ACTA UNIVERSITATIS AGRICULTURAE ET SILVICULTURAE MENDELIANAE BRUNENSIS

Volume 67 80 Number 4, 2019

GENETIC EVALUATION OF REPRODUCTIVE AND METABOLIC DISORDERS AND DISPLACED ABOMASUM IN CZECH HOLSTEIN COWS

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To link to this article: https://doi.org/10.11118/actaun201967040939
Received: 1. 7. 2019, Accepted: 1. 8. 2019

To cite this article: KAŠNÁ EVA, FLEISCHER PETR, ZAVADILOVÁ LUDMILA, ŠLOSÁRKOVÁ SOŇA. 2019. Genetic Evaluation of Reproductive and Metabolic Disorders and Displaced Abomasum in Czech Holstein Cows. *Acta Universitatis Agriculturae et Silviculturae Mendelianae Brunensis*, 67(4): 939–946.

Abstract

We estimated the genetic parameters of the most frequent reproductive and metabolic disorders as recorded on-line by 55 milk producers in Czech Holstein cows in the Diary of Diseases and Treatments. The dataset covered the period from July 2015 to May 2019. The coefficients of heritability were estimated for retained placenta (h^2 = 0.01), metritis (h^2 = 0.04), endometritis (h^2 = 0.03), cystic ovary disease (h^2 = 0.03), parturient paresis (h^2 = 0.01), ketosis (h^2 = 0.01) and displaced abomasum (h^2 = 0.03). Positive genetic correlations different from 0 were estimated between parturient paresis and displaced abomasum (r_{g1g2} = 0.75), retained placenta and metritis (r_{g1g2} = 0.61), displaced abomasum and endometritis (r_{g1g2}^2 = 0.49), metritis and endometritis (r_{g1g2} = 0.45), and metritis and displaced abomasum (r_{g1g2} = 0.41). Because each farmer recorded a slightly different portfolio of health data, the genetic correlations with metabolic disorders couldn't be estimated in most cases, since the number of observations was not sufficient.

Keywords: dairy cattle, retained placenta, metritis, endometritis, cystic ovary disease, ketosis, parturient paresis, displaced abomasum

INTRODUCTION

The long-term selection for higher milk production in dairy cattle has led to the deterioration of functional traits, including health traits. Taking functional traits into account in breeding programmes became indispensable, as these traits affect the profitability of the dairy herds, the welfare of the animals and the quality of the animal products. However, only a few countries have utilized some kind of disease-recording system at the population-wide level, which is a prerequisite for the incorporation of the traits into the breeding process. The longest tradition of systematic health traits monitoring within the framework of an animal recording system was established in Nordic countries, of which Norway started to record diseases in 1975–1976 (Østerås *et al.*, 2007). In other countries (Austria, Canada, France, Germany, Spain, USA), the health data monitoring and evaluation systems have quickly evolved over the last two decades, and in addition to veterinarian records, the producer-recorded data are also being used

(Egger-Danner *et al.*, 2012, Koeck *et al.*, 2012, Parker Gaddis *et al.*, 2014).

In the Czech Republic, the statistics show that health disorders are the most common reason (in 84–85%) for culling cows, with fertility disorders being the most frequent (Kvapilík *et al.*, 2018). However, health data at the population level were not collected until 2016. At that time the farmers then reported their health records retrospectively via two rounds of electronic surveys under the Q CZ Study. The Q CZ Study covered almost 80% of Czech dairy cows in the periods from July 2015 to June 2016, and from July 2016 to June 2017 (Fleischer *et al.*, 2018). At the same time, the national web application "The Diary of Diseases and Treatments" (the Diary) was being developed, which had been initiated in 2015 and finally it was launched in 2017. The Diary is designed for the continuous recording and evaluation of health data based on the farm records (Šlosárková *et al.*, 2017). Our aim was to evaluate the records of the most frequent reproductive and metabolic disorders and displaced abomasum entered in the Q CZ Study and the Diary and to estimate the genetic variability and genetic parameters for the Czech Holstein cattle.

MATERIALS AND METHODS

The data on the occurrence of reproductive and metabolic disorders and displaced abomasum were taken primarily from the Diary, and they were recorded by 55 milk producers in Holstein cows. This data were complemented with the records, which the participating farmers reported in both rounds of the Q CZ study. The size of herds was up to 1,620 with median of 400 calvings per herd and year. The data covered the period from July, 2015 to May, 2019. We considered only lactations from Holstein cows (≥ 75% of Holstein genotype), which started with calving from July 1, 2015 onward. Basic dataset included 118,021 observations (all records of 7 selected health traits together with records of lactations of cows calved at the same time in the same herds but without records of any of the selected health traits) of 74,038 lactations in 43,469 Holstein cows from 55 milk producers (60 herds).

These 7 selected health traits were retained placenta (REP), metritis (MET), endometritis (EMET), cystic ovary disease (CYS), parturient paresis (PAR), primary clinical ketosis (KET) and displaced abomasum (DA). The traits were scored as 0 (no case) or 1 (at least one case) in the interval from calving to 7 (REP, PAR), 20 (MET) or 100 (CYS, KET, DA) days in milk (DIM). For EMET, we used the interval of 21–100 DIM. The occurrence of each disease/disorder was then expressed as the lactational incidence rate (LIR), which is:

$$LIR = \frac{number\ of\ affected\ lactations}{number\ of\ lactations} \times 100\,(\%). \qquad (1)$$

Each farmer recorded a different portfolio of health events, so the basic dataset was divided into 7 subsets according to the observed disorder/disease. The minimum LIR = 1% for each trait and herd-year was used to exclude the herds without records in particular years. Another 21 subsets were created for the estimation of the genetic correlations among all the selected health traits.

We applied a single trait linear animal model for (co)variance components estimation:

$$\mathbf{y} = \mathbf{X}\beta + \mathbf{Z}_a a + e, \qquad (2)$$

where **y** is a vector of observation of the health trait; β is a vector of fixed effects (joint effect of herd-year-season of calving and the parity); **a** is a vector of random animal effects; **e** is a vector of random residuals; **X** and $\mathbf{Z}_a$ are the corresponding incidence matrices. The effect of year had 5 levels (2015–2019), the effect of season had 2 levels (January–June and July–December), and the effect of the parity had 5 levels (from 1st to 5th lactation). Random effects were assumed to be normally distributed with

$$Var\begin{bmatrix} a \\ e \end{bmatrix} = \begin{bmatrix} \boldsymbol{A}\sigma_a^2 & 0 \\ 0 & \boldsymbol{I}\sigma_e^2 \end{bmatrix} \qquad (3)$$

where $\boldsymbol{\sigma}_a^2$ and $\boldsymbol{\sigma}_e^2$ are the additive genetic and residual variances, $\boldsymbol{A}$ is the relationship matrix and $\boldsymbol{I}$ is the identity matrix. The heritabilities were calculated as $h^2 = \boldsymbol{\sigma}_a^2 / (\boldsymbol{\sigma}_a^2 + \boldsymbol{\sigma}_e^2)$.

Genetic correlations between the health traits were estimated with bivariate linear animal models with the same fixed and random effects as in the single trait model. The (co)variance structure of random effects was

$$Var\begin{bmatrix} a \\ e \end{bmatrix} = \begin{bmatrix} \boldsymbol{G}_0 \otimes \boldsymbol{A} & 0 \\ 0 & \boldsymbol{R}_0 \otimes \boldsymbol{I} \end{bmatrix}, \qquad (4)$$

where:

$$G_0 = \begin{bmatrix} \sigma_{a1}^2 & \sigma_{a1a2}^2 \\ \sigma_{a1a2}^2 & \sigma_{a2}^2 \end{bmatrix} \qquad (5)$$

is the (co)variance matrix between traits due to additive genetic effects, and:

$$R_0 = \begin{bmatrix} \sigma^2_{e1} & \sigma^2_{e1e2} \\ \sigma^2_{e1e2} & \sigma^2_{e2} \end{bmatrix} \quad (6)$$

is the residual (co)variance matrix.

All data editing and basic statistic procedures (frequencies, means, and generalized least squared means for fixed effects evaluation) were carried out with SAS 9.4. We used DMU v. 6, release 5.2 (Madsen and Jensen, 2010), module DMUAI with Average Information Restricted Maximum Likelihood (AI-REML) for (co)variance components and genetic parameters estimation.

RESULTS

The basic description of the data after editing is shown in Tab. I. The highest average LIR (9.3%) was found in MET, while the lowest one was found in PAR (1.9%). The variability among herds was high, when it ranged from 1.4 to 37.0% in MET and from 1.3 to 36.4% in CYS. The effect of herd-year-season (HYS) was evaluated as highly statistically significant ($p < 0.0001$) for all health traits.

Another fixed effect we evaluated was the parity (lactation number). It was also highly statistically significant for all health traits with the exception of displaced abomasum. Fig. 1 illustrates the LIRs according to the parity corrected for the effect of HYS. The occurrence of diseases/disorders tended to be the lowest in primiparous cows (except of MET) and increased with the parity. This trend was most distinct in PAR, where the LIR changed from a value close to 0 in the 1st lactation to nearly 11% in the 5th lactation. In contrast, the frequency of MET was the highest in primiparous cows and the lowest on the 2nd lactation and then gradually grew to the original level of 10% in the 5th lactation.

The estimated heritabilities were low and ranged from 0.01 (REP, KET, PAR) to 0.04 (MET). All estimates of h^2 with their standard errors (SE) are shown in Tab. II. The datasets used for the estimation of variance components with bivariate animal models are described in Tab. III. The genetic correlations with some metabolic disorders were non-estimable due to the low number of observations and the structure of the datasets. The strongest genetic relationships were found between PAR and DA (0.75), REP and MET (0.61), DA and EMET (0.49), MET and EMET (0.45), and MET and DA (0.41). The positive genetic correlation indicated that selection for one trait will lead to a favourable selection response in the other one. The negative correlations were too low (except of CYS and KET) and had very high SE, such that they practically did not differ from 0.

DISCUSSION

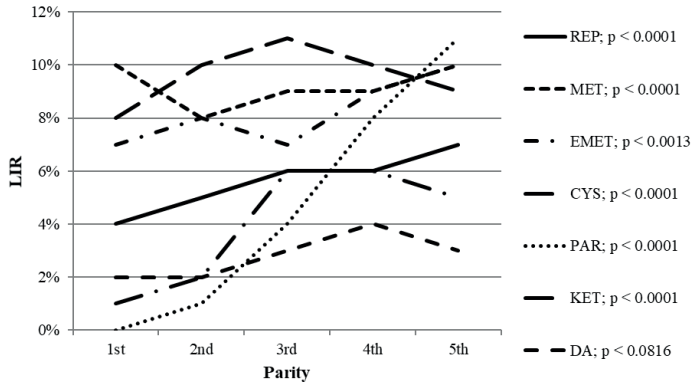
The comparison of health trait frequencies should be done in a careful manner, as different studies may use e.g. different methods of data collection, different calculation techniques and lengths of interval at risk. For example Kelton *et al.* (1998) suggested two approaches for retrospective and current analysis, and Østerås *et al.* (2007) defined the base population as the number of cow-years, while Pryce *et al.* (2016) or Benedet *et al.* (2019) pointed out the difference between the prevalence and incidence of disease, as these terms are not always used correctly, which made the interpretation of results difficult. Generally,

I: *Basic description of the datasets used for the heritability estimation of health traits*

Trait	LIR%			Number of herds	Number of observations
	average	min./herd	max./herd		
REP	4.7	1.3	12.1	45	36,379
MET	9.3	1.4	37.0	49	41,184
EMET	7.5	1.4	23.1	28	15,807
CYS	6.7	1.3	36.4	38	18,152
PAR	1.9	1.1	4.8	20	10,797
KET	2.7	1.2	8.4	17	10,256
DA	2.7	1.4	5.1	13	6,275

REP – retained placenta, MET – metritis, EMET – endometritis, CYS – cystic ovary disease, PAR – parturient paresis, KET – primary clinical ketosis, DA – displaced abomasum, LIR – lactational incidence rate

Fig. 1. Effect of the parity on LIR of health traits corrected on the herd, year and season of calving



1: *LIR of health traits according to the parity, corrected for the joint effect of herd, year and season of calving (HYS).* REP – retained placenta, MET – metritis, EMET – endometritis, CYS – cystic ovary disease, PAR – parturient paresis, KET – primary clinical ketosis, DA – displaced abomasum

II: *Heritabilities h^2 with their standard errors estimated with a single trait animal model*

Health trait	h^2	SE
REP	0.01	0.004
MET	0.04	0.005
EMET	0.03	0.008
CYS	0.03	0.007
PAR	0.01	0.007
KET	0.01	0.008
DA	0.03	0.013

REP – retained placenta, MET – metritis, EMET – endometritis, CYS – cystic ovary disease, PAR – parturient paresis, KET – primary clinical ketosis, DA – displaced abomasum, h^2 – heritability coefficient, SE – standard error of the estimate

the reported incidences of health traits show large variability.

Kelton *et al.* (1998) stated in their older meta-analysis that the LIR ranged between 0.03–22.3% with median 6.5% for PAR, 1.3–39.2% with median 8.6% for REP, 2.2–37.3% with median 10.1% for MET (different definition of the trait), 1.3–18.3% with median 4.8% for KET, 0.3–6.3% with median 1.7% for left DA, and finally 1.0–16.1% with median 8.0% for CYS. In a more recent study, Pryce *et al.* (2016) reported the median incidence of KET of 3.3% with a range 0.24–17.2%, the median incidence of DA of 2.7% (in our study the same average LIR), and the median incidence of PAR 2.8%. In comparison to these two meta-analyses our average LIR were lower for REP, CYS, PAR and KET. A very large inter-annual variability also exists in diseases/disorders incidence rates as demonstrated Østerås *et al.* (2007). Our dataset showed significant variability among herds, years and seasons, which probably reflects, aside from the inter-annual and seasonal changes in nutrition, technology and management, also the form of data recording (Q CZ retrospective survey vs. continuous Diary) and the fact, that the system of health recording is newly established and therefore not entirely stable. The consistency of data recording is often mentioned with regard to data quality (Zwald *et al.*, 2004; Egger-Danner *et al.*, 2012; Pryce *et al.*, 2016). Farmers recorded different portfolio of diseases, as is obvious from the number of herds with data (Tab. I). They were more likely to record diseases, where the diagnosis was unambiguous and the veterinary assistance or medication was needed (also Zwald *et al.*, 2004). Another statistically significant effect was the parity of cow. The occurrence of diseases is usually higher in older cows (Jamrozik *et al.*, 2016; Pryce *et al.*, 2016), with the exception of metritis, which is more frequent in primiparous cows (Egger-Danner *et al.*, 2012). Some studies treated the disease resistance as a genetically different trait in first and later parities (Jamrozik *et al.*, 2016) or focused on first-lactation cows to avoid

III: *Heritabilities and genetic correlations between health traits estimated with bivariate linear animal models*

Trait 1	Trait 2	No. of observations	LIR trait 1 LIR trait 2	$h_1^2 \pm SE$	$h_2^2 \pm SE$	$r_{g1g2} \pm SE$
REP	MET	26,828	4.9 6.9	0.01 ± 0.004	0.03 ± 0.006	0.61 ± 0.21
	EMET	10,658	4.4 7.4	0.01 ± 0.008	0.02 ± 0.008	-0.01 ± 0.35
	CYS	11,223	5.3 7.2	0.02 ± 0.009	0.03 ± 0.010	–0.04 ± 0.27
	PAR	7,730	5.7 1.6	x	x	x
	KET	7,189	5.4 3.2	x	x	x
	DA	3,304	5.4 2.2	x	x	x
MET	EMET	11,807	12.6 7.8	0.03 ± 0.008	0.03 ± 0.010	0.45 ± 0.20
	CYS	13,750	9.6 7.1	0.03 ± 0.008	0.02 ± 0.007	-0.08 ± 0.23
	PAR	7,071	8.5 1.5	x	x	x
	KET	8,250	11.4 3.0	0.02 ± 0.011	0.01 ± 0.009	0.25 ± 0.45
	DA	4,396	16.8 2.4	0.04 ± 0.016	0.03 ± 0.015	0.41 ± 0.30
EMET	CYS	10,634	7.0 6.8	0.04 ± 0.010	0.03 ± 0.010	0.38 ± 0.22
	PAR	4,244	10.1 1.3	0.03 ± 0.025	0.01 ± 0.018	0.48 ± 1.03
	KET	3,403	9.2 2.3	x	x	x
	DA	2,922	8.0 2.7	0.07 ± 0.023	0.03 ± 0.018	0.49 ± 0.38
CYS	KET	3,253	5.8 2.2	0.03 ± 0.022	0.02 ± 0.019	–0.65 ± 0.73
	PAR	3,875	10.2 1.7	x	x	x
	DA	3,070	9.0 2.6	0.04 ± 0.021	0.02 ± 0.018	0.16 ± 0.48
PAR	KET	2,392	1.9 2.2	x	x	x
	DA	2,060	3.0 3.3	0.06 ± 0.031	0.08 ± 0.038	0.75 ± 0.34
KET	DA	1,235	2.2 2.7	0.01 ± 0.030	0.03 ± 0.039	0.36 ± 1.43

REP – retained placenta, MET – metritis, EMET – endometritis, CYS – cystic ovary disease, PAR – parturient paresis, KET – primary clinical ketosis, DA – displaced abomasum, h^2 – heritability coefficient, r_{g1g2} – genetic correlation between trait 1 and trait 2; SE – standard error of the estimate, x – value was non-estimable

selection bias (Zwald *et al.*, 2004; Heringstad, 2010; Koeck *et al.*, 2012). In our study we preferred to keep as much data as was possible, so we treated the parity as one of the effects in model equation.

We applied a linear model for a (co)variance components estimation, even though the threshold model would better account for the binary nature of health traits. There are reasons for using

a linear instead of a threshold models, from lower computational demands, simpler interpretation of results, and difficulties computing threshold animal models (sire model is used instead, for example, in Heringstad *et al.*, 2005, and in Parker Gaddis *et al.*, 2014), to the possibility of combining the results with the routine evaluation of other traits, which is mostly linear. Some of these reasons were summarized by Koeck *et al.* (2010), who also compared results obtained from threshold sire, linear sire and linear animal models in their study and found, that the effects associated with applying an animal model instead of a sire model were more important than the differences resulting from the different methodologies (linear vs. threshold).

Our heritability estimates are in agreement with other studies, as most of them reported the heritabilities of health traits as < 0.10. According to Pryce *et al.* (2016), the heritabilities estimated with linear models ranged from 0.01 to 0.39 for KET, 0.00 to 0.08 for DA, and 0.01 to 0.08 for PAR. The heritabilities for reproductive disorders were usually < 0.05 (Koeck *et al.*, 2010; Koeck *et al.*, 2012; Jamrozik *et al.*, 2016).

Genetic parameters for metabolic diseases from bivariate models were non-estimable in our study due to the low number of observations, which did not provide enough information. For traits with low incidences, the composite traits are sometimes recommended for genetic evaluation. For example, Koeck *et al.* (2010) described the joining of all reproductive disorders, that occurred within 30 DIM (early reproductive disorders mainly associated with calving), within 31-150 DIM (late reproductive disorders, mainly hormonal and late infectious diseases), or within 150 DIM in three composite traits. This joint evaluation led to slightly lower heritability estimates, which indicated the loss of information, but the standard errors were lower, which made estimates more accurate. The joint evaluation of disorders/diseases is also used for routine genetic evaluation in Nordic countries (Johansson *et al.*, 2008), where the group "metabolic disorders" included ketosis, milk fever, other metabolic diseases, other feed-related disorders, and other diseases. For metabolic disorders, where under-reporting could be a challenge, a suitable predictor is often being suggested, which would be especially valuable, if it could be measured objectively (Pryce *et al.*, 2016). Different studies have already been aimed at fat-to-protein ratio, β-hydroxybutyrate concentration in milk, or body condition score (Jamrozik *et al.*, 2016; Benedet et al., 2019).

Estimated genetic correlations between health traits are usually from low to moderate. Many of them are positive, which may indicate that daughters of certain sires tend to be more susceptible to all health disorders, perhaps because they lack an adequate general immune response or because they experience an extreme negative energy balance in early lactation (Zwald *et al.*, 2004). The strongest genetic correlation was reported between DA and KET (Zwald *et al.*, 2004; Koeck *et al.*, 2012; Parker Gaddis *et al.*, 2014; Jamrozik *et al.*, 2016). Our estimate (0.36) was charged with an extremely high standard error, which made it uninformative. We found the highest genetic correlation between PAR and DA (0.75). This is in accordance with the study of (for example) Fleischer *et al.* (2001) who found out, that parturient paresis is most probably a risk factor for the displaced abomasum. The genetic correlation between REP and MET was of a similar value (0.61). This relationship was often reported as significant (Heringstad, 2010, Koeck *et al.*, 2012; Parker Gaddis *et al.*, 2014, Jamrozik *et al.*, 2016), though definition of metritis differ in other studies from ours, mainly in the length of the interval, in which the trait was recorded (usually from 0 to 30-305 DIM). Koeck *et al.* (2012) named as metritis a composite trait, which included cases of acute metritis, purulent discharge, endometritis and chronic metritis. These authors also described a very high genetic correlation between those traits, which did not differ from 1. However, our study showed only a moderate genetic correlation between MET and EMET (0.45). Other studies aimed on genetic evaluation did not differentiate between those two traits.

Our genetic correlations between CYS and other traits mostly did not differ from 0. This was in agreement with most of the other studies (Zwald *et al.*, 2004; Heringstad, 2010; Koeck *et al.*, 2010). The results for relationship between REP and CYS are contradictory: Heringstad (2010) found a genetic correlation of –0.26 between REP and CYS, while Koeck *et al.* (2012) estimated a value of 0.23 for the same parameter. The highest correlation we found was between EMET and CYS, with relatively high standard error (0.38 ± 0.22). Similar association found Fleischer *et al.* (2001) and cited a possible biological explanation. The narrower time overlap of EMET and CYS occurrence (compare to none or limited time overlap of REP or MET with CYS) gives to their relationship better opportunity to manifest itself.

CONCLUSION

The data of reproductive and metabolic disorders and DA recorded in "The Diary of Diseases and Treatments" showed sufficient genetic variability for the estimation of basic genetic parameters. The estimated heritabilities were in agreement with other studies. Genetic correlations for reproductive disorders were mostly positive, which implies that selection for one disorder will lead to a positive selection response in the other one. Genetic correlations for most of metabolic diseases were non-estimable or were not different from 0, as only some farms reported their occurrence, and the number of observations was not sufficient. The creation of composite trait, which would treat selected metabolic disorders as one, should be verified, as this step could lead to higher incidences and possibly more accurate estimates. However, continuity in recording and a sufficient number of reliable phenotypes are unnecessary for the estimation of genetic parameters and successful breeding for better resistance.

Acknowledgement

This paper was supported by the Ministry of Agriculture of the Czech Republic, institutional support MZE-RO0718, and by the National Agency for Agricultural Research, Project No. QK1910320.

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