INFLUENCE OF CLINICAL MASTITIS DURING THE PRE–CONCEPTION PERIOD ON MILK YIELD AND REPRODUCTIVE PERFORMANCE OF HOLSTEIN–FRIESIAN COWS

By

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SUMMARY

This study was undertaken to determine the impact of some risk factors on the incidence of clinical mastitis occurring during the open period of Holstein–Friesian cows in a large commercial dairy herd located near Amirya area at Alexandria–Cairo desert road. Also, the influence of clinical mastitis on milk yield and reproductive performance was investigated.

166 of 1152 (14.41%) cows were affected with clinical mastitis during the pre–conception period. The risk of acquiring clinical mastitis during this period was influenced by production level (higher risk for higher–producing cows), lactation number (higher risk for pluriparous cows) and season of calving (high risk for autumn– and winter–calving cows).

In general, the daily loss in milk yield during the first two weeks after the occurrence of mastitis was 2.40 kg (11.48%) and the 305–d milk loss was 480.11 kg (7.69%). The magnitude of these short– and long–term milk yield reductions depended on level of milk yield, season of calving and to a lesser extent on parity. High–yielding, winter–calving and pluriparous mastitic cows showed the highest milk losses.

Mastitic cows required, on the average, 9.90 more days from calving to the first insemination, 17.86 more days and 0.33 more inseminations to conceive compared to their unaffected matched controls. Inhibitory effects of clinical mastitis on fertility parameters of high–producing cows were large and significant ($P<0.05$), while those of low–yielding group were small and not significant. The disease caused a delay to first insemination both in primiparous and pluriparous cows. However, the delay to conception (days open) was significant ($P<0.05$) only in primiparous ones. The disease reduced the fertility of cows in all seasons, but; the severity of fertility inhibition varied among seasons with winter–calving cows were the most affected.

In conclusion, both productive and reproductive performance were adversely affected by clinical mastitis. The study, therefore, underlines the importance of mastitis control programs particularly for cows at higher risks.

INTRODUCTION

Mastitis is often described as the most prevalent and most important production disease facing the dairy industry. Recent reports demonstrated that mastitis remains a major problem for dairy cattle in Egypt (Hamouda, 2003) and worldwide (Fourichon et al., 2001; Bradley, 2002; Workineh et al., 2002; Dego and Tareke, 2003; Seegers et al., 2003). Its incidence varied between 14 and 44%. Wells et al. (1998) ranked health issues of dairy cows on the basis of their effect on production, zoonotic importance, international trade and animal welfare and have put mastitis, Salmonella, Johne's disease and bovine viral diarrhea at the top of the list. When production–related losses alone were considered, the key health issues were ranked as mastitis, reproductive diseases, lameness and metabolic diseases. In other words, mastitis came as first health disorder in dairy cows by both approaches.

Low milk yield and reproductive inefficiency are the two most important criteria causing economic losses, and are the primary reasons of culling dairy cows (Beaudeau et al., 1995; Gröhn et al., 1998; Alejandrino et al., 1999; Rajala and Gröhn, 1999; Smith et al., 2000; Workineh et al., 2002). Mastitis could be a reason of either or both conditions (Cullor, 1991; Lescourret and Coulon, 1994; Barker et al., 1998; Fourichon et al., 2000).

Mastitis may cause considerable economic consequences through reduced output (low milk yield and milk withheld from the market following antibiotic treatment), and increased costs (treatment, veterinary services, increased labor and cow culling), in addition to prevention costs of dry cow therapy (Beck et al., 1992; Bennett et al., 1999; Rajala et al., 1999; Fleischer et al., 2001). Kossaibati and Esslemont (1997) revealed that 38% of direct costs of ill health were due to mastitis. Under Egyptian conditions, Atallah (1997) similarly ranked mastitis as the most costly disease to dairy cattle. Furthermore, El–Sheikh et al. (1999) estimated losses to Egyptian dairy farmers as 3377.92 LE per cow culled due to mastitis. Besides the economic aspect, another important consequence of mastitis is the higher susceptibility to other

mastitis episodes in the same or next lactation, and to other diseases including infertility (van Drop et al., 1999; Loeffler et al., 1999; Gustafsson and Emanuelson, 2002; Lehtolainen et al., 2003).

Differences in the incidence and severity of clinical mastitis in dairy cows are associated with risk factors such as climate, season of calving, stage of lactation, parity, breed of cattle, level of production and management (Gröhn et al., 1995; Todhunter et al., 1995; Dego and Tareke, 2003; Lehtolainen et al., 2003). The objective of this study was to investigate the incidence and effect of clinical mastitis occurring during the open (non–pregnancy) period on milk yield and reproductive performance of Holstein–Friesian cows in relation to some risk factors.

MATERIALS AND METHODS

Animals and farm management:

The data of this study were obtained from the performance records of 1152 Holstein–Friesian cows that calved during the year 2001. These cows belonged to a large commercial herd located near Amirya area at Alexandria–Cairo desert road. All cows were machine–milked three times a day and milk yield was recorded for individual cows daily via computerized milking units. Lactating cows were grouped according to their milk production, and concentrate feeding was calculated accordingly. Green berseem, berseem hay, wheat or rice straw, and corn silage were also fed whenever available. Estrus detection was done by means of electronic pedometers on the cow's hind leg in addition to observation by herd personnel. Inseminations started almost 50 to 60 days postpartum, and pregnancy was checked via rectal palpation 40 to 60 days following insemination.

Mastitis data in this study were clinical cases detected at milking by the presence of visual abnormalities in milk and inflammatory condition of the udder. Only clinical mastitis occurring before confirmed pregnancy (during the open period) were considered in this investigation. Cases recorded after the confirmed pregnancy were outside the scope of this study.

Performance parameters:

Milk production parameters were average daily milk yield (DMY) during the 1st two weeks following the diagnosis of mastitis, and standardized 305–day milk yield (305–d MY). Reproductive

data included calving to first insemination interval (DFI), calving to conception interval (days open; DO) and number of services per conception (S/C). Cow characteristics e.g. date of calving and lactation number were recorded for each cow.

Selection of matched control cows and data analyses:

Control unaffected cows were chosen in such a way that they were comparable to mastitic cows in characteristics of interest except affection with mastitis. Matching criteria were milk production (above or below average), stage of lactation (nearly similar calving date, ± 7 days), and the same lactation number, e.g. mastitis–free, high–producing, first lactation cows were used as a control for mastitic, high–producing, first lactation cows milking at the same stage of lactation. The large number of unaffected cows in this survey (n = 986) enabled used to find out similar numbers of highly comparable control cows. This matching procedure was also used by Lescourret and Coulon (1994) and Menzies et al. (2003).

Of 166 mastitic cows revealed by the first analysis, 10 cows had no current lactation performance records because of death or very early culling for different reasons including severe mastitis. Therefore, the maximum number of observations available for the analysis of variance was 156. Furthermore, because of some missing values, not all 156 cows in the data file had complete information on performance parameters. Numbers of observations used were as follows: 156 for 305–d MY, 136 for DMY, 145 for DO and S/C. In order to estimate the effect of clinical mastitis on DFI, only cases occurring before first insemination (n = 125) were used.

Similar to the classification of Faye et al. (1998), parity was classified into two levels; primiparous (first lactation) and pluriparous (second or higher lactation). Four seasons of calving were defined by 3–month intervals; autumn (September to November), winter (December to February), Spring (March to May) and Summer (June to August). According to 305–d MY, cows were grouped as above (high–producing) or below average (low–producing). For those cows that had no 305–d MY records (n = 10), the milking potential was estimated from their initial production (mean production of the 4^{th}, 5^{th} and 6^{th} days of lactation) according to the method of Lescourret and Coulon (1994).

Incidence (categorical) data were analysed with the chi–square test. Production and reproduction variables were analysed using the least squares analysis of variance. Mastitis and unaffected cows were compared within production level, parity and season of calving. Statistical analysis system (SAS, 1996) was used for all calculations and analyses.

RESULTS AND DISCUSSION

Incidence of clinical mastitis in relation to some cow risk factors (Table 1):

166 out of 1152 (14.41%) Holstein–Friesian cows under investigation had contracted at least one episode of clinical mastitis during the period from calving to conception. Under the same Egyptian conditions, Hamouda (2003) obtained an incidence of 19.15% (90 of 470) for clinical mastitis during the first two months of lactation. Rajala et al. (1999) reported an incidence of 17% for clinical mastitis throughout the entire lactation of Ayrshire cows, and found that most cases occurred early in lactation. These estimates and others (Sviland and Waage, 2002; Workineh et al., 2002; Bradley, 2002) illustrate that mastitis remains a major challenge to the Egyptian and worldwide dairy industry.

The risk of clinical mastitis depended on level of milk yield as it increased with the production level (16.44% for high producers vs 12.32% for low producers; $P<0.05$). The present results support the conclusions of Bigras–Poulin et al. (1990), Houben et al. (1993), Gröhn et al. (1995) and Rajala and Gröhn (1998) that high milk producers are usually regarded as more predisposed to mastitis infection than cows yielding low or moderate amounts of milk.

Incidence of mastitis increased with lactation number. It was significantly ($P<0.05$) higher (16.46%) in pluriparous cows compared to 11.69% in primiparous ones. The higher susceptibility of older animals to infection than younger ones might be, partly, because with advancing age, the udder becomes more pendulous, thus increasing the risk of being injured, and because of higher milk yield of older cows. Similar findings were reported by Houben et al. (1993), Rajala et al., (1999), van Drop et al. (1999), Fleischer et al. (2001) and Dego and Tareke (2003) who concluded that age of the cow plays an important role in the genesis of mastitis, the older the cow the greater the incidence of mastitis.

Lactations initiated during autumn and winter were associated with higher incidence of the disease (16.67 and 15.66%, respectively) than those started during spring (11.25%) and summer (9.09%). Likewise, Dego and Tareke (2003) reported that bovine mastitis was significantly more prevalent in the wet season than in the dry season.

Short– and long–term effects of clinical mastitis on milk yield (Table 2):

Generally, mastitic cows produced significantly ($P<0.05$) less milk than unaffected controls. Losses during the first two weeks after the onset of clinical mastitis averaged 2.40 kg/day (11.48%), while total lactation losses averaged 480.11 kg (7.69%). Reduced 305–d MY indicates that mastitis has a long–lasting effect on milk yield, and affected cows could not totally recover from the disease but still yielding lower than their full production potential during the remaining of the lactation. Findings in the present study are comparable to those reported by Hamouda (2003) who revealed that a decrease in milk yield was the commonest sign in cows affected with clinical mastitis, and Rajala et al. (1999) who found that milk yield began to decline 4 weeks before the clinical onset of mastitis (during the subclinical phase) and dropped below the yield of healthy cows during the first two weeks after the diagnosis. They added that yield started to increase after this period but it did not reach the pre–mastitis level.

The magnitude of both short– and long–term milk losses depended on intensity of milk production. High–yielding mastitic cows suffered greater reductions in yield either daily (3.32 kg; 12.82% during the 1st 2 weeks after the onset of the disease) or throughout the lactation (673.17 kg; 8.57%) when compared to low–producing cows (1.48 kg/day; 9.30% and 287.05 kg/305 days).

Clinical mastitis adversely affected ($P<0.05$) milk yield of both primiparous and pluriparous cows; however, losses increased slightly with lactation number. Mastitic first–calf heifers produced 2.31 kg/day (11.06%) during the first two weeks after the clinical onset of mastitis, and 448.56 kg/305 days (7.26%) less than their unaffected controls. The respective losses for mastitic pluriparous cows were 2.5 kg/day (11.94%) and 511.66 kg/305 days (8.11%). In the same way, Rajala et al. (1999) recorded that short–term (1.1

to 2.5 kg/day) and total milk loss (294 to 552 kg/305 days) caused by mastitis increased with parity.

Daily milk losses during the first two weeks after the onset of an episode of clinical mastitis depended on season of calving. It varied between 1.08 kg (5.56%; P>0.05) in summer–calving cows to 3.20 kg (14.61%; P<0.05) for winter–calving cows. In a similar manner, total lactation milk losses were minimal and not significant for lactations initiated during summer (282.87 kg; 4.70%), while being maximal and significant (P<0.05) in those started during winter months (637.77 kg; 9.98%).

Effect of clinical mastitis on reproductive performance (Table 3):

Overall, occurrence of clinical mastitis prior to first service significantly (P<0.05) increased number of days to first insemination (89.46 ± 3.50) compared to unaffected control cows (79.56 ± 2.61). Furthermore, cows contracted clinical mastitis at any stage before conception required 17.86 days and 0.33 insemination to conceive more than cows without mastitis. Negative effects of mastitis on fertility were also observed by van Drop et al. (1999) who found that mastitis during early lactation increases the risk of cystic ovaries. Besides, Loeffler et al. (1999) and Schrick et al. (2001) who concluded that mastitis harmfully affects reproductive performance in terms of days open and conception rate.

Effects of clinical mastitis on reproductive parameters were dependent on milking ability of the cow. DFI, DO and S/C all were significantly (P<0.05) increased by mastitis in high–producing cows (+12.91 days, +28.22 days and +0.72 services, respectively) relative to unaffected cows. On the other hand, differences in fertility parameters between mastitic and unaffected low–producing cows were small and not significant (6.88 days, 7.5 days and –0.06 services for DFI, DO and S/C, respectively). Milk secretion in the dairy cow has a high metabolic priority and is maintained at the expense of other metabolic processes creating a negative energy balance (NEB) during the early lactation period (Radostits, 2001). NEB increases the severity of mastitis (Kremer et al., 1993; Oltenacu and Ekesbo, 1994), its occurrence (Bigras–Poulin et al., 1990; Heuer et al., 1999) and is associated with reduced fertility (Zurek et al., 1995; Senatore et al., 1996). These may explain why

fertility of mastitic high–producing cows was inhibited to a greater extent than low–producing cows.

Clinical mastitis seems to have more severe effects on reproductive efficiency of primiparous cows than on that of pluriparous cows. It significantly ($P<0.05$) delayed first insemination by 10.58 days (primiparous) and 9.21 days (pluriparous) compared to their unaffected controls. However, only first parity mastitic cows required significantly ($P<0.05$) 22.72 (16.56%) more DO than non–mastitic primiparous ones; meanwhile an increase of 13 (9.76%) DO in pluriparous cows due to mastitis was not statistically significant. S/C of cows in both parity groups have increased by clinical mastitis but the change did not prove statistical significance.

For clinical mastitis occurring before first service, the delay to first insemination was maximal and significant (15.32 days, $P<0.05$) for cows initiated their lactations in winter months followed by autumn–calvers (10.71 days; $P<0.05$); but was not significant in spring– and summer–calvers. In addition, the delay to conception (DO) was the greatest (28.20 days; $P<0.05$) when mastitis affected winter–calving cows followed by autumn–calvers (22.06 days; $P<0.05$). DO of cows that calved during spring and summer months were not altered significantly by mastitis. Winter–calving cows affected with clinical mastitis were the only season of calving group that required significantly more inseminations to conceive (3.14 ± 0.21) compared to their non–mastitic controls (2.29 ± 0.19). Differences in weather conditions and prevalence of some pathogens among seasons may partly affect the severity of mastitis leading to varying fertility responses among different seasons.

There is reasonable evidence in the literature explaining how can mastitis negatively affect reproduction. Clinical mastitis is an acute disease that often results in the release of bacterial endotoxins. Endotoxins may influence reproduction by altering endocrine profiles and follicular development. They significantly lower concentration of gonadotrophin–releasing hormone (GnRH) and leutinizing hormone (LH), elevate concentration of cortisol and body temperature (Battaglia et al., 1997). Suppression of LH in mastitis cows may affect the ability of oocyte to mature (Wise et

al., 1994). Cystic ovaries might be a result of LH suppression via elevated cortisol (Lopez–Diaz and Bosu, 1992).

A symptom that often associated with clinical mastitis is elevated body temperature (Hockette et al., 2000; Bekeer, 2002). Cows exposed to heat stress experience increased embryonic death (Thatcher and Hansen, 1993). In addition, Ealy et al. (1993) documented that effects of hyperthermia were greatest when occurring during early embryonic development. Edward and Hansen (1996) reported poor bovine oocyte development during elevated body temperature.

Prostaglandin F2alpha (PGF2α) release due to mastitis as a possible mechanism of reduced fertility has been hypothesized by Cullor (1991), Moore et al. (1991) and Schrick et al. (1993). Elevated PGF2α concentrations may decrease reproductive efficiency by decreasing luteal life span or by decreasing embryonic quality and development. Both mechanisms may lead to early termination of pregnancy, and ultimately could result in an increased number of services per conception and number of days open.

In conclusion, the results of the present study highlight the importance of strict mastitis control programs as it relates not only to milk losses, but also to reproductive inefficiency; in addition to the well–known costs associated with mastitis such as treatment costs, altered milk composition, discarded milk and culling of affected animals. Collectively, losses from mastitis are obvious and could affect returns from dairy cows. The novel approaches for controlling intramammary infections in the dairy cow call for dry cow therapy (antibiotic treatment at the end of lactation) to eliminate intramammary infections and prevent new infections (Bradley and Green, 2001; Berry and Hillerton, 2002), proper body conditioning during the dry period (Knight, 2001), supplementation with vitamin A, D and E in the late gestation period (Barnoiun and Chassagne, 1998; Bekeer, 2002) and the use of suitable teat dips (Boddie and Nickerson, 2002; Hamouda, 2003).

Abbreviation key: **DMY**: average daily milk yield during the first two weeks after the occurrence of mastitis; **305–d MY**: standardized 305–days milk yield. **DFI**: Days from calving to first insemination; **DO**: days open; **S/C**: number of services per conception.

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TABLE (1): Incidence of clinical mastitis during the pre–conception period of Holstein–Friesian cows in relation to some risk factors.

Factors	Total No.	Clinical mastitis	
		No.	%
Overall	1152	166	14.41
Milk Yield			
High–producing cows	584	96	16.44
Low–producing cows	568	70	12.32
Parity			
Primiparous cows	496	58	11.69
Pluriparous cows	656	108	16.46
Season of calving			
Autumn	420	70	16.67
Winter	396	62	15.66
Spring	160	18	11.25
Summer	176	16	9.09

Chi–square test for incidence of mastitis:
Difference due to level of milk yield: Chi–square value = 3.95; P=0.047.
Difference due to parity: Chi–square value = 5.21; P=0.022
Difference due to season of calving: Chi–square value = 7.57; P=0.055.

Table (2): Least squares means ± standard errors (LSM ± SE) for the effect of clinical mastitis during the calving–conception period on milk yield in relation to some risk factors.

Classifications	DMY (kg)		305–d MY (kg)	
	N*	LSM ± SE (kg)	N*	LSM ± SE (kg)
Overall				
Unaffected cows	136	20.91 ± 0.49 a	156	6245.01 ± 117.16 a
Mastitic cows	136	18.51 ± 0.51 b	156	5764.90 ± 107.52 b
Deviation**		–2.40 (11.48%)		–480.11 (7.69%)
Milk yield				
High–producing cows	79	25.90 ± 0.67 a	90	7857.09 ± 138.34 a
Unaffected	79	22.58 ± 0.68 b	90	7183.92 ± 99.02 b
Mastitic		–3.32 (12.82%)		–673.17 (8.57%)
Deviation				
Low–producing cows	57	15.91 ± 0.57 a	66	4632.93 ± 117.08 a
Unaffected	57	14.43 ± 0.59 b	66	4345.88 ± 124.47 a
Mastitic		–1.48 (9.30%)		–287.05 (6.20%)
Deviation				
Parity				
Primiparous cows				
Unaffected	44	20.88 ± 0.52 a	51	6182.32 ± 162.09 a
Mastitic	44	18.57 ± 0.55 b	51	5733.76 ± 158.21 b
Deviation		–2.31 (11.06%)		–448.56 (7.26%)
Pluriparous cows				
Unaffected	92	20.94 ± 0.53 a	105	6307.69 ± 119.32 a
Mastitic	92	18.44 ± 0.74 b	105	5796.03 ± 116.03 b
Deviation		–2.50 (11.94%)		–511.66 (8.11%)
Season of calving				
Autumn				
Unaffected	57	21.54 ± 0.59 a	66	6367.74 ± 134.08 a
Mastitic	57	19.07 ± 0.62 b	66	5739.72 ± 130.87 b
Deviation		–2.47 (11.47%)		–628.02 (9.86%)
Winter				
Unaffected	52	21.90 ± 0.61 a	59	6390.68 ± 137.67 a
Mastitic	52	18.70 ± 0.61 b	59	5752.91 ± 134.37 b
Deviation		–3.20 (14.61%)		–637.77 (9.98%)
Spring				
Unaffected	14	20.74 ± 1.01 a	16	6208.21 ± 254.52 a
Mastitic	14	17.89 ± 0.85 b	16	5836.41 ± 248.42 a
Deviation		–2.85 (13.74%)		–371.80 (5.99%)
Summer				
Unaffected	13	19.44 ± 1.16 a	15	6013.40 ± 282.47 a
Mastitic	13	18.36 ± 1.33 a	15	5730.53 ± 275.70 a
Deviation		–1.08 (5.56%)		–282.87 (4.70%)

DMY = average daily milk yield during the 1st two weeks after the onset of clinical mastitis.
305–d MY = standardized 305–day milk yield.
* Numbers of observation; unaffected cows were matched controls selected from a total of 986 cows.
** Deviation = absolute and relative deviation of mastitic cows from their unaffected controls.
Means of unaffected and mastitic cows followed by similar letter within a classification do not differ significant at P=0.05.

Table (3): Least squares means ± standard errors (LSM ± SE) for the effect of clinical mastitis during the calving–conception period on reproductive performance in relation to some risk factors.

Classifications	DFI N*	DFI LSM ± SE	DO N*	DO LSM ± SE	S/C N*	S/C LSM ± SE
Overall						
Unaffected cows	125	79.56 ± 2.61 b	145	135.24 ± 4.16 b	145	2.59 ± 0.10 b
Mastitic cows	125	89.46 ± 3.50 a	145	153.10 ± 5.48 a	145	2.92 ± 0.16 a
Deviation**		+9.9 (12.44%)		+17.86 (13.21%)		+0.33 (12.74%)
Milk yield						
High–producing cows						
Unaffected	73	75.51 ± 4.08 b	81	121.54 ± 5.01 b	81	2.23 ± 0.14 b
Mastitic	73	88.42 ± 3.69 a	81	149.76 ± 5.61 a	81	2.95 ± 0.16 a
Deviation		+12.91 (17.10%)		+28.22 (23.22%)		+0.72 (32.29%)
Low–producing cows						
Unaffected	52	83.62 ± 5.13 a	64	148.94 ± 7.88 a	64	2.95 ± 0.20 a
Mastitic	52	90.50 ± 5.90 a	64	156.44 ± 8.19 a	64	2.89 ± 0.21 a
Deviation		+6.88 (8.23%)		+7.50 (5.04%)		–0.06 (2.03%)
Parity						
Primiparous cows						
Unaffected	46	87.01 ± 3.01 b	49	137.22 ± 6.12 b	49	2.43 ± 0.21 a
Mastitic	46	97.59 ± 4.22 a	49	159.94 ± 9.85 a	49	2.81 ± 0.36 a
Deviation		+10.58 (12.16%)		+22.72 (16.56%)		+0.38 (15.64%)
Pluriparous cows						
Unaffected	79	72.12 ± 1.36 b	96	133.26 ± 8.15 a	96	2.75 ± 0.22 a
Mastitic	79	81.33 ± 3.65 a	96	146.26 ± 9.44 a	96	3.03 ± 0.28 a
Deviation		+9.21 (12.77%)		+13.00 (9.76%)		+0.28 (10.18%)
Season of calving						
Autumn						
Unaffected	52	80.44 ± 3.21 b	61	114.98 ± 6.92 b	61	2.53 ± 0.14 a
Mastitic	52	91.15 ± 4.10 a	61	137.04 ± 9.08 a	61	2.56 ± 0.23 a
Deviation		+10.71 (13.31%)		+22.06 (19.19%)		+0.03 (1.19%)
Winter						
Unaffected	46	79.34 ± 3.55 b	54	129.30 ± 8.19 b	54	2.29 ± 0.19 b
Mastitic	46	94.66 ± 5.69 a	54	157.50 ± 9.60 a	54	3.14 ± 0.21 a
Deviation		+15.32 (19.31%)		+28.20 (21.81%)		+0.85 (37.12%)
Spring						
Unaffected	14	78.98 ± 9.16 a	16	148.79 ± 11.21 a	16	2.54 ± 0.41 a
Mastitic	14	85.50 ± 10.17 a	16	162.87 ± 13.06 a	16	2.81 ± 0.51 a
Deviation		+6.52 (8.26%)		+14.08 (9.46%)		+0.27 (10.62%)
Summer						
Unaffected	13	79.50 ± 11.20 a	14	147.89 ± 10.51 a	14	3.00 ± 0.38 a
Mastitic	13	86.53 ± 10.84 a	14	154.99 ± 5.59 a	14	3.17 ± 0.41 a
Deviation		+7.03 (8.84%)		+7.10 (4.80%)		+0.17 (5.67%)

DFI = calving to first insemination interval; DO = days open; S/C = number of services per conception.

* Numbers of observation; unaffected cows were matched controls selected from a total of 986 cows.

** Deviation = absolute and relative deviation of mastitic cows from their unaffected controls.

Means of unaffected and mastitic cows followed by similar letter within a classification do not differ significant at P=0.05.

الملخص العربى

تأثير إلتهاب الضرع الإكلينيكي في فترة ما قبل الإخصاب على إدرار اللبن والكفاءة التناسلية في أبقار الهولشتاين فريزيان

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استهدفت هذه الدراسة تحديد تأثير بعض العوامل على معدل حدوث إلتهاب الضرع الإكلينيكي خلال فترة ما قبل الإخصاب في أبقار الهولشتاين فريزيان و كذلك دراسة تأثير هذا المرض على إدرار اللبن والكفاءة التناسلية لهذه الأبقار. أظهرت الدراسة أن 166 بقرة كانت مصابة بإلتهاب الضرع الإكلينيكي من أصل 1152 بقرة (14.41%) في فترة ماقبل الإخصاب. وقد تبين أن فرصة حدوث إلتهاب الضرع الإكلينيكي تتأثربمستوى إنتاج اللبن (فالأبقار عالية الإدرار كانت أكثر تعرضاً للمرض) وعدد مرات الولادة (التى ولدت اكثر من مرة كانت اكثر تعرضاً) وكذلك موسم الولادة (التى ولدت في الخريف والشتاء كانت اكثر تعرضاً للمرض).

بدراسة تأثير إلتهاب الضرع الإكلينيكى على إنتاج اللبن أظهرت النتائج أن كمية اللبن المفقودة خلال الأسبوعين التاليين لبداية حدوث إلتهاب الضرع كانت 2.4 كجم (11.48%) يومياً و أن معدل الفقد طوال موسم الحليب (305 يوم) كان 480.11 كجم (7.69%). و قد إختلفت شدة الفقد فى إنتاج اللبن على المدى القصير و البعيد تبعاً لمستوى إنتاجية البقرة و موسم ولادتها و بدرجة أقل على عدد الولادات حيث كانت الأبقار عالية الإنتاج و الأبقار التى تلد فى الشتاء و الأبقار متعددة الولادات أكثر فقداً للبن نتيجة لإلتهاب الضرع.

بدراسة تأثير إلتهاب الضرع الإكلينيكى على الخصوبة إتضح من الدراسة أن الأبقار التى تصاب بإلتهاب الضرع في فترة ما قبل الإخصاب تحتاج 9.9 يوم أكثر من الولادة حتى أول تلقيحة و 17.86 يوم أكثر من الولادة حتى الإخصاب و 0.33 تلقيحة أكثر مقارنة بالأبقار غير المصابة .

الخصـــوبة فـــي الأبقـــار عالية الإدرار كانت أكثر تضرراً عن تلك منخفضة الإدرار وقد زادت الفـــترة مـــن الولادة إلى أول تلقيحة في كلاً من الأبقار التى ولدت مرة واحدة والتى ولدت اكـــثر مـــن مـــرة. أمـــا الفـــترة من الولادة إلى الإخصاب فقد زادت معنوياً فقط في الأبقار التى ولـــدت مـــرة واحـــدة وكذلك فقد زادت مرات التلقيح في الأبقار المصابة بإلتهاب الضرع والتى ولـــدت مـــرة واحدة واكثر من مرة مقارنه بالأبقار غير المصابة . أيضا فان إلتهاب الضرع أدى إلـــى نقـــص خصـــوبة الأبقـــار المصابة خلال المواسم الأربعة ولكن تلك التى ولدت في الشتاء كانت الأكثر تأثرا.

الخلاصـــة :تبيـــن مـــن النـــتائج أن كـــل من الكفاءة الإنتاجية والتناسلية للأبقار قد تأثرت عكسيا بإلـــتهاب الضـــرع الإكلينيكي و لذلك تؤكد الدراسة على أهمية برامج الوقاية من إلتهاب الضرع خاصة للأبقار ذات العوامل المؤهلة للإصابة بالمرض.

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