

# INFLUENCE OF MASTITIS ON RESUMPTION OF OVARIAN ACTIVITY AND POSTPARTUM REPRODUCTIVE MEASUREMENTS IN BALADI COWS

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Submitted August 14, 2021; Accepted September 21, 2021; Published 1/10/2021

## SUMMARY

The current research aims to study the influence of mastitis on resumption of ovarian activity and postpartum reproductive measurements in Egyptian Baladi cows. Total of twenty-four postpartum cows, were split into two groups of twelve in each. The first group (12 cows) had mastitis, while the second group (12 cows) was healthy. The cows were monitored after calving directly and had their udders screened for subclinical mastitis using the California Mastitis Test (CMT). The present results indicated that the incidence rate of sub-clinical mastitis cases in cows were significantly ( $P < 0.05$ ) higher (75%) than clinical mastitis cases (25%). The interval from parturition to first ovulation, first service and conception was significantly ( $P < 0.05$ ) longer ( $48.3 \pm 7.8, 96.5 \pm 14.4$ , and  $153.2 \pm 12.5$ , days) in mastitic cows than ( $27.4 \pm 5.6, 72.3 \pm 11.2$ , and  $85.6 \pm 15.2$ , days) in healthy cows. The conception rate following the first service was significantly ( $P < 0.05$ ) lower (25%) in mastitic than in healthy cows (66.7%). **In conclusion**, the present results indicate a negative impact of mastitis by delaying the resumption of postpartum ovarian activity and reproductive measurements in Baladi cows. The current study recommends that small breeders must pay attention to the health of the udder, especially during the postpartum period, to reduce the incidence of mastitis and its harmful effects on the reproductive characteristics of lactating cows.

**Keywords:** Baladi cows, mastitis, ovarian activity, reproductive performance

## INTRODUCTION

During the transition period (3 weeks after and before calving) cows face many health problems and challenges, and elevated inflammatory markers have been well documented during this time (Bradford *et al.*, 2015). Mastitis is the most common disease found in lactating cows (NAHMS, 2007). Mastitis influences the consistency and quantity of milk by inducing physical, chemical, and typically bacterial changes in milk, as well as pathological changes in the glandular tissues of the udder (Radostits *et al.*, 2000; Sharma *et al.*, 2012). Identification of subclinical mastitis is a frustrating challenge for dairy farmers and veterinarians to solve, mostly due to the absence of signs, in order to maintain not only the animal's wellbeing but also the hygienic consistency of the milk generated (Halasa *et al.*, 2007; Zadoks *et al.*, 2011 and Idriss *et al.*, 2013). Several authors recorded that the percentage of sub-clinical mastitis cases was ranged between (46 and 64%) in dairy cows (Joshi and Gokale, 2006; Rahman *et al.*, 2010 and Jarassaeng *et al.*, 2012). In addition, Khokon *et al.* (2017) stated that 47 to 76% of cases were positive to California Mastitis Test (CMT) in dairy cows. Mastitis, whether clinical or subclinical, results in significant economic losses due to reduced milk production, low milk content, higher culling rates, and treatment costs (Cha *et al.*, 2011; Ali *et al.*, 2013 and Dahl *et al.*, 2018). There is a relationship between cows suffering from mastitis and the length of the period from calving until the first ovulation reported by (Lavon *et al.*, 2010; Nguyen *et al.*, 2011 and

Gindri *et al.*, 2019). The interval from post-partum first service and days open was longer in mastitic cows (Villa-Arcilaa *et al.*, 2017; Bouamra *et al.*, 2017 and Zigo *et al.*, 2019). In addition, lower conception rate (28-36%) recorded in mastitic cows compared to (41- 42%) in healthy cows (Mellado *et al.*, 2018 and Lavon *et al.*, 2019). May be there are limited researches on the effect of mastitis on resumption of ovarian activity and postpartum reproductive measurements in Egyptian Baladi cows. The current research was designed to study the effect of mastitis incidence post-calving on resumption of ovarian activity and postpartum reproductive measurements in Baladi cows.

## MATERIALS AND METHODS

### **Farm location and climatic conditions:**

This research was executed at a private farm in the Kom Ombou area in the province of Aswan, (32, 31' 23" east and 22, 28' 09" north). The cows were reared and housed in semi-shaded enclosures under traditional farm conditions. Table (1) displays the ambient temperature ( $^{\circ}\text{C}$ ), relative humidity (RH %), and calculated temperature humidity index (THI) over the course of the experiment. The experiment took place from November to April (20/21).

The temperature humidity index (THI) was calculated according to the formula of Mader *et al.* (2006):

$$\text{THI} = (0.8 \times \text{Tmax db}) + [(RH/100) \times (\text{T max db} - 14.4)] + 46.4$$

**Table 1. The ambient temperature (°C), relative humidity (RH %) and temperature humidity index (THI) during the experimental period**

Experimental month	Average ambient temperature (° C)		Average relative humidity (RH %)	THI value
	Min	Max		
November	21.4	30.2	19	73.6
December	16.1	28.3	21	70.5
January	13.2	20.3	21	63.8
February	15.9	24.2	23	68.1
March	18.3	33.1	22	77.0
April	20.1	34.0	17	76.9

**Animals and their feeding during the experiment:**

Twenty-four Baladi cows were included in this experiment. Cows' parities ranged from 3<sup>rd</sup> to 5<sup>th</sup>, and their live body weight at calving varied from 320 to 380kg. During the experiment, the animals were fed Barseem Higaze (*Alfa- alfa*) in addition to the concentrate feed, and hay wheat. All cows were housed in the same environmental and administrative conditions according to farm routine work.

**Experimental design:**

Twenty-four Baladi cows were split into two classes (each with 12 cows). The first group had mastitis (during fifteen days post-partum), while the second group served as a monitoring group (healthy cows). As part of routine practice, cows were examined with the California Mastitis Test (CMT) to see if they had subclinical mastitis and the findings were reported.

**Detecting cows with mastitis:**

Mastitis is a mammary gland infection that is commonly associated with physical, chemical, and bacteriological changes in the milk, as well as pathologic changes in the udder glandular tissue (Sharma *et al.*, 2007).

Clinical mastitis: is characterized by abnormal milk production (e.g., watery milk, flakes in milk, and so on) and/or mammary gland inflammation (e.g., redness, swelling, strength, and so on) (Chebel, 2007).

Subclinical mastitis: (i.e., the asymptomatic inflammation of mammary tissue) is the most common form of mastitis (Roy *et al.*, 2009)

The cows with sub-clinical mastitis were detected by California Mastitis Test and the manufacturer's steps were followed up.

According to the clinical symptoms:

Sub-clinical mastitic cows: The cows considered suffering from sub-clinical mastitis when they were not observed with any signs of mastitis, in both milk and udder, but the mastitis was known by a detector (California Mastitis Test) (Ebrahimi *et al.*, 2007; Kathiriya *et al.*, 2014).

California Mastitis Test (CMT) was used based on the amount of gel formed, the results were labeled as negative, 1 +, 2 +, or 3 + (Esron *et al.*, 2005).

**Reproductive disorders estimation:**

Anestrous cases: Those cows did not show any visible signs of heat during 60 days postpartum, smooth ovaries, no corpus luteum (CL) was detected and blood progesterone was persisted in the basal line (1ng/ml) were considered in anestrous according to Kamal *et al.* (2012).

Silent or quiet ovulation: Cows were considered with quiet ovulation when they did not show any visible signs of heat, CL was observed at least on one of the ovaries and plasma progesterone concentrations rose to the basal line (1ng/ml) (Kamal *et al.*, 2012).

Conception rate estimation as the following equation:

$$\text{Conception rate} = \frac{\text{Number of conceived cows} \times 100}{\text{Number of mated cows}}$$

**Detection of estrus, service and diagnosis of pregnancy:**

Cows were tracked visually on a daily basis, which proved to be successful. Cows were assumed to be in the heat process if one or more of the estrus symptoms, such as vaginal mucus discharge or standing behavior, were present. Natural service was performed and number of services per conception were recorded for each cow. After sixty days (without any signs of estrus) post-service, rectal palpation was performed to diagnose pregnancy.

**Hormones analysis:**

Blood samples (10 ml) were obtained from the jugular vein in heparinized tubes at estrus, 7 days, and 15 days after estrus. Samples were centrifuged at 3000 rpm for 20 minutes to collect plasma, which stored at -18 °C until assay time. The hormones progesterone (P4) and estradiol-17 $\beta$  (E2) were tested using a radioimmunoassay package. Progesterone and estradiol-17 susceptibility levels were 0.03ng/ml and 4.0pg/ml, respectively, according to manufacturer records. Progesterone (P4) and estradiol-17 $\beta$  (E2) had coefficients of intra-assay variation of 6.7 percent and 11.3%, respectively.

**Statistical analysis:**

Data were statistically analyzed by one-way ANOVA to study the effect of mastitis (clinical or subclinical mastitis) on resumption of ovarian activity and postpartum reproductive measurements of Baladi cows, using SAS (2002). The following model was used:

$$Y_{ij} = \mu + S_i + e_{ij}$$

Where:

$Y_{ij}$  = the observation of trait,

$\mu$  = overall mean,

$S_i$  = the fixed effect of  $i^{\text{th}}$  mastitis, where  $i=1,2$  (1=mastitis cows and 2= healthy cows), and

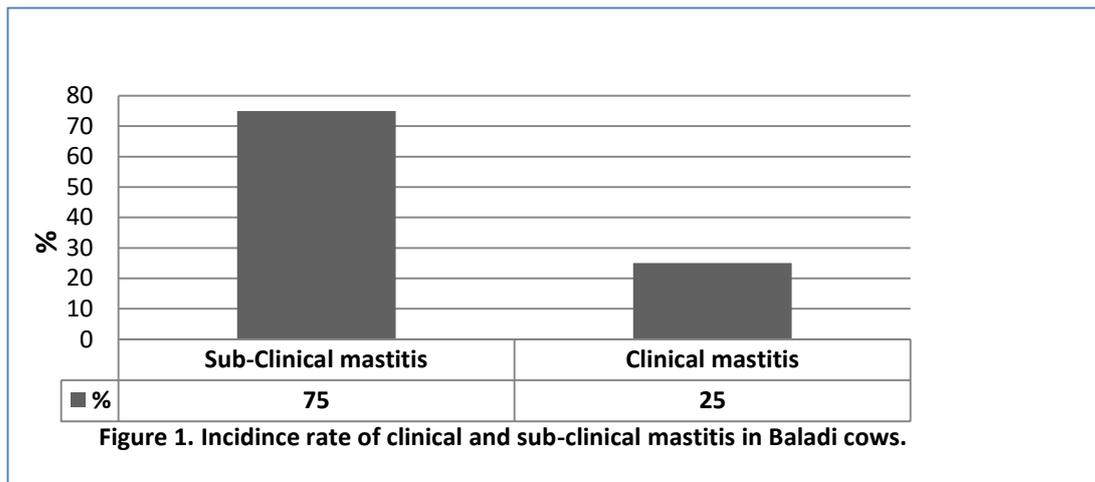
$e_{ij}$  = experimental error.

## RESULTS AND DISCUSSION

### Postpartum incidence rate of clinical and sub-clinical mastitis:

Figure (1) indicates that the percentage of sub-clinical mastitis cases in Baladi cows were significantly ( $P < 0.05$ ) higher (75%) than clinical

mastitis cases (25%). The current results agreed with that stated by Abera *et al.* (2012), Hameed *et al.* (2012) and Moges *et al.* (2012) who reported that sub-clinical mastitis ranged between 28 and 34%, while clinical mastitis ranged between 5 and 18%, in dairy cows. Higher percentage of sub-clinical mastitis cases (46- 64%) was reported by Joshi and Gokale (2006), Rahman *et al.* (2010) and Jarassaeng *et al.* (2012) in dairy cows. In addition, Khokon *et al.* (2017) found that 47 to 76%, cases was positive to California Mastitis Test (CMT) in dairy cows. Recently, Zigo *et al.* (2019) recorded that percentage of subclinical mastitis cases was 15.7 %, but clinical forms was 4.7 % in dairy cows.



### Effects of mastitis:

#### postpartum first ovulation interval:

Table (2) illustrates the time from parturition to first ovulation, being significantly ( $P < 0.05$ ) longer ( $48.3 \pm 7.8$ , days) in mastitic cows than in healthy ones ( $27.4 \pm 5.6$ , days). The present result was explained by Lavon *et al.* (2010) and Lavon *et al.* (2011), who found that cows that suffer from subclinical mastitis showed lower estrogen concentrations at estrus, which led to delay in ovulation. Chronic subclinical mastitis can trigger ovulation delay in up to 30% of cows (Lavon *et al.*, 2010). Due to the release of substances that inhibit the expression of receptors for gonadotropins and other reproduction-related hormones, clinical mastitis may affect the pattern of hormonal secretion and follicular growth (Moore *et al.*, 1991; Gilbert *et al.*, 2005). Clinical or sub-clinical mastitis leads to lowered feed intake and changes in metabolite levels and hormonal concentration resulting in altered follicular growth Oliver *et al.* (2000). Also, Wolfenson *et al.* (2015) reported in cows with subclinical mastitis a depression of steroid development in the preovulatory follicle, along with a low and delayed preovulatory luteinizing hormone increase, leading to delayed ovulation. Lipopolysaccharides (LPS), material generated by bacteria that trigger mastitis, cause a decrease in

follicle growth and delayed ovulations, and a decrease in the number of cows with ovulations (Gindri *et al.*, 2019). The effects of inflammatory mediators such as LPS and lipoteichoic acid (LTA) released during infections (mastitis), had a detrimental impact on pituitary and gonadal hormones (Hertl *et al.*, 2010; Hertl *et al.*, 2014). Moreover, LPS can interrupt ovarian follicular development by adversely affecting the hypothalamus and inhibiting the release of gonadotropins (Suzuki *et al.*, 2001; Williams *et al.*, 2007). Mammary infections (mastitis) interrupt ovarian functions during the estrous cycle, decreasing follicular growth and estradiol (E2) levels, disrupting luteal activity, and resulting in compromised ovarian functions (Sheldon *et al.*, 2002; Lavon *et al.*, 2008). Long-term mechanisms including follicular growth and development can be affected by subclinical forms of mastitis (Lavon *et al.*, 2011; Rahman *et al.*, 2012 and Roth *et al.*, 2013). Mastitis is linked to an increase in cytokine secretion, which can suppress LH secretion and lower  $P_4$  levels in the bloodstream (Hansen *et al.* 2004). Cortisol, a hormone whose secretion can be elevated during mastitis, can also block LH secretion (Hockett *et al.*, 2000). In this respect, Huszenicza *et al.* (2005) suggested that mastitis was correlated with delayed postpartum first ovulation or extended the follicular phase in cyclic

cows. The failure of GnRH release in the preovulatory period may be caused by cytokines released during the inflammatory process (mastitis infection) (Hockett *et al.*, 2005). In addition, Nguyen *et al.* (2011) showed that during the first month post-partum, cows with a

high somatic cell counts (SCC, >500,000) had a higher prevalence of delayed first ovulation than cows with those of less than 500,000.

**Table 2. Post-partum reproductive measurements in mastitis and healthy Baladi cows**

Items	Healthy cows	Mastitic cows
No. of cows	12	12
Post-partum first ovulation (days)	27.4 <sup>a</sup> ± 5.6	48.3 <sup>b</sup> ± 7.8
Post-partum first service (days)	72.3 <sup>a</sup> ± 11.2	96.5 <sup>d</sup> ± 14.4
Post-partum to conception (days)	85.6 <sup>a</sup> ± 15.2	153.2 <sup>b</sup> ± 12.5
Number of services per conception	1.6 <sup>a</sup> ± 0.4	2.5 <sup>b</sup> ± 0.8

a, b: values within the same row having different superscripts are significantly different at P < 0.05.

#### **Postpartum first service interval:**

Table (2) indicates that the interval from calving to post-partum first service in Baladi cows was significantly (P < 0.05) longer (96.5 ± 14.4, days) in mastitic cows than in healthy ones (72.3 ± 11.2, days). The present results agreed with those reported by Miller *et al.* (2001) and Villa-Arcilaa *et al.* (2017), who found that the post-partum first service in dairy cows was higher (86, 94, days) in those cows with mastitis (clinical or subclinical) than (71, 84, days) in healthy cows. Similar trend was observed by Zigo *et al.* (2019) who found that the first post-partum service was 61.5, 66, and 85 days in healthy cows, sub-clinical mastitis, and clinical mastitis in dairy cows, respectively. When compared to control cows, Jersey cows with clinical mastitis before first service had a 22.6 day increase in first service post-partum (Barker *et al.*, 1998). Schrick *et al.* (2001) recorded that cows with clinical or subclinical mastitis before first service had increased days to first service (77.3 d) compared with healthy dairy cows (67.8 d). In addition, Gunay and Gunay (2008), Boujenane *et al.* (2015) and Bouamra *et al.* (2017) reported that in mastitis-affected cows, the time from parturition to postpartum first service was longer than in healthy cows. The increased time between calving and first service in cows with mastitis may be due to changes in the hypothalamic-pituitary hormonal axis or postponed in recycling and ovarian activity, according to (Moore *et al.*, 1991, Huszenicza *et al.*, 1998 and Hansen *et al.*, (2004).

#### **Postpartum time from calving to conception:**

Table (2) clarified that the time from calving to be conceived was significantly (P < 0.05) longer (153.2 ± 12.5, days) in mastitic cows than in healthy ones (85.6 ± 15.2, days). The current findings correspond with those found by Villa-Arcilaa *et al.* (2017) who reported that in cows with subclinical mastitis the interval from calving-to-conception was longer (157, days) compared with healthy cows (119, days). The average days open ranged from 98 days in healthy cows to 105 days in subclinical mastitis and 121 days in clinical mastitis in dairy cows (Zigo *et al.*, 2019). Subclinical mastitis in cows linked to changes in ovarian physiology and steroidogenesis may explain the longer time between parturition and

conception (Sakumoto *et al.*, 2003; Lavon *et al.*, 2011). Scheid-Filho *et al.* (2012) stated that in dairy herds, the time between calving and conception was longer (164.2 days) in mastitis cows than (156.4 days) in healthy cows. Lavon *et al.*, (2019) found that longer intervals from calving to conceive (152, 159, days) in cows suffering from *Streptococcus* and *Escherichia coli* mastitis compared with (130, days) in healthy cows. Schrick *et al.* (2001) showed that cows with clinical or subclinical mastitis before first service had increased days to conception (110.0, days) compared to healthy cows (85.4, days). Manimaran *et al.* (2014) reported that mastitis prior to and/or after first service in dairy cattle and Murrah buffaloes caused extended days open. Prolonged days open in mastitis dairy cows may be linked to the release of endotoxins into the bloodstream from an infected udder, which promotes the production of prostaglandin F<sub>2α</sub> resulting in premature luteolysis (Huszenicza *et al.*, 2005). Mellado *et al.* (2018) found that in mastitis Holstein cows, the time from parturition to conception tended to be longer than healthy cows. Vacek *et al.* (2007), Chegini *et al.* (2016) and Bouamra *et al.* (2017) reported that the interval from calving to conception was longer (135 - 189, day) in cows had mastitis compared to healthy dairy cows (116- 147, day). Miller *et al.* (2001) reported that cows with clinical mastitis after first service had longer days open (137, d) than healthy cows (93, days).

#### **Number of services per conception:**

Table (2) illustrates that the number of services per conception in Baladi cows were significantly (P < 0.05) higher (2.5 ± 0.8, days) in mastitic cows than in healthy cows (1.6 ± 0.4, days). The present result is close to that reported by Zigo *et al.* (2019) and Lavon *et al.* (2019) who recorded higher number of services per conception (1.4 - 3.7, services) in mastitis dairy cows compared to (1.3 - 2.1, services) in healthy cows. Villa-Arcilaa *et al.* (2017) found that cows with positive subclinical mastitis had a greater number of services per conception (2.48, services) than cows with negative subclinical mastitis (1.91, services). Gunay and Gunay (2008) and Boujenane *et al.* (2015) reported that the number of services per conception was greater in mastitis-affected cows

(2.1-2.5 services) than in healthy cows (1.8–2.4 services). In addition, Kumar *et al.* (2017) reported that crossbred cows with mastitis had a higher number of services per conception (2.2, service) than healthy cows (1.2, services). Khokon *et al.* (2017) recorded that the number of services per conception were 3, 2.3 and 2 services in clinical mastitis, Sub-clinical mastitis and mastitis free of dairy cows, respectively. With the severity of udder infection in cows after the first service, the number of services per conception jumped from 1.8 to 3.4 (Gilbert *et al.*, 2005). Schrick *et al.* (2001) found that cows with clinical or subclinical mastitis before first service had increased services per conception (2.1) compared with healthy cows (1.6). Mellado *et al.* (2018) stated that higher number of services per pregnancy in cows with mastitis (3.1, services) than (2.9, services) in healthy Holstein cows.

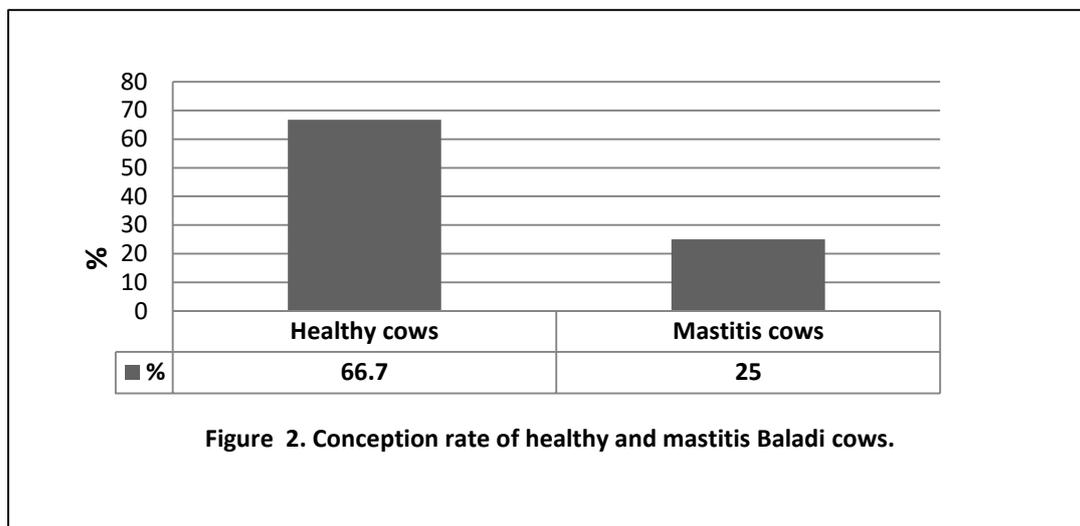
#### Conception rate:

Figure (2) shows the conception rate from first service in Baladi cows which was significantly ( $P < 0.05$ ) lower (25%) in mastitic cows than in healthy ones (66.7%). The present findings correspond with those reported by Mansour *et al.* (2016) who discovered that at day 45 post-insemination in buffaloes, pregnancy rates decreased to 60.9%, 44.5% and 16% in buffaloes without mastitis, subclinical mastitis, and clinical mastitis, respectively. Lavon *et al.* (2019) recorded that the pregnancy rates were less for mastitis cows (27-28%) compared to (42%) in healthy cows. Kelton *et al.* (2001) and Santos *et al.* (2004) found that lower conception rate in cows with mastitis (22, 38 %) compared with (29, 46%) of healthy cows. Marques *et al.* (2015) found that mastitis-affected cows had a lower conception rate (22%) than healthy cows (44%). The conception rate in mastitis-affected cows was 36%, compared to 41% in healthy Holstein cows (Mellado *et al.*, 2018). Whether the intra-mammary infection induced gram-positive or gram-negative bacteria, clinical mastitis after artificial insemination is linked to a low conception rate (Schrick *et al.*, 2001, Santos *et al.*, 2004 and Konig *et al.*, 2006). Mastitis, both clinical and subclinical, impairs oocyte competence, resulting in low blastocyst production (Wolfenson *et al.*, 2015). Mastitis may also cause prostaglandin  $F_{2\alpha}$  to be released, which causes luteolysis in the postovulatory period and early pregnancy (Risco *et al.*, 1999 and Hockett *et al.*, 2005). Intra-mammary infection (IMI) and linked toxins change or impair several reproductive processes, like as oocyte maturation (Asaf *et al.*, 2013), ovulation, fertilization and embryo development (Soto *et al.*, 2003b), corpus luteum growth (Huszenicza *et al.*, 2005) and hormone concentrations (Lavon *et al.*, 2011) in cattle. Similar to heat stress, the immune response during mastitis raises body temperature, which may restrict embryonic survival in cattle (Hansen *et al.*, 2004). When compared to cows without mastitis, pregnancy loss was 3.5 times higher in cows with subclinical

mastitis before gestation (Moore *et al.*, 2005) or 1.2 times higher in cows with subclinical mastitis during early gestation in dairy cattle (Pinedo *et al.*, 2009). Mastitis may result in failed fertilization and embryonic death in Holstein heifers (Peter *et al.*, 2004). At the time of service, cows with clinical or subclinical mastitis had a significantly lower pregnancy rate in dairy cows (Hudson *et al.*, 2012). Pregnancy loss in dairy cows has an interaction effect between clinical mastitis during early lactation and low body condition at 70 days postpartum in dairy cows (Hernandez *et al.*, 2012). Lipopolysaccharide from gram-negative bacteria including *E. coli* induces mammary inflammation and inhibits the formation and operation of the corpus luteum in cows (Herzog *et al.* 2012). It has been claimed that the most crucial risk period for mastitis to diminish conception success is at the time of or within a month after service in dairy cows (Schrick *et al.*, 2001; Ahmadzadeh *et al.*, 2009 and Hudson *et al.*, 2012). After intra-mammary *Escherichia coli* infection or endotoxin infusion, tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ) level in the blood are increased in cattle (Blum *et al.*, 2000; Hoeben *et al.* 2000 and Perkins *et al.* 2002). Soto *et al.* (2003a) suggested that (1) tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ) acts on the oocyte during maturation to diminish the percent of oocytes that cleave and evolve following fertilization, (2) exposure of embryos to tumor necrosis factor  $\alpha$  after fertilization lowers growth to the blastocyst stage and (3) tumor necrosis factor  $\alpha$  increases the percentage of blastomeres that undergo apoptosis in a stage-dependent fashion. Increases in prostaglandin  $F_{2\alpha}$  and potentially tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ) caused by mastitis have been linked to corpus luteum (CL) regression (Malinowski and Gajewski, 2010). Changes in uterine sensitivity to the action of prostaglandins  $F_{2\alpha}$  and  $E_2$  or oxytocin are a negative outcome of mastitis (Hertl *et al.*, 2010 and Rahman *et al.*, 2012). When compared to cows without clinical mastitis, pregnancy loss was 2.8 times higher in cows with clinical mastitis during the first 45 days of pregnancy (Chebel *et al.*, 2004). Clinical intra-mammary infection can result in embryonic death before the pregnancy is confirmed which can be caused by pro-inflammatory cytokines (Chebel *et al.*, 2004; Hansen *et al.*, 2004). In dairy cows, cytokines and prostaglandins  $F_{2\alpha}$  can cause luteolysis, which leads to pregnancy termination (Pate, 1994). Schams and Berisha, (2004) observed that on the corpus luteum, cytokines have cytotoxic effect in cattle. Nguyen *et al.* (2011) showed that in dairy cows with 50 somatic cell counts had a 47.6% conception rate from first service, while cows with 200-500 somatic cell counts had a 23.1 percent conception rate. In Holstein cows with subclinical mastitis, pregnancy loss was 1.4 times higher than in cows without mastitis (Dahl *et al.*, 2018). Mastitis during pregnancy or before breeding in dairy cows has been shown to increase the risk of pregnancy loss (Dahl *et al.*, 2017). Clinical mastitis was linked to a higher risk of pregnancy loss in dairy cows at any

time during lactation (Santos *et al.*, 2004; McDougall *et al.*, 2005). Endotoxin (LPS) produced by gram-negative bacteria increased serum prostaglandinF2 $\alpha$  levels and altered the estrous cycle or triggered abortion in cows due to its luteolytic action (Gilbert *et al.*, 1990; Jackson *et al.*, 1990). Lame cows experience pain and stress, which can lead to ailments like metritis and mastitis, as well as an

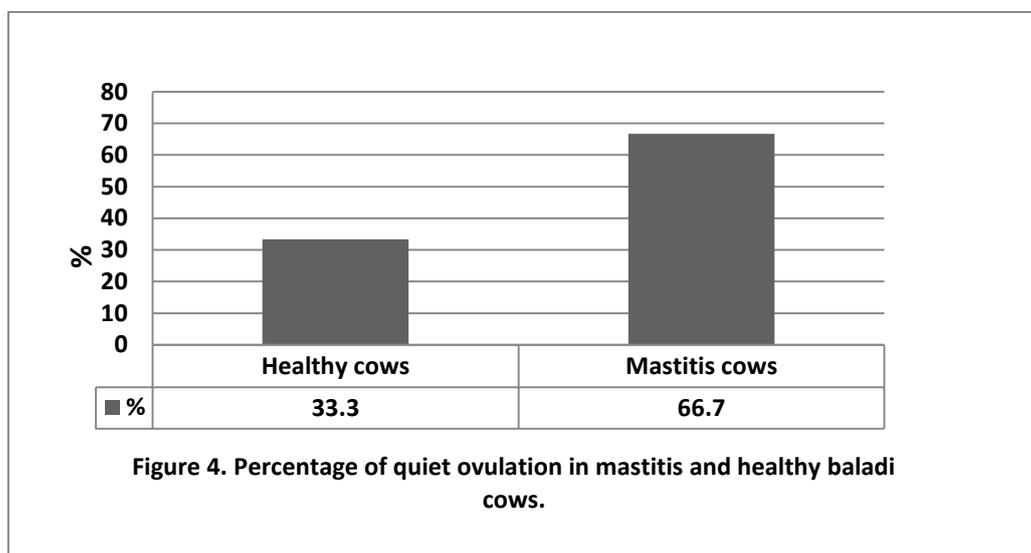
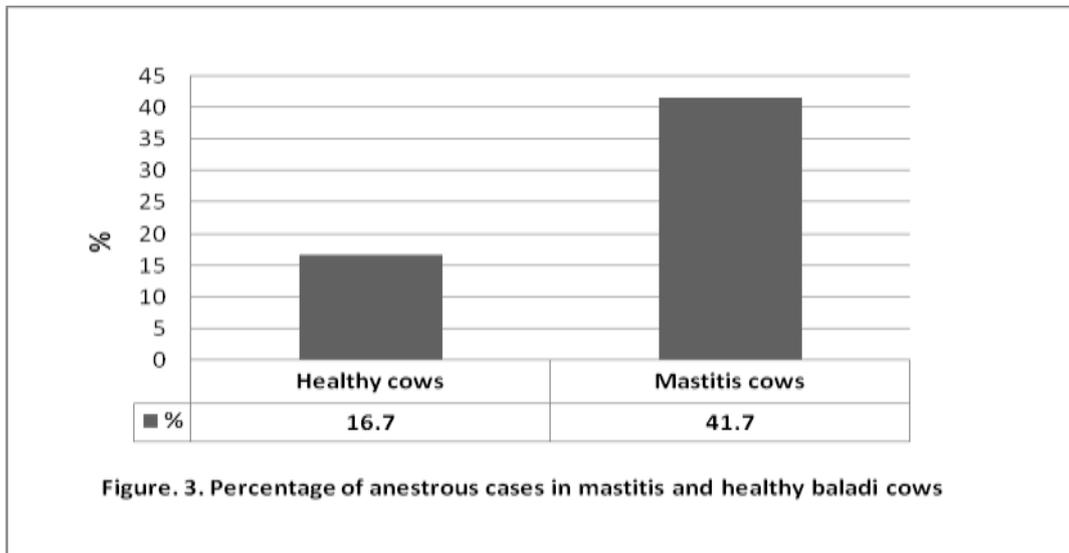
increase in glucocorticoids, which can lead to premature luteolysis in lactating dairy cows (Melendez *et al.*, 2003). Subclinical mastitis can cause a systemic inflammatory response, which can interfere with follicular development, oocyte quality, and embryo survival in dairy cows (Britt, 2008; Ribeiro *et al.*, 2016).



#### ***Postpartum anestrous and quiet ovulation cases:***

Figures (3, 4) indicate the percentage of anestrous and quiet ovulation cases which were significantly ( $P < 0.05$ ) higher (41.7, 66.7 %) in mastitic cows than (16.7, 33.3%) in healthy cows, respectively. The current finding correspond with that of Hansen *et al.* (2004) who reported that changes in endocrine profiles and follicular growth, inhibition of LH and FSH through cytokine release, and intrauterine embryonic survival are all possible mechanisms by which clinical or subclinical mastitis may communicate with conception. The disruption of the luteinizing hormone (LH) surge and ovulation failure are caused by increased plasma concentrations of lipopolysaccharides (LPS) as a result of infection (mastitis) (Peter *et al.*, 1989; Karsch *et al.*, 2002 and Sheldon and Dobson, 2004). Cows with subclinical mastitis recorded lower estrogen concentrations when they are in estrus, which might lead to a delay in ovulation (Lavon *et al.*, 2010; Lavon *et al.*, 2011). Lavon *et al.* (2010) and Rahman *et al.* (2012) stated that mastitis can cause atypical folliculogenesis, as

well as delayed ovulation, low estradiol levels, abnormal preovulatory LH surges. Clinical mastitis caused by gram-negative bacteria can disrupt the dairy cows reproductive cycle by modifying the inter-estrus interval and causing abortion by releasing inflammatory mediators (Moore *et al.*, 1991). Nguyen *et al.* (2011) reported that cows with higher somatic cell counts had a greater rate of extended luteal phase than cows with lower somatic cell counts. Inflammatory and immune responses in the udder may cause abnormal estrous cycle lengths, anovulation following estrus and fertilization failure (Huszenicza *et al.*, 2005; Moore *et al.*, 2005 and Pinzón-Sánchez and Ruegg, 2011). Reproductive problems, such as decreased estrus expression and irregular cyclicity, can occur when cows infected by mastitis (Dobson *et al.*, 2007). Endotoxin that produced from gram negative bacterial infection (mastitis) caused a reduced and delayed LH surge in cows during estrus. This has been linked to a delay in ovulation, which lowers the probability of fertilization success (Lavon *et al.*, 2008).



#### Concentrations of progesterone (ng/ml) and estradiol-17 $\beta$ (pg/ml):

Table (3) shows concentrations of progesterone and estradiol-17 $\beta$  during estrous cycle in cows with mastitis which were significantly ( $P < 0.05$ ) lower compared to those in healthy cows. Also, concentrations of progesterone and estradiol-17 $\beta$  during estrous cycle were higher in pregnant cows than non-pregnant cows ( $P < 0.05$ ) Table (3). Pregnant cows had higher progesterone serum concentrations on days 8 and 9 following insemination than non-pregnant cows (Perry *et al.*, 2005). When pre-ovulation estradiol-17 $\beta$  (E2) concentrations and post-ovulation progesterone concentrations were higher, all of which had a favorable impact on the percentage of pregnant postpartum cows (Ciernia *et al.*, 2021). A lower plasma estradiol-17 $\beta$  (E2) concentration before and after ovulation, as well as lower plasma progesterone concentration after ovulation, can result in insufficient preparation of the maternal tissues for pregnancy (Pohler *et al.*, 2012; Dickinson *et al.*, 2016). The two most significant factors influencing

the proportion of cows pregnant were estradiol concentrations prior to a GnRH-induced ovulation (d 0) and progesterone concentrations on d 7 after ovulation (Atkins *et al.*, 2013; Jinks *et al.*, 2013). Cows with higher plasma estradiol-17 $\beta$  (E2) concentrations had higher plasma progesterone concentrations as well (Vasconcelos *et al.*, 2001; Perry *et al.*, 2005 and Atkins *et al.*, 2013). Estradiol during the follicular growth cycle prior to ovulation, was necessary for the establishment of pregnancy after embryo transfer (Madsen *et al.*, 2015). The present findings agreed with those reported by Lavon *et al.* (2010) and Lavon *et al.* (2011) who found that cows with subclinical mastitis which are in estrus, had lower estrogen concentrations. Atypical folliculogenesis, as well as delayed ovulation, low estradiol levels, and irregular preovulatory LH spikes, may all be caused by mastitis (Lavon *et al.*, 2010; Rahman *et al.*, 2012). As a result of infection (mastitis), plasma concentrations of lipopolysaccharides (LPS) have increased (Karsch *et al.*, 2002 and Sheldon and Dobson, 2004). Lipopolysaccharides (LPS) were found in serum and

follicular fluid, suggesting that LPS influenced steroidogenesis (Herath *et al.*, 2007; Magata *et al.*, 2014). Suzuki *et al.* (2001) found that the basal LH pulsatility was disrupted, the plasma level of estradiol-17 was dramatically reduced, and the formation of the preovulatory LH peak was delayed or entirely blocked in heifers given an intravenous endotoxin (cytokines) like that produced by bacteria in mastitis infection. Mammary infections interrupt ovarian functions during the estrous cycle, decreasing follicular growth and estradiol (E<sub>2</sub>) development, disrupting luteal activity, and resulting in compromised ovarian functions (Sheldon *et al.*, 2002; Lavon *et al.*, 2008). Intravenous infusion of lipopolysaccharides in bovine in granulosa cells of the dominant follicle, LPS reduces the expression of steroidogenic genes and modulates the expression of toll-like receptor 4 (TLR4) and tumor necrosis factor (TNF) (Campos *et al.*, 2017). Estradiol (E<sub>2</sub>) synthesis was suppressed by lipopolysaccharide (LPS) in granulosa cells from small and large follicles in bovine (Shimizu *et al.*, 2012). *Escherichia coli* lipopolysaccharides (LPS) injection intravenously reduced progesterone levels in pregnant cows (Giri *et al.*, 1990). Lipopolysaccharide (LPS) reduces CL size and decreases plasma progesterone levels in cows (Herzog *et al.*, 2012). The presence of estradiol in the uterus of cattle has many implications, including regulating uterine pH (Perry and Perry, 2008), inducing endometrial progesterone receptors (Zelinski *et al.*, 1980) and regulating the timing of luteolytic secretion of prostaglandinF<sub>2α</sub> (Kieborz-Loos *et al.*, 2003). Embryo losses tended to occur after maternal pregnancy recognition, implying that an improvement in estradiol before ovulation may affect early placentation processes (Madsen *et al.*, 2015). Following ovulation, there was a higher rate

of conceptus elongation while plasma progesterone concentrations were higher (Mann *et al.*, 2006). Progesterone has direct effects on the uterus of cattle during the early stages of pregnancy (e.g., histotroph production) (Dorniak *et al.*, 2013; Brooks *et al.*, 2014). Exogenous progesterone treatment led to increase plasma progesterone concentrations during the luteal phase resulted in an increase of the pregnancy rate in dairy cows (Stronge *et al.*, 2005; MacNeil *et al.*, 2006). Yan *et al.* (2016) stated that the proportion of pregnant cows increased by around 5% while plasma progesterone concentrations were higher shortly after pregnancy, according to data from over 9000 cows. Greater concentrations of estradiol at the time of a GnRH-induced ovulation in cows were linked to higher concentrations of progesterone after ovulation (Vasconcelos *et al.*, 2001; Perry *et al.*, 2005). Estradiol is the main regulator of ovarian follicular development, and lipopolysaccharides (LPS) can invade follicular fluid, affecting steroidogenesis (Herath *et al.*, 2007; Williams *et al.*, 2008 and Magata *et al.*, 2014). Sheldon *et al.* (2009), Shimizu *et al.* (2012) and Magata *et al.* (2014) suggested that the lipopolysaccharides play a key function in inhibiting estradiol synthesis. Progesterone concentrations were higher in non-mastitis cows on days 9 through 25 after mating than in subclinical and clinical mastitis buffaloes (Mansour *et al.*, (2016). Mastitis may disrupt corpus luteum formation and regression, as well as progesterone secretion and embryonic growth, after cow mating (Mann and Lamming, 2001). Mastitis is linked to an increase in cytokine secretion, which can hinder LH secretion and lower progesterone levels in the bloodstream (Hansen *et al.*, 2004).

**Table 3. Concentrations of progesterone (ng/ml) and estradiol-17β (pg/ml) during estrous cycle in mastitic and healthy Baladi cows**

Hormones	Healthy cows		Mastitic cows	
	Pregnant	Non-pregnant	Pregnant	Non-pregnant
<b>Progesterone concentrations:</b>				
At estrus	0.34 <sup>a</sup> ± 0.03	0.22 <sup>b</sup> ± 0.02	0.25 <sup>c</sup> ± 0.01	0.14 <sup>d</sup> ± 0.02
7 days post-estrus	3.41 <sup>a</sup> ± 0.02	2.13 <sup>b</sup> ± 0.01	2.73 <sup>c</sup> ± 0.02	1.87 <sup>d</sup> ± 0.04
15 days post-estrus	5.31 <sup>a</sup> ± 0.04	3.5 <sup>b</sup> ± 0.02	4.2 <sup>c</sup> ± 0.05	2.20 <sup>d</sup> ± 0.03
<b>Estradiol-17β:</b>				
At estrus	38.15 <sup>a</sup> ± 0.44	22.01 <sup>b</sup> ± 0.22	23.24 <sup>c</sup> ± 0.21	13.13 <sup>d</sup> ± 0.23

a, b, c, d: values within the same row having different superscripts are significantly different at P < 0.05.

## CONCLUSION

The present results indicate a negative impact of mastitis by delaying the resumption of postpartum ovarian activity and reproductive measurements in Baladi cows. The current study recommends that small breeders must pay attention to the health of the udder, especially during the postpartum period, to reduce the incidence of mastitis and its harmful effects on the reproductive characteristics of lactating cows.

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## تأثير التهاب الضرع على إستئناف النشاط المبيضي والمقاييس التناسلية بعد الولادة في الأبقار البلدية

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يهدف البحث الحالي إلى دراسة تأثير التهاب الضرع على استئناف النشاط المبيضي والمقاييس التناسلية بعد الولادة في الأبقار البلدية. تم استخدام 24 بقرة بلدي بعد الولادة قسمت إلى مجموعتين كل منها اثني عشر بقرة. المجموعة الأولى (12 بقرة) كانت مصابة بالتهاب الضرع، بينما المجموعة الثانية (12 بقرة) كانت سليمة. تمت متابعة الأبقار بعد الولادة مباشرة، وتم فحص ضرورها لإكتشاف التهاب الضرع تحت السريري باستخدام إختبار كاليفورنيا لالتهاب الضرع. أظهرت النتائج الحالية أن معدل حدوث حالات التهاب الضرع تحت السريري في الأبقار البلدية كانت أعلى معنويًا 75% بالمقارنة بـ 25% حالات التهاب سريري. كانت الفترة من الولادة إلى التبويض الأول، التلقيح الأول و الحمل أطول معنويًا ( $P < 0.05$ ) (  $7.8 \pm 48.3$  يوم ) ، (  $14.4 \pm 96.5$  يوم ) و (  $12.5 \pm 153.2$  يوم ) في الأبقار التي تعاني من التهاب الضرع بالمقارنة بـ (  $5.6 \pm 27.4$  يوم ) ، (  $11.2 \pm 72.3$  يوم ) و (  $15.2 \pm 85.6$  يوم ) في الأبقار السليمة على الترتيب. كان معدل الحمل من التلقيح الأولى أقل معنويًا ( $P < 0.05$ ) ( 25 % ) في الأبقار المصابة بالتهاب الضرع مقارنة بـ 66.7 % في الأبقار السليمة. تشير النتائج الحالية إلى وجود تأثير سلبي لالتهاب الضرع على استئناف النشاط المبيضي والمقاييس التناسلية بعد الولادة في الأبقار البلدية. توصي الدراسة الحالية بضرورة إهتمام صغار المربين بصحة الضرع، خاصة خلال فترة ما بعد الولادة، للحد من تأثيره الضار على الخصائص التناسلية لأبقارهم.