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GENETIC PARAMETERS FOR CLINICAL MASTITIS, FERTILITY AND SOMATIC CELL SCORE IN CZECH HOLSTEIN CATTLE

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Abstract

Cases of mastitis (CM) from 38,236 lactations belonging to 16,497 cows were recorded on seven farms in the Czech Republic from 1996 to 2014. Clinical mastitis was analyzed with linear animal model as an all-or-none trait for each recorded lactation (CM305) and separately for each trimester of lactation (CM1, CM2, and CM3). Bivariate linear animal models were used to estimate the genetic correlation between these CM traits and lactation means for somatic cell score (SCS305), the interval between calving and first insemination (INT) and days open (DO). Factors included in the linear model were parity, herd, year of calving, calving season, fixed linear and quadratic regression on age at first calving, fixed linear and quadratic regression on milk production in the corresponding parity, permanent environmental effect of the cow, and additive genetic effect of the cow. Estimated heritabilities of the CM traits ranged from 0.01 to 0.03. Permanent environmental effects accounted for approximately two-thirds of the phenotypic variance. Genetic correlations of SCS305 with CM traits were 0.85±0.029, 0.81±0.086, 0.82±0.087, and 0.67±0.088 for CM305, CM1, CM2, and CM3, respectively. Genetic correlations of INT with CM305, CM1, CM2, and CM3, respectively, were 0.22±0.065, 0.19±0.084, 0.20±0.121 and 0.15±0.121: and genetic correlations of DO and the four CM traits were 0.28±0.079, 0.26±0.101, 0.43±0.134, and 0.15±0.131. For the 140 sires in the dataset, Spearman rank correlations among breeding values for the four CM traits and for SCS305 were uniformly high at 0.99±0.001.

Key words: mastitis, somatic cell score, fertility, genetic parameters, Holstein cattle

Because of the expense (Halasa et al., 2007; Wolfová et al., 2006) associated with veterinary treatment, discarded milk, reduced milk production, impaired reproduction, and increased risk of culling (Sharma et al., 2011; Fetrow, 2000), mastitis is the most common and costly disease in dairy cattle. Improving animal health in general is becoming increasingly important worldwide. Sender et al. (2013) concluded that selection programs will be designed to improve animal resistance to infection and inflammation of the udder using the cumulative impact of multiple genes and/or selecting animals which have favorable identifiable alleles.

Therefore, genetic selection programs are focusing on reducing diseases and improving functional traits (Fuerst et al., 2011; Zwald et al., 2006).

Often, the most effective selection criterion is direct measures of health or disease, but such information may not be included in recording, evaluation and selection schemes. Clinical mastitis is routinely recorded and used in selection against clinical mastitis susceptibility with beneficial impact on udder health of cows, for example in Scandinavia for many decades (Heringstad and Østerås, 2013), with positive impact on udder health of cows. Jamrozik et al. (2016) states that health recording system started in Canada in 2007. Because experience of collecting and analyzing udder health data is still limited in the Czech Republic, breeding values for somatic cell score, estimated using random regression models, have mainly been used as an indicator for udder health. In addition, breeding values have been calculated since 1999 for linear-type traits including udder conformation. Registration of every clinical mastitis (CM) occurrence has been obligatory on all dairy farms since 1997, mainly because direct evidence of CM is required prior to pharmaceutical treatment. Mandatory recording of each antibiotic treatment including the affected quarters is required on all farms. The resulting records are not transferred to the central database, however, and their availability for research is low.

Fortunately, there is a set of experimental CM data at the Institute of Animal Science, v.v.i., in Prague. This dataset is available for research and has been used in many investigations. For example, Wolf et al. (2010) and Zavadilová et al. (2015) estimated genetic parameters for mastitis traits and economic weights of CM traits were reported by Wolfová et al. (2006). Since 2010, the size of the clinical mastitis dataset has increased markedly, and therefore, it would be beneficial to validate or revise previous estimates. Zavadilová et al. (2015) suggested that CM as an all-or-none trait would be adequate in comparison to CM defined as number of cases per lactation or total number of days in CM per lactation for using in genetic analyses. In this investigation, we therefore concentrated on the most appropriate definition of the CM trait. Our aim was to determine the importance of different parts of lactation for CM traits genetic evaluation, because Negussie et al. (2007) suggested that clinical mastitis in early lactation accounted for most of the identified genetic variation for the trait. An alternative premise is that occurrences of clinical mastitis in different parts of lactation actually constitute different traits (Negussie et al., 2007).

The objectives of the present study were to estimate genetic parameters of clinical mastitis, considered here as an all-or-none trait during the entire lactation or separately in subsequent trimesters of lactation, and to determine the genetic correlations between clinical mastitis and somatic cell scores and fertility traits recorded on Czech dairy farms.

Material and methods

Animals and trait definition

Data on clinical mastitis incidence were collected from seven Holstein herds between 1996 and 2014. Duration of observation for these farms and other descriptive statistics are shown in Table 1.

Table 1. Herd characteristics

Herd	Data collection period	Cows (n)	Lactations (n) in sum	Cases (n) of mastitis
1	1998–2014	6 311	15 206	23 830
2	2002–2014	2 750	6 412	9 260
3	2000–2014	892	1 902	3 634
4	2002–2014	650	1 306	2 125
5	1997–2014	1 098	2 493	4 257
6	1996–2014	858	1 193	4 597
7	1997–2014	3 938	8 983	17 799
Total		16 497	38 236	65 502

Data from the same herds were utilized by Zavadilová et al. (2015), but only from years 2000 to 2012. These farms were not randomly chosen from the national population but rather were those willing to participate in the study. Herds differed in size and were from different regions. However, all used management, feeding, and housing systems commonly applied to dairy herds in the Czech Republic. On all farms, straw was used for bedding, cows were fed a balanced total mixed ration and cows were milked twice a day. Records collected on the farms included cow identification, date CM treatment began, date CM ended (i.e., the last day that milk from a treated cow was discarded), and identification of the treated quarter. Diagnosis of CM by farmers was on the basis of perceptible signs from the udder or milk. However, a detected mastitis case was recorded only if it was treated with antibiotics prescribed by a veterinarian. Thus, CM was defined as a veterinarian-treated udder disease.

Table 2. Number and percentage of lactations with recorded clinical mastitis (CM) by parity

		Parity					
CM 0/1		1	2	3	4	≥5	All
0	N	10 094	6 553	3 964	1 942	1 034	23 587
	%	70.44	62.64	56.64	52.43	37.70	61.69
1	N	4 235	3 909	3 034	1 762	1 709	14 649
	%	29.56	37.36	43.36	47.57	62.30	42.43

Traits of interest included occurrence of clinical mastitis (CM), considered as an all-or-none trait per 305 day lactation (CM305), in the first 100 days of lactation (CM1), the 101–200 days of lactation (CM2), and the 201–300 days of lactation (CM3), with values of 0 (no CM case) or 1 (at least 1 CM case). A new case of CM for the same cow was recorded when the period between the end of the previous case and the next occurrence was at least 8 d. The distribution of cows for the number of CM cases per lactation is shown in Table 2. Other analyzed traits included the interval between calving and first insemination (INT), days open (DO), and an udder health trait (lactation average somatic cell count during lactation, SCC). Somatic cell count was not analyzed as recorded but was first transformed to a somatic cell score (SCS305) according to the following formula, based on the formula by Ali and Shook (1980):

$$SCS305 = \log_2\left(\frac{SCC}{100\,000}\right) + 3$$

Records required for genetic evaluation of CM (birth date, calving date, parity, length of lactation, culling date, cumulative milk yield per lactation, lactation average of SCC together with the pedigree file) were made available from the national database for progeny testing. Only cows whose lactation began after the initiation of data collection, had a lactation length of 240 d or more, and a lactation yield of 1000 kg of milk or more were included in the analysis. For the analysis of fertility traits, only records in the interval between 21 to 280 days for INT and between 42 to 400 days for DO were used in the analysis. For cows without a subsequent calving, the length of INT and the length of DO were penalized by adding 21 days. If the length of the INT or DO were over the maximum value, then the value of the INT or DO was set to the maximum value (280 days INT, 400 days DO).

Table 3. Number of observations and descriptive statistics for CM and reproductive traits

Trait	Observations	Mean	SD	Min	Max
CM305	38 236	0.38	0.4861	0	1
CM1	38 236	0.24	0.426	0	1
CM2	38 236	0.08	0.277	0	1
CM3	38 236	0.06	0.240	0	1
SCS305	20 606	4.0	1.65	0.06	9.5
INT (days)	30 454	87.5	35.51	21	280
DO (days)	27 966	134.2	72.91	42	400

CM305 is considered as an all-or-none trait with values of 0 (no CM case) and 1 (at least 1 CM case); CM1 = CM in the 0th – 100th day of lactation; CM2 = CM in the 101st – 200th day of lactation; CM3 = CM in the 201st – 300th day of lactation. INT = interval between calving and the first insemination; DO = days open; SCS305 = lactation average SCS.

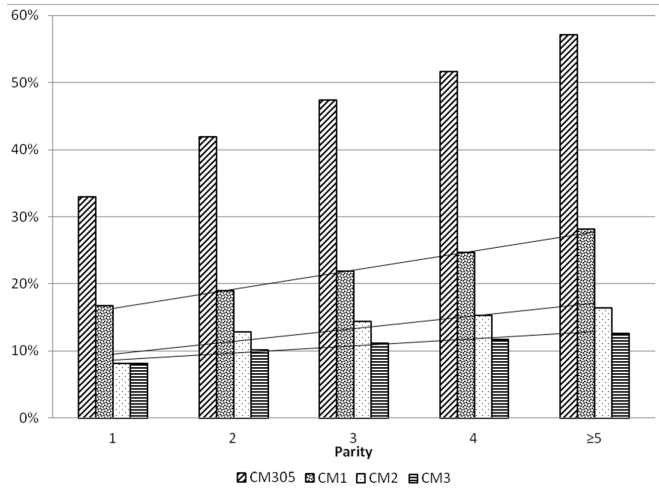

Figure 1. The ratios of the incidence of CM in particular parities

After editing, 16,497 cows with 38,236 lactations were in the analyzed dataset. Descriptive statistics are shown in Table 3. The cows were the daughters of 1,892 sires; and the number of daughters per sire ranged between 1 and 776 with an average of 34. The ratios of the incidence of CM in particular parities are shown in Figure 1.

Statistical methods

The following linear animal model was used for estimation of genetic parameters for CM traits, SCS305 and reproductive traits (INT, DO):

$$y_{ijklmn} = \text{parity}_i + \text{herd}_j + \text{year}_k + \text{season}_l + b_1 \times \text{aac}_{ijklmn} + b_2 \times \text{aac}^2_{ijklmn} + b_3 \times \text{mlk}_{ijklmn} + b_4 \times \text{mlk}^2_{ijklmn} + \text{pe}_m + a_n + e_{ijklmn}$$

where y_{ijklmn} is a recorded clinical mastitis case (CM), which was considered as an all-or-none trait during the 305 day lactation (CM305), in the 0–100 days of lactation (CM1), in the 101–200 days of lactation (CM2), and in the 201–300 days of lactation (CM3), with values of 0 (no CM case) or 1 (at least 1 CM case); SCS305; INT; and DO; parity_i is the effect of the parity i (5 levels); herd j is the effect of the herd j (7 levels); year k is the effect of the calving year k (19 levels); season l is the effect of calving season l (4 levels); aac_{ijklmn} is age at first calving, a fixed regression; aac^2_{ijklmn} is the squared age at first calving, a fixed regression; age of first calving was in the range from 500 to 1400 days; mlk_{ijklmn} is milk production in the corresponding lactation, a fixed regression; mlk^2_{ijklmn} is the squared milk production in the corresponding lactation, a fixed regression; b_1, b_2, b_3 and b_4 are the regression coefficients; pe_m is the permanent environmental effect of the cow m; a_n is the additive genetic effect of the cow n; and e_{ijklmn} is the residual effect.

The pedigree file contained 28,704 animals, 464 animals without parents, 2,965 sires and 17,338 dams, 19 generations. Data were analyzed using the DMU package (Madsen and Jensen, 2010).

The heritabilities and genetic correlations were estimated using bivariate analyses combining CM traits with other analyzed traits. Estimated genetic parameters were used in the prediction of breeding values for the CM traits using the same linear animal model equation, as well as the same dataset. Breeding values of bulls were estimated individually for each of the CM traits (CM305, CM1, CM2 and CM3) and then with SCS305 in a combined bivariate model. The employed model equations were the same as those used for genetic parameters estimation. Spearman rank correlations were performed between breeding values for the different CM traits and for SCS305 for the 140 sires with reliability of breeding value over 50% in the analyzed dataset.

Linear model for the analysis of binary trait is not optimal. On the other hand, this approach is common in livestock breeding. The significant advantage of linear model is the possibility of including traits (mastitis) in multitrait models when other traits are continuous, for example somatic cell score. By way of example, Govignon-Gion et al. (2016) or Jamrozik et al. (2016) prefer linear model over the threshold in the case of clinical mastitis. The detailed justification of using linear model in the place of threshold model can be found in Sasaki et al. (2015).

Results

Variances and heritability

Variance components, their standard errors, proportions of phenotypic variance attributable to heritability and repeatability for the analyzed CM traits are shown in Table 4. For all CM traits, additive genetic variances were lower than permanent environmental variances except for CM1, for which both variances were equal. Residual variance made up 93% of the total variance for CM305 and even higher proportions for the three sequential stages of lactation.

Table 4. Variance components and their standard errors (in parentheses) estimated for the clinical mastitis (CM) traits

	CM305	CM1	CM2	CM3
Variance component estimates				
Additive genetic	0.013 (0.011)	0.003 (0.0005)	0.001 (0.0003)	0.001 (0.0003)
Permanent environment	0.047 (0.010)	0.004 (0.0006)	0.004 (0.0004)	0.004 (0.0004)
Residual	0.212 (0.013)	0.150 (0.0009)	0.099 (0.0006)	0.083 (0.0005)
Variances as proportions of the total phenotypic variance				
Heritability	0.05 (0.007)	0.02 (0.0014)	0.01 (0.0012)	0.01 (0.0015)
Repeatability	0.07	0.04	0.04	0.06
Residual	0.93	0.96	0.95	0.94

CM305 is considered as an all-or-none trait with values of 0 (no CM case) and 1 (at least 1 CM case); CM1 = CM in the 0th – 100th day of lactation; CM2 = CM in the 101st – 200th day of lactation; CM3 = CM in the 201st – 300th day of lactation.

The heritability estimate for SCS305 was 0.21, and the repeatability estimate was 0.38. Heritabilities for INT and DO were 0.16 and 0.09, respectively; while repeatabilities for INT and DO were 0.04 and 0.07, respectively.

Genetic correlations

Estimates of genetic correlations among the evaluated traits and their standard errors are shown in Table 5. High positive genetic correlations were found (>0.90) between CM305 and CM1 and CM2, whereas the genetic correlation of CM305 with CM3 was 0.73. Genetic correlations among the CM traits in the different lactation trimesters were highest between consecutive periods (CM1–CM2 = 0.89; CM2–CM3 = 0.74); whereas the genetic correlation between CM1 (first 100 days of lactation) and CM3 (last third of lactation) was 0.42.

Substantial genetic correlations were reported between SCS305 and all CM traits. These correlations ranged from 0.81 to 0.85 except during the last third trimester of lactation, for which the genetic correlation estimate was 0.67.

Estimates of the permanent environmental correlations and their standard errors are shown in Table 6, and those of the residual correlations are in Table 7. Permanent environmental correlations were negative between CM1 and the other CM traits (from -0.37 to -0.84), residual correlations were negative between CM305 and the other CM traits. The permanent environmental correlations between the CM traits

and SCS305 were positive and significant (0.26–1.00). Among the fertility traits, INT showed the same trends in permanent environmental correlations as DO. The highest residual correlations occurred among the CM traits.

Table 5. Estimates of the additive genetic correlations between clinical mastitis (CM) traits, somatic cell scores (SCS) and reproductive traits and their standard errors (in parentheses)

Trait	CM305	CM1	CM2	CM3
CM1			0.89 (0.129)	0.42 (0.137)
CM2				0.74 (0.171)
CM305		0.92 (0.027)	1.00 (0.063)	0.73 (0.077)
SCS305	0.85 (0.029)	0.81 (0.006)	0.82 (0.087)	0.67(0.088)
INT	0.22 (0.065)	0.19 (0.084)	0.20 (0.121)	0.15 (0.121)
DO	0.28 (0.079)	0.26 (0.101)	0.43 (0.134)	0.15 (0.131)

CM305 is considered as an all-or-none trait with values of 0 (no CM case) and 1 (at least 1 CM case); CM1 = CM in the 0th – 100th day of lactation; CM2 = CM in the 101st – 200th day of lactation; CM3 = CM in the 201st – 300th day of lactation. INT = interval between calving and the first insemination; DO = days open; SCS305 = lactation average SCS.

Table 6. Estimates of permanent environmental correlations between the clinical mastitis (CM) traits, somatic cell scores (SCS), and milk production and their standard errors (in parentheses)

Trait	CM305	CM1	CM2	CM3
CM1			–0.44 (0.088)	–0.84 (0.099)
CM2				0.18 (0.056)
CM305		–0.37 (0.22)	0.85 (0.070)	0.48 (0.074)
SCS305	1.00 (0.059)	0.36 (0.051)	0.56 (0.036)	0.26 (0.039)
INT	0.18 (0.056)	–0.03 (0.050)	0.08 (0.035)	0.14 (0.024)
DO	0.12 (0.054)	–0.15 (0.052)	0.01 (0.034)	0.13 (0.096)

CM305 is considered as an all-or-none trait with values of 0 (no CM case) and 1 (at least 1 CM case); CM1 = CM in the 0th – 100th day of lactation; CM2 = CM in the 101st – 200th day of lactation; CM3 = CM in the 201st – 300th day of lactation. INT = interval between calving and the first insemination; DO = days open; SCS305 = lactation average SCS.

Table 7. Estimates of residual correlations between clinical mastitis (CM) traits, somatic cell scores (SCS), and milk production and their standard errors (in parenthesis)

Trait	CM305	CM1	CM2	CM3
CM1			–0.22 (0.004)	–0.16 (0.004)
CM2				–0.17 (0.004)
CM305		0.59 (0.003)	0.39 (0.003)	0.38 (0.004)
SCS305	0.014 (0.059)	0.07 (0.006)	0.08 (0.006)	0.03 (0.006)
INT	0.005 (0.005)	–0.05 (0.005)	0.01 (0.005)	0.01 (0.005)
DO	0.02 (0.005)	–0.04 (0.005)	0.02 (0.005)	0.03 (0.005)

CM305 is considered as an all-or-none trait with values of 0 (no CM case) and 1 (at least 1 CM case); CM1 = CM in the 0th – 100th day of lactation; CM2 = CM in the 101st – 200th day of lactation; CM3 = CM in the 201st – 300th day of lactation. INT = interval between calving and the first insemination; DO = days open; SCS305 = lactation average SCS.

Breeding values and ranking of sires

Breeding values of bulls were estimated individually for each of the CM traits (CM305, CM1, CM2 and CM3) and then with SCS305 in a combined bivariate model. Spearman rank correlation estimates among sire breeding values for the different CM traits and SCS were obtained. Only sires with daughters in the dataset were included in the correlation analysis. Correlations among the breeding values for the individual CM traits all exceeded 0.99; and correlations between breeding values for the CM traits and SCS305 all exceeded 0.97. The differences in rankings between those selected according to the breeding value for SCS and the analyzed CM traits are minimal.

Discussion

Heritability

Heritability estimates for CM traits were among the lower of those reported in the literature. Using the same population and data source as used in our study, heritability estimates reported by Zavadilová et al. (2015) were much lower than present results. They reported heritability for CM considered as an all-or-none trait to be 0.09 and for CM traits defined as days or number of cases per lactation a value of 0.10. Wolf et al. (2010), again using the same data source, reported heritabilities ranging from 0.11 to 0.13 for CM defined as the number of mastitis cases per lactation. Generally, when CM is defined as an all-or-none trait, heritability estimates have been lower than when CM is defined as number of cases per lactation. For Holstein cows, Pérez-Cabal and Charfeddine (2013) reported 0.04 to 0.05 heritabilities for an all-or-none CM trait. Estimates for all-or-none CM trait in studies by Heringstad et al. (2000), Carlén et al. (2004) and Negussie et al. (2006) varied from 0.01 to 0.03. Jamrozik et al. (2013) reported for all-or-none CM traits (clinical mastitis recorded from calving to 150 days after calving) heritability of 0.03 and 0.05 for first and later lactations, respectively. Govignon-Gion et al. (2016) found for the same CM trait as previous authors heritability of 0.02. For number of cases per lactation, Pérez-Cabal et al. (2009) and Vazquez et al. (2009) reported heritability estimates of approximately 0.10.

Our heritability estimates are lower than those of Zavadilová et al. (2015), probably because of differences in structure of the datasets. Ours included records collected over a longer interval than the dataset used by Zavadilová et al. (2015) and therefore we can assume that more random residual effects occurred in the analysis. This could increase the proportion of residual variance, because it is complicated to adjust for them during the analysis. Increased heritabilities for binary traits could be expected with threshold rather than linear models in an analysis. From threshold models and binary traits, Heringstad et al. (2003), Zwald et al. (2006), Negussie et al. (2008), and Pérez-Cabal et al. (2009) reported heritability estimates ranging from 0.06 to 0.12. Even so, linear models are more useful in multiple trait genetic evaluation than threshold models, and we therefore prefer linear analyses.

The heritability of 0.23 for SCS305 reported by Zavadilová et al. (2015) is almost the same as our result. Typical heritability estimates for lactation SCS are lower than our results, for example 0.10–0.14 in Carlén et al. (2004) and 0.14 in Buch et al. (2011). Zink et al. (2014) reported heritability of somatic cell score per lactation of 0.09 in Czech Holsteins. Negussie et al. (2006) reported heritabilities of SCS ranging from 0.11 to 0.14, and Jamrozik and Schaeffer (2012) and Jamrozik et al. (2013) reported heritability of 0.17 for SCC in early lactation (to 150 days in milk). Alam et al. (2015) presented heritability estimates from sire models in the range of 0.10 to 0.16 for complete lactation SCS.

For fertility traits in Czech Holsteins, Zavadilová et al. (2015) reported heritability of 0.04 for INT and 0.06 for DO, lower estimates than those in this report. In general agreement with our results, Zavadilová and Zink (2013) reported 0.05 for INT and 0.06 for DO; while estimates of Zink et al. (2012) were 0.04 for INT and 0.04 for DO. Other studies confirming low additive genetic variance for reproductive traits are Wall et al. (2003) – 0.04 for INT and 0.03 for DO; Kadarmideen et al. (2000, 2013) – 0.02 for INT, 0.03 for DO. Kadarmideen et al. (2000) reported a repeatability estimate of 0.09 for INT.

Genetic correlations

Contrary to our results, Zwald et al. (2006) found lower genetic correlations between CM trait records within segments of the first lactation (0.26 to 0.56), second lactation (0.33 to 0.64) and third lactation (0.38 to 0.55). Negussie et al. (2007) reported genetic correlations in CM traits in excess of 0.57 between subsequent or overlapping periods of lactation. They reported the lowest values 0.42 to 0.61 between early lactation and the second part of lactation. For us the most important agreement of the present study with findings of Negussie et al. (2007) was for genetic correlations between CM across the entire lactation with the first and second trimester periods of 0.92 and 1.00, respectively. Negussie et al. (2007) reported genetic correlations between CM at –30–300 days of lactation and 30–150 days of lactation to be 0.99 and 0.98. This suggests that recorded data about clinical mastitis occurrence in first part of lactation could replace data collected across the whole lactation period with little information loss. Negussie et al. (2006) used clinical mastitis data from only the first 150 days of lactation for analysis. The finding is important because there is little information loss possible for genetic evaluation when only information from first part of lactation instead of the whole lactation is used. Also better accuracy of data on CM in first part of lactation is assumed because of greater care of the cows in early lactation.

Substantial genetic correlations (0.81–0.85) were found between SCS305 and all the CM traits except CM3 (0.67), suggesting that many of the genes affecting somatic cell number affect CM incidence as well. From this point of view, those CM records collected during the last part of lactation, however, would be less valuable than earlier observations for genetic evaluation of CM traits. We propose that when the genetic correlation between those traits exceeds 0.68, the most appropriate method for the genetic evaluation of udder health traits is a combined analysis of somatic cell score and clinical mastitis incidence (Negussie et al., 2006).

Intermediate to high estimates of genetic correlations between SCS and CM as an all-or-none trait (0.72; 0.70; 0.68, in the first, second and third parity, respectively) were reported by Negussie et al. (2006) for the all-or-none CM trait. Zavadilová et al. (2015) reported genetic correlations of 0.80 between SCS and CM from the whole lactation period defined as number of CM cases per lactation; 0.79 for SCS and the number of days in CM per lactation and 0.83 for SCS and CM considered as an all-or-none trait, estimates very similar to those in present study. Jamrozik et al. (2013) reported genetic correlations between CM traits and SCS in both traits measured in early lactation (from 0 to 150 days in milk) to be 0.55 and 0.74 for first and later lactations, respectively. Govignon-Gion et al. (2016) found genetic correlation between CM trait (to 150 days in milk) and SCS to be 0.70 for Holstein.

Positive (unfavorable) genetic correlations between the CM traits and fertility traits ranging from 0.15 to 0.43 suggest that clinical mastitis may be associated with decreased reproductive efficiency. We conclude that collecting CM data beyond 200 days of lactation will not add substantially to accuracy or efficiency of genetic evaluation. The highest genetic correlation (0.43) was found between CM2 and DO. In contrast, the lowest genetic correlation was observed between both INT and DO and CM3. Zavadilová et al. (2015) estimated genetic correlations to be 0.12–0.20 between CM traits and INT, and 0.22–0.30 between CM traits and DO. CM traits were recorded across the entire lactation period and subsequently expressed as number of CM cases per lactation, number of days of CM per lactation, and CM considered as an all-or-none trait. Values presented in this study are in agreement with earlier analyses. Pérez-Cabal and Charfeddine (2013), for example reported genetic correlations between DO and all-or-none CM and number of CM cases throughout lactation of 0.34 and 0.40, respectively.

CM records collected on Czech dairy farms appear to be suitable input for the genetic evaluation of CM susceptibility. Comparison of genetic correlations among trimesters during lactation and the minimal difference in rankings of sires indicates that the first to the 100th day in milk is sufficient for genetic evaluation rather than evaluating the incidence of CM for the entire lactation. One disadvantage, however, is that clinical mastitis incidence in separate trimesters had much lower heritabilities than in the whole lactation.

Due to the relatively high incidence of mastitis in the dairy cattle population and the need to utilize information for breeding value estimation as quickly as feasible, we recommend using only the first trimester of lactation records (CM1) for breeding value prediction.

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