Ann. Anim. Sci., Vol. 13, No. 3 (2013) 435–448, DOI: 10.2478/aoas-2013-0030

A CONNECTION BETWEEN MASTITIS DURING EARLY LACTATION AND REPRODUCTIVE PERFORMANCE OF DAIRY COWS – A REVIEW*

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Abstract

The objective of this study was to present a relationship between mastitis in the post-calving period and fertility traits of dairy cows. The threshold of 200,000 cells/ml for somatic cell count is used as a reference for healthy quarters of the cow's udder. The genetic correlation between mastitis and somatic cell count is strong (from 0.7 to 0.8). Although heritability for fertility traits is low (from 0.01 to 0.02), the genetic standard deviation for mastitis varies from 1.2 to 7.0 percentage units, suggesting that genetic gain can be achieved by selecting for mastitis. Results of this study suggest that mastitis in the postpartum period can have a markedly negative impact on the reproductive performance of dairy cows. The synergistic effect of common conditions (somatic cell count, body condition score and lameness score) or other factors (e.g. heat stress, fertility management, the presence of repeat breeders) also lowers fertility of cows. Production of milk with fewer than 100,000 cells/ml leads to improved health and fertility in the cows.

Key words: ruminants, dairy cows, somatic cell count, mastitis, fertility

Effects of different diseases on reproduction of dairy cows have been described by many authors (Ahmadzadeh et al., 2009; Fourichon et al., 2000; Peter et al., 2009; Vacek et al., 2007). Their studies showed that milk fever, displaced abomasum and mastitis have no effect on reproduction. Clinical ketosis, dystocia and retained placenta were slightly associated with reproduction, while associations of locomotor disorders and metritis with reproduction were moderate; however, the strongest influence on reproduction was found for cystic ovaries and abortion (Fourichon et al., 2000). Significant effects of retained placenta, metritis and ovarian cysts on all reproduction parameters (days to first service, open days, number of services) were found by Vacek et al. (2007). Investigations showed that genetic correlations between health and fertility traits were low or moderate (Heringstad et al., 2009). The

*This study was financed from statutory activity, project No. 508.557.00.0.

strongest genetic correlation was observed between ketosis and days from calving to the first insemination (0.29), whereas genetic correlations between clinical mastitis and non-return rate within 56 days after the first insemination and between calving to the first insemination and non-return rate within 56 days after the first insemination were nil. In general, selection against disease is expected to slightly improve fertility (shorter period from calving to the first insemination and higher non-return rate within 56 days after the first insemination) as a correlated response and vice versa. Mastitis and lameness had also a significant effect on days open and the number of days from the first artificial insemination per conception, but milk fever occurrence was significantly associated with the period from calving to the first artificial insemination.

However, during the last 50 years there has been significant progress in the potential for intervention in reproduction of cattle (Berry et al., 2003; Dal Zotto et al., 2007; Malinowski and Gajewski, 2010; Peter et al., 2009). During this time there has been improvement in animal health and fertility by promoting good management rather than by relying on the widespread use of exogenous hormones. High capacity reproductive cows in the postpartum period require the prevention of metabolic diseases in the periparturient period and after calving should be provided high dry-matter intake to meet the demands of milk production. In farms during conversion to organic farming calving interval did not change; however, age at first calving increased by 1.75 months (Nauta et al., 2006).

At present, however, the prevailing trend is associated with control of mastitis, especially during early lactation. Clinical mastitis is a common and costly disease in dairy cattle farms not only because it reduces the production and quality of milk, but also due to increased involuntary culling rates and discarded milk (Barker et al., 1998; Dal Zotto et al., 2007; Olson et al., 2011; Suriyasathaporn et al., 2000). Mastitis costs are associated mainly with milk loss, increased culling rates and treatment costs. The highest incidence of clinical mastitis is observed in early lactation and has a negative impact on the reproductive performance of dairy cows (Barker et al., 1998; Buch et al., 2011; Chebel et al., 2004; Gunay and Gunay, 2008; Hansen et al., 2004; Heringstad and Larsgard, 2010). The number of days to the first artificial insemination was greater ($P<0.01$) for cows with clinical mastitis before the first artificial insemination (93.6±5.6 days) than for all the other cows (for cows with clinical mastitis between the first artificial insemination and pregnancy, and for cows with clinical mastitis after confirmed pregnancy and for control cows (71.0±2.2 days) (Barker et al., 1998). Moreover, cows experiencing mastitis between the first artificial insemination and pregnancy confirmation had a greater number of services per conception and days not pregnant when compared with cows without mastitis. It is of interest to indicate that cows with clinical mastitis or subclinical mastitis before the first artificial insemination also affected reproductive performance by increasing days in milk at first service, days not pregnant, and services per conception (Schrick et al., 2001).

In high production herds of cows increasing problems with fertility and health are being observed (Berry et al., 2003). High-yielding cows often experience prolonged periods of negative energy balance (NEB) resulting in an increased use of

body reserves, which can have detrimental effects on fertility and health (Olson et al., 2011; Peter et al., 2009; Pryce et al., 2004). Cows in the negative energy balance may preferentially save nutrients typically required for reproduction by limiting the number of ovarian follicles, growth and maximum dimensions of the dominant follicle, as well as delaying the first ovulation, interfering with ovulation, hindering the expression of oestrus, and lowering plasma progesterone concentrations (Peter et al., 2009). It is assumed that crossbreds have fewer health and lameness problems than purebred Holsteins; however, Olson et al. (2011) found no differences between the genetic groups (Holstein × Jersey, Jersey × Holstein, and Holstein) in terms of the incidence of ketosis, displaced abomasum and pregnancy. Cows with higher cumulative energy balance estimates of expenditures at week 15 of lactation (less severe negative energy balance) tended to have higher odds of becoming pregnant. Fertility of cows is important regardless of the length of lactation. At any scheduled time the cow should be capable of conception. Metabolic load high performance cows should develop nutrition strategies important for new genotypes (Pryce et al., 2004). More recent data show that fertility is higher in organic herds of cows in comparison with conventional herds; however, somatic cell count was found to be higher in the organic system (Sundberg et al., 2009).

The objective of this study was therefore to present a relationship between mastitis in the post-calving period and fertility traits of dairy cows.

Genetic association between mastitis and fertility, and genetic relationship between fertility traits

Somatic cell count (SCC) is commonly used as an indicator trait for mastitis, which in many countries is used as a criterion for indirect selection for improving mastitis resistance (Berry et al., 2011; Carlén et al., 2004; De Vliegher et al., 2005; Heringstad et al., 2006 a; Heringstad et al., 2006 b). The heritability estimates for mastitis show a variation across studies (Carlén et al., 2004; Heringstad et al., 2006 b; Negussie et al., 2008). Heritability rates of mastitis are low: 0.03 for the first lactation and 0.01 for the second and third lactations (Carlén et al., 2004). Heringstad et al. (2006 b) assessed the heritability of mastitis and services per conception for first lactation Norwegian Red cows, amounting to 0.08 and 0.03, respectively. The genetic correlation between these traits was 0.21. For lactation average somatic cell score (LSCS), heritability rates were considerably higher than those of mastitis, but here also estimates decreased slightly with increasing parity, from 0.14 to 0.10. Dal Zotto et al. (2007) estimated heritability for somatic cell score (SCS) at 0.06. Selection of cows to increase milk production was related to an adverse response for lactation average somatic cell score; however, selection against mastitis resulted in a favourable response correlated selection (Heringstad et al., 2008). Estimated genetic correlations between mastitis and lactation average somatic cell score were about 0.7 to 0.8, with the highest estimate found for the third lactation. Similarly, a strong correlation was found by Buch et al. (2011) between mastitis and somatic cell score (0.73±0.06). Heritability of somatic cell score was 0.03 for mastitic cows and 0.08 for healthy cows, while the genetic correlation between these traits was 0.78 (Heringstad et al., 2006 b). Heritability of lactation average somatic cell count

was 0.11. Negussie et al. (2008) estimated heritability in the first lactation for mastitis 1 (occurrence from 7 to 30 days after calving), and mastitis 2 (occurrence from 31 to 300 days after calving) at 0.03 and 0.02, respectively. According to those authors heritability of test-day somatic cell score during the first lactation ranged from 0.07 to 0.12. Recent estimates of the genetic correlation between mastitis and somatic cell count ranged between 0.53 and 0.77 (Carlén et al., 2004). These variability coefficients for heritability reflected variation of many factors, such as the person undertaking the recording (i.e. the farmer or veterinarian) and how they interpret the clinical signs, the completeness of data recording (i.e. some observations not recorded), as well as the pathogen and the environment, including exposure, which may influence the expression of an animal's genotype. The genetic standard deviation for mastitis varies from 1.2 to 7.0 percentage units, suggesting that genetic gain can be achieved by selecting for clinical mastitis.

Cows genetically higher, wider, deeper and more angular with high pins may have lower values of genetic deviation for pregnancy rate both in the first service and 63 days after the start of breeding. These animals also tend to require more services (Berry et al., 2004). Similarly, cows with genetically tighter fore udders and higher, shallower udders with a stronger support also have a lower pregnancy rate to first service, and require more services. According to Berry et al. (2004), the genetic correlation between body condition score (BCS) and interval from calving to the first artificial insemination, number of services, pregnancy rate to first service, pregnant 63 days after the start of breeding, and somatic cell count have been calculated at –0.53, 0.13, –0.14, 0.35 and –0.57, respectively. Previous results reported by those authors were similar and indicate that body condition score at different stages of lactation correlated favourably with improved fertility, while genetic correlations between body condition score and pregnant 63 days after the start of breeding ranged from 0.29 to 0.42 (Berry et al., 2003). The genetic correlations of calving interval with yield and most linear type traits were positive, whereas the correlation between calving interval and body condition score was negative, amounting to −0.35 (Dal Zotto et al., 2007). Heritability for fertility traits is low and it is calculated to be 0.01 to 0.02 for interval to first service, first service to conception interval, pregnant to first service, number of services per cow and pregnant 63 days after the start of the breeding season. Body condition score was not significantly related to mastitis, but heavier cows had a greater probability of mastitis (Berry et al., 2007). Body condition score and its relationship with fertility in first parity Italian Brown Swiss cows were analysed in another study (Dal Zotto et al., 2007). The genetic correlation of calving interval or body condition score with somatic cell score was moderately low, but favourable (0.19 and −0.26, respectively). Heritability of the interval between calving and the first ovulation was 0.20, while heritability of conception rate was 0.05 (Schneider et al., 2005). No genetic correlation was found between those traits. Genetically, days from calving to the first insemination was more strongly correlated with clinical mastitis than the number of inseminations. Probably the reason may be that these traits primarily are observed in the early part of the lactation period when the cows are likely to be in negative energy balance, whereas number of inseminations is usually recorded when the cows start to regain body resources (Buch et

al., 2011). The genetic correlation between number of inseminations and days from calving to the first insemination was low (–0.002±0.05), which indicates that these traits are not affected by the same gene. The h^2 estimate for the mean natural logarithm somatic cell count (ln SCC) was intermediate (0.14), whereas that for calving interval was low (0.04). Survival was genetically negatively correlated with calving interval (–0.33), also with mean natural logarithm somatic cell count (–0.13), and with the slope of natural logarithm somatic cell count (–0.13). This means that the cows with a high level of somatic cell count and the increasing somatic cell count in late lactation may more likely be culled from a herd in comparison to other cows (Haile-Mariam et al., 2003).

Risk factors for clinical mastitis in postpartum period and its effect on reproductive performance

Variables affecting changes of natural logarithm somatic cell count are explained in 50.2%. Infection status was the predominant factor explaining variation in natural logarithm somatic cell count. Other factors that explained the variation in natural logarithm somatic cell count are as follows: herd, cow nested within herd, month of sampling, quarter, stage of lactation, parity, interaction between stage of lactation and parity, and clinical mastitis, but cow and stage of lactation explained >5% of the variation in natural logarithm somatic cell count (Schepers et al., 1997). According to these authors, a fixed somatic cell count threshold of 200,000 cells/ml should be adopted as a reference and its sensitivity can be slightly improved, taking into account parity and stage of lactation. The status of healthy quarters of the udder was similarly defined by Suriyasathaporn et al. (2000). The authors assume an udder-inflammation free state to be the state in which a cow had a somatic cell count of $<200 \times 10^3$ cells/ml. An udder-inflammation state was defined as either somatic cell count $\geq 200 \times 10^3$ cells/ml or clinical mastitis. Lavon et al. (2011) adopted the somatic cell count threshold of 150,000 cells/ml of milk to distinguish between uninfected cows and cows with mastitis. However, too low numbers of somatic cells in bulk milk during the non-infection state (somatic cell count <200,000 cells/ml) were negatively associated with an increased risk of clinical mastitis as udder inflammatory responses. A majority of infections was caused by environmental mastitis pathogens, primarily coliforms, coagulase-negative staphylococci (CNS), environmental streptococci (Barker et al., 1998; Hertl et al., 2010; Santos et al., 2004), yeast and algae of *Prothotheca* genus (Dworecka-Kaszak et al., 2012; Jagielski et al., 2011; Wawron et al., 2010). Many of these infections originate during the non-lactating period and result in clinical mastitis during the first 30 to 60 days after calving (Barker et al., 1998) or in the first 90 days in milking (DIM) (Schrick et al., 2001). Some previous studies examined gram-positive versus gram-negative mastitis and reproduction (Barker et al., 1998; Santos et al., 2004; Schrick et al., 2001), while some examined mastitis occurring in the intervals before artificial insemination, artificial insemination to pregnancy diagnosis, and after pregnancy diagnosis (Baker et al., 1998; Santos et al., 2004) or other intervals (Ahmadzadeh et al., 2009). Hertl et al. (2010) analysed the type of mastitis and timing of mastitis in 6 weekly intervals before artificial insemination and 6 weekly intervals after artificial insemination. The

results suggest that additional information on clinical mastitis (e.g. its timing with respect to artificial insemination and whether the causative agent is gram-positive, gram-negative, or other) is beneficial to farmers in determining why some cows have trouble conceiving. It was found that the gram-negative and gram-positive pathogens have similar mechanisms leading to reproductive failure during early lactation (Barker et al., 1998; Hertl et al., 2010).

Clinical mastitis during early lactation markedly influenced reproductive performance of lactating Jersey cows (Barker et al., 1998). No differences in measures of reproductive performance were detected between mastitis caused by gram-positive or gram-negative pathogens. Cows with mastitis occurring at different times during early lactation (before the first artificial insemination, between the first artificial insemination and pregnancy, after confirmed pregnancy) and control cows with no clinical mastitis were examined for fertility traits. It appeared that the number of days to first artificial insemination was greater for cows with clinical mastitis before the first artificial insemination than for all the other cows, amounting to 93.6±5.6 days and 71.0±2.2 days, respectively. Significant differences were found in the number of artificial inseminations per conception for cows with clinical mastitis after the first artificial insemination than before the first artificial insemination and cows with no clinical mastitis, or cows with clinical mastitis after confirmed pregnancy, amounting to 2.9, 1.6, and 1.7, respectively. The number of days from calving to conception for cows with clinical mastitis before the first artificial insemination and for cows with clinical mastitis after the first artificial insemination was greater (113.7±10.8 days and 136.6±13.3 days, respectively) than this parameter for control cows and cows that developed clinical mastitis after confirmed pregnancy (92.1±4.6 days). Similarly, negative associations were found between mastitis in early lactation and cows' reproductive performance (Gunay and Gunay, 2008). The occurrence of mastitis delayed days from calving to the first insemination, increased the calving to conception interval and elevated the services per conception. Klaas et al. (2004) claimed that during the postpartum period 11.0% cows developed clinical mastitis, and 33.6% subclinical mastitis in the period up to 42 days in milking. In their study mean days from calving to the first insemination were 81.1±1.3 days and the following factors have a significant impact on the length of the days from calving to the first insemination: herd × season, parity, milk yield, reproductive and metabolic disorders. Cows with subclinical mastitis had prolonged days from calving to the first insemination by approximately 12 days, but clinical mastitis had no effect. The authors explained the fact that cows with clinical mastitis were usually treated locally and/or parenterally with specific antibiotics, whereas cows with subclinical mastitis in general were not treated. The first report on the harmful impact of clinical mastitis occurrence before the first service on reproductive performance under tropical conditions was presented by Nava-Trujillo et al. (2010). The mean days from calving to the first insemination in cows with clinical mastitis was 136.31 days and 98.53 days in control cows. Similarly, the open period (days to conception) was significantly prolonged in mastitis cows when compared with healthy cows, amounting to 187.21 and 143.95 days, respectively. The number of services per conception did not differ in both groups of cows (2.21 and 2.35), similarly to the first service conception rate (56.10 and 49.72).

The clinical mastitis had a greater negative impact on reproductive performance in primiparous cows than multiparous cows. Heifers, which have an increased number of somatic cells measured between 5 and 14 days in milk were at an increased risk of being culled during the first lactation (De Vliegher et al., 2005). Part of the effect was associated with the consequential elevation of test-day somatic cell count and suppression of milk yield at test-day (kg) measured after 14 days in milking. In general, however, heifers in high-yielding herds are protected against culling, even at increased somatic cell count between 5 and 14 days in milking. Many authors, however, noted that clinical mastitis increases involuntary culling rates (Rougoor et al., 1999; Santos et al., 2004; Schrick et al., 2001).

Connection of mastitis with occurrence of oestrus, follicular growth and ovulation

In cows, clinical mastitis was experimentally induced before ovulation (on day 8; oestrus = d 0) by *Streptococcus uberis* to determine the effect of clinical mastitis on endocrine function, follicular growth, or ovulation (Hockett et al., 2005). The results suggest that clinical mastitis has multiple actions that hinder normal endocrine function. Luteinizing hormone pulsatility was significantly reduced, the production of estradiol-17β decreased and some animals had a blocked expression of oestrus. This explains the lack of increase of luteinizing hormone in cows with acute clinical mastitis and why ovulation was blocked. The results obtained by Huszenicza et al. (2005) may explain clinical mastitis related ovarian abnormalities in the postpartum period in dairy cows. In that study milk samples were collected three times weekly for 95–100 days for progesterone assay. Individual profiles were used to monitor ovarian cyclicity. Individual profiles do not show all mastitis induced changes that cause ovarian alterations in the first two weeks after calving. However, if mastitis occurred later, i.e. between days 15 and 28 after calving, then it delayed the first postpartum ovulation or lengthened the follicular phase and induced premature luteolysis. The authors suggested, however, that the results of their study did not clarify the influence of clinical mastitis on the pregnancy rate or calving-to-conception interval. Subclinical long-term mastitis and short-term clinical mastitis in early lactation induced delayed ovulation in about 30% of the cows (Lavon et al., 2010). It is associated with low estradiol concentrations, and then low or delayed luteinizing hormone secretion, indicating that mastitis directly affects follicular functioning. Nguyen et al. (2011) suggested that cows with high somatic cell count (from 200,000 to 500,000 cells/ml) had a significantly higher incidence of a prolonged luteal phase (abnormal resumption of ovarian cyclicity postpartum) than cows with a somatic cell count of 50,000 to 100,000 cells/ml. High somatic cell count in the postpartum period leads to reduced reproductive performance. The increase in the number of services per conception caused by mastitis suggests that mastitis is associated with either anovulation at oestrus, fertilization failure, or embryonic mortality (Hansen et al., 2004). Observations of cows with mastitis suggest that an activation of inflammatory or immune responses external to the reproductive tract can lead to embryonic mortality. According to Hansen et al. (2004), invasion of the mammary gland leads to the release of lipopolysaccharide, proteoglycans and other molecules of bacterial

origin that activate inflammatory and immune responses. This results in an increased cytokine synthesis from the mammary gland, lymph nodes draining the mammary glands. Certain cytokines are directly inhibitory to oocyte and embryonic function.

Relationship between mastitis, somatic cell count and fertility traits

The results obtained for test-day somatic cell count through lactation showed the presence of test-day SCC greater than 250,000 cells/ml with 21, 16, and 35% of early, mid, and late lactation stages having at least 1 test-day somatic cell count greater than 250,000 cells/ml (Berry et al., 2007). However, the incidence of clinical mastitis was greatest in early lactation (10%) and lowest in mid-lactation (3%), while it was intermediate in late lactation (8%). In another study somatic cell count levels above 400,000 cells/ml indicated mastitis, where 10% of cows were affected (König et al., 2006). Cows with chronic clinical mastitis throughout lactation, which have in the first test-day somatic cell count after the insemination above 400,000 cells/ml, showed about 4% lower pregnancy rates when compared with cows in somatic cell count classes below 150,000 cells/ml. Lavon et al. (2011) evaluated the impact of mastitis (determined by the pattern and level of somatic cell count around the first artificial insemination) on conception rate. The threshold of somatic cell count in uninfected cows was established to be below 150,000 cells/ml on all 3 monthly test days around the first artificial insemination. Mastitis is associated with a significant reduction in the probability of conception. The degree of the reduction of conception rate is related to clinical mastitis or subclinical mastitis to the level of somatic cell count elevation in response to different bacteria, and to its exact timing of elevation relative to artificial insemination. In Jersey cows no significant relation was found for somatic cell score before the first artificial insemination to the rate of non-return to oestrus by 70 days after the first service. In contrast, in Holstein cows relatively small linear regressions of non-return to oestrus by 70 days after the first service were found for preceding test-day somatic cell score from 2 to 3 weeks before insemination (Miller et al., 2001). Polish Holstein-Friesian cows were assessed for an association between somatic cell score and two fertility traits: non-return rate within 56 days after the first insemination and days from calving to the first insemination (Morek-Kopeć et al., 2009). It was found that in the beginning days from calving to the first insemination increased with somatic cell score, but later, with higher values of somatic cell score, days from calving to the first insemination slightly declined. This indicates that high somatic cell score might delay the first insemination. In another study the relationship between somatic cell count and fertility was assessed in Polish Holstein-Friesian cows using records from the first 7 test-days (months) after calving (Skrzypek et al., 2007). The analysed fertility traits included the number of artificial insemination services per confirmed pregnancy and the calving to conception interval. The results showed that the lowest values of both parameters were found in cows in which somatic cell count on the test-day preceding the first artificial insemination service was below 50,000 and between 201,000–400,000 cells/ml of milk, whereas they were highest in cows in which somatic cell count exceeded 1,000,000 cells/ml. The first artificial insemination conception rate was 34.0%, and

75% of the cows had their first artificial insemination within 100 days after calving (Yusuf et al., 2011). Cows, which became pregnant in an earlier stage of lactation, showed better reproductive performance in subsequent seasons. In Chilean dairy cattle subclinical mastitis measured by natural logarithm somatic cell count ≥4.5, had a significant impact on reproductive performance (Pinedo et al., 2009). This resulted in increased days from calving to the first insemination, calving to conception interval and services per conception values. High natural logarithm somatic cell count in the proximity of first breedings had a detrimental effect on conception. The odds of occurrence of abortion for cows with high natural logarithm somatic cell count during the first 90 days of gestation were increased by 1.22 times when compared with unaffected cows. Similarly, it was found that in the Tunisian Holstein population the test-day somatic cell score levels were high in the first three lactations and this could be due to high mastitis (sub-clinical and clinical) infection rates (Rekik et al., 2008). These infections reduced not only milk and protein yields, but also extended days from calving to the first insemination (mean interval = 94.9 days) and calving to conception interval (mean interval = 161 days), mainly in the first parity of cows. The first service would be delayed by approximately 2 days for each unit increase in test-day somatic cell scores recorded before the first service has occurred.

Synergistic effect of some factors and association of other factors with reduced fertility

In recent years many authors investigated the synergistic effect of common conditions (somatic cell count, body condition score and lameness score) or other factors (e.g. heat stress, fertility management, the presence of repeat breeder) influencing ovarian follicular growth and ovulation in dairy cows (Chebel et al., 2004; Gehrke and Zbylut, 2011; Morris et al., 2009; Peake et al., 2011; Rougoor et al., 1999; Yusuf et al., 2010; Yusuf et al., 2011). Morris et al. (2009) analysed cows with different conditions: healthy, high somatic cell counts only, lame only, lame and high somatic cell counts, lame and low body condition score. A cell count <100,000 cells/ml was classified as low somatic cell count and a count ≥100,000 cells/ml was classified as high. Body condition scores were determined using a 1–5 system incorporating 0.5 scores. Cows with a mean body condition score <1.5 were classified as low body condition score. The cows were also scored for lameness at the same time as body condition score, using a standardized 1–5 system. The authors suggested that three common conditions (high somatic cell count, low body condition score and lameness) reduce fertility in the dairy cow. Mechanisms for this phenomenon do not cause changes in the follicular growth rate or maximum follicle diameter. The results indicate that lame cows are less likely to ovulate, although the same is not true for animals with either high somatic cell score alone or low body condition score alone. The results showed for the first time a synergistic effect of lameness and high somatic cell count that further reduces the likelihood of ovulation. Peake et al. (2011) also monitored cows in the pre-breeding postpartum period for the presence of three production stressors, i.e. lameness, subclinical mastitis and body condition score loss. The authors concluded that combinations of these, often overlooked, conditions cause a delay in the interval from calving to the onset of the first luteal phase;

however, this does not affect fertility traits of cows; these subclinical conditions do have a synergistic detrimental effect on progesterone profiles.

Cows, particularly high-yielding, are sensitive to heat stress and cows exposed to heat stress (by at least 1 day of maximum temperature above 29ºC) prior to artificial insemination had a lower conception rate than cows not exposed to heat stress (Chebel et al., 2004). The effect of heat stress on decreased conception rate is associated with harmful effects of high ambient and body temperatures on follicle and oocyte competence. However, at 31 days after artificial insemination in case of pregnancy, previous or subsequent exposure to heat stress seems to have little effect on pregnancy maintenance, at least in the following 14 days. As a result of intensive metabolism in organism high-yielding cows produce free radicals. Oxidation processes lead to oxidative stress, which especially during the perinatal period can cause mastitis and other health disturbances in high-yielding cows (Jóźwik et al., 2012). In assessing the relationships between mastitis and fertility management it can be concluded that bulk somatic cell count and calving interval are general indicators of the farmers' attitude, and milk production for instance under 100×10^3 cells/ml leads to an enhanced health of animals (Rougoor et al., 1999). However, it appears that there is a negative relationship between milk production and fertility, and hence the resulting differences in fertility between herds of cows. There are cows, which do not show signs of reproductive disorders, which did not become pregnant after three inseminations and this is defined as a repeat breeder (Yusuf et al., 2010; Yusuf et al., 2011). In such cases very poor reproductive performance is recorded in cows despite repeated insemination. Risk factors for repeat breeding are the following: lower parity, abnormal resumption of postpartum ovarian cycles, and shorter days in milk at first artificial insemination.

Conclusion

Infection status was the predominant factor explaining variation of somatic cell counts in early lactation. A majority of infections was caused by environmental mastitis pathogens (e.g. *E. coli*, coagulase-negative staphylococci or *Strep. uberis*). The threshold of somatic cell count at 200,000 cells/ml was applied as a reference for healthy quarters of the udder; however, this threshold in uninfected cows is also assumed to be below 150,000 cells/ml on three monthly test days around the first artificial insemination. Estimated genetic correlations between mastitis and lactation average somatic cell scores are strong (from 0.7 to 0.8); hence, somatic cell count is commonly used as an indicator trait for mastitis, which in many countries is used as a criterion in indirect selection for improving mastitis resistance. Results of this study suggest that clinical mastitis in the postpartum period can have a markedly negative impact on the reproductive performance of dairy cows. Mastitis hinders and often blocks expression of oestrus, causes a prolonged luteal phase, interferes with endocrine function, follicular growth and ovulation. This is reflected in the extended period from calving to the first artificial insemination, and the period from calving to conception, an increase in the number of services to conception, reduced conception rates and lower pregnancy rates.

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Accepted for printing 20 XII 2012

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Związek mastitis we wczesnej laktacji z użytkowością reprodukcyjną krów mlecznych – artykuł przeglądowy

STRESZCZENIE

Celem opracowania jest przedstawienie związku między *mastitis* po wycieleniu z cechami płodności u krów mlecznych. Granica 200 000 komórek/ml jest referencyjną liczbą komórek somatycznych dla zdrowych ćwiartek wymienia krów. Genetyczna korelacja pomiędzy *mastitis* i liczbą komórek somatycznych jest silna (od 0,7 do 0,8). Chociaż odziedziczalność dla cech płodności jest niska (od 0,1 do 0,2), genetyczne odchylenie standardowe dla *mastitis* jest zmienne i wynosi od 1,2 do 7 jednostek procentowych wskazując na możliwość selekcji w kierunku *mastitis*. Dane piśmiennictwa wskazują, że *mastitis* w okresie powycieleniowym ma wyraźnie ujemny wpływ na użytkowość reprodukcyjną krów mlecznych. Synergiczny efekt wspólnych uwarunkowań (liczby komórek somatycznych, kondycji i kulawizny) lub innych czynników (np. stres termiczny, organizacja i zarządzanie płodnością, obecność tzw. krów powtarzających ruję) również obniża płodność krów. Produkcja mleka z liczbą komórek somatycznych poniżej 100 000/ml prowadzi do poprawy zdrowia i płodności krów.