Comparison of different models for genetic analysis of clinical mastitis in Austrian Fleckvieh dual-purpose cows

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ABSTRACT

The performance of different models for genetic analyses of clinical mastitis in Austrian Fleckvieh dual-purpose cows was evaluated. The main objective was to compare threshold sire models (probit and logit) with linear sire and linear animal models using REML algorithm. For comparison, data were also analyzed using a Bayesian threshold sire model. The models were evaluated with respect to ranking of sires and their predictive ability in cross-validation. Only minor differences were observed in estimated variance components and heritability from Bayesian and REML probit models. Heritabilities for probit and logit models were 0.06 and 0.08, respectively, whereas heritabilities for linear sire and linear animal models were lower (0.02). Correlations among ranking of sires from threshold and linear sire models were high (>0.99), whereas correlations between any sire model (threshold or linear) and the linear animal model were slightly lower (0.96). The worst sires were ranked very similar across all models, whereas for the best sires some reranking occurred. Further, models were evaluated based on their ability to predict future data, which is one of the main concerns of animal breeders. The predictive ability of each model was determined by using 2 criteria: mean squared error and Pearson correlation between predicted and observed value. Overall, the 5 models did not differ in predictive ability. In contrast to expectations, sire models had the same predictive ability as animal models. Linear models were found to be robust toward departures from normality and performed equally well as threshold models.

Key words: clinical mastitis, model comparison, linear and threshold model

INTRODUCTION

Breeding for improved mastitis resistance is becoming increasingly important because of its effect on farm economy and animal welfare and because there is an increase in consumer demands for healthy and natural products. Further, it is well known that unfavorable genetic correlations exist between milk production and clinical mastitis (CM). Estimates of the genetic correlation based on Nordic field data range from 0.24 to 0.55 (Heringstad et al., 2000).

A nationwide health monitoring system for cattle was started in Austria in 2006 in which diagnoses from veterinarians are recorded (Egger-Danner et al., 2007). Recording of health data on farms is currently voluntary. The main project aims are to provide support for herd management and data that can be used for prediction of breeding values for health traits. Based on this data, CM is the most frequent disease besides reproductive disorders in Austrian Fleckvieh dual-purpose cows.

For an efficient genetic evaluation of CM, it is important to use the most appropriate methodology. In the Scandinavian countries direct selection for improved mastitis resistance has been carried out for more than 30 yr, with routine genetic evaluations based on linear models. Because CM is commonly defined as a binary trait for genetic evaluation, scored as 1 if the cow had at least 1 treatment within a defined time period and 0 otherwise, the assumption of normally distributed data is not fulfilled. In the early 1980s, threshold models were developed based on Wright's threshold concept for analysis of categorical data in animal breeding (Gianola and Foulley, 1983; Harville and Mee, 1984; Gilmour et al., 1985). For genetic analysis of CM, several studies have applied threshold models using likelihood procedures (De Haas et al., 2002; Vazquez et al., 2009). In recent years, threshold models applied in a Bayesian framework were widely used (e.g., Heringstad et al., 2004).

A potential disadvantage of the threshold model methodology is that the application of animal models is not always possible because of the extreme category

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Table 1. Number of records, mastitis frequency, and proportion of culled cows within and across lactations

Lactation, n	Records, n	Mastitis frequency, ¹ %	Culled cows, %	Cows culled because of udder problems, %
1	10,841	5.2	6.5	0.5
2	8,525	4.7	2.4	0.3
3	6,573	5.6	3.0	0.6
4	4,918	6.5	4.4	0.7
5	2,998	7.9	5.7	1.0
Overall	33,855	5.6	4.4	0.6

¹Percentage of cows with at least 1 record of veterinary treatment of clinical mastitis or culled because of udder problems in the period from 10 d before to 50 d after calving.

problem, which occurs if all observations for some subclasses are in the same category (Hoeschele and Tier, 1995). The animal model requires fewer assumptions, particularly with regard to randomness in mating.

The objective of this study was to evaluate different models for genetic analysis of CM in Austrian Fleckvieh cows. Threshold sire models (probit and logit) were compared with linear sire and linear animal models with respect to ranking of sires and their predictive ability in cross-validation. This is the first genetic analysis of CM based on data from the Austrian health recording system.

MATERIALS AND METHODS

Data

Diagnoses data from the Austrian project “Health monitoring in cattle” (Egger-Danner et al., 2007) were used for genetic analysis of CM. Because not all herds report health data continuously, it was a challenge to distinguish between farms with low frequencies and farms with incomplete documentation and recording. To ensure that all cows were from herds with reliable health recording, only farms with at least 1 recorded diagnosis (any disease) per 10 cows and year were considered. Furthermore, only data from veterinarians who recorded at least 500 diagnoses distributed over the whole period were considered in this analysis to rule out inconsistent data collection. About 25% of the farms had to be excluded by data editing assuming unreliable documentation and recording.

Records of 41,793 Austrian Fleckvieh cows calving between January 1, 2007 and February 28, 2009 were available for this study. Cows from first to fifth lactation with an age at first calving between 19 and 43 mo were considered. Animals with a calving interval shorter than 300 d or longer than 800 d were excluded. Data were further restricted to cows from herd classes with at least 10 cows in the data set and sires with a minimum of 30 daughters. The final data set had

33,855 records of 23,866 cows, daughters of 201 sires. A total of 1,462 herds were represented in the data.

Clinical mastitis was defined as a binary trait (0 = no mastitis, 1 = mastitis) based on whether the cow had at least 1 veterinary treatment of CM in the period from 10 d before to 50 d after calving. All cows culled because of udder problems within 50 d after calving were considered as diseased even if they did not have a record of veterinary treatment. Cows culled because of other reasons were included as healthy if they had no CM treatment. The culling reason was provided by the farmers. The overall mean CM frequency was 5.6% across lactations. The number of records, frequency of CM, and proportion of culled cows by lactation is given in Table 1.

The sire pedigree file had information on sires and maternal grandsires of the 201 bulls with daughters in the data set, traced back 7 generations, and included a total of 873 males. An animal pedigree file was generated by tracing the pedigrees of cows with data 7 generations back. The resulting pedigree file contained the relationship of 101,521 animals.

Statistical Models

Clinical mastitis was analyzed using threshold sire models (probit and logit) and linear sire and linear animal models. All the models applied belong to the class of generalized linear mixed models, which can be used to analyze data with different distributions from the exponential family (e.g., normal, binomial). These models use a link function relating the expected value $E(y_{ijkl}) = \mu_{ijkl}$ to the linear predictor η_{ijkl} . The following linear predictor was common to all models:

$$\eta_{ijkl} = \varphi + AGE_i + YS_j + h_k + g_l$$

where η_{ijkl} is a function of the expected CM; φ is an intercept; AGE_i is the fixed effect of age at calving-parity interaction ($i = 1, 2, \dots, 11$); YS_j is the fixed effect of year-season of calving ($j = 1, 2, \dots, 13$); h_k is a random

effect for herd of calving ($k = 1, 2, \dots, 1,462$); and g_l is a random effect for sire ($l = 1, 2, \dots, 873$) or animal ($l = 1, 2, \dots, 101,521$).

Four calving age classes were formed for each of the first 2 parities. Age at first calving was grouped into <27, 27 to 28, 29 to 30, and >30 mo and age at second calving was grouped into <40, 40 to 41, 42 to 44, and >44 mo. For older cows, age-parity classes were parity 3, 4, and 5. Year-season of calving, from January 2007 to February 2009, were formed by combining 2 mo (January and February, and so on). All threshold model analyses were based on a sire model because the extreme category problem (i.e., all observations in individual subclasses are either 0 or 1) tends to cause difficulties in animal models (Sorenson and Gianola, 2002). Linear models were carried out with both a sire and an animal model. The permanent environmental effect, which accounts for the correlation between repeated measures on the same animal, was not included in the models. A preliminary linear animal model analysis including this effect resulted in an estimated permanent environmental variance (SE) of 0.4×10^{-3} (0.6×10^{-3}). This indicates that the permanent environmental effect is negligible so far. This is probably because recording began recently and only a small proportion of the animals had health information from more than 1 lactation.

Random effects were assumed to be normally distributed with zero means, and the covariance structure was

$$\begin{bmatrix} h \\ g \end{bmatrix} = \begin{bmatrix} \mathbf{I}\sigma_h^2 & 0 \\ 0 & \mathbf{A}\sigma_g^2 \end{bmatrix},$$

where σ_h^2 and σ_g^2 are the herd and genetic (sire or animal) variances, respectively; $\mathbf{I}$ is an identity matrix with order equal to number of herd classes (1,462); and $\mathbf{A}$ is the additive genetic relationship matrix of the 873 sires or 101,521 animals.

The following link functions were applied:

$$\text{probit model, } \eta_{ijkl} = \Phi^{-1}(\mu_{ijkl}),$$

where $\Phi^{-1}(\cdot)$ is the inverse cumulative standard normal distribution function;

$$\text{logit model, } \eta_{ijkl} = \log_e \frac{\mu_{ijkl}}{1 - \mu_{ijkl}}; \text{ and}$$

$$\text{linear model, } \eta_{ijkl} = \mu_{ijkl}.$$

The threshold model postulates an underlying continuous variable, liability, such that the observed binary

response takes value 1 if liability exceeds a fixed threshold and 0 otherwise. The threshold and the residual variance (σ_e^2) are not identifiable, so these parameters were set equal to 0 and 1, respectively. Variance components were estimated with generalized linear mixed models using the AI-REML algorithm in the DMU package (Madsen and Jensen, 2008).

Bayesian Analysis

For comparison, the data were also analyzed with a Bayesian probit threshold model using Gibbs sampling. The RJMC routine implemented in the DMU software (Madsen and Jensen, 2008) was used. Independent uniform priors were assumed for each of the fixed effects and normal prior distributions were assigned for sire (s) and herd (h) effects $s | \mathbf{A}, \sigma_s^2 \sim N(0, \mathbf{A}\sigma_s^2)$ and $h | \sigma_h^2 \sim N(0, \mathbf{I}\sigma_h^2)$, respectively, where σ_s^2 and σ_h^2 are the sire and herd variances, respectively; $\mathbf{A}$ is the additive genetic relationship matrix of the 873 sires; and $\mathbf{I}$ is an identity matrix with order equal to number of herd classes (1,462). Inference was based on a chain length of 200,000 samples collected after a burn-in of 5,000 iterations. The sample size and length of burn-in were decided based on the convergence diagnostics of Raftery and Lewis (1992) and on visual inspections of trace plots using the BOA software package (Smith, 2007).

Heritability Estimates

Heritabilities for the different models were calculated as follows:

$$\text{probit model, } h^2 = \frac{4\sigma_s^2}{\sigma_s^2 + \sigma_h^2 + 1};$$

$$\text{logit model, } h^2 = \frac{4\sigma_s^2}{\sigma_s^2 + \sigma_h^2 + \pi^2/3};$$

$$\text{linear sire model, } h^2 = \frac{4\sigma_s^2}{\sigma_s^2 + \sigma_h^2 + \sigma_e^2}; \text{ and}$$

$$\text{linear animal model, } h^2 = \frac{\sigma_a^2}{\sigma_a^2 + \sigma_h^2 + \sigma_e^2},$$

where σ_s^2 , σ_a^2 , σ_h^2 , and σ_e^2 are the estimated sire, animal, herd, and residual variances, respectively. In case

of the logit link function, heritabilities were calculated by means of a residual variance of $\pi^2/3$ (Southey et al., 2003). Heritability estimates from the linear models were transformed from the observable (0/1) scale to the underlying scale using the classical formula of Dempster and Lerner (1950).

Cross Validation

A 4-fold cross-validation was carried out to assess the predictive ability of the models. The entire data set was split randomly into 4 subsets, with the restriction that in each subset every herd was represented. For cross-validation, 3 of the 4 subsets were used for fitting and prediction (training set) and predictive ability was tested on the remaining subset (testing set). Variance components and solutions of fixed and random effects of each model were reestimated for each of the 4 training sets.

Two criteria were used to compare the predictive ability of the models, the mean squared error (**MSE**) and the Pearson correlation ($\rho_{\hat{y},y}$) between predicted and observed value. The MSE was calculated as

$$\text{MSE} = \frac{1}{n} \sum_{i=1}^n (y_i - \hat{y}_i^{(-f)})^2,$$

where y_i is the observed value (0 or 1) in the f th fold; $\hat{y}_i^{(-f)}$ is the predicted value based on data from the other 3 folds; and n is the number of observations in a testing subset. Because the predicted values were on different scales for the different models, they were back transformed to the original scale (probability) using the inverse link function. In the probit model $\hat{y}_i = \Phi(\hat{\eta}_i)$, in the logit model $\hat{y}_i = \frac{\exp(\hat{\eta}_i)}{1 + \exp(\hat{\eta}_i)}$, and in the linear model $\hat{y}_i = \hat{\eta}_i$.

Pearson correlation ($\rho_{\hat{y},y}$) was computed as

$$\rho_{\hat{y},y} = \frac{\text{cov}(\hat{y}, y)}{\sigma_{\hat{y}} \sigma_y},$$

where $\text{cov}(\hat{y}, y)$ is the estimate of covariance between predicted and observed values and $\sigma_{\hat{y}}$ and σ_y are the estimates of standard deviations of predicted and observed values, respectively.

RESULTS AND DISCUSSION

Trait Definition and Phenotypic Frequency

In this study CM was considered in the interval from 10 d before to 50 d after calving because preliminary

analysis showed that most genetic variation is found in early lactation, which is in agreement with Lund et al. (1999). As shown in Table 1, mastitis frequencies within lactations ranged from 4.7 to 7.9%. Heringstad et al. (2004) and Negussie et al. (2008b) found higher mastitis frequencies based on veterinary treatments and similar trait definitions. Heringstad et al. (2004) reported mastitis frequencies of 8.8, 9.9, and 13.0% in the interval from 1 d to 30 d after calving for first-, second-, and third-lactation Norwegian Red cows, respectively. In first-lactation Ayrshire cows, Negussie et al. (2008b) found a mastitis frequency of 7.2% in the period from 7 d before to 30 d after calving. In Austria only veterinarians are allowed to initiate mastitis treatments using antibiotics. Also, several measures were applied to ensure reliable reporting within herds. Thus, the lower mastitis frequency in Fleckvieh cattle may partly be caused by differences in the recording system or underreporting of mastitis treatments. As mentioned by Valde et al. (2004), mastitis frequencies may vary considerably depending on the infection level (which is attributed to exposure or lack of exposure to risk factors) and the farmers' treatment strategy (e.g., frequent milking). Another possible explanation is breed differences. Fleckvieh is also the breed with the lowest average SCC (194,167 cells/mL) in Austria (ZuchtData, 2009).

Heritabilities

The results from the Bayesian probit threshold model are given in Table 2, where mean, SD, and 95% credibility intervals of the posterior distributions of variance components and heritability of liability to CM are shown. Differences between herds accounted for about 12.2% of the variation on the underlying scale. This highlights the importance of environmental factors, such as management and hygienic conditions on farms, for mastitis incidence. Although lower than the herd variance, the variation between sires was considerable. The posterior mean (SD) of heritability of liability to CM was 0.06 (0.02). This confirms that in Fleckvieh cattle sufficient genetic variation exists for selection. As illustrated in Figure 1, the posterior distribution of heritability of liability to CM was reasonably sharp and symmetric, but with a longer tail to the right. The heritability estimate is in agreement with results from other studies using probit threshold models, ranging from 0.05 to 0.09 (Heringstad et al., 2004; Negussie et al., 2008a). The corresponding variance components and heritability from the probit threshold model using REML procedures are presented in Table 3. Almost identical results for estimates of variance components and heritability were obtained. With the logit model

Table 2. Posterior mean, SD, and 95% CI (2.5 and 97.5 percentiles) of posterior distribution of sire variance, herd variance, and heritability of liability to clinical mastitis from a Bayesian probit model

Parameter	Mean	SD	95% CI
Sire variance	0.018	0.006	0.008–0.030
Herd variance	0.141	0.015	0.112–0.169
Heritability ¹	0.063	0.020	0.028–0.102

¹Heritability: $h^2 = 4\sigma_s^2 / (\sigma_s^2 + \sigma_h^2 + 1)$ where σ_s^2 and σ_h^2 are the estimated sire and herd variances, respectively.

(Table 3) a higher heritability of 0.08 was found for CM. However, this result cannot be compared directly with the probit model because the estimates are on a different scale. The estimates of the probit model are on the underlying normal scale, whereas the estimates of the logit model are on the underlying logistic scale. Using logit models, De Haas et al. (2002) and Vazquez et al. (2009) obtained heritabilities of 0.04 and 0.11, respectively, for CM. As expected, the heritability estimates of 0.02 from linear sire and linear animal models (Table 3) were lower. When transformed to the assumed underlying liability scale, heritability estimates were 0.08 and, thus, fairly similar to the threshold model estimates. Overall, the heritability estimates from the linear model analysis were in the range of previous studies. In a review, Heringstad et al. (2000) reported that most heritability estimates of CM from analyses with linear models were between 0.02 and 0.03 based on data from the Nordic health recording systems.

Ranking of Sires

Rank correlations between random effects of sires, with progeny in the data set, from the different models are shown in Table 4. The correlations between sire evaluations among threshold and linear sire models were almost 1 (>0.995). Boettcher et al. (1999) also showed that EBV of sires from threshold and linear sire models were highly correlated ($r = 0.98$). Further, Heringstad et al. (2003) compared ranking of bulls for CM and found correlations greater than 0.99 between

Figure 1. Posterior distribution of heritability [$h^2 = 4\sigma_s^2 / (\sigma_s^2 + \sigma_h^2 + 1)$, where σ_s^2 and σ_h^2 are the estimated sire and herd variances, respectively] of liability to clinical mastitis.

threshold and linear sire models. A slightly lower correlation of 0.94 between sire effects for CM from logit and linear sire models was obtained by Vazquez et al. (2009).

Correlations between any sire model and the linear animal model (Table 4) tended to be slightly lower ($r = 0.96$). This implies that the effects associated with applying an animal model instead of a sire model were more important than the differences resulting from threshold versus linear model. This is in agreement with results by Boettcher et al. (1999), where correlations of breeding values from sire models (threshold or linear) and linear animal models were considerably lower ($r = 0.86$ to 0.87) for survival traits. Sun et al. (2009) found rank correlations ranging from 0.95 to 0.97 between linear sire and linear animal models for different fertility traits.

The 10 best and the 10 worst sires from the Bayesian threshold model and their ranking from the other 4 models are presented in Table 5. Selecting the 10 best sires based on any threshold model would have almost no consequence on selection decisions. However, small differences in ranking were observed by applying a linear sire model instead of threshold models. Sires ranked as 12, 13, and 16 in the linear sire model analysis were among the top 10 sires in the Bayesian threshold model. As shown before, the largest difference

Table 3. Restricted maximum likelihood estimates (SE) of variance components and heritability for clinical mastitis from probit and logit threshold sire models and linear sire and linear animal models

Parameter	Probit	Logit	Linear sire	Linear animal
Sire/animal variance	0.017 (0.005)	0.081 (0.024)	0.252×10^{-3} (0.706×10^{-4})	0.102×10^{-2} (0.246×10^{-3})
Herd variance	0.109 (0.011)	0.483 (0.048)	0.178×10^{-2} (0.153×10^{-3})	0.174×10^{-2} (0.153×10^{-3})
Residual variance	—	—	0.505×10^{-1} (0.397×10^{-3})	0.498×10^{-1} (0.434×10^{-3})
Heritability ¹	0.060 (0.018)	0.084 (0.024)	0.019 (0.005)	0.019 (0.005)

¹Probit model: $h^2 = 4\sigma_s^2 / (\sigma_s^2 + \sigma_h^2 + 1)$; logit model: $h^2 = 4\sigma_s^2 / (\sigma_s^2 + \sigma_h^2 + \pi^2/3)$; linear sire model: $h^2 = 4\sigma_s^2 / (\sigma_s^2 + \sigma_h^2 + \sigma_e^2)$; linear animal model: $h^2 = \sigma_a^2 / (\sigma_a^2 + \sigma_h^2 + \sigma_e^2)$. σ_s^2 , σ_a^2 , σ_h^2 , and σ_e^2 are the estimated sire, animal, herd, and residual variances, respectively.

Table 4. Rank (Spearman) correlations between random effects of sires (n = 201) for clinical mastitis based on different models

Item	Probit	Logit	Linear sire	Linear animal
Bayesian	0.999	0.998	0.996	0.961
Probit		0.999	0.997	0.962
Logit			0.998	0.963
Linear sire				0.964

was found between sire models and the linear animal model. Although sires 3, 5, and 6 performed very well in all sire models, they were not among the top 10 sires in the animal model analysis. Generally, the ranking of sires across models agreed better among the worst sires than among the best sires. A similar result was obtained by Fuerst-Waltl et al. (2009), who compared the ranking of sires for inter- and cross-sucking in calves between threshold and linear animal models. Further, Carlén et al. (2006) observed in a simulation study that sires with a larger proportion of daughters with CM got more precise breeding values and were ranked more correctly across models (linear, threshold, and survival analysis).

Cross Validation

Posterior means of heritability of liability to CM over training sets ranged from 0.042 to 0.092 for the Bayesian model. Heritabilities using REML procedures were from 0.043 to 0.085 for the probit model, from 0.063 to

0.118 for the logit model, and from 0.014 to 0.029 for linear sire and linear animal models.

Table 6 shows the average (over testing sets) MSE and Pearson correlation between observed and predicted CM, by model. All threshold and linear sire models showed the same predictive ability in terms of both MSE and correlations. This is in accordance with previous studies. Andersen-Ranberg et al. (2005) compared a threshold-linear with a linear-linear model for 56-d nonreturn rate and interval from calving to first insemination and observed very small differences in ability to predict 56-d nonreturn rate between the 2 bivariate models. Similar predictive performance for CM between logit and linear models was also obtained by Vazquez et al. (2009). In a simulation study, Carlén et al. (2006) demonstrated that little was gained by replacing a linear model with a threshold model or even with a survival analysis, where culled cows and incomplete and ongoing records are considered more correctly. Further, Negussie et al. (2008a) showed that the use of multiple-trait models provides a higher increase in accuracy than is achieved by using a threshold model instead of a linear model for CM. However, by comparing a threshold-linear with a linear-linear model for CM and SCC, they found that the threshold-linear model performed slightly better, especially for smaller progeny groups.

In contrast to our expectations, sire and animal models did not differ in predictive ability (Table 6). In Danish Holstein cattle, Sun et al. (2009) showed that

Table 5. The best and worst 10 sires for clinical mastitis based on a Bayesian probit model and their corresponding ranks from probit and logit threshold sire and linear sire and linear animal models using REML

Item	Bayesian	Probit	Logit	Linear sire	Linear animal
Best	1	1	1	1	1
	2	2	2	2	3
	3	3	3	9	22
	4	4	6	4	9
	5	5	5	8	17
	6	8	8	3	28
	7	6	4	13	16
	8	7	7	10	4
	9	10	10	12	21
	10	11	9	16	8
Worst	192	193	195	192	186
	193	192	190	186	183
	194	194	194	193	190
	195	195	193	194	193
	196	196	196	196	198
	197	197	197	197	192
	198	198	199	198	197
	199	199	198	199	199
	200	200	200	200	201
	201	201	201	201	200

Table 6. Average mean squared error (MSE) and Pearson correlation between predicted and observed occurrence of clinical mastitis over testing sets by model

Item	Bayesian	Probit	Logit	Linear sire	Linear animal
Average MSE	0.052	0.052	0.052	0.052	0.052
Average Pearson correlation	0.129	0.136	0.129	0.127	0.128

linear animal models are superior to linear sire models to predict breeding values for fertility traits.

Model Assumptions and Limitations

A disadvantage of the binary response approach is that records in progress cannot be included in the analysis. Also, cows that were culled in the time period under investigation cannot be considered correctly. Survival analysis would be an alternative to overcome these problems. However, in a simulation study only minor differences were found between linear and threshold models and survival analysis (Carlén et al., 2006).

Previous studies also showed that CM cannot be regarded as the same trait throughout lactation (Lund et al., 1999). Therefore, in recent years longitudinal multivariate threshold models (Heringstad et al., 2004) and random regression models (Chang et al., 2004; Carlén et al., 2009) were applied. Analyzing CM throughout lactation in Austrian Fleckvieh cows will be the subject of further studies. Other alternative models for genetic analyses of CM that have been suggested are censored threshold model (Heringstad et al., 2006) and zero-inflated Poisson model (Rodrigues-Motta et al., 2007) for analyses of number of CM cases.

A possible shortcoming of this study is that records from the first 5 lactations were analyzed together because the available data set was small. Although not all cows start with their first parity, it is not expected that the potential selection bias will act differently in the different models. Further, only information from elite sires, which represent a selected group of sires, was available. However, although the correlated trait SCC is included in the total merit index in Austrian Fleckvieh cattle, it is not the main selection criteria. Thus, the known reduction of the genetic variance within the selected group is expected to be limited for CM. Further, in Austria the progeny groups of test bulls are quite small and range between 60 and 80 daughters. In this study the average number of daughters per sire was about twice as high. As shown by Negussie et al. (2008a), threshold models may perform slightly better for smaller progeny groups. However, heritabilities and incidences of CM are rather low. Therefore, to obtain accurate breeding values, testing of larger progeny groups will be more important than the applied methodology and the choice of linear or threshold model.

CONCLUSIONS

The present study shows that the data from the Austrian project “Health monitoring in cattle” based on records of veterinary treatments can be used for genetic evaluation and selection toward better udder health. Generally, heritability of CM is low regardless of whether it is treated as binary or continuous. Similar overall predictive performance was obtained for all tested models. Estimated variance components as well as ranking of sires and predictive ability were similar for probit models carried out with Gibbs sampling and REML procedures. Although linear models ignore the binary nature of disease traits, genetic evaluation of sires obtained with linear and threshold models were highly correlated. Overall, linear models were found to be robust toward departures from normality and performed equally well as threshold models for genetic evaluation of CM in Fleckvieh.

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