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SOME STUDIES ON BACTERIAL CAUSES ASSOCIATED WITH CASES OF SWOLLEN HEAD SYNDROME IN CHICKENS

(With 4 Tables and 14 Figures)

By

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(Received at 23/12/2004)

بعض الدراسات عن المسببات البكتيرية المصاحبة لحالات ظاهرة تورم الرأس فى الدجاج

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تم تجميع ١٦٨ دجاجة حية ونافقة حديثاً من بعض مزارع التسمين بمحافظة أسيوط. وكانت أعمارهم تتراوح ما بين ٤-٦ أسابيع، وكانت نسبة كبيرة من هذه الطيور بها تورم فى الرأس والباقى سليم ظاهريا. وقد اسفر الفحص الظاهرى عن وجود التهابات فى الجفون مع انتفاخ الجيب التحت عينى وتورم الوجه مع وجود بعض الحالات كانت مغلقة العينين تماماً، مع رشح من الأنف وصعوبة فى التنفس وكانت هناك حالات أخرى تعانى من إسهال شديد بالإضافة للأعراض السابقة. وكانت الصفة التشريحية متمثلة فى أحتقان القصبة الهوائية وعتامة فى الأكياس الهوائية وكانت الحجرات الهوائية والأنف تحتوى على سائل مخاطى. وتمت محاولة عزل مسببات المرض البكتيرية وامكن عزل ١٥٠ عترة منهم ٧٦ عترة من الميكروب القولونى و٢٤ عترة من ميكروب الميكوبلازما جاليسبتكم وكذلك عزل ٢٠ عترة من ميكروب الهيموفيليس باراجاليثيرم و١٤ عترة من ميكروب السيدوموناس وعدد ٨ عترات من ميكروب الكلبسيللا وعدد ٦ عترات من ميكروب البروتيس وعدد ٢ عترة من ميكروب إستاف أوريس. وقد تم فحص هذه العترات من خلال الشكل الظاهرى للميكروب والخواص البيوكيميائية والسيرولوجية. ثم تم القيام بعمل عدوى صناعية فى كتاكيت تسمين عمر ٤ أسابيع بالعترات المعزولة. فقد تم حقن المجموعة الأولى داخل الجيب الأنفى لميكروب الهيموفيلس باراجلينيرم بمفرده. وحقن المجموعة الثانية داخل الجيب الأنفى بالميكوبلازما جاليستيكم والمجموعة الثالثة بالميكروب القولونى عن طريق الفم وحقن مجموعة رابعة بميكروب الميكوبلازما جاليسبتكم والميكروب القولونى كل على حده ووضعت طيور هذه المجموعة فى ظروف بيئية سيئة من حيث سوء التهوية وزيادة كثافة الطيور وإرتفاع نسبة الرطوبة فى الجو وكذلك نقص فى كمية الغذاء وقد تبين من هذه التجربة أن الطيور التى حقنت بميكروب الميكوبلازما جاليسبتيكم والميكروب القولونى ووضعت فى ظروف بيئية سيئة قد ظهرت عليها اعراض إصابة معقدة ونسبة عالية من النفوق وكانت فترة حضانة المرض حوالى ٢٤ ساعة. أما الأعراض فكانت أشد خطورة وضراوة والصورة التشريحية كانت إنعكاساً لهذه الأعراض. تم وصف ومناقشة التغيرات الباثولوجية الميكروسكوبية فى الجفون

والرئتين والكبد والأمعاء مما يلقى الضوء على خطورة العدوى المعقدة بميكروبات شديدة الضراوة مثل الميكوبلازمات والميكروب القولونى. مما أضعف مقاومة الطائر وأفسح المجال لهذه الميكروبات أن تنتقل إلى أجزاء أخرى من جسم الطائر وما يتبع هذا من أعراض أشد خطورة وخسائر أقتصادية فادحة.

SUMMARY

One hundred and sixty eight birds were collected from 4 broiler flocks in Assiut farms (governmental and private)from both live and freshly dead birds, aging between 4-6 weeks. The clinical signs showed swelling of heads with conjunctivitis and some birds were with closed eyes, nasal discharge and diarrhea was also noticed in some cases. The causative bacterial agents could isolated only from the sinus and the upper respiratory tract. 150 isolates were recovered from 168 cases, 76 isolates, 24, 20, 14 isolates *Pseudomonas aeruginosa,* 8 *Klebsiella,* 6 isolates Proteus, and 2 isolates *Staphylococcus aureus.* The pathogenicity of *E. coli, Mycoplasma gallisepticum* and *Haemophilus paragallinarum* to 4 weeks old broiler birds free from any bacterial infection revealed appearance of depression, conjunctivitis, sneezing, nasal discharge within 3-4 days post inoculation. Injection of birds by *Mycoplasma gallispeticum* and *E. coli* in combination with bad hygienic condition, birds showed signs within 24 hours post inoculation in the form of nasal discharge, sneezing, abnormal sounds, facial oedema, swelling in the infraorbital sinuses, conjunctivitis, abnormal ocular secretion and diarrhea. The postmortum of dead cases revealed septicaemia, severe enteritis, perihepatitis, congestion of the lung, air saculitis, sinusitis, tracheitis, pericarditis and caseous material in the nasal passages. Histopathological studies of the chickens experimentally infected with *M. gallisepticum* and *E. coli* revealed pathognomonic changes in the cutaneous and subcutaneous tissue of periocular skin and eyelids, liver, lung and intestine.

Key words: *Swollen head, chickens.*

INTRODUCTION

Swollen head syndrome (SHS) is recently described as an acute respiratory disease observed in two to six weeks old broiler chickens. SHS was first described by Morley and Thomson (1984) who attributed

the disease to a corona virus with *E.coli* and the disease seen in broiler chickens between 4 and 6 weeks of age in Southern Africa.

O'Brien (1985) observed a number of broiler parent flocks in East Anglia which shown unusual signs as swollen heads and severe depression. These symptoms are the result of periorbital oedema which is often unilateral. He isolated pure culture of *E.coli* from the head lesions and meninges. Litjens *et al.,* (1989) reported a case of SHS in guinea fowl and could isolate *E.coli* and staphylococci during the course of the disease. Treatment of these cases with Baytril produced satisfactory results. Qureshi (1991) recorded the disease in broiler farms in parts of Saudi Arabia and the microbiological examination revealed *E.coli* in some birds. Arns and Hafez (1992) described the disease in Brazil that it mostly occurs in broilers between 4 and 6 weeks of age. *E.coli* was isolated from the subcutaneous exudate over the head and from some tracheal swabs. Both Goodwin and Wattman and Droual and Woolcock (1994) investigated cases of SHS and isolated many bacteria as *Pseudomonas aeruginosa, E.coli, Proteus, Clostridium* and *Staphylococcus aureus*. Tanaka *et al.* (1995) stated the first report of an outbreak of SHS occurred in a commercial broiler farm in Miyzaki. *E.coli* and Proteus mirabilis were isolated from these cases. Georgides *et al.* (2001) examined 50 commercial flocks in Greece suffering from respiratory disease and signs of swollen head syndrome. The trachea and head were collected from each bird for laboratory investigation. Bacteriological examinations of the affected birds were made from the infraorbital sinuses resulted in the isolation of *E.co*li, *staphylococcus* spp. *Mycoplasma synoviae* and *Mycoplasma gallisepticum*. They concluded that the TRT virus did not play a causal role in SHS in commercial broiler flocks in Greece but *Mycoplasma* or other bacteria and environmental conditions seem to be essential for the occurrence and severity of the disease.

Murakami *et al.* (2002) tested a farm of Japanese quail for egg production, the birds showed swelling head, nasal discharge, increased lacrimation, decreased egg production and mortality rate of 5.7% per day. They isolated *M. gallisepticum, Pasteurella multocida, E.coli, Staphylococcus* spp., *Streptococcus* spp. and *Haemophilus paragallinarum (H. paragallinarum)*. They concluded that the swelling heads in birds due to mixed infection with *M. gallisepticum* and the high level of ammonia fumes promoted infection and multiplication of *M. gallisepticum.*

MATERIALS and METHODS

(1) Samples:

Four broiler farms of 4-6 weeks of age at Assiut governorate were manifested by swelling of the face, eyelids and the head. After 72 hours from the first signs, some birds showed severe oedema of the head and the face with total closure of the eyes and oedema of the tissues in lower jaw and neck. The wattles were swollen only in a small number of birds as well as variable rates of morbidity and mortality. The clinical data concerning these flocks are shown in table (1)

A total of 168 birds with SHS were observed for clinical signs , post mortem and bacteriological examination.

Antisera:

- *E.coli* "O"antisera from Bhring Werk Marburg, Lahn, Germany, were used for serotyping of the isolated strains. The procedure outlined by using polyvalent and monovalent *E. coli* antisera (Edward and Ewing, 1972).
- Reference *M.gallisepticum* antisera were kindly supplied by prof. Dr. Adel M. Soliman, Dept. of Poultry Diseases Assiut University.
- Standared strains of *H. paragalinarum* (Strain 221, Spross and H-18) represnting H. paragalinarum serovars A, B, and C respectively were kindly supplied by Dr M. Aly, Prof. of Poultry Diseases, Assiut University.

(2) Isolation of bacterial agent:

(i) Isolation of *H. paragallinarum*:

Samples from infraorbital sinuses, exudate from the subcutis of swollen head, trachea, air sac exudates and eye content were collected and cultured on tryptose agar supplemented with 10% sterile sheep blood with 2.5 mg/mL reduced form of Nicotinamide Adinine Dinucleotide (NAD) and incubated at 37°C under 10% CO_2 tension using candle jar for 24-48 hours.

(ii) Isolation of other bacterial agents:

Samples were inoculated into nutrient broth at 37°C for 24 hours then, streaked on nutrient agar and MacConkey's agar and incubated aerobically at 37°C for 24-48 hours, according to the method of Koneman *et al*. (1994) and Quinn *et al*. (1994).

(iii) Isolation of Mycoplasma

Samples obtained from trachea, air sacs and lung were cultured on brain heart infusion broth at 37°C for 3 days then subcultured on

brain heart infusion agar plates at 37°C in moist candle jar under reduced oxygen tension for 7 days, as described by Sabry (1968).

(3) Identification of the isolated organisms:

Colonial and cellular morphology:

Colonial morphology were studied and cellular examination from these colonies were done using Gram stain and Indian Ink staining.

Biochemical examination:

All isolates were subjected to sugar fermentation tests (glucose-lactose - mannitol and sucrose). These tests were done according to Cruickshank *et al*. (1975). Indol production, oxidase test, nitrate reduction test and arginin were carried out according to Cruickshank *et al*. (1975), Kume *et al*., 1978; Erno and Stipkovits, 1973.

(4) Serological identification:

Isolates that produced biochemical reaction simulating *H. paragallinarum* were subjected to serological identification by using rapid plate agglutination test as described by Kume *et al.*, (1978). Isolates that produced biochemical reaction simulating *E.coli* were serologicoly identified after their purification by determination of the group antigens using slide agglutination test against the *E.coli* antisera. Isolates that produced biochemical reaction simulating *M. gallisepticum* were serologically identified by growth inhibition test (Clyde, 1964). On the other hand, other organisms either Gram-ve or Gram+ve were identified only by biochemical tests as their respective immune sera were not available.

(5) Pathogenicity test:

A number of 50 broiler chickens of 4 weeks old were divided into 5 equal groups (10 birds each) and used for testing the pathogenicity of each isolate.

The first group was inoculated via intrasinus route with 0.2 ml of 24 hours broth culture containing 10^8 CFU/ml of *H. paragallinarum* (Rimler, 1979).

The second group was inoculated via intrasinus route with 0.1 ml of 24 hours broth culture containing 10^8 CFU/ml *M. gallispeticum* (Rocke *et al*. 1988).

The third group was inoculated orally with 0.1 ml of 24 hours broth culture of *E.coli* (Gross, 1956).

The fourth group was inoculated via intrasinus route with 0.1 ml of 24 hours broth culture containing 10^8 CFU/ml *M. gallisepticum* and

orally with 0.1 ml of 24 hours broth culture of *E.coli* and kept under bad hygienic condition (Gross, 1957).

- The fifth group was kept as non infected control.

Birds were observed for 6 weeks for clinical signs and the deaths were recorded. At the end of the 6 weeks the survivors were sacrified and the postmortem lesions were recorded.

(6) Histopathology:

Representative samples from air sacs, traches, liver, heart, spleen, eyelid were obtained from the group which inoculated with *E. coli* and *Mycoplasma.* Samples were fixed in 10% neutral buffered formalin. The fixed tissue samples were processed routinely for paraffin embedding technique. The embeded tissues were sectioned at 3 um and stained with Haematoxylin and Eosin (HE) (Bancroft and Stevens, 1982).

RESULTS

Microbiological studies:

A total of 168 live and freshly dead broiler chickens of 4-6 weeks of ages suspected to be suffering from SHS were obtained from different chickens farms (Table 1) and subjected to clinical, postmortem and bacteriological examination. Cultures from these cases were identified morphologically, biochemically and serologically. Out of 150 isolates, 76 (50.7%) isolates were identified as *E.coli*. 24 isolates (16%) were identified as *M. gallisepticum,* 20 isolates (13.3%) were identified as *H. paragallinarum.,* 14 isolates (9.3%) were identified as Pseudomonas, 8 isolates (5.3%) were identified as *klebsiella,* 6 isolates (4%) were identified as *Proteus* and 2 isolates (1.3%) were identified as *Staphylococcus* spp. are shown in (Table 2).

Thirty one cases out of 168 examined cases were showed mixed infection with more than one type of bacteria. the most prominent organism that share in most cases of SHS was *E.coli*, but in EL Mabda *M. gallisepticum* was found in high incidence more than other localities. In Banyzed *E.coli* and *H. paragallinarum* found in high incidence. In Mangabad found *E.coli* and *M. gallisepticum.* Lastly in Bany Hessen *Pseudomonas* with *E.coli* the most prominent bacteria. These showed in (Table 3).

The pathogenicity test :

The pathogenicity test results are shown in (Table 4). In the first group that infected with *H. paragallinarum,* the clinical signs were

observed 2 days post-infection in the form of nasal discharge, slight swelling of the infraorbital sinuses with reduced food and water consumption.

The second group that infected with *M. gallisepticum* the clinical signs were observed after 3 days post-infection in the form of rales, dyspnea, facial oedema and nasal discharge. Postmortem examination revealed presence of caseous material in the nasal passages, congestion of the lungs and the air sacs were thickened.

The third group that infected with *E.coli* clinical signs were observed 3 days post-infection in the form of diarrhea, decreased water and food consumption, emaciation and nasal discharge. Postmortem examination revealed congestion and slight swelling of the liver and kidneys, congestion of lungs, pericarditis and enteritis.

The fourth group which was combinedly infected with *E.coli* and *M. gallisepticum*. The clinical signs appeared after 24 hours as depression, purulent nasal discharge, abnormal sounds, swelling of the infraorbital sinuses and eyelids with prominent facial oedema, conjunctivitis and diarrhea, emaciation and decreased water and food consumption (Figures 1 and 2).

Dead cases showed septicaemia, airsacculitis which were severe in some cases. Affected air sacs were thickened, opaque and flecked with numerous yellowish white foci, Perihepatitis, peritonitis, tracheitis and pericarditis were also observed.

The fifth group which kept as non infected control showed no clinical signs.

Morphopathological studies:

Histopathological changes was observed in different organs of experimental birds. However, the most servere changes were seen in the periocular skin and eyelids and the liver of broiler experimentaly infected with *E. coli* and *M. gallisepticum.*

The pathological changes of the periocular skin and eyelids manifested the followings: edema and necrosis of subcutanous tissue (Fig.3), abscesation in the periocular subcutanous tissue (Fig.4), cellular reaction including macrophage, lymphocyte and heterophilic cells (Fig.5). Thickening of derms and perivascular cuff consists of lymphocyte, somtimes abscesation, esinophilic cells and lymphocyte in cellular reaction (Fig. 6,7) perivascular cuff and thrombosis in blood vessels, the inflammatory process sometimes extend to subcutanous muscular tissue (Fig. 8). Lung showed pneumonia, congestion, cellular

reaction in septa of alveoli.

Liver showed focal area of inflammation (Fig.9), dilatation of central vein, portal vein and sinusoid which was ingorged with blood (Fig.10). Severe congestion and cellular reaction around central and portal veins (Fig.11). Increase amount of haemosedren pigment and cellular reaction in the liver (Fig.12).

Intestine showed severe inflammatory changes with accumulation of exudate desquamated epithelial cells in the lumen of the intestine (Fig.13) the blood vessels of the cure of the villi is congested and have cellular reaction (Fig.14) .

DISCUSSION

Swollen head syndrome appears to affect the broiler flocks causing significant losses in many countries. The swelling is the result of oedema and inflammation of periorbital cutaneous and subcutaneous tissue and the swelling extend over the skull. The eyes of the birds closed due to swelling of lacrimal gland and eyelids. So the birds were blind show no desire for food or water and died due to starvation and dehydration.

The objective of the present work is to study the bacterial causes of SHS in broiler flocks which become obvious problem in many farms in Assiut governorate. A number of 168 broiler chickens showing typical SHS from 4 flocks were subjected to clinical, and Post mortem examination. The clinical signs revealed depression, swelling of heads, conjunctivitis, some birds with closed eyes, sneezing, lacrimation, abnormal respiratory sounds and decreased food consumption. Similar finding were observed by Molrey and Thomson (1984), Qureshi (1991), Arne and Hafez (1992) and Droual and Woolcock (1994).

The postmortem findings of examined birds were observed as oedema under the skin of the head region and mild to purulent conjunctivitis, congestion of the trachea, lungs and sinuses. The nasal passages contained frothy mucous with cloudiness of air sacs. These lesions are similar with those reported by Morley and Thomson (1984) and Qureshi (1991) who found beside the above lesions that larynx was contained mucous and food particles and perihepatitis and pericarditis in complicated cases.

Results of isolation indicated that 150 isolates were recovered from broiler chickens. The isolates were identified as *E.coli* 76 isolates

(50.7%), *M. gallisepticum* 24 isolates (16%), *H. paragallinarum* 20 isolates (13.3%), *Pseudomonas aeruginosa* 14 isolates (9.3%) *Klebsiella* 8 isolates (5.3%), *Proteus* 6 isolates (4%) and *Staphylococcus aureus* 2 isolates (1.3%). These results are in agreement with those reported by Morley and Thomson (1984), Zellen (1988), Qureshi (1991), Aydin *et al.* (1993), Droual and Woolcock (1994), Cookson & Shivaprasad HL (1994), Goodwin and Waltman (1994), Georgiades *et al.* (2001) and Murakami *et al.* (2002). High incidence of *E.coli* may be attributed to environmental conditions which play a significant role in interacting with infectious agents in the production of respiratory diseases in poultry (Kleven and Glisson 1997).

Mixed infection showed in 31 cases out of 168 examined cases with more than one type of bacteria. The most prominant bacteria was *E.coli* which isolated from all cases, this result simillaer with Litjens *et al.*, (1989), Goodwin and Waltman (1994), Tanaka *et al.* (1995), Murakami *et al.* (2002).

The pathogenicity tests were designed to reproduce the SHS in broiler chickens using the isolated bacteria. The choice of 4 weeks old broilers is preffered due to the absence of maternal antibodies at this age (Hafez, 1993). The present investigations have confirmed the pathogenicity of *H. paragallinarum* for the experimentally infected birds, the clinical signs appeared at 2 days post intrasinus inoculation. Signs were nasal discharge, slightly swelling in the infraorbital sinuses with decrease food and water consumption, Postmortem examination revealed presence of mucus exudate in the sinuses, congestion of trachea and nasal passages. These observation come in accordance with those observed by Sawata *et al.* (1985), Aly (1987), Sandoval *et al.* (1994), Aly *et al.* (1995) and Blackall (1999).

Regarding the second group which was infected with *M. gallisepticum,* the clinical signs begin at the 3rd day post intrasinus inoculation. These signs were mainly respiratory. These observed signs were similar to those reported by El-Ebeedy (1976). Postmortem findings revealed congestion of lungs, slight turbidity of air sacs, swelling of the infraorbital sinuses and presence of mucous exudate in the trachea. These results are similar to those recorded by Reece *et al.* (1986) and Cookson and Shivaprasad (1994).

In the third group which was infected with *E.coli.* The clinical signs appeared after 3 days of oral inoculation. These signs were slightly nasal discharge, ruffled feather, diarrhea, increased body temperature and

decreased food and water consumption. Postmortem examination revealed congestion and slight swelling of the liver and kidneys. Congestion of the lungs, pericarditis and enteritis. These findings were similar to those reported by Gross (1957) and Gross (1961).

The fourth group which was infected orally with *E. coli* and via intrasinus route with *M. gallisepticum* and kept under bad hygenic conditions such as bad ventillation, over crowdness, mal-nutrition. Showed clinical signs at 24 hours post infection in the form of nasal discharge, sneezing, abnormal sounds, facial oedema, swelling in the infraorbital sinuses, conjunctivitis, abnormal ocular secretion and diarrhea. The examined dead cases revealed septicaemia, severe enteritis, perihepatitis, congestion of the lungs, airsacculitis, sinusitis, tracheitis, pericarditis and caseous material in the nasal passages. These observations were similar to those reported by Gross (1957), Gross (1961), Murakami *et al.* (2002)who reported that *E. coli* can not invade the air sacs without *M. gallisepticum* infection .

The histopathological examination showed cellulitis in the cutaneous and subcutaneous tissue of periocular skin and eyelids. The liver showed widence of acute hepatitis with dilatation of the hepatic vasculture indicating toxic chock. The intestine manifest a moderate to severe degree of catarrhal enteritis. These results are similar to those observed by Gross (1957), Tetsuo Nunoya *et al* (1991) Droual and Woolcock (1994) and Nakamura *et al* (1997).

From the results of this work we can conclude that the bacterial agents are the main cause of SHS. Management errors act as predisposing causes which influence the appearance of the disease. So it must be emphasized that all aspects of hygiene management especially the ventilation, stocking density, litter condition and general hygiene, should be improved this beside drug therapy with suitable vaccines and vaccination programs should be also used.

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ACKNOWLEDGMENT

Great thanks to Prof. Dr. A. Z. MAHMOUD Head Department of pathology and clinical pathology faculty of Vet. Med. Assiut University.

LEGEND OF FIGURES

Fig. 1: Experimentally infected birds with *E.coli* and *M.gallisepticum* show depression and ruffling feathers.

Fig. 2: Experimentally infected birds with *E.coli* and *M.gallisepticum* showing facial oedema and swelling of the eylid.

Fig. 3: Eye of infected birds showing edma , necrosis of subcutaneous tissue. H&E. 10X.

Fig. 4: Eye of infected birds showing wall of chronic abscess in the periocular subcutaneous tissue H&E. 10X.

Fig. 5: Eye of infected birds showing necrosis of the blood vessels and diffuse heterophil cell reaction in periocular subcutaneous tissue. H&E. 10X.

Fig. 6: Eye of infected birds showing edema, thrombosis of blood vessels in periocular subcutaneous tissue. H&E. 10x.

Fig. 7: Eye of infected birds showing cellular reaction of the dermis in periocular subcutaneous tissue. H&E. 10X.

Fig. 8: Showing extension of inflammation to subcutaneous musscular tissue H&E. 25X

Fig. 9: Liver of infected birds showing focal hepatitis. H&E. 10X.

Fig. 10: Liver of infected birds showing inflammatory reaction and dilatation of the hepatic vasculture H&E. 10X.

Fig. 11: Liver of infected birds showing cellular reaction in the portal area. H&E. 10X.

Fig. 12: Liver of infected birds showing inflammatory cellular reaction and congestion of the liver. H&E.10X.

Fig. 13: Intestine of infected birds showing exudate consists of desquamated epithelial cells and inflammatory cells. H&E. 10X.

Fig. 14: Intestine of infected birds showing hyperemia and thickening of the intestinal villi. H&E. 10X.

Table 1: Clinical data of 4 broiler chicken farms in Assiut Governurate.

Locality	No. of Birds	Age (weeks)	No. of		Morbidity %	Mortality %
			Diseased	Dead		
El-Mabda	150	6	4	10	9.3	6.7
Banyzed	100	4	21	7	28	7
Mangabad	200	6	47	8	27.5	4
Bany Hessen	150	5	30	5	23.3	3.3
Total	600		102	30		

Table 2: Recovery rates of different organisms from cases showed SHS.

Locality	No. of examined cases	No. of isolates	Bacterial agents						
			E. coli	M. G.	H. para.	Pseudomonas	Proteus	Klebsella	Staph.
El-Mabda	50	35	15	8	5	3	-	4	-
Banyzed	28	24	13	-	5	3	-	1	2
Mangabad	55	56	30	11	6	3	4	2	-
Bany Hessen	35	35	18	5	4	5	2	1	-
Total	168	150	76	24	20	14	6	8	2

Table 3: Recovery rates of mixed infection :

Locality	No.of examined cases	No . of mixed cases	%	The mixed bacteria
G. El-Mabda	50	9	18%	E coli + M .G
Banyzed	28	8	28.6%	E .coli + H. para .
Mangabad	55	10	18.2%	E . coli + M.G
Bany Hessen	35	4	11.4%	E . coli + Pseudomonas
Total	168	31	18.5%	

Table 4: Results of experimental infection of 4-week-old chickens with different isolated bacter

Group	Infection	Route	Incubation period	Course of the disease
1	H. paragallinarum	Intrasinus	2 days	10 days
2	M. gallisepticum	Intrasinus	3 days	4 weeks
3	E. coli	orally	3 days	4 weeks
4	M. gallisepticum + E. coli in bad hygienic conditions	Intrasinus + orally	24 h.	6 weeks

Table 4: Results of experimental infection of 4-week-old chickens with different isolated bacteria.