dose (EID₅₀) was calculated according to (Reed and Munch 1938).

ELISA test procedures. ELISA test was carried out according to manufacture instructions.

Histopathological Examination. Tissue specimens from bursa of experimentally infected and control chicks were fixed in 10% neutral formosaline, stained (H and E) according to (Culling, 1974). Bursal lesion score was adapted according to (Muskett *et al.*, 1979).

Bursa: body weight index. Bursa/body weight ratio, bursal index and bursa/ body weight index of 7 day old infected chickens were calculated according to (Sharma *et al.*, 1989). Chicks with bursa: body weight index lower than 0.7 was considered suffering from bursal atrophy (Lucio and Hitchner, 1979).

Challenge test. Each chick received 10^4 EID₅₀ /0.2 ml of the virus that was previously titrated via eye drop instillation at 10 days of age with

5 days interval until 45 days of age (Giambrone and Closser, 1990).

Experiment. Two hundred and forty 1-day old chicks were used for each breed. Birds of each breed were grouped into 8 groups; 30 chicks / group prepared for challenge and for each group 10 chicks as control.

Ten chicks were randomly collected for sera to measure IBDV MDAbs level by ELISA, at the time of IBDV challenge, Chicks were challenged at 10, 15, 20, 25, 30, 35, 40 and 45 days of age each group inoculated with 0.2 ml of 10^4 EID₅₀ /chick via eye instillation, and observed daily for a period of 7 days with record of Clinical signs, morbidity and mortality rates and Post-mortem examination of dead chicks.

The remaining live chicks at the end of observation individually weighted scarified. Also Bursae were removed, weighed for calculation of bursa: body weight ratio, index and bursal index. Bursa was fixed in 10% formosaline for histopathological lesion score. Results are shows in Table (1-3).

Results

Clinical sing started to appear after 2 days post infection (P.I) as ruffled feathers incoordination, weakness and recumbancy while at 4th day P.I profused yellowish diarrhea, was seen at 4th d P.I lesion were enlarged bursa and hemorrhagic muscles in thigh. . Morbidity rates were observed as $\geq 30\%$ in Fayoumi and Dandrawi infected at 15 days of age and in Senawi and Baladi and Lohmann at 20 days of age. All breeds showed clinical sings of infection occurred at 30-35 days of age, where Senawi breed showed the highest values (65 and 70%) followed by Fayoumi (55 and 55%), Dandrawi (50%), Baladi (55-45%) and Lohmann (50-45%). Mortality rates due to IBDV infection varied from 0 to 35% in respective to age, in Fayoumi and Lohmann breeds where maximum 35 and 40% occurred at 30 day of age; respectively .Mortality in Dandrawi and Senawi varied from 5 to 40% and pass in close manner at all intervals with the highest value at 30 days of age while Baladi chicks showed same values but lower only at 20 and 25 days. Mean lesion scores in Fayoumi were the lowest at all intervals followed by Lohmann, Senawi, Baladi and Dandrawi. (Table 2 and Fig. 2)

Results of ELISA titers at time of infection showed that Senawi chicks having the highest

Table (1) Mean Prechallenge ELISA antibodies titers of all breeds.

Age/days	Breeds	Mean Prechallenge ELISA titers				
		Fayoumi	Dandrawi	Senawi	Baladi	Lohmann
10		2993±318	3189±422	4225±455	3808±488	3986±474
15		1594±255	2962±321	3167±366	3260±364	3320±328
20		1061±118	2120±127	2866±274	2540±321	2740±341
25		833±106	1571±138	2578±301	2161±243	2262±221
30		638±98	1226±119	1740±237	1730±231	1736±156
35		426±103	576±98	1479±154	1360±198	1198±123
40		299±89	313±64	870±215	810±233	730±144
45		69±36	293±56	470±75	320±89	340±73

titers followed by Lohmann, Baladi, Dandrawi and Fayoumi at most intervals (Table 1 and Fig.1). Control non infected birds showed no signs, mortalities or lesions at end of observation period as well as no detectable bursal tissue changes in histopathological examination at all intervals of the experiment. All infected chicken breeds chicks with virulent IBDV virus showed a Bursal weight and ratios at 7 days lower than their control non infected at all intervals. Bursal index in Fayoumi gradually decreased from 0.7 at 10 day infected bird to reach 0.4 in those infected at 25 and 30 days and reincreased at 40-45 days to reach 0.6. All these values were indicative for bursal atrophy but it varies from mild at 10, 15, 40 and 45 days of infection to moderate at 20, 35 days to severe at 25 and 30 days. The infected Dandrawi chicks showed Bursal index in infected birds were decreased from 0.6 at 10 day infected bird to reach 0.5 in 40 days infected and further decreased to 0.4 at 45 days of age. Bursal index of Senawi were gradually decreased from 0.7 at 10 day infected bird to reach 0.6 in those infected at 15-35 days and decreased to reach 0.5 at 40 and 45 days of age. The Baladi infected chicks showed Bursal weight and ratios of 7 days infected birds were lower than their control non infected at all intervals. Bursal index of infected birds were 0.6 at 10 day infected bird till 35 days of infected birds and decreased in those infected at 40 and 45 days of age. The infected Lohmann chicks with virulent IBD virus showed Bursal index of infected birds were 0.6 at 10 day infected bird till 35 days of infected birds and decreased in those infected at

40 and 45 days of age.

Histopathologically. The histopathological finding recorded in (Plate 1) proved that the examined bursal section showed the following:

The bursa of control -ve showed normal tissues and normal follicular distribution (0). Microscopic examination of the Bursae showed mild bursal lesion represented by follicular lymphoid necrosis and depletion of 5-25% of lymphocytes (score 1) especially at medulla of lymphoid follicles. The group challenged at 30,35 days of age (3) the Bursae showed moderate follicular lymphoid necrosis and depletion of 5-25% of lymphocytes (score 2) in addition to slight proliferation in the intrfollicular fibrous connective tissue (i.e. fibroplasia) more than 50% of follicles damaged (score 3). The bursa lesion score was, (4), the cortex of lymphoid follicles there were infiltration of few numbers of heterophils 50-75% of lymphoid follicles were damaged, Intrfollicular connective tissue showed fibrosis. (Score 4) and some lymphoid follicles showed cystic cavitations and others were atrophied and plicae as 75-100% follicle damaged and fibroplasia (score 5).

Discussion

IBDV infection causing serious losses in young chickens, since 1986, Europe has experienced the emergence of "very virulent" (vv) strains of IBDV, which can cause up to 70% flock mortality in laying pullets (Chettle and Wyeth 1989; van den Berg and Meulemans, 1991), are antigenically similar to the "classical" strains, (Etteradossi *et al.*, 1992). Remarkably, however, vvIBDV can establish infection in the

Fig. (1)

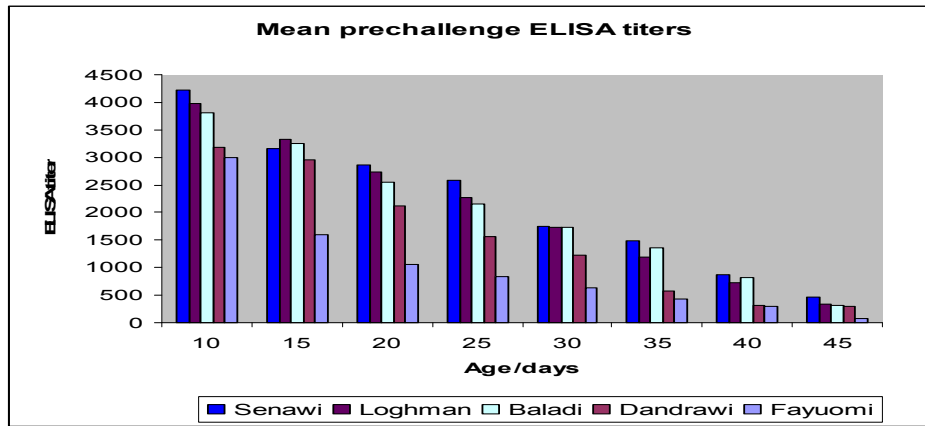


Fig. (2)

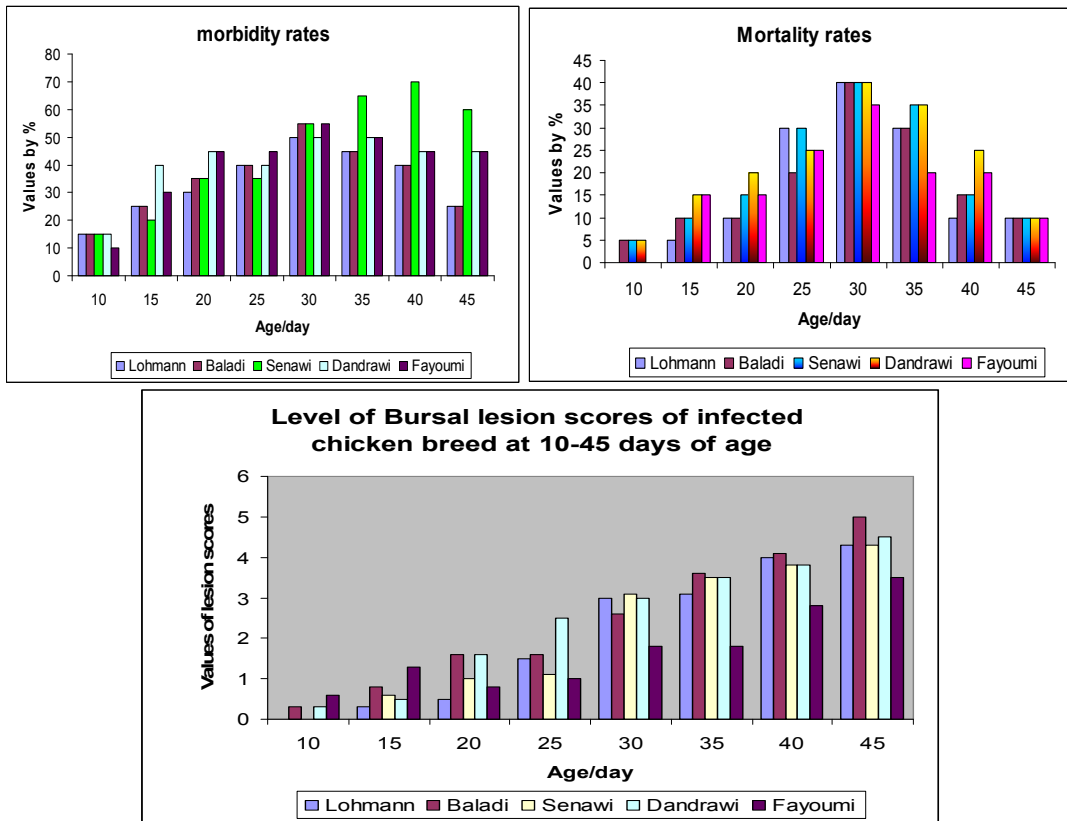


Table (2) Morbidity, Mortality and Lesions scores of IBD infected Different chicken breeds:

Age	Fayoumi			Dandrawi			Senawi			Baladi			Lohmann		
	Mb%	Mt%	L.S	Mb%	Mt%	L.S	Mb%	Mt%	L.S	Mb%	Mt%	L.S	Mb%	Mt%	L.S
10	10	0	0.6	15	5	0.3	15	5	0	15	5	0.3	15	0	0
15	30	15	1.3	40	15	0.5	20	10	0.6	25	10	0.8	25	5	0.3
20	45	15	0.8	45	20	1.6	35	15	1	35	10	1.6	3	10	0.5
25	45	25	1	40	25	2.5	35	30	1.1	40	20	1.6	40	30	1.5
30	55	35	1.8	50	40	3	55	40	3.1	55	40	2.6	5	40	3
35	50	20	1.8	50	35	3.5	65	35	3.5	45	30	3.6	45	30	3.1
40	45	20	2.8	45	25	3.8	70	15	3.8	40	15	4.1	40	10	4
45	45	10	3.5	45	10	4.5	60	10	4.3	25	10	5	25	10	4.3

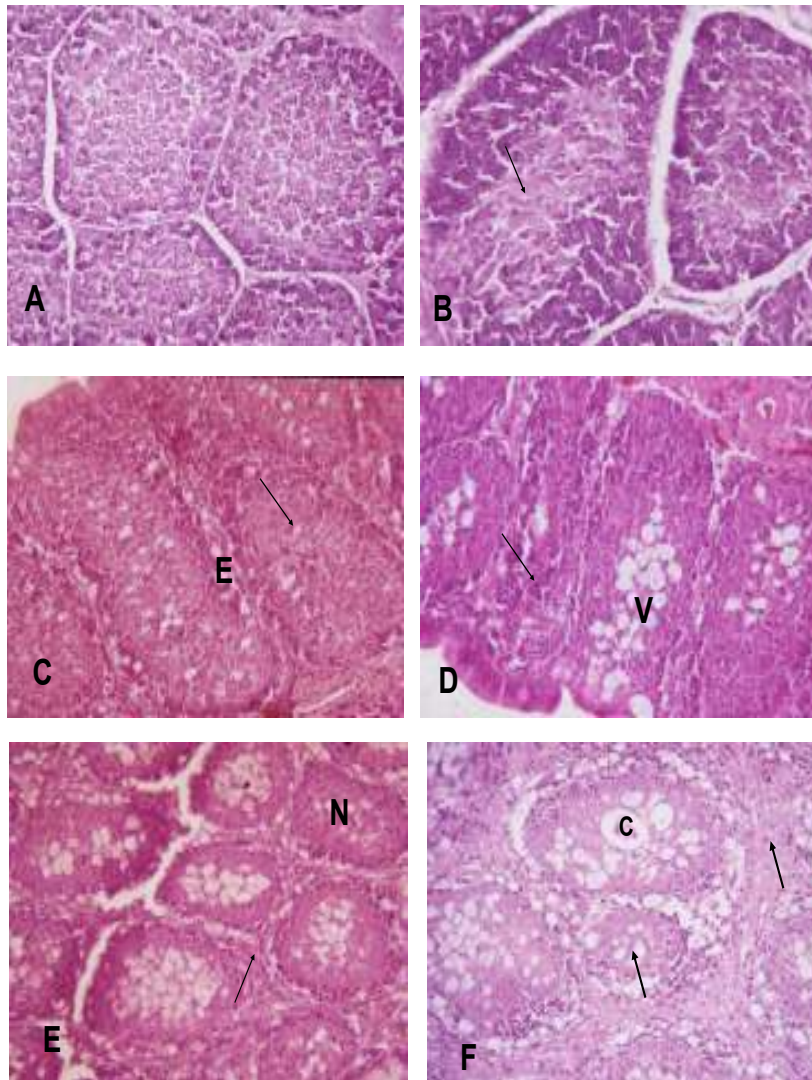
Mb--morbidity rate, Mt—mortality rate, L.S—bursa lesion score.

Table (3) Average body and bursal weights as well as bursal index and bursal body weight index of IBD infected all breeds group

Bwt==body weight,Bu.wt==bursal weight,B.I==bursal index,B.B.I==bursal body weight index

Age/ Day	Lohmann				Baladi				Senawi				Dandrawi				Fayoumi			
	Bwt	Bu.wt	B.I	B.B.I	Bwt	Bu.wt	B.I	B.B.I	B.wt	Bu.wt	B.I	B.B.I	Bwt	Bu.wt	B.I	B.B.I	Bwt	Bu.wt	B.I	B.B.I
10	82	0.211	2.8	0.7	90	0.185	2.2	0.6	77	0.158	2.4	0.7	85	0.165	2.0	0.6	105	0.189	1.2	0.8
15	98	0.169	1.9	0.6	100	0.170	1.9	0.6	85	0.146	2.0	0.6	96	0.150	1.7	0.6	182	0.178	1.0	0.7
20	105	0.162	1.8	0.5	110	0.218	2.2	0.6	97	0.162	1.8	0.6	103	0.167	1.8	0.6	210	0.160	0.8	0.6
25	118	0.158	1.5	0.4	130	0.246	2.1	0.6	112	0.158	1.5	0.6	115	0.159	1.5	0.6	238	0.177	0.8	0.6
30	125	0.175	1.5	0.4	142	0.248	2.0	0.6	128	0.175	1.5	0.6	133	0.154	1.2	0.6	281	0.241	0.8	0.5
35	138	0.198	1.6	0.5	158	0.229	1.7	0.6	147	0.168	1.3	0.6	142	0.177	1.3	0.6	389	0.211	0.7	0.6
40	175	0.201	1.2	0.6	233	0.244	1.0	0.5	210	0.193	0.9	0.5	177	0.147	0.9	0.5	420	0.214	0.5	0.5
45	189	0.221	1.3	0.6	308	0.248	0.8	0.4	295	0.221	0.7	0.5	276	0.179	0.7	0.6	475	0.193	0.4	0.4

Plate (1): Bursal sections of 7 days post infection with IBDV stained with H and E.



A: Bursa of non infected control: Normal bursal tissue (lesion score: 0) X200.

B: Infected bursa showing: Slight lymphoid necrosis (arrow) and depletion with edematous connective tissue between follicles (lesion score:1) X200.

C: Infected bursa showing: Atrophied follicles (arrow) and edematous intrafollicular tissue (Lesion score: 2) X200.

D: Infected bursa showing: Vacuolation of medullary cells (V) and necrosis in cortical cells (arrow) (Lesion score: 3) X200.

E: Infected bursa showing: Atrophied bursas with necrosis (N) with interfollicular fibrosis infiltrated with lymphocytes (arrow) (Lesion score: 4) X100.

F: Infected bursa showing: Severe lymphocytic depletion and necrosis (arrow) and medulla of lymphoid follicles showed vacuolated reticular cells cyst formation (C) (lesion score:5) X200.

face of levels of maternally derived antibodies that were previously protective against "classical" strains. While, vvIBDV infections also have been observed in Africa, Asia and, only recently, in South America (Ikuta *et al.*, 2001). Clinical signs and morbidity rates were observed as $\geq 30\%$ in Fayoumi and Dandrawi infected at 15 days of age and in Senawi and Baladi and Lohmann at 20 days of age, so signs increased in severity with the increase of age and reduction of antibodies titer. (Hitchner, 1971), while (EI-Batrawi and EI-Kady, 1991) reported the Difference was relative to difference in neutralizing antibodies level transferred to the progeny, dose and virulence of challenge virus. Comparing the bursal index and bursa: body weight index of challenged chicks at different ages with control non challenged groups challenged at 10 days of age and older a lower bursal index than control suggesting bursal atrophy (>0.7) (Luico and Hitchner, 1971). All breeds showed clinical signs of infection at 30-35 days of age, Senawi breed showed the highest values (65 and 70%) followed by Fayoumi (55 and 55%), Dandrawi (50%), Baladi (55-45%) and Lohmann (50-45%). Mortality rates due to IBD infection varied from 0 to 35% in respective to age, comparing results of bursal ratio and index with the reported prechallenge ELISA titers (Table 2 and 3), Even when maternal immunity was effective in protecting against mortalities, it didn't prevent bursal damage as judged by histopathological lesions (Van Den Berg and Meulemans, 1991). Under our experimental conditions moderate to extensive histopathological lesions were seen in chicks challenged at 15 days of age and older despite protection against mortalities. The severity of histopathological lesions increased with age of birds and waning of MAbs. The challenge virus induced 60 - 65% mortalities in 38 days old chicks indicating maximum age of susceptibility as judged by severe clinical signs in all affected bird, typical post mortem lesions, bursal atrophy (bursal index lower than control and bursa: body weight index was 0.4) and extensive histopathological bursal lesions. However less mortality rates observed before this age were attributed to the passive protection conferred by MAbs (Van Den Berg and Meulemans, 1991).

Bursal lesion score in 10 days infected birds was 0.3 and increased in distribution to be stronger with age to reach maximum at 45 days of age. and these results agreed with the

recorded of native chickens play an important role in household food supply in rural Africa (Kitalyi, 1998) and recently have been raised in semi-intensive systems with more efficient output per bird. The control of immunosuppressive diseases is of prime importance for the nascent poultry industry in developing countries. In this regard, selection and enhancement of genetic resistance to economically important diseases should be considered. Variation in breed susceptibility has been documented for many poultry diseases (Bumstead *et al.*, 1991). In the case of IBD, brown and white Leghorn breeds are more susceptible than broiler breeds (Bumstead *et al.*, 1993). However, little information is known about the general genetic resistance of native Egyptian breeds to IBD (Hassan *et al.*, 2002). This result may indicate that Fayoumi chicks MDABs may be age resistance or genetic resistant. The differences of maternal antibody decay of IBDV with regard to breed variation and relative susceptibilities of local Egyptian breeds to vvIBDV were tested. The Dandrawi Fayoumi and Senawi were susceptible while the Baladi and Lohmann breeds was particularly resistant or of intermediate susceptibility. The variation in mortality rates between breeds did not correlate with the vvIBDV-induced bursal lesions or the humoral antibody response to IBDV. All breeds had high titers of serum antibody at 7 days p.i. ($P > 0.05$), which declined in all breeds by 14 days p.i. This rapid response may be due to the highly acute nature of the disease. The antibody titers in vaccinated chickens before and after challenge with vvIBDV did not correlate with the mortality rates observed. It is difficult to identify specific innate or acquired immune responses that are responsible for IBD resistance. The B haplotype has been shown to influence the level of complement (Chanh *et al.*, 1976), and complement has been implicated in the formation of immune complexes and development of clinical IBD (Skeeles *et al.*, 1979). Chickens that lacked sufficient complement did not develop lesions. (Cook *et al.*, 1992). In contrast, (Gelb *et al.*, 1998) reported that antibody production in tears of vaccinated birds was not an accurate indicator of IBDV immunity as determined with challenge studies. It was suggested that mechanisms other than antibody mediated-immunity in tears are important in IBDV resistance. Selection experiments on various

components of the immune response in livestock have provided some evidences that variation in maternal antibody transmission is at least partially genetically based (Grindstaff *et al.*, 2003). Maternal antibody transmission is influenced by genes expressed in both females and offspring (Cheverud and Moore, 1994). Such evidence may lead to variable antibody titer patterns in dam, hen and yolk. IgY begins to be transported into the embryonic circulation by embryonic day 7, with low levels of transmission initially. IgY concentration in embryonic plasma increases slowly until embryonic day 14, (Kowalczyk *et al.*, 1985) The level of MDABs may be determined by interactions between the maternal genome and the offspring genome, or Chicken line variation may be related to different inherent abilities (Muggli *et al.*, 1984; Linder *et al.*, 2000; Kölliker and Richner, 2001; Hager and Johnstone, 2003). Native chickens play an important role in household food supply in rural Africa (Kitalyi, 1998) Selection and enhancement of genetic resistance to economically important diseases should be considered, in breed Variation susceptibility has been documented for many poultry diseases (Bumstead *et al.*, 1991). Variation in maternal antibody transmission is at least partially genetically based (Grindstaff *et al.*, 2003). Prediction equation was calculated for each breed that allows the forecast of antibody titer decay. High correlation was observed between dependant and independent parameters of the regression analysis. Different breeds showed differences in the slope value that indicate differences in the decay of maternal antibodies and the predication of antibody titer for each breed as the Loghman breed showed highest value then Dandrawi breed while the Fayoumi and Baladi were as the same values and on the other hand the Senawi showed the lowest value. Fayoumi breeds are more susceptible than other native breeds. Little information is known about the general genetic resistance of native Egyptian breeds to IBD. In search of more effective control measures to IBD, we investigated the resistance and susceptibility of unvaccinated native chickens to vvIBDV. The differences of maternal antibody decay of IBDV with regard to breed variation were evaluated. The relative susceptibilities of local Egyptian breeds to vvIBDV, There fore other attentions were directed toward breeding and genetics as a tool in disease prevention to select resistant breeds.

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دراسات للتعرف على قابلية السلالات المحلية واللوهمان الأبيض البياضه لفيروس التهاب غدة فابريشس المعدى (فيوم ٩٧)

تم استخدام عترة حقلية شديدة الضراوة من فيروس مرض الجمبورو (FY79)، وذلك لدراسة مدى قابلية السلالات ضد العدوى بهذه العترة شديدة الضراوة من فيروس مرض جمبورو وذلك كل خمسة أيام من العمر بداية من العمر ١٠ أيام - حتى العمر ٤٥ يوم ودراسة مستوى معدل الأجسام المناعية الحماني لكل سلالة وذلك باستخدام اختبار الأليزا وتوقع الوقت المناسب والمثالي للتحصيل، حيث أظهرت الأعراض الاكلينيكية ومعدلات الإصابة في سلالة الفيومي والندراوي أقل ٣٠% عند عمر ١٥ يوم من العدوى وكانت لكل من سلالة السيناوي، وسلالة البلدي، وسلالة اللوهمان ٣٠% عند عمر ٢٠ يوم من العدوى. وقد قامت هذه الدراسة لتقييم مدى امتداد الحماية للأجسام المناعية الأمية ضد العدوى بالعترة شديدة الضراوة بفيروس مرض الجمبورو، كل السلالات أظهرت أعراض اكلينيكية للعدوى عند عمر ٣٠، ٣٥ يوم. وكانت سلالة السيناوي بأعلى قيم لمعدلات الإصابة (٦٥% و ٧٠%)، يليها بعد ذلك سلالة الفيومي (٥٠% و ٥٥%)، سلالة الندرناوي (٥٠%) وسلالة البلدي (٥٥% و ٤٥%)، وأخيرا سلالة اللوهمان الأبيض (٥٠%) و (٤٥%). وكانت معدلات الوفيات نتيجة الإصابة بفيروس شديد الضراوة بمرض الجمبورو يختلف من (٠% إلى ٣٥%) بالمقارنة بالعمر، حيث أن سلالة الفيومي واللوهمان الأبيض حققا أقصى معدلات لنفوق (٣٥% و ٤٠%) على الترتيب وذلك عند عمر ٣٠ يوم. وكانت معدلات النفوق في كل من سلالة الندرناوي وسلالة السيناوي تتراوح ما بين (٥% إلى ٤٠%) على الترتيب، وذلك بطريقة متقاربة عند كل فترات العدوى ووصلت أعلاها عند عمر ٣٠ يوم بينما سلالة البلدي أوضحت نفس القيم ولكن بمعدل أقل فقط عند عمر ٢٠ و ٢٥ يوم. وكان متوسط نتيجة الإصابة الهيستولوجية (Lesion Score) في سلالة الفيومي أقل نتيجة (Score) في كل فترات العمر يليها بعد ذلك سلالة اللوهمان الأبيض، سلالة السيناوي، سلالة البلدي وأخيرا سلالة الندرناوي. وكانت نتائج اختبار الأليزا لقياس معدل الأجسام المناعية عند بداية العدوى أظهرت أن كفايت السيناوي هي أعلى المعدلات في الأجسام المناعية يليها بعد ذلك اللوهمان، البلدي، الندرناوي، وأخيرا الفيومي عند أغلب الفترات. لذلك من الضروري أن تكون دراسات أكثر لتوضيح أسباب هذه الظاهرة ودور الجينات الوراثية في الحماية ضد العدوى بفيروس مرض الجمبورو.