Bacterial causes of decrease in performance of the breeder chicken flocks

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This study was carried out to investigate the possible bacterial causes affecting the productivity of the breeder hens. The hens under test were positive for *Mycoplasma gallisepticum* (*MG*) and *Mycoplasma synoviae* (*MS*) and negative for *Salmonella gallinarum-pullorum* (*S. g-p*) as tested by serum agglutination test. Ovaries with lesions were bacteriologically and serologically proven to have 12 bacterial isolates including three un-typed *E. coli*, one O11, and one O78; three *Staphylococcus aureus*; one *S.g-p*; two Proteus and one *Pseudomonas aeruginosa* (*P. aeruginosa*) isolates. The antibiogram of the two identified *E. coli* strains, *S. g-p* as well as *P. aeruginosa* showed variable sensitivity. The tested organisms were very sensitive to colistin and enrofloxacin. The selected *E. coli*, S.g-p and *P. aeruginosa* strains proved to be pathogenic to 3-day-old chicks, with induction of clinical signs, mortalities, post mortem lesions and adverse effect on the body weight gain. On the other hand, *E. coli* O78 was more pathogenic than O 11. The use of the abovementioned drugs for controlling of those infections in chicks was effective as indicated by the results of the in vitro test. It is important to carry out bacteriological examination to the breeder flocks in order to investigate the bacterial affections with estimation of changes in their sensitivity to the used antibiotics.

As poultry rearing developed from backyard to an organized industry, many bacterial be pathogens had been incriminated as the cause of ovarian affection of laying hens. These affections including salmonellosis (Elleman, 1960; Sokker et al., 1975; Netherlands, 1990; Al-Nakhal et al., 1999; Shivaprasad, 2000), colibacillosis (Gross and Siegel, 1959; Harry, 1964; Abd El-Nasser, 1976; Azzam, 1983; Ibrahim and Sheha, 1985; Montogomery et al., 1999; Srivasan et al., 2003), and pseudomoniasis (Sharma et al., 1980; Batra et al., 1982; Kheir El-Din et al., 1986; Shehata et al., 1988; Sidu et al., 1989). These bacterial affections resulted in ovarian lesions including misshaped and discolored ovarian follicles, caseated and degenerated ova and egg peritonitis (Saif et al., 2003). Drop in egg production varies according to the nature and type of such infection as S. g-p (Shivaprasad, 2000) and E. coli (Bisgaard and

Dam, 1981; Gazdziniski and Barnes, 2002) infections can cause 10-30%, while *P. aeruginosa* can cause 20-80% (Kaul *et al.*, 1992).

Pathogenicity to 1-3-day-old chicks was reported in *S. g-p* (Sieburth and Johanson, 1957; Kosugi *et al.*, 1985; Gorham *et al.*, 1994), *E. coli* (Awaad, 1972; Khalid, 1990; Andreatti *et al.*, 1993; Johnson *et al.*, 2001) and *P. aeruginosa* (Ray and Baujeri, 1969; Kheir El-Din *et al.*, 1986; Hamouda *et al.*, 1987).

Antibiogram is recommended for detection of suitable drugs for controlling of such pathogens (Saif *et al.*, 2003) as they acquired drug resistance by pervious, long and hazard one use of these antibacterial drugs.

Infection of chickens with *Mycoplasma* gallisepticum and *Mycoplasma* synoviae increase susceptibility to pathogenic and potentially pathogenic organisms like *E. coli* (Gross, 1990; Nakamura et al., 1994).

This work was carried out to investigate the possible bacterial causes of ovarian lesions in broiler and layer breeder flocks, the antiobiogram of the isolated bacterial strains, the pathogenicity

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of the strains to 3-day-old chicks and the treatment of experimentally infected birds as recommended with the results of the *in vitro* sensitivity test.

Material and Methods

Chickens history. Two broiler breeder flocks aged 35 and 56 weeks with total number of 30,500 and 29,300 birds, respectively and one layer breeder flock aged 35 weeks with total number of 22,800 birds showed lower egg production than the farm standard by 8%, 12% and 21% respectively. The hatchery parameters including fertility and hatchability were also reduced.

Samples.

1. Ovaries. Thirty ovaries (10/flock) with misshapen, discolored and long stalk caseated cystic ovules were aseptically collected for bacteriological examination.

2. Blood samples. From each flock, 100 blood samples were randomly collected from the wing vein and the sera were separated to be tested using slide agglutination test to confirm the results of the farm test.

Bacteriological examination. Aseptically collected ovaries were subjected to individual testing. One ml of the follicular content was aspired by a sterile syringe, inoculated into selenite F broth and incubated at 42°C for 18 h for Salmonella spp. isolation, another 1 ml. was inoculated in nutrient broth and incubated at 37°C for 24 h. Each of the inoculated broth medium was sub-cultured onto selective agar medium [5% Shigella sheep blood, Salmonella (SS), MacConkey, nutrient and pseudomonas agar) plates, then incubated at 37°C for 48 h. All the plates were examined for bacterial growth according to (Cruickshank et al., 1975).

Bacterial identification. The obtained bacterial growth was purified, examined for colonial morphology, staining characters (Cruickshank et al., 1975) and subjected to biochemical identification (Edwards and Ewing, 1972; MaCffaddin, 1980; Quinn *et al.*, 1994).

Serological typing.The obtained *E. coli* and Salmonella spp. strains were serotyped using slide agglutination test against polyvalent and monovalent standard serum obtained from Behring Werk Institute, Germany, using methods of (Neville and Brgant, 1986; Lee and Arp, 1998).

Antibiogram. All the isolated strains were tested for their in vitro sensitivity to the following chemotherapeutic discs: neomycin $(30\mu g)$, oxytetracycline $(30\mu g)$, trimethoprim $(25\mu g)$, streptomycin $(10\mu g)$, ampicillin $(25\mu g)$, nalidixic acid $(30\mu g)$, colistin $(10\mu g)$ and enrofloxacin $(10\mu g)$ adopting method of Cruickshank *et al.*, (1975) and the results were interpreted according to (Bio-Merieux, 1980).

Broth culture.Twenty-four h broth cultures from *S. g-p, E. coli* O11 and O78 as well as *P. aeruginosa* were separately prepared and subjected to plate counting as described (Collins and Lynn's 1989).

Mycoplasmas and S.g-p antigens. Slide agglutination colored antigen against both *MG* and *MS* as well as *S.g-p* were obtained from Intervet Co., Boxmeer, Holland. Serum agglutination tests for Mycoplasma spp. and *S.g-p*. were carried out as stated in NPIP.

Antibiotics used for treatment. According to the results of the in-vitro sensitivity test colistin sulphate 10% liquid in water (lot No. 1050020) was obtained from Ascor chimici, Forli, Italy and enrofloxacin 10% (lot No. 604160) from Memphis Co. for Med. Ind., were used for the treatment of experimentally infected chick groups in rate of 6 mg/kg for colistin and 10 mg/kg of enrofloxacin in drinking water for 6 days each.

Experimental chicks. Three hundred and twenty two, day-old male LCL chicks were obtained from commercial hatchery. Ten chicks were sacrificed and bacteriologically examined for freedom of any bacterial pathogen. The remaining chicks were floor reared and fed on commercial ration without antimicrobial feed additives and water was given *ad libitum*.

Experimental infection. The used three-day-old chicks were orally infected each with 0.2 ml containing $4X10^4$ CFU/ bird for *S. g-p* (El-Kady, 1986), $3X10^7$ CFU/bird for *E. coli* (Awaad, 1972) and $3X10^9$ CFU/bird (Kheir El- Din, 1986) for *P. aeruginosa*.

Statistical analysis. The obtained results were statistically analyzed using ANOVA test (Snedecor and Corchran, 1980).

Experimental design. At the 3rd day of life, the remaining 312 chicks were randomly divided into 13 equal groups (1-13), 24 chicks per group. Chick groups were treated as follows: groups 1-3, 4-6, 7-9 and 10-12 were orally infected with *S. g-p, E. coli* O11, *E. coli* O78 and *P. aeruginosa*, respectively, while chicks of group 13 were kept as non-infected negative control. The infected chicks

were observed daily for clinical signs and/or mortalities. At the first detection of clinical signs (5-days of age), birds of groups 1, 4, 7 and 10 were treated with colistin, while those of groups 2, 5, 8 and 11 were treated with enrofloxacin. The medicaments were given in the drinking water for 6 days. Birds of groups 3, 6, 9 and 12 were left as infected non-treated controls. All the groups were daily observed for 10 days with recording clinical signs, mortality rates and post-mortem P.M lesions. Bacterial reisolation was tried from dead birds. At the 10th day post-infections (3 days after stop medication), 10 randomly selected chicks/group were weighed, sacrificed and examined for (P.M) lesions. Samples from liver, spleen and lung were collected from dead and sacrificed birds and subjected to bacterial reisolation.

Results

Results of slide agglutination test revealed that the tested sera were positive to *M. gallisepticum*, *M. synoviae and S. g-p* in rates of 44.35%, 63% and 0 %; respectively.

The bacteriological examination of ovaries with lesions showed isolation of 12 bacterial isolates. The obtained isolates were morphologically, staining and biochemically identified to be (5) E. coli, (3) Staphylococci, (2) Proteus, (1) Pseudomonas and (1) non-motile Salmonella. These isolates were further identified to be (3) un-typed E. coli, (1) was O11 and the other was O78; the (3) Staphylococcal isolates Staphylococcus aureus, (2) Proteus. were Salmonella isolate was S. gallinarum pullorum and the last one was P.aeruginosa. Identified strains of E. coli, S .g-p and P. aeruginosa were isolated from the most severe ovarian lesions.

Isolated strains showed variable sensitivity to the available antibiotics when tested *in vitro*. The tested organisms were totally sensitive to colistin and enrofloxacin, resistant to oxytetracyclin and trimethoprim and variable to the others.

The infected chicks showed signs of illness at the 2^{nd} day post oral infection with rates of 1/24 (4.16%) for each *E. coli* O11 and *S. g-p* infected groups, while it was 2/24 (8.33%) *E. coli* O78 infected group (Table 1). The most predominant signs were off food and ruffled feather. One chick was found dead in *S. g-p* group at the 2^{nd} day post-infection. At the 3^{rd} day of infection, the signs progressed to be 4, 2, 2 and 2 (16.66%, 8.33%)

8.33% and 8.33%) for *E. coli* O11, *E. coli* O78, *S* .*g-p* and *P. aeruginosa* infected groups respectively, while the mortality was 2, 2, 2 and 1 (8.32%, 8.32%, 8.32% and 4.16 %) for *E.coli* O11, *E. coli* O78, *S. g-p* and *P. aeruginosa* respectively (Table 1).

The detected signs were diarrhea, pasty vent, off food and huddling together for *E. coli* O11 and *S. g-p* infected groups, while the signs were more severe in *E. coli* O78 group. Group infected with *P. aeruginosa* showed ataxia, incoordination, off food and diarrhea.

All dead birds revealed lesions of septicaemia and enteritis except for *E. coli* O78 dead chicks showed slight airsaculitis.

At the end of the 10th day post-infection, the total morbidity rates were 62.5%, 95.83%, 91.66% and 79.16% (Table 1, Fig. 1) and the mortality rates reached 20.83%, 45.83%, 58.53% and 50% in groups infected with *E. coli* O11, *E. coli* O78, *S. g-p* and *P. aeruginosa* respectively. The control non-infected group showed no signs or mortality (Table 1, Fig. 2). Dead birds proved positive results for bacterial reisolation.

Non-treated infected groups showed higher morbidity and mortality rates (Table 1) than those treated either with colistin or with enrofloxacin (Table 2, Fig. 1, 2).

Results of the mean body weight at the end of observation showed that the infected recovered groups stunted than the non-infected control negative group (Table 3, Fig. 3). The recorded mean weights were the highest 71.32 gm for control, followed by 61.41, 58.16, 56, 36 and 51.83 gm in descending manner for groups 5, 3, 2 and 4 respectively.

Results of body weight in infected non-treated groups (Table 3, Fig. 3) proved significant difference between non-infected group and infected groups 2, 3, and 4 at P>0.05, While the treated groups (Table 4, Fig. 3) proved significant difference in the mean body weight only between group 2 (73.54) and groups 4 (54.80) and 5 (54.63) at P > 0.05.

Groups received colistin or enrofloxacin in drinking water at the 3^{rd} day post-infection showed the following results (Table 2, Fig. 1, 2): Clinical signs began to subside at the 5^{th} day post-treatment in all treated groups except those of group 2 and 9, while signs were completely disappeared in all groups at the 6^{th} day. Total mortality rates in each

Group	Infection	Parameters	Days post-infection								Cumulative	Positive %		
No.		rarameters	1	2	3	4	5	6	7	8	9	10	positive	1 USILIVE /0
1	Negative	S	0	0	0	0	0	0	0	0	0	0	0	0
		Μ	0	0	0	0	0	0	0	0	0	0	0	0
2	E. coli O11	S	0	1	4	5	3	2	0	0	0	0	15	62.50
		Μ	0	0	2	0	2	1	0	0	0	0	5	20.83
3	E. coli 078	S	0	2	2	3	5	3	4	4	0	0	23	95.83
		Μ	0	0	2	1	2	0	3	1	2	0	11	45.83
4	S.g-p	S	0	1	2	2	2	2	4	4	5	0	22	91.66
		Μ	0	1	2	1	5	1	3	1	0	0	14	58.53
5	Р.	S	0	0	2	2	2	3	3	3	4	0	19	79.16
	aeruginosa	Μ	0	0	1	1	3	3	2	1	1	0	12	50.00

Table (1): Distribution of morbidity and mortality rates of 3-day-old infected chicks (n=24).

S: Number of birds with signs.

M: Number of dead birds.

S. g-p: Salmonella gallinarum pullorum.

Table (2): Distribution of morbidity and mortality rates in 5-day-old infected and the	reated chicks
(n=24).	

Crown	Infection	Treatment	Parameters	Days post-treatment						No. of	% of
Group No.				1	2	3	4	5	6-7	positive	positive
1	Negative	Negative	S	0	0	0	0	0	0	0	0
-			Μ	0	0	0	0	0	0	0	0
2		Colistin	S	4	5	3	2	1	0	15	62.50
2	E. coli O11		Μ	1	2	0	1	0	0	4	16.67
3		Enrofloxacin	S	1	4	5	2	0	0	12	50.00
3			Μ	2	3	1	0	0	0	6	25.00
4	4 <i>E. coli 078</i> 5	Colistin	S	1	3	3	2	0	0	9	37.50
4			Μ	2	2	1	0	0	0	5	20.83
5		Enrofloxacin	S	3	2	2	1	0	0	8	33.33
5			Μ	1	0	1	1	0	0	3	12.50
6		Colistin	S	3	3	2	1	0	0	9	37.50
U		Constin	Μ	2	2	0	0	0	0	4	16.67
7	S. g-p	Enrofloxacin	S	3	4	2	1	0	0	10	41.67
1		Emonoxaciii	Μ	2	1	1	0	0	0	4	16.67
8	P. aeruginosa	Colistin	S	3	3	4	1	0	0	11	45.83
o			Μ	2	1	2	1	0	0	6	25.00
0		Enrofloxacin	S	3	3	2	0	1	0	9	37.50
9			Μ	1	2	2	0	0	0	5	20.83

S: Number of birds with signs.

M: Number of dead birds.

S. g-p: Salmonella gallinarum pullorum.

Crown No.	Infostion		Body weight/gm	
Group No.	Infection –	Minimum	Maximum	Mean ± SD
1	Negative	44.4	95.92	$71.32* \pm 16.27$
2	E. coli 011	46.8	64.85	$56.36* \pm 5.58$
3	E .coli 78	44.45	67.28	$58.16* \pm 5.97$
4	S.g-p	38.10	60.70	$51.83* \pm 7.19$
5	P. aeruginosa	53.00	67.57	61.41 ± 4.61

Table (3): Mean body weight of infected non-treated chicks (n=10).

S.g-p: Salmonella gallinarum pullorum.

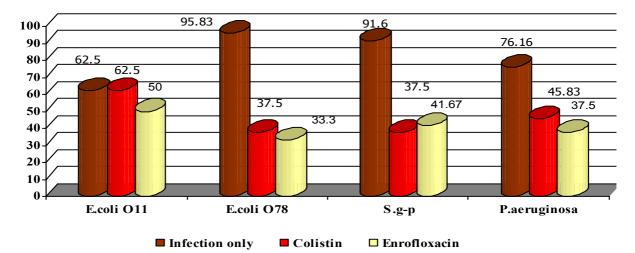
*: Significant difference at p>0.05.

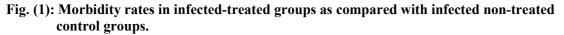
Crown No.	Infection	Treatment	Body weight/gm					
Group No.	Infection	Treatment	Minimum	Maximum	Mean ± SD			
1	Negative	Negative	44.4	95.92	71.32 ± 16.27			
2	$\mathbf{E} = 0 \mathbf{E} 0 1 1$	Colistin	45.70	92.75	$73.54* \pm 13.05$			
3	E. coli 011	Enrofloxacin	56.50	82.84	68.41 ± 7.57			
4	E. coli 078	Colistin	37.90	74.85	$54.80* \pm 9.36$			
5	<i>E. Coll</i> 0/0	Enrofloxacin	34.56	72.95	$54.63* \pm 10.37$			
6	San	Colistin	53.96	73.30	59.86 ± 11.69			
7	S.g-p	Enrofloxacin	38.86	69.58	54.75 ± 8.54			
8	P. aeruginosa	Colistin	44.70	71.88	59.31 ± 8.19			
9		Enrofloxacin	50.20	62.86	59.31 ± 3.66			

Table (4): Mean body weight of infected and treated chicks (n=10).

S.g-p: Salmonella gallinarum pullorum.

*: Significant difference at p>0.05.





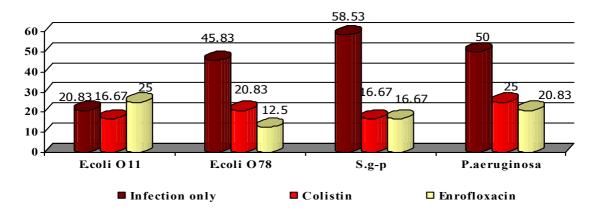


Fig. (2): Mortality rates in infected-treated groups as compared with infected non-treat control groups.

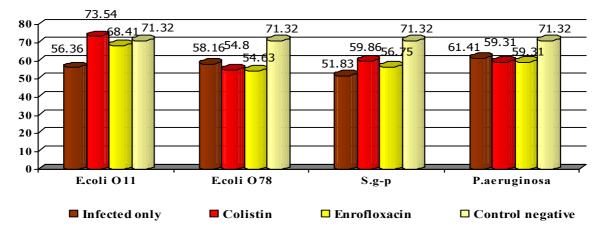


Fig. (3): Mean body weight in infected treated groups as compared with infected non-treated and non-infected control groups.

of group 3 and 8 was (25%) higher than those of groups 4 and 9 (20.83%) followed by 16.67% in groups 2, 6 and 7 while group 5 showed the lowest mortality rate (12.5%).

Discussion

Many bacterial infections have economical importance in laying breeder flocks including *E. coli* (Bisgaard and Dam, 1981; Gazdziniski and Barnes, 2002), *Salmonella gallinarum pullorum* (Shivaprasad, 2000) and *P. aeruginosa* through their effects on the ovary inducing; pathological lesions, and lowered fertility, hatchability and egg production (Saif *et al.*, 2003).

The positive results of serum plate agglutination test to *Mycoplasma* spp. indicated that the flocks under test were infected. It was found that *MG* and *MS* increased the susceptibility

to infection with *E.coli* as stated by (MacOwan *et al.*, 1982; Gross, 1990; Nakamura *et al.*, 1994; van de Zande *et al.*, 2001). The negative results of serum plate agglutination test to *S.g-p* can be attributed to that reported by Gast and Beard, (1990) where hens infected with antigenically intermediate or variant strains of *S.pullorum* were detected as sero-positive less often than hens infected with antigenically standard strains. Therefore, ovaries with lesions could be involved in sampling for isolation as recommended by Shivaprasad (2000).

Results of bacteriological isolation and identification proved the isolation of *E.coli* as reported (Abd El-Nasser, 1976; Osklokov and Saltykov, 1976; Zahdeh, 1982; Azzam, 1983). Serologically, those isolates were typed to be O11

(Abd El-Nasser, 1976; Andrawis, 1980); while O78 was also reported (Karmy *et al.*, 1987; Singab, 1987).*S.g-p* was identified as an ovarian isolate of laying chickens (Saif *et al.*, 2003). On the other hand, *P.aeruginosa* was detected from ovaries with obvious lesions (Sharma *et al.*, 1980; Batra *et al.*, 1982; Kheir El-Din *et al.*, 1986; Riad, 1994).

The *in vitro* sensitivity test of the isolated strains gave variable results to the used drug discs. This point was reported (Azzam, 1983; Riad 1994). The most effective antibacterial drugs were colistin and enrofloxacin as detected previously (Riad, 1994), while Abd El-Ghafar (1979) proved resistance of all tested strains to colistin. Abd El-Wahab (1977), Welsh *et al.*, (1997) and Salmon and Watts, (2000) reported the sensitivity of the isolates to enrofloxacin.

For studying the pathogenicity of the identified organisms to 3-day-old chicks, oral route was used for the experimental infection with 24 h broth cultures. *E.coli* infected birds showed clinical signs and lesions similar to those reported (Abd El-Wahab 1977; Bassiouni *et al.*, 1979; Azzam 1983).

Results of infection with *E.coli* O11 and O78 and the control non-infected group showed that mortality percentage in groups infected with *E.coli* O11 was 20.83%, while it was 45.83% in-group infected with *E.coli* O78. These results were higher than those of Davis (1938) where as *E.coli* induced losses ranged from 15-40% in chicks less than 10-days of age. The detected lesions were similar to those previously reported by Azzam (1983) and Singab (1987) who observed gross lesions including pericarditis and perihepatitis at 3-7 days post infection; while airsaculitis was also reported (Batra *et al.*, 1982).

Morbidity and mortality rates were higher ingroup received *E.coli* O78 than those of group received O11. O78 was reported to be more commonly pathogenic to chickens than O11 (Harry, 1964; Sojka, 1965). Moreover, Bassiouni *et al.*, (1979) concluded that *E.coli* O78 was highly pathogenic to 3-day-old chicks and it was more pathogenic than O11. Alian (1978) recorded 50% mortality rate in day-old chicks orally infected with *E.coli* O78.

The group infected with *S.g-p* showed signs, lesions, 91.66 % morbidity rate and 58.53 % mortality rate. El-Kady (1986) reported similar

signs and lesions in 2-day-old chicks but only with 8.54% mortality rate.

The recorded signs and lesions in chicks of group infected with *P.aeruginosa* were similar to those noticed by Hamouda *et al.*, (1987). The recorded mortality rate (50.0 %) was lower than that of Hamouda *et al.*, (1987) which was 56% while, Lin *et al.*, (1996) sated that *P.aeruginosa* could cause 50-100% mortality rate in experimentally inoculated 4-week-old chickens.

The mean body weight in group infected with *P.aeruginosa* was lower than that of non-infected group. Kheir El-Din et al., (1986), reported similar result.

Colistin and enrofloxacin were used in the treatment as recommended by the results of the *in vitro* test. Both drugs were effective in reducing morbidity, mortality rates and restoring body weight in the treated groups regardless to the type of the organism as compared with negative and non-treated control.

This study pointed out that *E.coli*, *s*,*g*-*p* as well as *P.aeruginosa* can cause reduction in the breeder flocks performance, especially when they are serologically positive to *Mycoplasma* spp. The antibiogram is necessary for controlling such infections.

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الإصابات البكتيرية وإجراء إختبار الحساسية للمضادات الحيوية قبل التدخلات العلاجية

المسببات البكتيرية لإنخفاض إنتاجية قطعان أمهات الدجاج

أجريت هذه الدراسة لإستبيان المسببات البكتيرية المحتملة المسببة لإنخفاض إنتاجية قطعان أمهات الدجاج، الدجاجات التى تم إختبار ها كانت إيجابية للفحص المصلى لكلا من الميكوبلازما جاليسيبتيكم و الميكوبلازما سينوفى وسالبة للفحص المصلى للسالمونيلا بللورم بإختبار الترسيب المصلى على شريحة الميكروسكوب ، عناقيد البيض ذات التغيرات المرضية تم فحصها بكتريولوجياً وأوضح الإستبيان المصلى للعترات البكتيرية المعزولة الإثنى عشرة إشتمالها على ٣ عترات للميكروب القولونى غير معروفة وواحدة 101 وواحدة 708 وشلاث للميكروب العنقودى وواحدة للسالمونيلا جالينيرم بللورم وكذا إثنان لميكروب البروتيس وواحدة 101 وواحدة 708 إيروجينوزا. أظهر إختبار حساسية الميكروبات المعزولة إختلافا شدياً فى حساسيتها للمضادات الحيوية عامة وكانت جميعها حساسة لكل ويراث للائ للميكروب العنقودى وواحدة 100 للمعزولة إختلافا شدياً فى حساسيتها للمضادات الحيوية عامة وكانت جميعها حساسة لكل إيروجينوزا. أظهر إختبار حساسية الميكروبات المعزولة إختلافا شدياً فى حساسيتها للمضادات الحيوية عامة وكانت جميعها حساسة لكل من الإنروفلوكساسين و الكوليستين. كما أوضحت الدراسة أن العترات المختارة من نوع الميكروب القولونى 011 و من الإنروفلوكساسين و الكوليستين. كما أوضحت الدراسة أن العترات المختارة من نوع الميكروب القولونى 011 و جالينيرم بللورم و السيدوموناس إيروجينوزا كانت ضارية لكتاكيت عمر ٣ أيام عند إجراء العدوى بالفام على الغار مرضية. نفوق, اقات تشريحية وتأثير شديد على معلات حساب الوزن. كما وجد أن الميكروب القولونى 011 كان مرافية و07. اقات تشريحية وتأثير شديد على معلات حساب الوزن. كما وجد أن الميكروب القولونى 011 كان مراوة من عترة 708.

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