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INfIUENCE OF SUBCLINICAL MASTITIS ON SOME REPRODUCTIVE PARAMETERS IN HOLSTEIN-FRIESIAN DAIRY COWS

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SUMMARY

Over a period of one year (Nov.,2007- Oct.,2008) a total of 1757 Holstein –Friesian cows belonging to a commercial dairy farm in Giza –Egypt were included in this investigation. Somatic cell count (SCC) as a measure of subclinical mastitis was recorded for three times in three consecutive months. Bacteriological examination was also done for milk sample. Reproductive data including days to first insemination, number of inseminations per conception (S/C) and days open (DO) were individually recorded ,also the percentage of cows conceived by 305 days in milking (DIM) and percentage of culled cows for reproductive failure were recorded. The obtained data were recorded .The animal status (parity and reproductive status) as well as the establishment and degrees of affection were also considered. The obtained results were recorded and statistically analyzed. Results revealed that, the incidence of subclinical mastitis was 31.75 and 23.79 % of the total lactating non pregnant and lactating pregnant cows, respectively,. Among cases of subclinical mastitis, 75 % were bacteriolo-gically positive. Coagulase-negative staphylococci, Staphylococcus aureus, Streptococcus uberis and Streptococcus dysgalactiae were the prevalent isolates.The adverse effects of subclinical mastitis on the reproductive performances in dairy herd included prolonged days to first insemination; Days open; increased number of inseminations per conception and culling rate for infertility. The establishment of subclinical mastitis during the interval to first insemination prolonged this stage while, the establishment from the first insemination to the fertile one prolonged the days open and increase the number of inseminations per conception.It could be concluded that, subclinical mastitis was associated with unfavourable effects on reproductive performance of dairy cows. Therefore, the appropriate management of lactating dairy cows to minimize the incidence of mastitis should increase the profitability of dairy herds not only by improving milk quality, reducing the use of antibiotics, reducing the amount of milk discarded, and reducing involuntary culling , but also by improving reproductive performance.

INTRODUCTION

Mastitis is one of the most costly and common diseases affecting dairy cows throughout the world (DeGraves and Fetrow, 1993;Tsuruta et al., 2004 and Losinger,2005). Somatic cell count (SCC) is often used as an indirect measure of mammary infection status (Shook and Schutz, 1993, Mrode and Swanson, 1996). The SCC mean level for Holstein cows without clinical mastitis was around 200,000 cells/ml (Coulon et al., 1996; Rupp et al., 2000). Mastitis infections affect milk yield and quality (Heringstad et al., 2003; Ikonen et al., 2004; Miller et al., 2004; Hagnestam et al., 2007). Furthermore, other studies (Barker et al., 1998; Miller et al., 2001; Moore et al. 2005;Windig et al., 2006 and Rekik et al.2008) confirmed antagonistic relationships between the cow's mammary infection and its reproductive performances. Barker et al. (1998) found that the interval from calving to first service was longer for cows having clinical mastitis before first service than for other cows (71 vs 94 days). Likewise, these authors found that mastitis when occurring after the first service delays conception. Two extensive epidemiological studies indicated that both Gram-positive and Gram-negative bacteria, associated with clinical and sub-clinical mastitis, are involved in the disruption of reproductive performance in dairy cattle (Schrick et al.2001 and Santos et al.2004). In several studies, mastitis was associated with low fertility in cattle due to endotoxin induction of PGF2 α production (Hockett et al.2000) and luteolysis of the CL of pregnancy (Moore et al.1991).Other studies indicated that a variety of stressors, including mastitis-induced endotoxin, can disrupt the timing of ovulation and consequently prevent successful fertilization. Several studies showed that stressors activate the hypothalamic-pituitary-adrenal axis and enhance the secretion of glucocorticoids that can mediate suppression of reproductive processes (Sapolsky et al 2000)]. Acute restraint or isolation reduces plasma LH concentration and pulsatile LH secretion in the rat, sheep, and monkey (Tilbrook et al.2000). Immune/inflammatory stress that was induced by Gram-negative lipopolysaccharide(LPS) administration by intravenous or intrauterine injection in ewes and cows, during the follicular phase, suppressed pulsatile LH secretion and delayed or blocked the preovulatory LH surge [Battaglia et al 2000; Suzuki et al .2001and Daniel et al .2003).For these reasons, SCC is used in studies on mammary health status and for reproduction purposes (Miller et al.,2004 and Caraviello et al., 2005) .However, little is known about the effects of subclinical mastitis during lactation period on reproductive performance. Thus, the objective of the present study was to determine the effects of subclinical mastitis during lactation on reproductive performance of Holstein-Friesian dairy cows under Egyptian management circumstances.

MATERIALS AND METHODS

Animals

Over a period of one year (Nov., 2007- Oct., 2008) a total number of 1757 Holstein –Friesian cows belonging to Al -Alamia dairy farm located at Kilo,26 Cairo Alexandria Desert Road (Abu Rawash – Giza - Egypt) were included in the current study. The animals included in this investigation were classified as shown in table 1.

RESULTS

Overall 31.75 and 23.79 % of the total lactating non pregnant and lactating pregnant cows, respectively, were found to be affected with subclinical mastitis (table 2). As shown in table 2, subclinical mastitis in lactating non pregnant animals had detrimental effects on some reproductive performance (S/C, days open and percentage of pregnant cows by the 305th DIM).Otherwise, controversial effects of high SCC on reproductive performance were recorded in the lactating pregnant cows.A total number of 194 (11.04 ± 0.75%) of the overall cows were culled for infertility, out of these, 150 cows (77.32 ± 3.01 %) were of normal SCC (< 200,000) and 44 cows (22.68 ± 3.01 %) were of high SCC (≥ 200,000). Among the subclinical mastitis cases, 75 % were found to be bacteriologically positive. The majority of infections were due to coagulase-negative Staphylococci, Staphylococcus aureus and the Streptococcus species including Streptococcus uberis and Streptococcus dysgalactiae. Cows of the control group (< 200,000 SCC) had 2.92, 226.17 and 74.67 S/C, days open and percent of pregnant cows by the 305th DIM, respectively (table 3). As a function of degrees and timing of affections (table 3), cows affected before the first insemination showed a non significant difference of their reproductive performance, while those affected from the first to the fertile insemination showed adverse effects on their reproductive performances specially in the 2nd and 3rd groups (≥ 200,000 – 1000,000 SCC) .In the 4th group (> 1000,000 SCC), the reproductive performances were slightly improved.In general, subclinical mastitis adversely affects the reproductive performance in primiparous and pleuriparous cows specially those affected from the first to the fertile insemination .However, the adverse effects of subclinical mastitis were apparently higher in pleuriparous cows (table 4).

DISCUSSION

The somatic cells of milk are mainly leukocytes, which include macrophages, lymphocytes and neutrophils (Harmon and Reneau, 1993). The goal of the leukocytes is to embody and digest the invading microorganism of the mammary gland. Although subclinical mastitis does not present apparent signal, it limits the economical exploration of the cow (Torres, 1985). In the current study the overall mean of SCC/ ml milk was significantly higher in pleuriparous than primiparous cows (287.47± 25.61 vs 195.26 ± 23.10, table 4). These results agreed with those reported by Miller (1982) Mackie and Rodgers (1986) and Nickerson et al .(1995).The SCC level under 200.000 cells/ml of milk considered normal, although it may be low in the first lactation (Ltavo et al.2001). Generally, SCC increases with the age and number of lactation in infected cows. On the contrary the SCC in non-infected udder, does not seem to vary with the age (Monardes, 1984 and Rekik et al.2008). All the animals, free from infection, had elevated SCC immediately after parturition; so, a fast decrease was observed after birth in non-infected animals or quarters (Monardes, 1994; Harmon and Reneau, 1993). Sheldrake (1983) reported that, the SCC from milk of non-infected animals increased from

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83.000 on the 35 day after the birth to 160.000 cells/ml milk on the 285 day. However, in animals infected with Staph. aureus, it increased from 234.000 to 1.000.000 cells/ml of milk in the same period. Our results revealed that, the incidence of subclinical mastitis was 31.75 and 23.79 % of the total lactating non pregnant and lactating pregnant cows, respectively. These results agreed with those of DeGraves and Fetrow (1993); Tsuruta et al., (2004) and Losinger (2005).Percentage of cows culled in the current study (11.04 ± 0.75%) was lower than that recorded by Shook and Schutz (1993) who reported a culling percentage of 30 % due to high SCC. The low culling rate in the current study referred to the policy of culling due to the high prices of imported pregnant heifers and the restrictions of heifer's importation in Egypt over the last five years.The results of the current study revealed that, the occurrence of subclinical mastitis during early interval to first insemination (140.46± 16.7 days) was recorded in primiparous cows. These results agreed with that of Oltenacu et al. (1990) who observed a significant influence of mastitis on interval to first insemination in primiparous cows.Our results revealed that, the most prominent negative effects of subclinical mastitis on the reproductive performance were recorded for the 2nd and 3rd groups (≥ 200,000 –1000,000 SCC).These results agreed with Matos et al. (1991), Barker et al. (1998) ; Schrick et al. (2001) and Santos et al.(2004).The mechanism(s) by which subclinical mastitis may influence reproductive performance is unknown. Therefore, potential mechanisms through which mastitis may affect reproductive efficiency will be discussed here.One of the possible mechanisms for the reduction in fertility of lactating dairy cows that develop mastitis is the elevated body temperature (fever), which can result from infections of the mammary gland (Wenz et al., 2001). In vitro studies had demonstrated a smaller proportion of oocytes and embryos cultured under heat stress develop to the blastocyst stage (Edwards and Hansen, 1997; Krininger et al., 2002). Furthermore, when lactating dairy cows were exposed to heat stress, the fertilization rate and the proportion of excellent/good quality embryos were dramatically decreased (Sartori et al., 2002). This indicates that exposure of oocytes and embryos to heat stress compromises fertilization and development.

lactation resulted in detrimental effects on the subsequent reproductive performance in dairy cows. The interval to first service was significantly prolonged in cows established subclinical mastitis before the first insemination .Moreover the number of inseminations per conception and days open were found to be significantly higher in cows established subclinical mastitis after the first insemination. These results were consistent with those reported by Barker et al. (1998); Schrick et al.(2001); Santos et al.(2004) ; Moore et al 2005 and Ahmadzadeh et al.(2009). Loeffler et al. (1999), Barker et al.(1998) and Moore et al. (2005) had pointed out the importance of the time of subclinical mastitis occurrence for the effect on fertility. In their studies, mastitis reduced conception rate significantly, if it occurred after the first insemination. In the current study, the higher value of

Aside the direct effect of elevated body temperature on oocyte, embryo quality and development, feverish cow have decreased feed intake and body condition (Maltz et al., 1997 and Buttler, 2000).

Another possible mechanism by which mastitis may affect fertility in lactating dairy cows is through the production of substances that affect oocyte and embryo quality and development, uterine environment, and ovarian function. These substances are called cytokines and amongthem interleukin (IL)-1α, IL-1β, IL-6, IL-10, IL-12, and tumor necrosis factor-α (TNF-α) have been isolated from milk-derived cells from infected mammary glands (Riollet et al., 2001). Furthermore, challenge of lactating cows with components of the cell wall (lypopolysaccharide, LPS) of *Escherichia coli* (gram-negative bacteria) resulted in increased milk concentrations of IL-1β, IL-8, and TNF-α (Nakajima et al., 1997 ; Blum et al., 2000; Hoeben et al., 2000 and Waller et al., 2003). Mastitis is also correlated with increased concentrations of nitric oxide (NO) and prostaglandin F2α (PGF2α) in milk and mastitic cows had increased blood concentrations of PGF2α metabolite (Blum et al., 2000; Bouchard et al., 1999; Giri et al., 1984;Hockett et al., 2000).Moreover, negative bacteria result in increased blood concentrations of cortisol, a hormone that blocks the release and the peak of LH (Stoebel et al., 1982; Li et al., 1983; Padmanabhan et al., 1983). The decrease or lack of LH secretion may result in compromised follicle and oocyte development, failure of ovulation, and suboptimal luteal function. Some of the cytokines produced during mastitis also have a direct effect on the maturation of bovine oocytes in the presence of TNF-α resulted in reduced proportion of fertilized oocytes developing to the blastocyst stage (Soto et al., 2003). It was recorded that embryos cultured in the presence of TNF-α, PGF2α, or NO had either increased number of apoptotic cells(dead cells) or compromised development to the blastocyst stage (Pampfer et al., 1994; Wuu et al., 1999; Soto et al., 2003; Chen et al., 2001; Hobbs et al., 1999). Furthermore, administration of PGF2α to cows supplemented with progesterone resulted in poorer quality embryos and decreased pregnancy rates, reinforcing the idea that PGF2α may have a negative effect on embryo development (Buford et al., 1996).It has been demonstrated that production of PGF2α which is responsible for luteolysis can be stimulated by cytokines such as TNF-β and IL-1c (Davidson et al., 1995;Skarzynski et al.). Therefore, mastitis can lead to an increase in secretion of PGF2α and consequently premature luteolysis, which could result in embryonic/fetal death.It has been demonstrated that certain cytokines such as IFN-β decrease the secretion of LH (McCann et al., 2000). Furthermore, mastitis and exposure of cows to endotoxins secreted by gram- concentrations of Conclusion:It was clear that, ovaries. Interleukin-6, for example, blocks the secretion of estradiol (Alpizar et al., 1994), which can lead to reduced LH secretion, while TNF-β and IFN-δ are cytotoxic to the corpus luteum (Fairchild et al., 1991; Petroff et al., 2001) and could cause reduction in subclinical mastitis exerted great deteriorated effects on reproductive performance of dairy cows. The establishment of subclinical mastitis before the first insemination, prolonged this period. Meanwhile the

establishment of subclinical mastitis after first insemination increased the S/C and resulting in prolongation of days open. Therefore, the appropriate management of lactating dairy cows to minimize the incidence of mastitis could increase the profitability of dairy herds not only by improving milk quality, reducing the use of antibiotics, reducing the amount of milk discarded, and reducing involuntary culling but also by improving the reproductive performan

REFERENCES

Ahmadzadeh, F. Frago , B. Shafii ,J.C. Dalton , W.J. Price , and M.A. McGuire (2009): Effect of clinical mastitis and other diseases on reproductive performance of Holstein cows. Animal Reproduction Science ;(112),273–282.

Alpizar, E., and L.J. Spicer (1994): Effects of interleukin-6 on proliferation and follicle- stimulating hormone-induced estradiol production by bovine granulose cells in vitro: dependence on size of follicle. Biol. Reprod. 50: 38.

Barker, A.R., F.N. Schrick, M.J. Lewis, H.H. Dowlen, and S.P. Oliver. (1998):Influence of Clinical Mastitis During Early Lactation on Reproductive Performance of Jersey Cows. J. Dairy Sci. 81:1285.

Battaglia, DF, Krasa HB, Padmanabhan V, Viguie C, Karsch FJ. (2000): Endocrine alterations that underlie endotoxin-induced disruption of the follicular phase in ewes. Biol Reprod.;62:45–53.

Blum, J.W., H. Dosogne, D. Hoeben, F. Vangroenweghe, H.M. Hammon, R.M. Bruckmaier, C. Burvenich. . (2000): Tumor necrosis factor-b and nitrite/nitrate responses during acute mastitis induced by Escherichia coli infection and endotoxin in dairy cows. Dom. Anim. Endocrinol.19: 223.

Bouchard, L., S. Blais, C. Desrosiers, X. Zhao, and P. Lacasse. (1999): Nitric oxide production during endotoxin-induced mastitis in the cow. J. Dairy Sci. 82: 2574.

Buford, W.I.,N. Ahmad, F.N. Schrick, R.L. Butcher, P.E. Lewis, and E.K. Inskeep.(1996): Embryotoxicity of a regressing corpus luteum in beef cows supplemented with progesterone. Biol.Reprod.54: 351.

Buttler, W.R.(2000): Nutritional interactions with reproductive performance in dairy cattle. Anim. Reprod. Sci. 60:449.

Caraviello, D.Z., Weigel, K.A., Shook, G.E., Ruegg, P.L., (2005): Assessment of the impact of somatic cell count on functional longevity in Holstein and Jersey cattle using survival analysis methodology. J. Dairy Sci. 88, 804–811.

Chen, H.W., W.S. Jiang, and C.R. Tzeng. (2001): Nitric oxide as a regulator in preimplantation embryo development and apoptosis. Fertil. Steril. 75:1163.

Coulon, J.B., Dauver, F., Garel, J.P. (1996):. Facteurs de variation de la
numération cellulaire du lait chez des vaches laitières indemnes de
mammites cliniques. INRA Prod. Anim., 9, 133–139.

Daniel JA, Abrams MS, deSouza L, Wagner CG, Whitlock BK, Sartin JL. (2003): Endotoxin inhibition of luteinizing hormone in sheep. Domest Anim Endocrinol;25:13–9.

Davidson, J.A., U. Tiemann, J.G. Betts, and P.J. Hansen. (1995): DNA synthesis and prostaglandin secretion by bovine endometrial cells as regulated by interleukin-1. Reprod. Fertil. Dev. 7:1037.

DeGraves, F.J., Fetrow, F., (1993): Economics of mastitis and mastitis control. The Veterinary Clinics of North America: Update on Bovine Mastitis, vol. 9, pp. 421–434

Edwards, J.L., and P.J. Hansen. (1997): Differential responses of bovine oocytes and preimplantation embryos to heat shock. Mol. Reprod. Dev. 46: 138.

Fairchild, D.L., and J.L. Pate. (1991): Modulation of bovine luteal cell synthetic capacity by interferon-d. Biol.Reprod. 44: 357.

Giri, S.N., Z. Chen, E.J. Carroll, R. Mueller, M.J. Schiedt, and L. Panico. (1984): Role of prostaglandins in pathogenesis of bovine mastitis induced by Escherichia coli endotoxin. Am. J. Vet. Res. 45: 586.

Hagnestam, C., Emanuelson, U., Berglund, B., (2007): Yield losses associated with clinical mastitis occurring in different weeks of lactation. J. Dairy Sci. 90, 2260–2270.

Harmon, R.J.; RENEAU, J.K. (1993): Factors affecting somatic cell count in milk. Proc. Natl. Mast. Counc., v. 33, p. 33.

Heringstad, B., Rekaya, R., Gianola, D., Klemetsdal, G., Weigel, K.A.,(2003): Genetic change of clinical mastitis in Norwegian cattle: a threshold model analysis. J. Dairy Sci. 86, 369–375.

Hobbs, A.J., A. Higgs, and S. Moncada. (1999): Inhibition of nitric oxide synthase as potential therapeutic target. Annu. Rev. Pharmacol. Toxicol. 39:191.

Hockett, M.E., F.M. Hopkins, M.J. Lewis, A.M. Saxton, H.H. Dowlen, S.P. Oliver, and F.N. Schrick.(2000): Endocrine profiles of dairy cows following experimentally induced clinical mastitis during early lactation. Anim. Reprod. Sci. 58:241.

Hoeben, D., C. Burvenich, E. Trevisi, G. Bertoni, J. Hamann, R.M. Bruckmaier, and J.W. Blum. (2000): Role of endotoxin and TNF-b in the pathogenesis of experimentally induced coliform mastitis in periparturient cows. J. Dairy Res. 67: 503.

Ikonen, T., Morri, S., Tyrisevä, A.M., Ruottinen, O., Ojala, M., (2004):
Genetic and phenotypic correlations between milk coagulation properties, milk production traits, somatic cell count, casein content, and pH of milk. J. Dairy Sci. 87, 458–467.

Krininger, C.E. III, S.H. Stephens, and P.J. Hansen. (2002): Develpomental changes in inhibitory effects of arsenic and heat shock on growth of preimplantation bovine embryos. Mol. Reprod. Dev. 63: 335.

Li, P.S., and W.C. Wagner. (1983): In vivo and in vitro studies on the effect of adrenocorticotropic hormone or cortisol on the pituitary response to gonadotropin releasing hormone. Biol. Reprod. 29:25.

Loeffler, S.H., de Vries, M.J., Schukken, Y.H, (1999): The effects of time of disease occurrence, milk yield, and body condition on fertility of dairy cows. J. Dairy Sci. 82, 2589–2604.

Losinger WC. (2005): Economic impacts of reduced milk production associated with an increase in bulk-tank somatic cell count on US dairies. J Am Vet Med Assoc 2005;226:1652–8.

Ltavo ,L.C.V; Santos,G.T.; de Toledo,V.A.and Camila Celeste (2001): Milk quality and subclinical mastitis detection through somatic cells counting . Acta Scientiarum , 23, (4) ,p. 1065-1068.

Mackie, D.P. and Rodgers, S.P.(1986): Mastitis and cell content in milk from Scottish blackface ewes. Vet. Rec., 118: 20-21.

Maltz, E., S. Devir, J.H.M. Metz, H. Hogeveen. (1997): The body weight of the dairy cow. I. Introductory study into body weight changes in dairy cows as a management aid. Livest. Prod. Sci. 48:175.

Mccann, S.M., M. Kimura, S. Karanth, W.H. Yu, C.A. Mastronardi, and V. Rettori. (2000): The mechanism of action of cytokines to control the release of hypothalamic and pituitary hormones in infection. Ann. N. Y. Acad. Sci. 917: 4.

Miller, R.H.(1982): Genetics of resistance to mastitis. In: CL. de Cuenca (Editor), Proc. II World Congress on Genetics Applied to Livestock Production, 4-8 October, Madrid, Spain, pp. 186-198.

Miller, R.H., Clay, J.S., Norman, H.D., (2001): Relationship of somatic cell sore with fertility measures. J. Dairy sci. 84, 2543–2548.

Miller, R.H., Norman, H.D., Wiggans, G.R., Wright, J.R., (2004): Relationship of test day Somatic cell score with test day and lactation milk yields. J. Dairy sci. 87, 2299–2306.

Monardes, H.G. (1984): Genetic and phenotypic parameters of lactation cell count in different lactation of Holstein cows Thesis (Doctoral in Animal Science) - McDonald Campus of McGill Univ., Ste. Anne de Bellevue, Quebec, Canada.

Monardes, H.G.(1994): Somatic cell counting and genetic improvement of resistance to mastitis. In: Symposium of International Ruminant Production , 31, p. 1-19.

Moore, D.A., J.S. Cullor, R.H. Bondurant, and W.M. Sischo. (1991): Preliminary field evidence for the association of clinical mastitis with altered interestrus intervals in dairy cattle. Theriogenology 36: 257.

Moore, D.A., M.W. Overton, R.C. Chebel, M.L. Truscott, and R.H. BonDurant. (2005): Evaluation of factors that affect embryonic loss in dairy cattle. J. Am. Vet. Med. Assoc. 226: 1112.

Mrode, R.A., Swanson, G.J.T., (1996): Genetic and statistical properties of somatic cell count and its suitability as an indirect means of reducing the incidence of mastitis in dairy cattle. Anim. Breed. Abstr. 64, 847–857.

Nakajima, Y., O. Mikami, M. Yoshioka, Y. Motoi, T. Ito, Y. Ishikawa, M. Fuse, K. Nakano, and K. Yasukawa. (1997): Elevated levels of tumor necrosis factor-b (TNF-b) and interleukin-6 (IL-6) activities in the sera and milk of cows with naturally occurring coliform mastitis. Res. Vet. Sci. 62: 297.

National Mastitis Council (NMC),(1999): Laboratory Handbook on Bovine Mastitis. National Mastitis Council, Madison, WI.

National Research Council (NRC, 2001): Nutrient Requirements of Dairy Cattle, Seventh revised edition. National Academy of Science, Washington, DC, USA, pp. 258–265.

Nickerson, S. C., W. E. Owens, and R. L. Boddie.(1995):Mastitis in dairy heifers: Initial studies on prevalence and control. J. Dairy Sci. 78:1607

Oliver, S. P., B. E. Gillespie, M. J. Lewis, T. L. Ingle, and H. H.

Dowlen. (1994):. Evaluation of chlorhexidine as a premilking teat disinfectant for the prevention of intramammary infections during lactation. J. Food Prot. 57:614618.

Oltenacu, P.A., Frick, A., Lindhe', B., (1990): Epidemiological study of several clinical diseases, reproductive performance and culling in primiparous Swedish cattle. Prev. Vet. Med. 9, 59-74.

Padmanabhan, V., C. Keech, and E.M. Convey. (1983): Cortisol inhibits and adrenocorticotropin has no effect on luteinizing hormone-releasing hormone-induced release of luteinizing hormone from bovine pituitary cells in vitro. Endocrinology 112:1782.

Pampfer, S.,Y.D. Wuu, I. Vanderheyden, and R. De Hertogh.(1994): Expression of tumor necrosis factor-b (TNF-b) receptors and selective effect of TNFb on the inner cell mass in mouse blastocyst. Endocrinology 134: 206.

Petroff, M.G., B.K. Petroff, and J.L. Pate. (2001): Mechanisms of cytokine-induced death of cultured bovine luteal cells. Reproduction 121:753.

Rekik B., N. Ajili, H. Belhani, A. Ben Gara, H. Rouissi (2008): Effect of somatic cell count on milk and protein yields and female fertility in Tunisian Holstein dairy cows.Livestock Science 116 (2008) 309–317

Riollet, C., P. Rainard, B. Poutrel. (2001): Cell subpopulations and cytokine expression in cow milk in response to chronic Staphylococcus aureus infection. J. Dairy Sci. 84: 1077.

Rupp, R., Boichard, D., Bertrand, C., Bazin, S., (2000): Bilan national des numérations cellulaires dans le lait des différentes races bovines laitières françaises. INRA Prod. Anim. 13 (4), 257–267.

Santos, J.E.P., R.L.A. Cerri, M.A. Ballou, G.E. Higginbotham, and J.H. Kirk. (2004): Effect of timing of first clinical mastitis occurrence on lactational and reproductive performance of Holstein dairy cows. Anim. Repro. Sci. 80:31.

Sapolsky RM, Romero LM,Munck AU. (2000): How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocrin Rev.;21:55–89.

Sartori, R., R. Sartor-Bergfelt, S.A. Mertens, J.N. Guenther, J.J. Parrish, and M.C. Wiltbank. (2002): Fertilization and Early Embryonic Development in Heifers and Lactating Cows in Summer and Lactating and Dry Cows in Winter. J. Dairy Sci. 85: 2803.

SAS User's Guide (1996) :SAS User Guide. SAS Inst. Inc., Cary, NC.

Schrick, F.N., M.E. Hockett, A.M. Saxton, M.J. Lewis, H.H. Dowlen, and S.P.Oliver. (2001): Influence of Subclinical Mastitis During Early Lactation on Reproductive Parameters. J. Dairy Sci.84:1407.

Sheldrake, R.F.(1983): Lactation stage, parity, and infection affecting somatic cells, electrical conductivity, and serum albumin in milk. J. Dairy Sci.,66, p.542.

Shook, G.E., Schutz, M.M.(1993): Selection on somatic cell score to improve resistance to mastitis in the United States. J. Dairy Sci. 77, 648–658.

Skarzynski, D.J., Y. Miyamoto, and K. Okuda. (2000): Production of prostaglandin F 2b by cultured bovine endometrial cells in response to tumor necrosis factor b: cell type specificity and intracellular mechanisms. Biol.R eprod. 62:1116.

Soto, P., R.P. Natzke, and P.J. Hansen. (2003):Actions of tumor necrosis factor-b on oocyte maturation and embryonic development in cattle. Am. J. Reprod. Immunol. 50:380 .

Stoebel, D.P., and G.P. Moberg. (1982):Effect of adrenocorticotropin and cortisol on luteinizing hormone surge and estrous behavior of cows. J. Dairy Sci. 65: 1016.

Suzuki C, Yoshioka K, Iwamura S, Hirose H.(2001): Endotoxin induces delayed ovulation following endocrine aberration during the proestrous phase in Holstein heifers. Domest Anim Endocrinol 2001;20:267–78.

Tilbrook AJ, Turner AI, Clarke IJ (2000): Effects of stress on reproduction in non-rodent mammals: the role of glucocorticoids and sex differences. Rev Reprod 2000;5:105–13.

Torres, C.L.A. (1985): Mamite bovina. EMPASC.

Waller, K.P., I.G. Colditz, K. Östensson. (2003): Cytokines in mammary lymph and milk during endotoxin-induced bovine mastitis. Res. Vet. Sci. 74:31.

Wenz, J.R., G.M. Barrington, F.B. Garry, K.D. McSweeney, R.P. Dinsmore, G. Goodell, and R.J. Callan. (2001): Bacteremia associated with naturally occurring acute coliform mastitis dairy cows. J. Am. Vet. Med. Assoc. 219:976.

Windig, J.J., Calus, M.P.L., Beerda, B., Veerkamp, R.F., (2006): Genetic correlation between milk production and health and fertility

Wuu, Y.D.,S. Pampfer, P. Becquet, I. Vanderheyden, K.H. Lee, and R. De Hertogh. (1999): Tumor necrosis factor b decreases the viability of mouse blastocyst in vitro and in vivo. Biol. Reprod. 60: 479.

Table 2: Interrelationship between subclinical mastitis and some fertility indices in Holstein-Friesian cows (M± SE)

Reprod. Indices / SCC (X 1000/ml) & preg.status		Avenge SCC (X 1000/ml)	Parity	Nº. of inseminations per conception	Interval to 1st insemination	Days Open	Pregnant cows within 305 DIM (%= SE)
LNP	< 200	65.44 ± 1.86 ^{a}	1.96 ± 0.05	2.39 ± 0.06 ^{a}	83.93 ± 2.04 ^{a}	143.46 ± 3.82 ^{a}	(398/533) (74.76 ± 1.88) ^{a}
	≥200	886.79 ± 72.63 ^{b}	2.62 ± 0.10	3.92 ± 0.18 ^{b}	89.50 ± 3.66 ^{ab}	227.07 ± 8.55 ^{b}	(160/248) (64.51 ± 3.04) ^{a}
Over all mean		261.35 ± 12.51	2.12 ±0.01	2.76± 0.03	85.26± 1.68	163.40±1.27	(558/781) (71.44± 1.61)
LP	<200	62.32 ± 1.93 ^{a}	2.04 ± 0.05	4.22 ± 0.10 ^{a}	93.69 ± 2.33 ^{b}	242.93 ± 5.22 ^{b}	(349/596) (58.55 ± 2.01) ^{a}
	≥ 200	1058.5 ± 138.67 ^{b}	2.11 ± 0.09	2.38 ± 0.11 ^{a}	89.52 ± 3.16 ^{ab}	153.67± 6.44 ^{a}	(176/186) (94.62 ± 1.65) ^{a}
Overall mean		301.48±36.52	2.06 ±0.04	3.78 ± 0.05	92.96 ± 1.74	221.62 ± 3.81	(525/782) (67.13 ± 1.68)
culled cows	< 200	64.55 ± 3.72 ^{a}	2.67 ± 0.14	5.68 ± 0.27 ^{d}	98.84 ± 5.08 ^{bc}	----	0/ 150 (0.00±0.00)
	≥200	883.65 ± 113.75 ^{b}	2.75 ± 0.18	6.82± 0.37 ^{e}	94.39 ± 6.61 ^{ab}	----	0/44 (0.00±0.00)
	Over all	315.63± 43.91	2.70 ± 0.11	6.03 ± 0.22	97.48 ± 4.06	----	0 / 194 (0.00±0.00)

Preg.= pregnancy B = before the 1st insemination A = from the 1st insemination to the fertile one DIM =days in milking

LNP = lactating non pregnant LP = lactating pregnant

Means within the same column with different alphabetical are significantly different at $p < 0.05$

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Table 3: Effect of degree and establishment of subclinical mastitis (SCC) on the subsequent fertility indices in Holstein –Friesian cows (Means ± SE)

Reprod. Indices / SCC(1000/ml)-(N)			Average SCC (X 1000/ml)	Interval to 1st insemination	NO. of inseminations per conception	Days open	Pregnant cows within 305 DIM (% ± SE)
Gp.1 (< 200)	(n=533)		73.75 ± 2.24 [a]	92.89 ± 2.44 [b]	2.91 ± 0.11 [a]	226.17 ± 5.45 [a]	398 / 533 (74.67±1.88) [b]
Gp.2 (≥200 - <400)		B (n=50)	271.65 ± 7.15 [b]	113.46 ± 8.72 [c]	3.57 ± 0.37 [a]	228.53 ± 14.26 [a]	38/50 (76.00 ± 6.10) [b]
		A (n=88)	280.80 ± 6.25 [b]	80.98 ± 4.21 [ab]	5.46 ± 0.21 [b]	284.32 ± 7.86 [b]	58/88 (65.9 ± 5.08) [b]
Gp.3 (≥400 - < 1000)		B (n=30)	680.00 ± 32.75 [d]	122.33 ± 12.99 [c]	2.60 ± 0.33 [a]	198.00 ± 25.25 [a]	24/30 (80.00 ± 7.42) [b]
		A (n=48)	578.52 ± 24.84 [c]	80.04 ± 6.33 [ab]	5.95 ± 0.37 [b]	355.25 ± 18.95 [b]	17/48 (35.41 ± 6.97) [a]
Gp.4 (≥ 1000)		B (n=16)	2281.06 ± 356.51 [e]	92.12 ± 10.34 [b]	2.94 ± 0.53 [a]	180.37 ± 34.07 [a]	13/16 (81.25± 10.07) [b]
		A (n=16)	2081.12 ± 252.42 [e]	64.37 ± 6.90 [a]	5.06 ± 0.56 [ab]	244.87 ± 26.08 [ab]	10/16 (62.5 ± 12.5) [ab]

B = before the 1st insemination A = from the 1st insemination to the fertile one DIM =days in milking
Means within the same column with different alphabetical are significantly different at $p < 0.05$

Table 4: Fertility indices of Holstein-Friesian cows in relation to parity and establishment of subclinical Mastitis (M ± SE)

Reprod. Indices / Parity - SCC(1000/ml) -Time			Average SCC (X 1000/ml)	Interval to 1st insemination	N0. of inseminations per conception	Days open	Pregnant cows within 305 DIM (% ± SE)
Primiparous	< 200 SCC	(n=238)	62.79 ± 2.92^{a}	99.86 ± 4.08^{c}	4.21 ± 0.19^{c}	252.3 ± 8.79^{b}	155/238 (65.12± 3.09)^{a}
	≥ 200 SCC	B (n=28)	692.6 ± 141.6^{e}	140.46 ± 16.7^{d}	3.78 ± 0.53^{bc}	259.75 ± 25.1^{bc}	19/28 (67.85 ± 8.98)^{ab}
		A (n=58)	501.2 ± 84.6^{e}	87.86 ± 6.18b^{c}	5.37 ± 0.24^{d}	313.63 ± 11.89^{c}	31/58 (53.44 ± 6.60)^{a}
	Over all mean	(n=324)	195.26 ±23.10^{c}	101.22± 3.52^{c}	4.38 ± 0.15 c	263.92 ± 7.23^{d}	[illegible]/324 (63.27 ± 2.68)
Pleuriparous	< 200 SCC	(n=295)	82.73± 3.21^{b}	87.27 ± 2.91^{b}	3.66 ± 0.13^{b}	205.09 ± 6.6^{a}	[illegible]/295 (82.37 ±2.22)^{b}
	≥ 200 SCC	B (n=68)	753.3 ± 119.3^{e}	102.0 ± 6.03 c	2.94 ± 0.26^{a}	193.07 ± 13.5^{a}	56/68 (82.35 ± 4.65)^{b}
		A (n=94)	593.1 ± 62.92^{e}	73.12 ± 3.4^{a}	5.7 ± 0.25^{d}	293.9 ± 11.6^{c}	54/94 (57.44 ± 5.12)^{a}
	Over all mean	(N=457)	287.47 ± 25.61^{d}	86.55 ± 2.23^{b}	3.97 ± 0.11^{bc}	221.85 ± 5.55^{a}	353/457 (77.24 ± 1.96)

B = before the 1st insemination A = from the 1st insemination to the fertile one DIM =days in milking
Means within the same column with different alphabetical are significantly different at $p < 0.05$

تاثير إلتهاب الضرع تحت السريرى على بعض المقاييس التناسلية فى الأبقار الهولستين – فريزيان الحلابة

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أجريت هذه الدراسة على عدد ١٧٥٧بقرة هولستين – فريزيان بمزرعة الشركة العالمية للإنتاج الحيوانى – ابورواش – الجيزة خلال الفترة من نوفمبر ٢٠٠٧حتى اكتوبر ٢٠٠٨، تم تقدير عدد الخلايا الجسدية فى لبن هذه الأبقار كوسيلة لمعرفة تأثر هذه الحيوانات بإلتهاب الضرع تحت السريرى من عدمه على مدى ثلاث شهور متتالية ، أخذت عينات لبن للفحص البكتيرى ، تركزت مقاييس الكفاءة التناسلية على عدد الأيام من الولادة حتى أول تلقيحه ، عدد التلقيحات اللازمة للعشار ، وعند الأيام من الولادة حتى التلقيحة المخصبة وكذا نسبة العشار فى هذه الحيوانات خلال ٣٠٠ يوم حليب ، هذا بالإضافة إلى نسبة الإستبعاد من هذه الحيوانات بسبب المشاكل التناسلية ، رتبت بيانات هذه الحيوانات حسب الحالة التناسلية لها سواء كانت عشار أو غير عشار وكذا حسب الموسم الإنتاجى لها وتم اجراء التحليل الإحصائى بعد ذلك و لقد تبين من هذه الدراسة ما يلى :-

بلغت نسبة الحيوانات التى تعانى من إلتهاب ضرع تحت سريرى ٣١.٧٥ و٧٩ ٢٣% فى الابقار الحلابة وغير عشار والحلابة العشار على التوالى مع العلم أن نسبة ٧٥% فقط من هذه الحيوانات المصابة أظهرت إصابات بكتيرية تم عزلها وتصنيفها ، كان معظم هذه المعزولات تنتمى إلى عائلتى الميكروب العنقودى والسبحى .

لوحظ من هذه الدراسة أن لإلتهاب الضرع تحت السريرى تأثيرات سلبية عالية فى الخصوبة تلخصت فى زيادة الفترة من الولادة حتى أول تلقيح خصوصا عند إصابة هذه الحيوانات قبل هذه الفترة .

لوحظ أيضا من هذه الدراسة أن إصابة الأبقار بإلتهاب الضرع تحت السريرى بعد أول تلقيحه يؤدى إلى زيادة معنوية فى عدد التلقيحات اللازمة للعشار وكذا زيادة معنوية فى الأيام من الولادة حتى التلقيحة المخصبة .

الخلاصة :- يستخلص من هذه الدراسة أن لإلتهاب الضرع تحت السريرى أثار بالغة السوء على الكفاءة التناسلية لهذه الأبقار

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