

PREVALENCE OF BUFFALO MASTITIS IN DAKAHLIA GOVERNORATE

EL-NAKER Y.F.*; SAYED-AHMED M.**; SAAD Z.***; REIAD E.**** and YOUNIS** E.E.

* Department of animal medicine, (Infectious Diseases) Faculty of Veterinary Medicine New Valley, Assiut Univ.

** Department of Internal Medicine and Infectious Diseases, Faculty of Veterinary Medicine, Mansoura University.

*** Veterinarian in Veterinary Medicine Directorate of Dakahlia.

**** Animal Health Research Institute, Dokki, Giza 12611, Egypt.

Email: yassereInaker@yahoo.com

Assiut University Email: www.aun.edu.eg

ABSTRACT

Received at: 23/2/2015

Accepted: 15/3/2015

This study surveyed the mastitis prevalence and risk factors in buffalo's population in Dakahlia Governorate. A total of 471 Water buffaloes (*Bubalus bubalis*) brought to the Veterinary Teaching Hospital, Mansoura, Dakahlia Governorates from 2009 to 2012 were analyzed to determine, the seasonal occurrence of mastitis, prevalence in relation to the lactation stage. Isolation and identification of causative agent using PCR and other serological technique were applied. The prevalence of buffalo's mastitis was 19.9% and 5.9% for clinical and subclinical mastitis respectively. A high incidence of clinical mastitis (51.6%) was observed in animal during their early lactation stage, while high incidences of subclinical mastitis recorded (12.9%) in late lactation stage. And the most isolated microorganism is *E-coli spp.* The Statistical analysis of our results revealed that a significant variance between the occurrence of mastitis and lactation season, $P < 0.001$.

Key words: Buffalo, lactation, mastitis, prevalence.

INTRODUCTION

Buffalo's population shared by about 54.5% of milk production in 1990. This share increased to 56% in 2005. The annual increase rate in buffalo milk production was about 3.8%, which was the highest rate among other types of livestock producing milk in Egypt (CAPMAS, 2007). Mastitis is a highly prevalent disease in dairy herds, and one of the most important diseases affecting the world's dairy industry as it causes reduced milk yields and have deleterious effects on the chemical and cytological composition of milk. In addition, it may result in the presence of bacteria and other infectious agents which may be harmful to humans. (Costa *et al.* (1997) and Beheshti *et al.* (2010).

Clinical and subclinical mastitis recorded in buffalo and it considered one of the most economically important deadly diseases of milky animals, and causes the changes in glandular tissues affecting quality and quantity of the milk Nagahata *et al.* (1992) and Sharma and Sindhu, (2007). The Prevalence of intramammary infection in buffalo was 66%. Since the mammary gland is highly susceptible to infection during the periparturient period, the incidence was highest during the 30 days after calving. Bacterial pathogens that caused mastitis classified into contagious pathogens and environmental pathogen Moroni *et al.* (2006).

Mastitis generally results from interaction between a variety of microbial infections and host responses in the udder, and it is influenced by management practices. Factors which predispose to mastitis include mostly environmental aspects such as poor hygiene, poor husbandry, overcrowding, bad ventilation, poor milking technique and malfunction of milking machines. Besides, factors which adversely affect the normally efficient barriers to infection of the udder such as teat skin, teat canal and mammary cistern, predispose udder to mastitis Fagiolo and Lai (2007). In buffalo cows mastitis is quite always caused by bacteria Mastitis-causing bacteria can be classified in contagious as *Streptococcus agalactiae*, *Stapylococcus aureus*, *Arcanobacter piogenes*, *Micoplasma*; environmental as *Streptococcus uberis*, and *dysgalactiae*, *Escherichia coli*, *Enterobacteriacee*, yeasts and moulds (*Prototheca zoophii*) and opportunist as coagulase negative Staphilococcus (Galiero, 2002). In buffalo Incidence of subclinical mastitis more prevalent than clinical mastitis in housed buffaloes in percentages 18.5% and 9% respectively. *S. aureus*, *E. coli*, *St agalactia* and *St. dysgalactia* were the most common isolates in clinical mastitis. *E. coli*, *S. aureus*, C.N.S, *Pseudomonas*, *St agalactia*, and *St. dysgalactia* were the most common isolates in subclinical mastitis. Mixed infection by *S. aureus* and *E. coli* common cause in clinical mastitis 24.4% and

S. aureus and *C.N.S* common cause in subclinical mastitis 18.9% Abd-Elrahman (2013).

The objectives of this study were aimed to record the prevalence of mastitis in buffalo and discuss some risk factors associated with mastitis in buffalo in buffalo farms in Dakahlia governorate.

MATERIALS and METHODS

• Animals

During a period from May 2009 to December 2012 a total of 471 lactating buffalo's cow brought to Veterinary teaching hospital, Fac. of Vet. Med. Mansoura University, the buffaloes cow were examined for subclinical and clinical mastitis and analyzed for the prevalence of clinical and subclinical mastitis during different seasons. Quarter milk from udder was categorized as subclinical mastitis based on the following criteria: absences of visible abnormalities of milk secretions, California mastitis test (CMT) Gonzalez *et al.* (1990).

• California Mastitis Test (CMT)

The CMT was used alongside the physical examinations and the test was carried out as described. Equal volume of milk and the CMT reagent (2ml of each quarter) and was mixed thoroughly in a cup of black plastic paddle; the mixture was gently rotated for 10 seconds, and then results were recorded Moroni *et al.* (2006).

• Milk sampling

The infected teat end was disinfected with cotton soaked in 70% ethyl alcohol. The first few streams of foremilk were discarded. Samples for bacteriological analysis were then collected into screw capped sterile McCartney bottle and were held on ice until delivery to the laboratory within 20 to 30 min of collection the samples were preserved at -20 °C till send to Microbiology Department, Animal Health Research Institute Dokki, for Bacterial isolation and identification.

• Isolation of bacteria and bacterial count

5 ml of milk were centrifuged at 3000 r.p.m. for 20 min. The cream and supernatant fluid were discarded. A loopfull from the sediment incubated into nutrient broth for 24 hrs at 37°C, then streaked onto MacConkey (*Coliform spp.*), 5% ovine blood agar fortified with potassium tellurite 3.5% (*Arcanobacterium spp.*), Barid-Barker media (*Staphylococcus aureus*), EMB, *Pseudomonas* agar (*Pseudomonas spp.*), Mannitol salt agar, and Edward's media (*Streptococcus spp.*) for bacterial culture and isolation. The colonies were counted after 24 - 48hr of incubation at 37°C. Bacterial numbers > 25 cfu/100 µl were the standard of the presence of mastitis infection. Pure colonies from the respective

plates were identified on the basis of Gram stain, morphological findings, and colony characteristics. Enumeration of mastitis microorganisms were performed by serial dilution of milk samples and poured plated according to standard methods of American Public Health Association (APHA 1993).

• PCR Assay for the major isolate:

1- E-coli

Detection of E.coli (verotoxin 2) by real time pcr

Extraction kit Gf1 (vivantis) lot no. :12192c

Mix kit Titangene lot no. : EM1821

Primer of verotoxin 2:

Sequence:

F: 5'- TGTGGCTGGGTTTCGTTAATACGGC - 3'

R: 5'- TCCGTTGTCATGGAAACCGTTGTC - 3'

2 - Staph Aureus

Staphylococcus aureus culture for DNA extraction:

According to Stephens (2008), bacterial growth for the purpose of DNA extraction was prepared as follows: 20 µl of stock solution was streaked onto a Brain Heart Infusion (BHI) agar plate (prepared as specified, Oxoid Australia Pty Ltd, Adelaide) and cultured overnight at 37°C. the following day a single colony was selected and suspended in a 5 ml falcon tube (Becton Dickinson, New South Wales, Australia) containing BHI broth (prepared as specific, Oxoid Australia Pty Ltd, Adelaide) and cultured overnight at 37°C, with shaking.

DNA Extraction:

According to Stephens (2008), from the overnight BHI broth culture, 1 ml was extracted using the Qiagen DNA extraction Kit (Qiagen, Victoria, Australia), as per manufacturer's instructions, including lysostaphin at 200 µg/ml for the lysis step. Purified DNA samples were eluted using ddH₂O and stored at -20°C.

DNA amplification and Analysis

Polymerase Chain Reaction (PCR)

Standard PCR amplifications were performed using a Bio-Rad Research Thermocycler in 0.2 ml PCR tubes.

mecA duplex PCR

Multiplex Polymerase Chain Reaction for detection of *Staphylococcus aureus* species specific 16S rRNA and (SCCmec) type IV genes (responsible for methicillin resistance): Two sets of primer pairs were used, the first one was *Staph756F*

(5'-AACTCTGTTATTAGGGAAGAAC-3') and *Staph750R*

(5'-CCACCTTCCTCCGGTTTGTACC-3')

primers which could amplify 756 base pair fragments specific for 16S rRNA of *S. aureus*; the second one was SCCmec

4a1 (5'-TTTAATGCCCATGAATAAAAT-3') and SCCmec
 4a2 (5'-AGAAAAGATAGAAGTTCGAAAGA-3') primers which could amplify 450 base pair fragments specific for SCCmec subtype IVa gene according to Ryffel *et al.* (1990).

The reaction mixtures consisted of 5 µl of the extracted DNA template of the bacterial isolates, 5 µl 10× PCR buffer (75 mM Tris-HCl, pH 9.0, 2 mM MgCl₂, 50 mM KCl, 20 mM (NH₄)₂SO₄), 1 µl dNTPs (40 µM), 1 µl (1U Ampli Taq DNA polymerase), 1 µl (50 pmol) from the forward and reverse primers. The two sets of primer pairs were used in each reaction mixture and the volume of the reaction mixture was completed to 50 µl using DDW. 40 µl paraffin oil was added and the thermal cycler was adjusted as follows: 94°C for 5 min, followed by 10 cycles of 94°C for 1 min, 55°C for 1 min., and 72°C for 1.5 min, and 25 cycles of 94°C for 1 min, 50°C for 1 min, and 72°C for 1.5 min, followed by final extension at 72°C for 1.5 min, and the PCR products were stored in the thermal cycler at 4°C until they were collected.

PCR product were stained with ethidium bromide and visualized on 1.5% agarose gel with a UV light transilluminator. Control marker with molecular mass of 100bp was used (fermentase, Lithuania).

All previously identified phenotypically as *S. aureus* with bacteriological examination were used in PCR run accompanied with 10 isolates identified as *S. aureus*. All strains are positive for amplification of 756 base fragments specific for 16S rRNA of *S.*

aureus using *Staph756 F* and *Staph750 R* primers, while 4 strains showed positive amplification of 450 base pair fragments specific for SCCmec subtype IVa genes using SCCmec 4a1 and SCCmec 4a2 primers, as shown in Figure 1.

Statistical analysis

The cumulative data was entered in Microsoft Excel for analysis of the data, and P<0.05 was regarded as significant. Incidence risks of clinical mastitis were computed by dividing the number of occurrences of clinical mastitis during a defined period by average number of lactating cows during that period. The seasonal variation of clinical mastitis incidence was tested with Edward's test corrected for changing size of the population at risk in different months Edwards (1961).

RESULTS

Prevalence of clinical and subclinical mastitis in buffaloes in relation to lactation stage

In (Table 1), the clinical and subclinical mastitis in buffaloes was recorded in which 19.9% of buffaloes had clinical mastitis, while 5.9% had subclinical one. A greater incidence of clinical mastitis (51.6%) was observed in animal during their post parturient (early lactation stage), with subsequent reduction in mid (16.1%) and late (12.9%) lactation stage. While incidences of subclinical mastitis in relation to stage of lactation were (2.3%, 2.3%, and 12.9%) in early lactation stage, mid lactation stage and late lactation stage respectively (Fig.1).

Table 1: The effect of stage of lactation on mastitis incidence in buffaloes

Stage of lactation	No. of animals at risk	Clinical Mastitis		Sub clinical mastitis	
		No.	%	No.	%
Recent parturition	62	32	51.6	2	3.2
Mid lactation	254	41	16.1	6	2.3
Late lactation	154	20	12.9	20	12.9
Dry period	1	1	-	-	-
Total	471	94	19.9	28	5.9

Early lactation stage means 2 months post parturition.
 Mid lactation stage means 3-5 months post parturition.
 Late lactation season means 6-8 months post parturition.

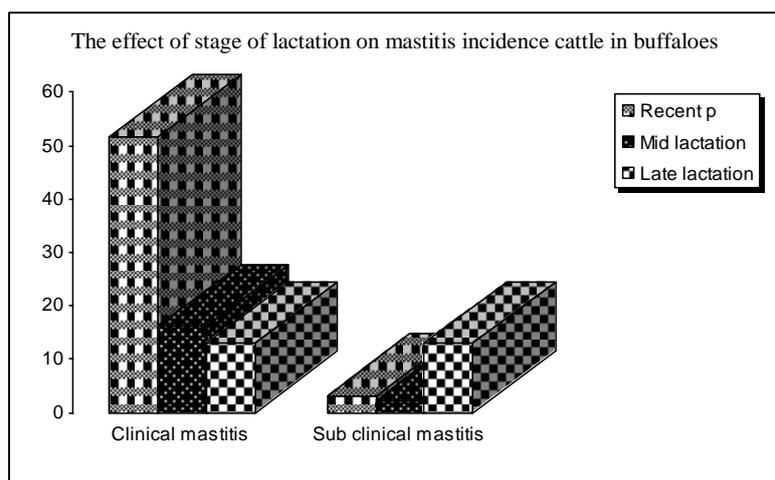


Fig 1: The effect of stage of lactation on mastitis incidence in buffaloes

In studying lactation season Table (2), the percent of the clinical mastitis was 12.5% in the 1st lactation season of the examined animals, 14.5% in the 2nd lactation season, 21.3% in the 3rd lactation season and 34.6% in the 4th lactation season while the percent of sub clinical mastitis was 2.5%, 3.4%, 7.2% and 9.6% in the 1st, 2nd, 3rd, and 4th lactation season respectively.

Table 2: The effect of lactation season (age) on mastitis incidence in buffaloes.

Lactation Season*	No. of animals at risk	Clinical mastitis		Sub clinical mastitis	
		No.	%	No.	%
1	40	5	12.5	1	2.5
2	144	21	14.5	5	3.4
3	235	50	21.3	17	7.2
4	52	18	34.6	5	9.6
Total	471	94	19.9	28	5.9

- *1st lactation season means a buffalo has 3 years old
- *2nd lactation season means a buffalo has 4 years old
- *3rd lactation season means a buffalo has 5 years old
- *4th lactation season means a buffalo has 6 years old

Effect of season (weather) on mastitis incidence

The occurrence of clinical and subclinical mastitis in buffaloes during different months was recorded (Table 3). The high percent of clinical mastitis was observed during spring season (57.14%), followed by autumn season (29.2%), then winter (27.8%) and summer season (12.7%). While in the high percent of subclinical mastitis observed in winter months (40.90%) followed by summer months (25.71%).

Table 3: The effect of weather (season) on mastitis incidence in buffaloes.

Season	No. of animals	Clinical mastitis	Sub clinical mastitis
Summer	275	35 (12.72%)	9 (25.71%)
Autumn	24	7 (29.16%)	-
Winter	158	44 (27.84%)	18 (40.90%)
Spring	14	8 (57.14%)	1 (7.14%)
Total	471	94 (19.9%)	28 (5.9%)

Isolation of different bacteria causing Mastitis in buffaloes

In (Table 4) from 122 clinical and subclinical cases of buffalo mastitis, bacterial isolates revealed that, *E-coli* is the most isolated pathogen 37 isolates (30.32%) followed by 25 isolate for *Strept.agalactia* (20.49%) followed by 24 isolates (19.67%) for *Staph. aureus*, 5 isolates (4.09%) for *Pseudomonas*, 2 isolates 1.63% for *Proteus* and at last one isolates (0.81%) for *Klebsiella*, From 28 acute mastitic cases in buffaloes were 10 *E-coli*, 5 *Staphylococcus aureus*, 4 *Streptococcus agalactia*, one *Klebsiella pneumoniae*, 4 *Pseudomonas aerogenosa*, one *Proteus*

vulgaris and 3 isolates of *Corynbacterium bovis* where the isolated bacteria from 56 subacute mastitic cases were *E-coli* (18), *Staphylococcus aureus* (8), *Streptococcus agalactia* (10) and *Pseudomonas aerogenosa* (1). out of 10 chronic mastitic buffaloes the isolated bacteria were *E-coli* (3), *Staphylococcus aureus* (3), *Streptococcus agalactia* (6) and one *Proteus vulgaris*, while the isolated bacteria from 28 subclinical mastitis were, 6 isolate for *E-coli*, 8 isolate for *Staph*, 5 isolate *Strept*. Putting in consideration that there were a mixed isolates contain more than one isolate.

Table 4: Clinical examination (types of mastitis in buffaloes and the isolated microorganism)

Type of symptom	No. of mastitic cases	Bacterial isolation							
		E-Coli	Staph aureus	Strept agalactia	Klebsiella pneumoniae	Pseudomonas aerogenosa	corny bovis	Proteus vulgaris	
Clinical mastitis	Acute	10	5	4	1	1	3	1	
	Sub- acute	18	8	10	-	1	-	-	
	Chronic	3	3	6	-	-	-	1	
Subclinical mastitis	28	6	8	5	-	-	-	-	
Total	122	37	24	25	1	2	3	2	
	%	30.32	19.67	20.49	0.81	1.63	2-45	1.63	

Identification of some common isolates using (*Staph* and *E-coli* Toxin) Using PCR



Figure 2: Agarose gel electrophoresis showing, lane 1 100 bp ladder. Lanes 2, 3, 4, 5, 6, 7 and 8 showing amplification of 756 bp fragments of 16S rRNA. While lanes 5, 6, 7, and 8 showing amplification of 450 bp fragments of SCC mec IVa gene.

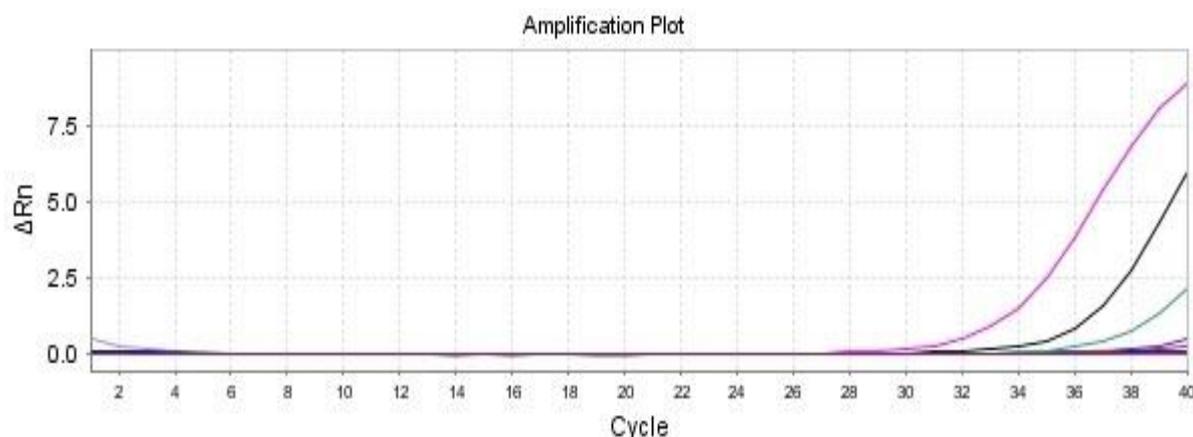


Fig. 3: Detection of *E.coli* (verotoxin 2) by real time pcr.

DISCUSSION

In this study a total of 471 lactating buffalo were examined for clinical and subclinical mastitis, 19.9% of buffaloes had clinical mastitis, while 5.9% had subclinical one. A greater (51.6%) incidence of clinical mastitis was observed in animal during their early lactation stage, with subsequent reduction in mid (16.1%) and late (12.9%) lactation stage. While incidences of subclinical mastitis in relation to stage of lactation were (2.3%, 2.3%, and 12.9%) in early lactation stage, mid lactation stage and late lactation stage respectively (Table 1, Fig.1). The obtained result are in agreement with Yas *et al.* (1983) who found that the incidence of mastitis was higher during the first 2 months of lactation and declined in subsequent months. The reason for this remains to be elucidated: It may be related to the characteristics of decreased host defense capability during the post-parturient period in buffaloes (unpublished observation), as observed widely in dairy cows Moroni *et al.* (2006). Also Kavitha *et al.* (2009). Concluded that, Buffaloes in the first stage of lactation (1-4 months) and the last part of dry period (10-12 months) are found to be more prone to mastitis.

In studying lactation season (Table 2), the percent of the clinical mastitis was 12.5% in the 1st lactation season of the examined animals, 14.5% in the 2nd lactation season, 21.3% in the 3rd lactation season and 34.6% in the 4th lactation season while the percent of sub clinical mastitis was 2.5%, 3.4%, 7.2% and 9.6% in the 1st, 2nd, 3rd, and 4th lactation season respectively, the result obtained are going hand by hand with Pal and Verma (1988) who reported the highest incidence at 3rd calving, and agree with Kavitha *et al.* (2009) and Sharma *et al.* (2007) who found that, As the parity increases, an increase in the incidence of mastitis is seen (also found higher

prevalence of subclinical mastitis in 5 to 9 years old animals and in 3rd and 4th parities. The difference found among reports may be due to various factors such as breed, season, and husbandry system Moroni *et al.* (2006).

On studying seasonal effect on prevalence of mastitis, the occurrence of clinical and subclinical mastitis in buffaloes during different months was recorded (Table 3). The high percent of clinical mastitis was observed during spring season (57.14%), followed by autumn season (29.2%), then winter (27.8%) and summer season (12.7%). While in the higher percent of subclinical mastitis observed in winter months (40.90%) followed by summer months (25.71%). The obtained results are nearly different from Godden *et al.* (2003), who mentioned that, Heat and humidity may increase the pathogen load in the environment (field or housing), resulting in a greater incidence of mastitis in warm weather, Shathele (2009) reported that, the incidence of mastitis decreased with increasing ambient temperature but increased with decreasing ambient temperature. Dhakal *et al.* (1997) showed that 37.3% of buffaloes had clinical mastitis during the summer season followed by the autumn season (31.7%) and minimum (7.83%) during spring season (February, March and April) in Nepal. Incidence of staphylococcal mastitis was found to be significantly higher during summer than winter in Chennai (Thennarasu *et al.*, 2009). The difference in results may be attributed to the season of calving had a significant effect in the incidence of mastitis in buffaloes. The animals calved in rainy season had the highest incidence of mastitis as reported by Chand *et al.* (1995).

From 122 clinical and subclinical cases of buffalo mastitis, bacterial isolates revealed that, (Table 4), *E.coli* is the most isolated pathogen 37 isolates (30.32%) followed by 25 isolate for *Strept.agalactia*

(20.49%) followed by 24 isolates (19.67%) for *Staph aureus*, 5 isolates (4.09%) for *Pseudomonas*, 2 isolates 1.63% for *Proteus* and at last one isolates (0.81%) for *Klebsiella*, From 28 acute mastitic cases in buffaloes were 10 *E- coli*, 5 *Staphylococcus aureus*, 4 *Streptococcus agalactia*, one *Klebsiella pneumoniae*, 4 *Pseudomonas aerogenosa*, one *Proteus vulgaris* and 3 isolates of *Corynebacterium bovis* where the isolated bacteria from 56 subacute mastitic cases were *E- coli* (18), *Staphylococcus aureus* (8), *Streptococcus agalactia* (10) and *Pseudomonas aerogenosa* (1). Out of 10 chronic mastitic buffaloes the isolated bacteria were *E-coli* (3), *Staphylococcus aureus* (3). *Streptococcus agalactia* (6) and one *Proteus vulgaris*, while the isolated bacteria from 28 subclinical mastitis were, 6 isolate for *E-coli*, 8 isolate for *Staph*, 5 isolate *Strept*. Putting in consideration that there were a mixed isolates contain more than one isolate, the rates of environmental pathogens found in milk from buffaloes with clinical mastitis were also similar to that reported by Moroni *et al.* (2006), Minor Pathogens and coliform are often isolated from the skin of the udder due to contamination with soil and feces to the mammary gland of buffaloes are still unclear. Improvement of the production environment, good milking hygiene and proper handling of buffaloes appear to be important. The obtained result are parallel to result obtained by Abd-Elrahman (2013), who found that *S. aureus*, *E. coli*, *St agalactia* and *St. dysgalactia* were the most common isolates in clinical mastitis. *E. coli*, *S. aureus*, *C.N.S*, *Pseudomonas*, *St agalactia*, and *St. dysgalactia* were the most common isolates in subclinical mastitis. Mixed infection observed in our study in which *S. aureus* and *E. coli* common cause in clinical mastitis 24.4% and *S.aureus* and *C.N.S* common cause in subclinical mastitis 18.9%. The occurrence of clinical mastitis in buffaloes fed on normal feeding was nearly similar to animal fed in low quality feed although it effect on milk production. In spite of increase of subclinical mastitis in low quality feed group and this supported by the season as low feeding usually found in summer (dry season).

In conclusion, we got preliminary information about the prevalence and risk factors of mastitis in buffaloes, Therefore, the result could suggest the main risk factors associated with buffalo mastitis in Dakahlia to establish the appropriate method for prevention. Different species of bacteria could exist in the Egyptian buffaloes.

REFERENCES

Abd-Elrahman Amir Hamed (2013): Mastitis in housed dairy buffaloes: incidence, etiology, clinical finding, antimicrobial sensitivity and different medical treatment against *E. coli* mastitis. Life Science Journal; 10 (1).

- American Public Health Association (APHA) (1993): Standard Methods for the examination of dairy products 16th Ed American Public Health Association, Washington.
- Beheshti, R.; Shayegh, J.; Eshratkhan, B. and Ghiasi Ghalekandi, J. (2010): Prevalence and etiology of subclinical mastitis in ewes of the Tabriz region, Iran. Global veterinaria 4(3): 237-241.
- CAPMAS: Central Agency of Public Mobilization and Statistics of Egypt, (2007): "Animal Wealth Statistics", several numbers.
- Chand, P.; Behra, G.D. and Chand, P. (1995): Factors influencing occurrence of mastitis, genetic and environmental factors. Indian J. Dairy Sci., 48: 271-273.
- Costa, E.O.; Garino, F.; Watanabe, Jr. E.T.; Ribeiro, Vezon P.; Baruselli, P.S. and Paske, A. (1997): Study of mastitis among ten dairy buffaloes herds (*Bubalus bubalis*) in the Vale do Ribeira (Ribeira River Valley) Sao Paulo. Brazil. Proc. 5th World Buffalo Congress, Caserta, Italy. 635-638.
- Dhakal, I.P. (1997): Drug selection and use on clinical mastitis in buffaloes at Chitwan Valley of Nepal. 11: 56-70.
- Edwards, J.H. (1961): Seasonal incidence of congenital disease in Birmingham. Ann. Human Gen. 25: 89-93.
- Fagiolo, A. and Lai, O. (2007): Mastitis in buffalo. Ital. J. Anim. Sci. 6(2): 200-206.
- Galiero, G. (2002): The control of environmental mastitis. *Bubalus bubalis* I: 26-28.
- Godden, S.; Rapnicki, P.; Stewart, S.; Fetrow, J.; Johnson, A.; Bey, R. and Farnsworth, R. (2003): Effectiveness of an internal teat seal in the prevention of new intramammary infections during the dry and early-lactation periods in dairy cows when used with a dry cow intramammary antibiotic. J. Dairy Sci. 86 (12): 899-911.
- Gonzalez, R.N.; Jasper, D.E.; Kronlund, N.C.; Farver, T.B.; Cullor, J.S.; Bushnell, R.B. and Dellinger, J.D. (1990): Clinical mastitis in two California dairy herds participating in contagious mastitis control programs. J. Dairy Sci. 73: 648-660.
- Kavitha, K.L.; Rajesh, K.; Suresh, K.; Satheesh, K. and Sundar, N.S. (2009): Buffalo mastitis - risk factors. Buffalo Bull, 28: 135-137.
- Moroni Moroni, P.; Sgoifo Rossi, C.; Pisoni, G.; Bronzo, V.; Castiglioni, B. and Boettcher, P.J. (2006): Relationships between somatic cell count and intramammary infection in buffaloes. J. Dairy Sci. 89 (3): 998-1003.
- Nagahata, H.; Ogawa, A.; Sanada, Y.; Noda, H. and Yamamoto, S. (1992): Peripartum changes in antibody producing capability of lymphocytes from dairy cows. Vet. Quart. 14: 39-40.
- Pal, B. and Verma, B.B. (1988): Preliminary trials with kanamycin acid sulphate in the treatment

- of subclinical mastitis in buffaloes. *Indian Vet. J.* 65: 346-347.
- Ryffel, C.; Tesch, W. and Birch-Machin, I. et al (1990):* Sequence comparison of *mecA* genes isolated from methicillin resistant staphylococcus aureus and staphylococcus epidermis. *Gene* 94 (1): 137–138.
- Sharma, A. and Sindhu, N. (2007):* Occurrence of clinical and subclinical mastitis in buffaloes in the State of Haryana (India). *Ital. J. Anim. Sci.* 6(2): 965-967.
- Shathele, M.S. (2009):* Weather Effect on Bacterial Mastitis in Dairy Cows. *International Journal of Dairy Science.* 4(2): 57–66.
- Stephens, Alex, J. (2008):* The development of rapid genotyping methods for methicillin-resistant *Staphylococcus aureus*. Ph. D. thesis, Queensland University of Technology. Ph.D. School of life Sciences, Institute of Health and Biomedical Innovation, Queensland University of Technology. Brisbane, Australia.
- Thenarasu, A.; Muralidharan, R.; Murugan, M. and Thamilvanan, T. (2009):* Effect of season and microclimatic variables on the incidence of bovine mastitis. *Ind. Vet. J.* 86(4): 393.
- Yas, A.A.; Kalara, D.S. and Khalaf, A.M. (1983):* Studies on mastitis in buffaloes in Iraq. Prevalence rate and etiology. *Tropical Vet. Anim. Sci. Res.* 1: 23-28.

معدل انتشار التهاب الضرع الجاموسي في محافظة الدقهلية

ياسر الناقر ، محمد زكريا ، زكريا سعد ، عماد رياض ، عماد يونس

Email: yasserelnaker@yahoo.com

Assiut University Email: www.aun.edu.eg

اجريت هذه الدراسة في الفترة من عام ٢٠٠٩ وحتى عام ٢٠١٢ علي عدد ٤٧١ حالة من الجاموس والتي اتت الي المستشفى البيطري التعليمي بشها التابعة لكلية الطب البيطري بجامعة المنصورة. حيث تم فحص هذه الحالات لمرض التهاب الضرع باشكاله وتم تسجيل البيانات الخاصة بالحالات. وتبين من النتائج ان معدل التهاب الضرع في الجاموس بلغ ١٩.٩% و ٥.٩% لالتهاب الضرع الاكلينيكي وتحت الإكلينيكي على التوالي. ولوحظ وجود نسبة عالية من التهاب الضرع في الجاموس (٥١.٦%) وتتواجد في الحيوان أثناء مرحلة الرضاعة المبكرة، في حين سجلت حالات التهاب الضرع تحت الإكلينيكي نسبة عالية (١٢.٩%) في أواخر مرحلة الرضاعة. وكشف التحليل الإحصائي للنتائج بأنه يوجد تباين كبير بين حدوث التهاب الضرع وموسم الرضاعة، وفصول السنة حيث ان اعلي نسبة لحدوث التهاب الضرع كانت في فصل الربيع وبلغت ٥٧.١٤% وتم عزل الميكروبات المختلفة المسببة لالتهاب الضرع في الجاموس وكانت اعلي نسبة للعزل هي لميكروب *E-coli*.