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Relationship between clinical lameness and somatic cell counts, and fat and protein contents in the milk of dairy cows

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Summary

The aim of the study was to investigate the impact of clinical lameness on the somatic cell count (SCC) and percentage contents of fat and protein in cow's milk. Clinically lame cows (n = 12) were selected from a herd of 125 Polish Holstein-Friesian cows (located in the Wielkopolska region, Poland). The cows were housed in tie stalls with access to a pasture and a cow-yard throughout the year. Another group of 12 cows was included in the control on the basis of analogues (parity and stage of lactation). During the three weeks of the study one milk sample was collected from cows of both groups at weekly intervals (1 day after the assessment of locomotion in cows). Fat and protein contents in milk were determined with a MilkoScan device, SCC with a Fossomatic 90 apparatus. Somatic cell count values were transformed to a common logarithm (log SCC). Clinically lame cows and cows with a normal gait had a similar number of somatic cells (log SCC 5.58 and log SCC 5.63, respectively) and fat content (4.32% and 4.16%, respectively), but lower protein contents in milk (3.13% and 3.27%, respectively). It has been concluded that there were no statistically significant differences in SCC between clinically lame cows and healthy cows. The results of this study concerning of SCC, and fat and protein contents in milk of healthy and clinically lame cows should be treated as preliminary. Examinations should be repeated in large herds of cows taking into account interaction between the milk traits of cows (clinically lame and healthy) and parity, stage of lactation, as well as environment (housing system).

Keywords: dairy cows, clinical lameness, SCC, fat and protein content

The etiology of lameness is complex and multifactorial and it is currently only partly understood (7). The mean prevalence of lameness in dairy herds in England and Wales is 36.8% (range = 0-79.2%) and should be a significant warning to the dairy industry, especially taking into account those farms in which prevalence of lameness is over 70% (4). Ettema et al. (6) reported that hoof lesions were diagnosed in as many as 80% of all cows in Danish dairy herds. In Slovak farms the mean prevalence of lameness varies from 12 to 47%, at a mean value of 26 ± 8% (18). Important risk factors include not only optimal environmental conditions, but also early detection and treatment of lame cows (4). Lameness in cows is associated with a reduction of milk yield (3, 6, 7, 13, 14, 16), with decreased reproductive performance of cows (8-10), as well as a deterioration in their welfare (7, 13). Effects of lameness on a decrease of milk production and fertility have been fairly well described in literature; however, little is known of their impact on somatic cell counts (SCC) and milk composition (2, 12, 13). Somatic cell count (SCC) is a good tool indicating the likelihood of infection among quarters of the udder, and SCC of 200,000 cells/ml is the generally accepted level indicating a bacterial infection (15). Individual cow factors such as parity, month of lactation and body condition score (BCS) may also be risk factors for increased SCC > 199,000 cells/ml (5). However, the relationship between *mastitis* and lameness in cows is unclear. Based on the results for 2368 dairy cows in 102 Swedish herds, Hultgren et al. (8) found a strong association between teat injuries and *mastitis*; however, no relationship was found between sole ulcer (SU) and clinical *mastitis* or high milk-cell counts. Conversely, the results obtained by Pavlenko et al. (13) showed that only SU-affected

cows had significantly higher cell counts during the 5th and 6th weeks after claw trimming (WACT) when compared with the healthy cows. The effect of lameness, measured by the locomotion score (LS), on SCC confirms truly negative associations between LS (assessed in a 4-point scale), although lame cows on some farms produce milk with lower geometric mean SCCs than unaffected cows (2). In that study this concerned the cows with LS 2 on 2 of the 7 studied farms within 10 d of LS assessment, and for severely lame cows (LS 3) six months later.

The aim of the study was to investigate the relationship of clinical signs of lameness at the time of their occurrence with somatic cell count and the percentage of fat and protein contents in milk when compared to healthy cows.

Material and methods

Animals and management. The study was conducted on Polish Holstein-Friesian dairy cows (n = 24), kept on a farm (n = 125) located in the Wielkopolska region. The cows were housed in tie stalls with access to a pasture and a cow-yard throughout the year. Of all the cows in the herd on the pasture on September 2, 2011, 12 cows with clear clinical signs of lameness (17) were selected, comprising experimental animals in this study. In this group there were 3 moderately lame cows (25%), 7 lame cows (58.3%), and 2 severely lame cows (16.7%). The other 12 healthy cows (normal gait) were selected on the basis of analogues, taking into account parity and month of lactation of cows from the experimental group. In each group there were 2 primiparous cows and 10 multiparous cows. Table 1 shows the distribution of clinically lame and healthy cows according to the mean parity and mean days from calving to assessment of lameness (September 2, 2011). Differences in average values for parity and days from calving to assessment of lameness in both groups of cows were similar and not statistically significant. The previous study shows that the main hoof diseases in cows in this herd causing clinical lameness include SU, digital dermatitis (DD) and interdigital dermatitis (ID) (11). The average milk yield for approximately 125 cows was 8,054 L/yr per cow in the herd. In the summer period cows were fed as follows: pasture from 8:00 AM in the morning up to 1:00 PM in the afternoon, feeding with a total mix ration (15 kg of corncob silage, 8 kg of beet pulp – 24% dry matter, 6 kg of lucerne haylage, 5 kg of grass haylage/day/cow). Apart from that, cows received 8 kg of fresh green lucerne fodder/day/cow. After calving the cows were additionally fed a concentrate (7 kg/d/cow). Every day new bedding was provided in the tie stalls before the cows returned from pasture. After completion of the experiment (after 3 weeks) the claws of clinically lame cows were temporarily trimmed, disinfected with Bioval (Biovet Drawalew SA) and treated with an antibiotic spray. Generally, hoof trimming in the herd was performed twice a year, in November and April.

Lameness recording. In September 2011 individuals were scored for lameness (score 1-5) by the same experienced observer 3 times at weekly intervals. The scores were based on gait and posture while walking and standing, using methods suggested by Sprecher et al. (17). During this period both groups of cows were examined. Experimental cows were evaluated in each case as 3 being moderately lame, 4 – lame and 5 – severely lame. Cows in the control group in each case were evaluated as 1 – non-lame (normal gait). The cows were observed and scored when they were standing or walking a distance of 5 to 10 m.

Sampling and analysis of milk samples. Single milk samples were collected from cows of both groups during the three weeks at weekly intervals (1 day after assessment of locomotion of cows). At each milk recording before the morning milking, after fore stripping, washing and drying of the teats, approx. 50 ml of milk was collected as one milk sample from all quarters of the udder during the milking by a milk meter. Samples were preserved with a CC preparation, which ensures the maintenance of chemical parameters of milk samples for 30 days at room temperature (0.25 g/50 ml milk). Milk samples were analyzed at the Laboratory of Milk Evaluation in Kolo (Poland). Fat and protein contents in milk were determined with a MilkoScan device, while a Fossomatic 90 apparatus was used to evaluate SCC in milk. Somatic cell count values were transformed to a common logarithm (log SCC) in order to obtain a distribution close to normal (1).

Statistical analysis. Data analysis was performed using the Statistica statistical package (Version 10). Results were statistically analyzed using the multivariate analysis of variance (ANOVA). The main factors considered in the analysis of variance were as follows: group of cows (experimental and control), replicates (1, 2, and 3), parity (1 and 2; 3 and 4; > 4) and stage of lactation (early: 1-100 d; medium: 101-200 d; late > 200 d). The significance of differences between means was set at $P < 0.05$ and $P < 0.01$.

Tab. 1. The distribution of clinically lame and healthy cows according to parity and days from calving to assessment of lameness

Factor	Number of cows	Parity $\bar{x}$ ± s.d.	Minimum	Maximum	Days from calving to assessment of lameness $\bar{x}$ ± s.d.	Minimum	Maximum
The average general	24	3.67 ± 1.76	1	8	145 ± 77	5	274
Group of cows:							
Clinical lameness	12	3.75 ± 1.96^{ns}	1	8	137 ± 77^{ns}	5	269
Normal gait	12	3.58 ± 1.62^{ns}	1	6	154 ± 80^{ns}	45	274

Explanations: September 2, 2011; ^{ns} – non-significant

Results and discussion

Table 2 presents the results of the effect of clinical lameness, replicates, parity and stage of lactation on log SCC and percentage contents of fat and protein in milk of cows. Clinical lameness in cows did not significantly affect the log SCC and milk fat percentage, but a significantly lower protein content ($P < 0.05$) was recorded in the milk of those cows compared to healthy animals. No significant differences in the studied traits were found between successive replications of the experiment. After the first and second parity statistically significantly fewer somatic cells were recorded in comparison with a further parity. In the milk of cows over 4 parity a significantly higher protein content ($P < 0.05$) was found when compared to the content in the milk of cows in the first two parities. In early lactation there were significantly fewer somatic cells in milk, as was the percentage content of protein in milk when compared to the content of these components in the milk of cows in medium and late lactations.

Somatic cell count. In this study clinically lame cows and healthy cows had similar log SCC, amounting to 5.58 and 5.63, respectively. When compared with the geometric mean SCC for these cows (385,000 cells/ml, and 430,000 cells/ml, respectively), the level in case of lame cows was lower by 45,000 cells/ml than in healthy cows. In this study we did not compare the SCC before and after the occurrence of lameness, but during their occurrence. Archer et al. (2) studied the association between LS, test day yield (TDY), and SCC records collected from 1,397 cows in a convenience sample of 7 dairy herds. Potential confounding factors affecting the relationship between LS and log SCC were as follows: farm of origin, parity, season, month of lactation, and milk yield at the current test day (geometric mean SCC decreased by 6,000 cells/ml per kilogram increase in TDY). The authors indicate that the LS had an influence on $\log_{10}$ SCC within 10 days of a test day at monthly intervals for 12 months between August 2008 and July 2009. Increased TDY was associated with decreased SCC. In all the seven herds cows with LS 3 (severely lame) six months later produced milk with 16,000 fewer cells/ml compared with the geometric mean for cows that would have LS 1 in 6 months' time. For cows assessed from August 08 to October 08, parity 1, lactation month 1, and with TDY of 20 kg this was equivalent to a 10% decrease in SCC for cows that would have LS 3 in 6 months' time when compared with those that would have LS 1 in 6 months' time. Pavlenko et al. (13) reported that somatic cell counts in milk during 2 to 3 WACT of DD-affected and Su-affected cows were lower than in milk of healthy cows (4.35; 6.34, and 7.79 cells × 10^4, respectively). During 5 to 6 WACT SU-affected cows had significantly higher SCC than healthy cows (21.42 and 5.51 cells × 10^4, but differences in SCC between DD-affected and healthy cows were insignificant during those WACT (6.06 and 5.51 cells × 10^4). In the first WACT period a similar trend was observed in comparison to our results, where lame cows had a lower number of somatic cells in milk than healthy cows; however, somatic cell counts in the cited study were lower than our results.

Tab. 2. The impact of clinical lameness, replicates, parity and stage of lactation on log SCC, and fat and protein contents in milk

Factor	The level of factor	Number of milk samples	Log SCC	Fat (%)	Protein (%)
The average general		72	5.60 ± 0.72	4.24 ± 1.16	3.20 ± 0.39
Group of cows:					
Clinical lameness	1	36	5.58 ± 0.73	4.32 ± 1.29	3.13 ± 0.37^{a}
Normal gait	2	36	5.63 ± 0.72	4.16 ± 1.02	3.27 ± 0.41^{b}
Replicates:	1	24	5.54 ± 0.52	4.17 ± 0.88	3.18 ± 0.38
	2	24	5.67 ± 0.72	4.47 ± 1.60	3.16 ± 0.37
	3	24	6.60 ± 0.91	4.08 ± 0.85	3.26 ± 0.44
Parity:					
1 and 2	1	18	4.90 ± 0.69^{A}	4.25 ± 0.75	3.12 ± 0.23^{a}
3 and 4	2	30	5.92 ± 0.53^{B}	4.18 ± 1.35	3.18 ± 0.40
> 4	3	24	5.73 ± 0.61^{BC}	4.30 ± 1.19	3.29 ± 0.48^{b}
Stage of lactation (average in days)					
Early (73 ± 24)	1	21	5.15 ± 0.57^{A}	3.87 ± 0.59	2.91 ± 0.15^{A}
Medium (150 ± 31)	2	24	5.68 ± 0.70^{B}	4.40 ± 1.44	3.18 ± 0.34^{B}
Late (245 ± 32)	3	27	5.89 ± 0.71^{BC}	4.38 ± 1.19	3.44 ± 0.41^{AC}

Explanations: ab – means in the same column with different superscript letters differ significantly at $P < 0.05$, AB – $P < 0.01$

Somatic cell count is a good indicator of health of udder quarters, with SCC of 200,000 cells/mL being generally regarded as suggestive of bacterial infection (15). This threshold was exceeded in our results. In our previous study in the same herd during early lactation of Polish Holstein-Friesian, cows which were never lame log SCC was 5.15, but in case of lame cows (score = 3) for a month it was log SCC 5.47 ($P < 0.01$). The same log SCC values were recorded for cows which were lame > one month (15). This is in agreement with the results of the present study, with the exception of log SCC for healthy cows (log SCC 5.63 and log SCC 5.15).

Fat and protein percentage content. The results of our study show that clinically lame cows had a higher fat content in milk than cows with

a normal gait – 4.32 and 4.16%, respectively ($P > 0.05$), but a lower protein content than healthy cows – 3.13 and 3.27%, respectively ($P < 0.05$). In a study by Pavlenko et al. (13) in both study periods (2 to 3 WACT and 5 to 6 WACT), the percentages of fat and protein in milk between DD-affected cows, SU-affected cows, and healthy cows were insignificant statistically. Olechnowicz and Jaśkowski (12) reported that in early lactation mean monthly milk production – as well as fat, protein, and lactose production – were significantly lower in cows with clinical lameness when compared to cows which were never lame or that were mildly lame.

Conclusion

There were no statistically significant differences in SCC and milk fat contents between clinically lame cows and healthy cows; however compared with healthy cows lame cows had a significantly lower protein content. The results of this study should be treated as preliminary. Examinations should be repeated in large herds of cows taking into account interaction between milk traits of cows (clinically lame and healthy) and parity, stage of lactation, and environment (housing system).

References

1. *Ali A. K. A., Shook G. E.*: An optimum transformation for somatic cell concentration in milk. J. Dairy Sci. 1980, 63, 487-490.
2. *Archer S. C., Green M. J., Madouasse A., Huxley J. N.*: Association between somatic cell count and serial locomotion score assessments in UK dairy cows. J. Dairy Sci. 2011, 94, 4383-4388.
3. *Archer S. C., Green M. J., Huxley J. N.*: Association between milk yield and serial locomotion score assessments in UK dairy cows. J. Dairy Sci. 2010, 93, 4045-4053.
4. *Barker Z. E., Leach K. A., Whay H. R., Bell N. J., Main D. C. J.*: Assessment of lameness prevalence and associated risk factors in dairy herds in England and Wales. J. Dairy Sci. 2010, 93, 932-941.
5. *Breen J. E., Bradley A. J., Green M. J.*: Quarter and cow risk factors associated with a somatic cell count greater than 199,000 cells per milliliter in United Kingdom dairy cows. J. Dairy Sci. 2009, 92, 3106-3115.
6. *Ettema J. F., Capion N., Hill A. E.*: The association of hoof lesions at claw trimming with test-day milk yield in Danish Holsteins. Prev. Vet. Med. 2007, 79, 224-243.
7. *Green L. E., Borkert J., Monti G., Tadich N.*: Associations between lesion--specific lameness and the milk yield of 1,635 dairy cows from seven herds in the Xth region of Chile and implications for management of lame dairy cows worldwide. Anim. Welfare 2010, 19, 419-427.
8. *Hultgren J., Manske T., Bergsten C.*: Associations of sole ulcer at claw trimming with reproductive performance, udder health, milk yield, and culling in Swedish dairy cattle. Prev. Vet. Med. 2004, 62, 233-251.
9. *Morris M. J., Kaneko K., Walker S. L., Jones D. N., Routly J. E., Smith R. F.*: Influence of lameness on follicular growth, ovulation, reproductive hormone concentrations and estrus behavior in dairy cows. Theriogenology 2011, 76, 658-668.
10. *Morris M. J., Walker S. L., Jones D. N., Routly J. E., Smith R. F., Dobson H.*: Influence of somatic cell count, body condition and lameness on follicular growth and ovulation in dairy cows. Theriogenology 2009, 71, 801-806.
11. *Olechnowicz J., Jaśkowski J. M.*: Hoof measurements related to locomotion scores and claw disorders in dairy primiparous cows. Bull. Vet. Inst. Pulawy 2010a, 54, 87-92.
12. *Olechnowicz J., Jaśkowski J. M.*: Impact of clinical lameness, calving season, parity, and month of lactation on milk, fat, protein, and lactose yields during early lactation of dairy cows. Bull. Vet. Inst. Pulawy 2010b, 54, 605--610.
13. *Pavlenko A., Bergsten C., Ekesbo I., Kaart T., Aland A., Lidfors L.*: Influence of digital dermatitis and sole ulcer on dairy cow behaviour and milk production. Animal 2011, 5, 1259-1269.
14. *Reader J. D., Green M. J., Kaler J., Mason S. A., Green L. E.*: Effect of mobility score on milk yield and activity in dairy cattle. J. Dairy Sci. 2011, 94, 5045-5052.
15. *Schepers A. J., Lam T. J. G. M., Schukken Y. H., Wilmink J. B., Hanekamp W. J.*: Estimation of variance components for somatic cell counts to determine thresholds for uninfected quarters. J. Dairy Sci. 1997, 80, 1833-1840.
16. *Singh Y., Lathwall S. S., Chakravarty A. K.*: Effect of lameness (hoof disorders) on productivity of Karan Fries crossbred cows. Anim. Sci. J. 2011, 82, 169-174.
17. *Sprecher D. J., Hostetler D. E., Kaneene J. B.*: A lameness scoring system that uses posture and gait to predict dairy cattle reproductive performance. Theriogenology 1997, 47, 1179-1187.
18. *Tongel P., Brouček J.*: Influence of hygienic condition on prevalence of mastitis and lameness in dairy cows. Slov. J. Anim. Sci. 2006, 43, 95-99.

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