

The effect of *Salmonella paratyphoid* infection on immune response to Avian Influenza Vaccine in ducks

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Abstract

The present study was performed to evaluate the possible role of *paratyphoid salmonella* (*Salmonella Enteritidis*) infection as immunosuppressive agent to AI inactivated vaccine in white pekin ducks. Two hundreds of 1-day-old white pekin ducks were randomly allocated to four treatment groups, each 50 duckling: group 1 (**G1**), 1-day-old ducklings were inoculated orally with 10^5 CFU of *Salmonella Enteritidis* and vaccinated s/c with 1 ml of AI inactivated vaccine at ten days of age; **G2**, 1-day-old ducklings were inoculated orally with 10^5 CFU of *S. Enteritidis* and non-vaccinated; **G3**, non-infected, vaccinated with 1 ml of AI inactivated vaccine at ten days of age; **G4**, non-infected, non-vaccinated birds as a control. Evaluation parameters included immune response to AI inactivated vaccine as measured by haemagglutination inhibition (HI) test, organs/body weight ratio and index. Also, clinical sign, morbidity and mortality percentages, postmortem lesions and re-isolation of *Salmonella Enteritidis* were recorded. The obtained results denoted that first and second group revealed 100% morbidity (illness) and 10% mortalities. In third and fourth group neither morbidity nor mortalities were recorded. The shedding of the inoculated *Salmonella Enteritidis* detected via cloacae swabbing at 1-63 day post infection (PI). Evaluation of the experimental data revealed that non-infected, vaccinated group (**G3**) had significantly higher geometric mean of HI titers and positivity percent as compared with infected, vaccinated Group (1) at 3 wk, 5wk and 7wk post vaccination. Whereas, GMTs were 3.0 ± 1.04 , 3.8 ± 0.81 and 2.8 ± 1.23 and positivity percent were 20,60 and 30 respectively in the infected, vaccinated Group (1). While, the GMTs, in non-infected, vaccinated group (3) were 3.8 ± 0.31 , 6.4 ± 0.22 and 5.6 ± 0.50 and positivity percent were 40,100 and 90 respectively. On the other hand, *Salmonella Enteritidis* infection induced variable degree of bursal atrophy and spleen hypertrophy in infected ducks at 4th and 10th PI which explain possible a immunosuppressive effect of *Salmonella Enteritidis* infection on immune response to AI inactivated vaccine in white pekin ducks.

Key words: *Highly pathogenic avian influenza virus-paratyphoid, Salmonella Enteritidis, ducks, inactivated vaccine.*

Introduction

The H5N1 highly pathogenic avian influenza (HPAI) viruses continue circulating in poultry in Asia and have also spread to countries in the Middle East, Europe and Africa, causing great losses to the poultry industry. Highly pathogenic avian influenza (HPAI) viruses of the H5N1 subtype descending from A/goose/Guangdong/1/96 lineage were first detected in

Africa in 2006 (**Chen et al., 2006**). Egypt was the second African country affected by H5N1 HPAI, which reported cases of infection in poultry in early 2006. Initial attempts to control the epidemics by implementing stamping out and movement control measures were largely unsuccessful. In March 2006, vaccination of poultry was authorized essentially using inactivated vaccines derived from low patho-

genic H5N2 Mexican or H5N1 Asian strains (**Peyre *et al.*, 2009**). H5N1 HPAI virus infections are constantly monitored worldwide not only because of the high mortality they produce in poultry but also because of the spread to humans and fear of a pandemic. Both wild and domestic ducks have been implicated in the dissemination of H5N1 HPAI viruses (**Gilbert *et al.*, 2006** and **Songserm *et al.*, 2006**).

Domestic ducks not only can be affected by H5N1 HPAI, but also serve as key intermediates in the transmission of the virus and therefore are included in control programs including vaccination (**Li *et al.*, 2004** and **Swayne, 2006**). Normally avian influenza (AI) viruses, including the highly pathogenic H5 and H7 strains, do not cause disease or death in ducks (**Swayne and Halvorson, 2008**). Mortality among ducks naturally infected with AI viruses was first reported in 2001 during an outbreak of H7N1 HPAI virus in Italy, and then in 2002, with Asian-origin H5N1 HPAI viruses causing mortality in wild birds and waterfowl (**Capua and Mutinelli, 2001** and **Ellis *et al.*, 2004**). Since then, a number of H5N1 HPAI viruses have been found to cause disease in ducks (**Pantin-Jackwood and Swayne, 2009**). Vaccination has proven effective in protecting ducks against H5N1 HPAI and is being used in several Asian countries to control the disease (**Sims, 2007**); however, virus circulation may still occur in clinically healthy vaccinated populations, which may contribute to endemic situations (**Capua and Alexander, 2008**).

Economically, Salmonella paratyphoid infections are among the bacterial diseases that are most important to the hatching industry, with an estimated annual loss of \$77 million. (**Hassan *et al.*, 1993**). Salmonella paratyphoid infections commonly occur in poultry at different ages, but seem to have maximum impact in younger birds. Colonization by Salmonella results in invasion of the gut wall and the further dissemination to internal organs (**Porter, 1998**). Young birds are more susceptible to Salmonella infections when compared to older birds. This may be partially attributed to the

fact that the gastrointestinal tract (GIT) is not well colonized at hatch and is, therefore, amenable to colonization by pathogens such as Salmonella (**Porter, 1998**).

Additionally, Salmonella paratyphoid infection at hatch causes significant damage to the gut lining early on (**Porter, 1998**). The immunologic naivety of young birds may also lead to less robust immune responses and, therefore, slower clearance from the host as compared to older birds (**Beal *et al.*, 2004**). **Hassan and Curtiss, (1994)** demonstrated significant perturbation of lymphocyte development and atrophy of lymphoid organs and have an adverse effect on chicken immunocompetence in day-old chicks challenged with highly virulent *S. typhimurium* and may encourage the development of a Salmonella carrier status in addition to persistent shedding. The objective of the present study was to evaluate the possible role of Paratyphoid Salmonella infection as immunosuppressive agent to AI inactivated vaccine in white pekin ducks.

Materials and Methods

Experimental ducks:

two-hundreds 1-day-old newly hatched salmonella free commercial unsexed white pekin ducks were employed. The birds were divided into 4 groups, each 50 ducklings and transferred to separate deep litter rooms to prevent contact between birds from different treatment groups. The isolation rooms were decontaminated by suitable disinfectants. All birds received drinking water and feed *ad libitum*.

Inactivated Oil-emulsion AIV H5N2 vaccine: Was used obtained from the local agency. The AIV strain was chicken/Mexico/232/94/CPA.

Reference AIV H5N2 Antigen:

AIV hemagglutinating antigen H5N2: which present the homologous antigen of the above mentioned vaccine. The antigen was obtained from local agency and was used in HI test.

Standard positive and negative AIV antisera: imported from Intervet International, B. V. Boxmeer Holland were obtained from local agency and was used in HI test.

Haemagglutination test: Procedures were

performed according to **OIE terrestrial manual (2009)**.

The haemagglutination inhibition assay (HI): was performed with 4HAU/25 ml of the inactivated homologous H5N2 antigen after heat pretreatment of sera (sera were inactivated at 56°C for 30 minutes), and consistent with standard methods of **OIE (2009)**. The HI titer was expressed as the reciprocal of the highest dilution that caused complete inhibition of agglutination. A HI titer of ≥ 16 is considered a positive result in ducks under OIE guidelines

Infective inoculum preparation:

Salmonella Enteritidis strain was kindly provided by Prof. Dr. Hany Fawzy Ellakany, Professor of poultry diseases. Faculty of Vet. Med. Damanhur Univ. Bacterial cells were suspended in the broth plus mixture of 40% wv shalk, 43% wv light kaolin and 17% wv magnesium hydroxide to prevent strain from acidic environment of duck stomach (**Smith, 1956**). The Salmonella Enteritidis is approximately 10^5 colony forming units (CFU) / ml.

Experimental design:

At 1 day of age, the birds were divided into four groups, each 50 duckling. Group one, birds were infected orally with approximately 1×10^5 CFU of Salmonella Enteritidis at one day of age and vaccinated s/c by 1 ml of inactivated oil-emulsion AIV H5N2 vaccine at ten days of age; group two, birds were infected orally with same dose of Salmonella Enteritidis at one day of age and non-vaccinated. Group three, birds were non-infected and vaccinated s/c by 1 ml of inactivated oil-emulsion AIV H5N2 vaccine at ten days of age; in group four, birds were non-infected and non-vaccinated. All ducks were kept under observation throughout the experimental period.

Organ/body weight ratio and index: At 4, 10 days of age, 5 birds from each group were taken randomly and sacrificed then, spleens and bursae were collected and weighed individually, bursa/body weight ratio or spleen/body weight ratio was calculated for each bird according to the formula of **Sharma et al. (1989)**. Then, bursa/body weight index or spleen/body weight index was calculated for

each bird according to the formula of **Lucio and Hitchner (1979)**.

Blood samples for serology:

At 4, 10 days of age, 5 birds from each group were taken randomly and sacrificed for blood samples collection as well as five to ten blood samples were collected from each of the four groups of ducks throughout the experiment at three weeks, five weeks and seven weeks post vaccination. Sera were inactivated at 56°C for 30 minutes then antibody titers to AIV H₅N₂ were determined by HI as described previously by **OIE (2009)**.

Bacteriological analysis :for re-isolation of Salmonella Enteritidis which was carried out as a follow:

a– Swabs: The shedding was tested by rectal swabbing according to **Bhatia and McNabb (1980)** at 24, 48, 72. 96 hours post-inoculation and repeated at the end of 1st week, then weekly till 9th week post inoculation. Individual rectal swabs were directly inoculated onto seleniet of broth and rubabart vaseliades broth and incubated overnight at 37°C the broth then cultured on S agar and X L D agar also incubated overnight at 37°C. Suspected colonies were picked and identified biochemically according to **Gast, et al. (1993)** and **Yuno, et al. (1995)**

b– Tissues: After dissections of freshly dead or sacrificed ducks at 4 & 10 days of age, the collected tissues including liver, heart, blood, spleen brain, yolk sac if present were minced in buffered peptone broth incubated overnight at 37°C. Then small portion was carried of seleniet of broth and rubabart vaseliades broth incubated overnight at 37°C. Then cultured on S S agar and X L D agar also incubated overnight at 37°C. Suspected colonies were picked and identified biochemically according to **Christensen, et al., (1992)**.

Statistical analysis: All data were collected and statistical analysis was carried out using analysis of variance (ANOVA) according to **SAS (1987)**.

Results

Clinical signs: The affected birds in-groups (1&2) showed marked depression, weakness, depressed appetite, and adherence of chalky white material to the vent. The affected birds exhibit lassitude and an inclination to huddle together under heaters, having droopy wings, and distorted body appearance. Labored breathing or gasping observed as a result of extensive involvement of the lungs. Few number of survivors greatly retarded in their growth and appear underdeveloped and poorly feathered, nervous manifestation appeared in one case at 8th day PI. First and second groups showed 100% morbidity (illness) and 10% mortalities. In third and fourth group neither morbidity nor mortalities were recorded.

PM lesion: Affected birds in-groups (1&2) showed slightly enlarged and congested liver, spleen, and kidneys, enteritis, the yolk sac contents had creamy or caseous consistency. Birds with respiratory signs had congested lung. Severely atrophied thymus and bursa in almost infected ducks.

Re-isolation of Salmonella enteritidis: The shedding of the inoculated Salmonella Enteritidis were detected via cloacae swabbing at 24, 48 72 and 96 hours PI. Also. Salmonella Enteritidis were detected from cecci and yolk sacs of freshly dead cases

At 4th and 10th day PI, Salmonella Enteritidis were detected via cloacae swabbing, heart blood gall bladder, liver and cecci of scarified cases. Salmonella Enteritidis were detected from cultured brain tissue of one affected duck suffering from severe nervous manifestation at 7th day PI. Shedding of Salmonella Enteritidis were detected via cloacae swabbing at 14, 21, 28, 35, 42, 49th, 56th and 63th day PI i.e. till the end of experimental period.

Effect of Salmonella Enteritidis infection on immune response of white pekin ducks to inactivated avian influenza vaccine (H5N2) at different period of experiment: The Geometric mean of HI titers were shown in table (1) and fig.(1. a & b), the results revealed that 4- day-old ducklings had maternal antibody

levels ranged from (7.8 ± 0.37 HI unit) to (8.0 ± 0.23 HI unit) which gradually declined to reach a mean of (2.6 ± 1.42 HI unit) to (2.6 ± 1.47 HI unit) by 10 days of age. Then, all ducklings in non-vaccinated groups (2 & 4) were seronegative by HI at different intervals of experiment. Antibody response to AIV vaccine was seen by 3 wk, 5wk and 7wk post vaccination with variety in geometric mean of HI titers and positivity percent. the GMTs in the infected, vaccinated group (1) at 3 wk, 5wk and 7wk post vaccination were 3.0 ± 1.04 , 3.8 ± 0.81 and 2.8 ± 1.23 and positivity percent were 20, 60 and 30 respectively. While, the GMTs, in non-infected, vaccinated group (3) at 3 wk, 5wk and 7wk post vaccination were 3.8 ± 0.31 , 6.4 ± 0.22 and 5.6 ± 0.50 and positivity percent were 40, 100 and 90 respectively. The results showed significantly lower ($p \leq 0.05$) AIV geometric mean HI titers and positivity percent in the infected, vaccinated group (1) at different intervals PV than the non-infected, vaccinated group (3). On other hand, the vaccinated groups either infected (G.1) or non-infected (G.3) had significantly higher ($p \leq 0.05$) AIV geometric mean HI titers and positivity percent than that of non-vaccinated group (2 & 4) at different intervals PV.

Effect of salmonella enteritidis infection on bursa/body weight ratios and indexes:

As presented in table (2) and fig. (2), the average mean of bursa/body weight ratios and indexes in infected groups (1 & 2) were significantly ($p \leq 0.01$) reduced at 4 and 10 days PI as compared with the non-infected groups (3 & 4).

Effect of salmonella enteritidis infection on spleen/body weight ratios and indexes:

As presented in table (3) and fig. (3), the average mean of spleen/body weight ratios and indexes in infected groups (1&2) were significantly ($p \leq 0.01$) increased at 4 and 10 days PI as compared with the non-infected groups (3&4).

Discussion

In the present study a special concern was taken to study the effect of Salmonella enteri-

tidis infection on immune response of white pekin ducks. All infected ducks in groups (1&2) showed clinical signs and gross lesions which were agreed with that previously recorded by (**Shivaprasad, et al., 1990; Hoop and Pospischil. 1993**). While, non-infected ducks in groups (3 & 4) remain normal throughout the experimental period.

In the poultry industry, field challenges could be more severe and cause more damage to the gastro-intestinal tract than that seen in this experimental model. The microbial diversity of the gastro-intestinal tract dramatically changes when ducks were infected with *Salmonella enteritidis*. So that, the shedding of *Salmonella enteritidis* continued till end of the experimental period (63 days of age). This findings agreed with records of **Gast and Holt, (1998)** who mentioned that after oral inoculation of 1-day-old chicks, *S. Enteritidis* was shed in the feces of nearly half of these birds at 6 months of age; **Phillips and Opitz, (1995)** reported that ParaTyphoid infection has been persisted for 64 weeks after the exposure of young chicks and **Skoy et al., (2002)** who reported that some internal tissues remained *Salmonella*-positive for 1 year after inoculation of 1-day-old chicks.

The results in table (1) showed that there was a significant interaction between the infection with *Salmonella enteritidis* and the immune response to AIV inactivated vaccine (H_5N_2) manifested by significantly lower ($P \leq 0.05$) AIV GM HI titers and positivity percent in the infected, vaccinated group (1) than the non-infected, vaccinated group (3) at different intervals PV. Our findings agreed with previous reports (**Youssief et al., 1996; Hegazy et al., 1999 and Hamouda et al., 2001**).

The average mean of bursa/body weight ratios and indexes in the infected group (1&2) were significantly ($p \leq 0.01$) reduced and indicating variable degrees of bursal atrophy as compared with the non-infected groups (3&4) at 4 and 10 days PI with *Salmonella enteritidis*. Our findings agreed with results of **Hassan and Curtiss (1994)**, who found a bursal atrophy in chickens infected with *Salmonella typhi-*

murium at 1- day and 3 weeks of age and with results of **Hamouda et al., (2001)** who found a bursal atrophy in chickens infected with *Salmonella pullorum* at 4 days of age.

The average mean of spleen/body weight ratios and indexes in the infected groups (1 & 2) were significantly ($p \leq 0.01$) increased as compared with the non-infected groups (3 & 4) at 4 and 10 days PI. The spleen in these groups enlarged as a compensatory mechanism of the damaged bursa, which is in agreement results of **Hassan and Curtiss (1994)**, who found a spleen hypertrophy in chickens infected with *Salmonella typhimurium* at 1-day and 3 weeks of age.

Conclusion

The *Salmonella enteritidis* infection used in this study share in the immunosuppressive condition that commercial duck flocks face daily. So that, the duck flocks must be free from *Salmonella enteritidis* and duck flocks should be protected by good management practices, using of immune stimulant and several antibiotics to treat *Salmonella enteritis* to subside the immunosuppression effect which may result from the infection.

Table (1). Effect of *salmonella enteritidis* infection on immune response of white Pekin ducks to inactivated avian influenza vaccine(H5N2) at different period of experiment (Arithmetic mean).

Group No.		G1 : infected, Vaccinated		G2: Infected, non-vaccinated		G3: Non-infected, vaccinated		G4:Non-infected, non-vaccinated	
Age (day)	No*	Geometric mean $\pm$ SE	positive percent	Geometric mean $\pm$ SE	positive percent	Geometric mean $\pm$ SE	positive percent	Geometric mean $\pm$ SE	positive percent
4 th -d	5	Aa 7.8 $\pm$ 0.37	100 A	Aa 7.8 $\pm$.39	100 A	Aa 8.0 $\pm$ 0.23	100 A	Aa 8.0 $\pm$ 0.13	100 A
10 th d	5	Da 2.6 $\pm$ 1.42	20 D	Ba 2.6 $\pm$ 1.47	20 B	Ea 2.6 $\pm$ 1.41	20 D	Ba 2.6 $\pm$ 1.41	20 B
31d. (3 rd w pv)	5	Cb 3 $\pm$ 1.04	20 D	Cc 0	0 C	Da 3.8 $\pm$ 1.31	40 C	Cc 0 $\pm$ 0	0 C
45d. (5 th w pv)	5	Bb 3.8 $\pm$ 0.81	60 B	Cc 0	0 C	Ba 6.4 $\pm$ 0.22	100 A	Cc 0 $\pm$ 0	0 C
59d. (7 th w pv)	10	Db 2.8 $\pm$ 1.23	30 C	Cc 0	0 C	Ca 5.6 $\pm$ 0.50	90 B	Cc 0 $\pm$ 0	0 C

Capital letters: Means within the same column of different letters are significantly different at ($P \leq 0.05$).

Small letters: Means within the same row of different letters are significantly different at ($P \leq 0.05$).

*: Number of tested serum

Table (2). Effect of *Salmonella enteritidis* on body weight, bursa weight, bursa/body weight ratio and bursal index of white Pekin ducks at different periods of experiment.

Groups	Age	No	Body weight (gm)	Bursa weight (gm)	Bursa/body weight ratio	Bursal index
			Mean Std. Error	Mean Std. Error	Mean Std. Error	Mean Std. Error
Infected ,vaccinated	4 th -day	5	E 111.80 $\pm$ 2.08	D 0.12 $\pm$ 0.03	F 1.07 $\pm$ 0.26	C 0.51 $\pm$ 0.12
Infected, non-vaccinated		5	E 114.20 $\pm$ 2.29	D 0.12 $\pm$ 0.03	F 1.04 $\pm$ 0.24	C 0.49 $\pm$ 0.12
Non-infected, vaccinated		5	C 186.20 $\pm$ 7.69	B 0.40 $\pm$ 0.03	C 2.13 $\pm$ 0.10	A 1.01 $\pm$ 0.05
Non-infected, non-vaccinated		5	C 188.00 $\pm$ 7.20	B 0.40 $\pm$ 0.03	C 2.11 $\pm$ 0.09	A 1.00 $\pm$ 0.04
infected ,vaccinated	10 th - day	5	D 150.46 $\pm$ 36.23	C 0.22 $\pm$ 0.05	D 1.51 $\pm$ 0.08	B 0.61 $\pm$ 0.04
Infected, non-vaccinated		5	D 160.21 $\pm$ 33.52	C 0.22 $\pm$ 0.04	E 1.40 $\pm$ 0.05	B 0.63 $\pm$ 0.02
Non-infected, vaccinated		5	B 303.00 $\pm$ 11.39	A 0.71 $\pm$ 0.07	A 2.31 $\pm$ 0.15	A 1.04 $\pm$ 0.07
Non-infected, non-vaccinated		5	A 315.80 $\pm$ 15.89	A 0.71 $\pm$ 0.07	B 2.22 $\pm$ 0.14	A 1.00 $\pm$ 0.06

Means within the same column of different letters are significantly different at ($P \leq 0.01$).

Table (3). Effect of *salmonella enteritidis* on body weight, spleen weight, and spleen/body weight ratio and spleen index of White Pekin ducks at different periods of experiment.

Groups	Age	No	Body weight (gm)	spleen weight (gm)	spleen /body weight ratio	spleen index
			Mean Std. Error	Mean Std. Error	Mean Std. Error	Mean Std. Error
infected,Vaccinated	4 th - day	5	111.80±2.08 E	0.099±0.03 D	0.88±0.24 C	1.06±0.30 B
Infected, non-vaccinated		5	114.20±2.29 E	0.10 ±0.03 D	0.87 ±0.24 C	1.05±0.27 B
Non-infected, vaccinated		5	186.20±7.69 C	0.154 ±0.03 C	0.83±0.14 D	0.92±0.18 D
Non-infected, non-vaccinated		5	188.00±7.20 C	0.154 ±0.03 C	0.81±0.13 D	0.92±0.17 D
Infected, vaccinated	10 th – day	5	150.46±36.23 D	0.166±0.02 B	1.23±0.17 A	1.21±0.16 A
Infected, non-vaccinated		5	160.21±33.52 D	0.170±0.02 B	1.22±0.13 A	1.20±0.12 A
Non-infected, vaccinated		5	303.00±11.39 B	0.34±0.04 A	1.11±0.10 B	1.03±0.09 C
Non-infected, non-vaccinated		5	315.80±15.89 A	0.35±0.04 A	1.08±0.08 B	1.00±0.08 C

Means within the same column of different letters are significantly different at ($P \leq 0.01$).

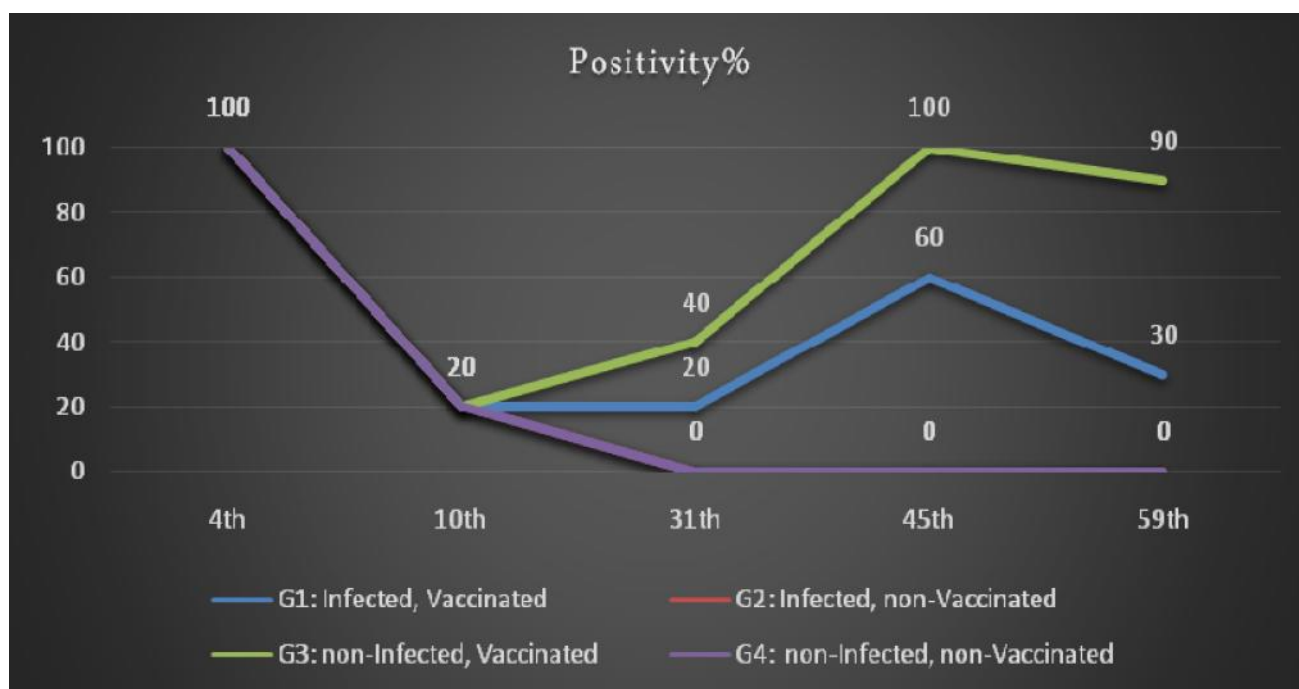
Fig. (1.a). Effect of *salmonella enteritidis* infection on positivity percent of AIV HI titres of white Pekin ducks to inactivated avian influenza vaccine (H5N2) at different period of experiment.

Fig. (1.b). Effect of *salmonella enteritidis* infection on geometric mean AIV HI titres of white Pekin ducks to inactivated avian influenza vaccine (H5N2) at different period of experiment

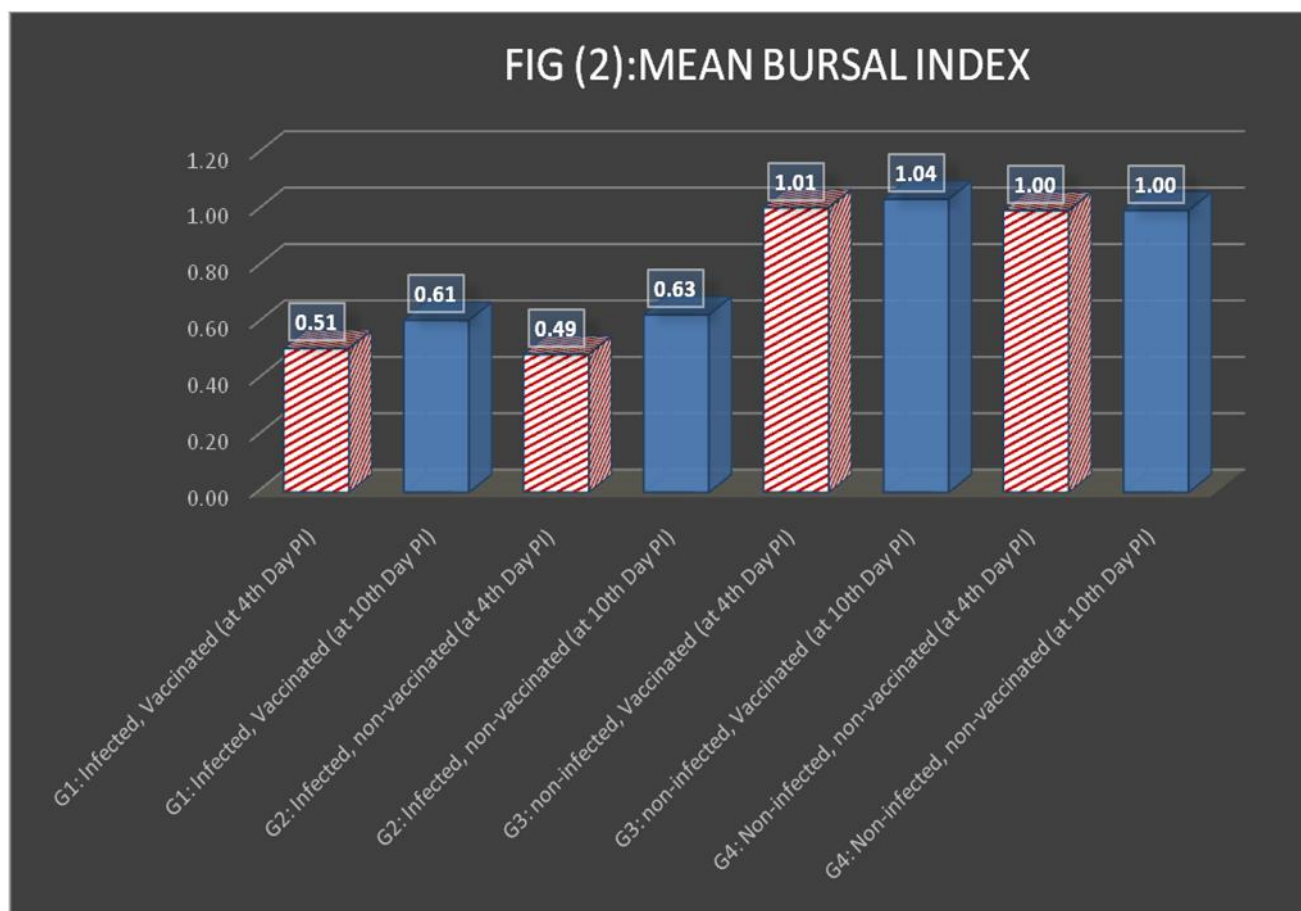
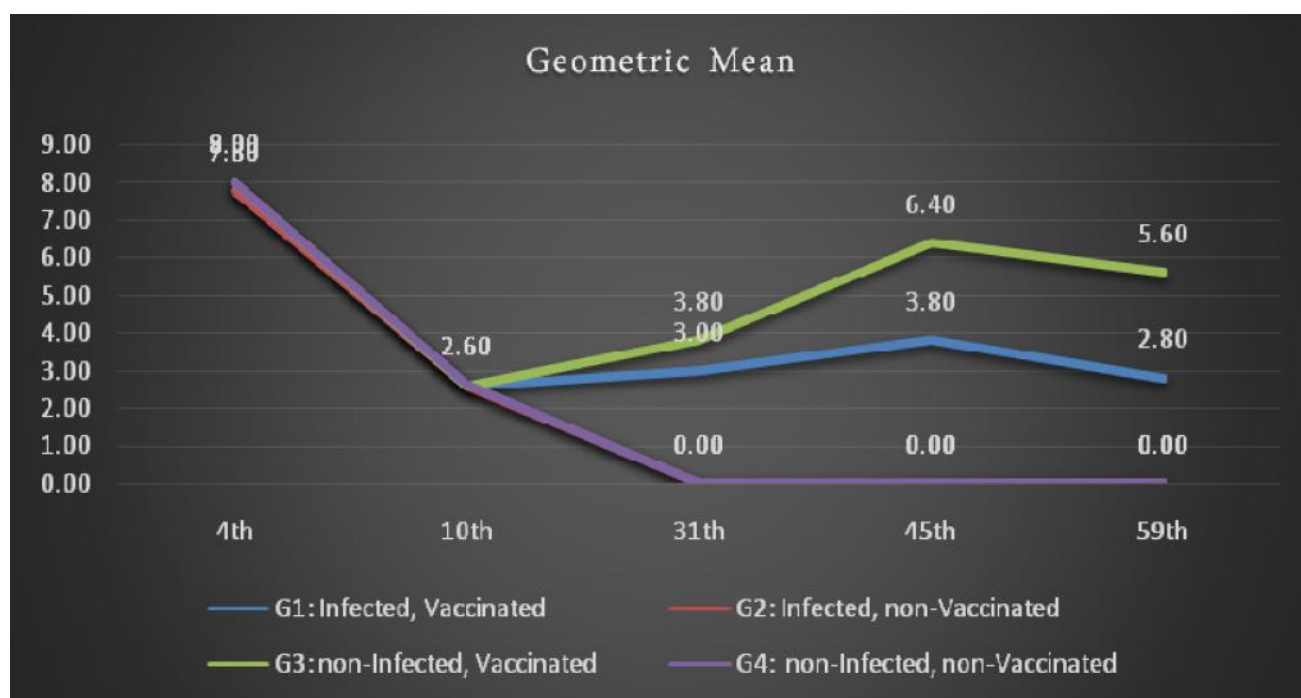
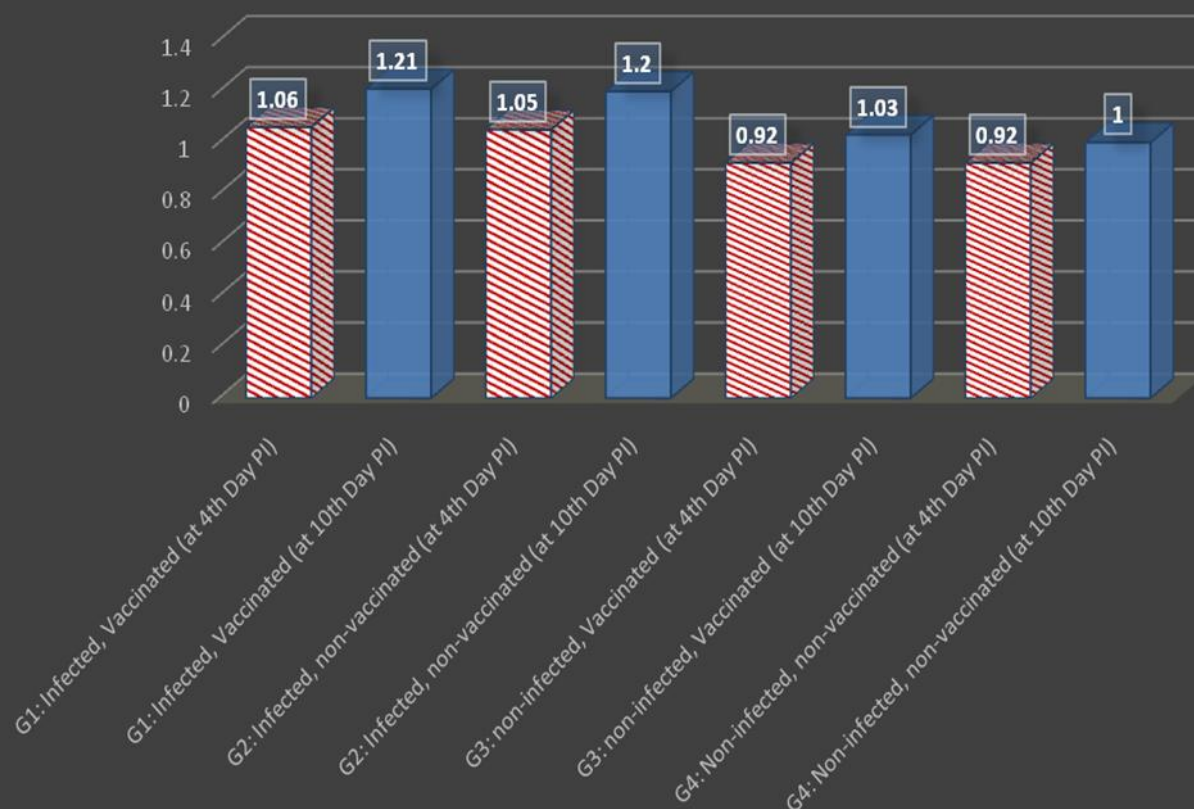


FIG.(3):MEAN SPLEEN INDEX



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تأثير عدوى السالمونيلا باراتيفويد على الاستجابة المناعية للقاح أنفلونزا الطيور في البط عيد جمعه شحاته حسين* ، هانى فوزى اللقانى** ، أحمد أبو المجد بخيت*

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المخلص العربى

أجريت هذه الدراسة على ٢٠٠ بطة بكينى أبيض مختلط الجنس عمر يوم واحد لأستبيان تأثير عدوى السالمونيلا باراتيفويد على الاستجابة المناعية للقاح أنفلونزا الطيور. حيث تم تقسيم ٢٠٠ بطة الى أربعة مجموعات متساوية كل مجموعة ٥٠ بطة عند عمر ١ يوم. المجموعة الأولى: تم عدواها بميكروب السالمونيلا باراتيفويد 10×10^8 (cfu) ومحصة ب (مللى من لقاح أنفلونزا الطيور الميت؛ الثانية: تم عدواها بميكروب السالمونيلا باراتيفويد 10×10^8 (cfu) وغير محصة؛ المجموعة الثالثة: لم يتم عدواها بميكروب السالمونيلا باراتيفويد ومحصة بنفس الجرعة؛ المجموعة الرابعة: مجموعة ضابطة ولم يتم عدواها بميكروب السالمونيلا باراتيفويد وغير محصة. وقد تم التقييم بناء على قياس الاستجابة المناعية للقاح أنفلونزا الطيور بواسطة اختبار مانع التلازن (HI) وتأثير عدوى السالمونيلا باراتيفويد على اوزان الطحال و غدة كيس فابريشى، وكذلك تم دراسة الأعراض الاكلينيكية الظاهرة، نسب النفوق، الأفات التشريحية الناتجة عن العدوى بميكروب السالمونيلا باراتيفويد وإعادة عزل ميكروب السالمونيلا باراتيفويد من البط المصاب. وأظهرت الدراسة التأثير السلبى للعدوى بميكروب السالمونيلا باراتيفويد متمثلاً فى انخفاض معنوى فى نسبة وقيمة الاستجابة المناعية للقاح أنفلونزا الطيور، ضمور فى غدة كيس فابريشى، زيادة فى وزن الطحال فى البط المصل. وكانت نسبة الأعراض الاكلينيكية الظاهرة (١٠٠%)، نسبة النفوق (١٠%) بالمجموعتين المصابتين (٢&١) وقد تم إعادة عزل ميكروب السالمونيلا باراتيفويد من ٢٤ ساعة-٦٣ يوم بعد العدوى من البط المصاب بالمجموعتين (٢&١). لذا يجب أن يكون البط خالى من ميكروب السالمونيلا باراتيفويد وننصح بتطبيق نظم الرعاية الأساسية من جودة المياه والتغذية المثلى واستخدام منشطات المناعة واستخدام العلاجات الكيميائية المناسبة والفعالة ضد ميكروب السالمونيلا باراتيفويد لتجنب تأثيره الضار على الجهاز المناعى.