A multi-trait approach combining clinical mastitis and indicator traits to predict mastitis resistance.

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Introduction

Selection on clinical mastitis can be direct, requiring clinical mastitis records, or indirect using predictors correlated to mastitis. Due to a lack of data on clinical mastitis in Australia, in common with many other countries, the selection criterion has been indirectly selecting for somatic cell count (SCC). Somatic cell count has a genetic correlation of around 0.6-0.7 with mastitis, implying that selection for reduced SCC will also reduce mastitis (Kadarmideen & Pryce, 2001). A source of clinical mastitis data is now available in Australia from around 100 herds selected because they have detailed phenotypic data that have also been genotyped and form the genomic information nucleus (Ginfo) (Abdelsayed *et al*., 2017). There are also opportunities to improve the reliability of mastitis breeding values by using combinations of predictor traits including, SCC, and udder conformation traits (Koeck *et al*., 2012). Accordingly, the objectives for this study were: 1) perform bi-variate analyses between clinical mastitis, SCC, udder type traits and milking speed to identify the best predictors of mastitis resistance to be used in the multi-trait analysis. 2) perform genetic prediction using both pedigree and genomic data by combining clinical mastitis and the best predictors identified above in a multi-trait approach to predict mastitis resistance.

Materials and Methods

Data and traits

On farm clinical mastitis records were accessed from dedicated Australian genomic reference herds (Ginfo) (Abdelsayed *et al*., 2017). Herd test day SCC records, and for udder traits, pre-corrected phenotypes (trait deviations) were obtained from DataGene (Bundoora, Victoria, Australia). A total of 26,937 cow-lactations from 1st parity Holsteins were used in the pedigree analysis. There were 1,419 sires with daughter records and of those 449 sires had greater than 10 daughters with records. The pedigree file consisted of 63,350 cows from 3,188 sires and 42,694 dams. For the genomic analysis, a total of 6,744 cows out of 26,937 had both genotypes and first parity records available. Of those genotyped cows, they had 739 genotyped sires.

The following traits were used in the analysis, clinical mastitis (MAST) (0/1 absence/presence based on treatments), and various SCC traits were created; average (Av) and standard (SD) deviation SCC over whole 305 day (all), early (first 150 d) and late (150 to 305 d) lactation, infection trait (INFEC) (coded 0 or1 for absence/presence of infection where at least 1 test day within lactation > 250,000 cells per ml), and severity trait (SEV) calculated as the number of test days with SCC > 250,000 divided by total number of test days. The SCC threshold of >250,000 was chosen based on Australian dairy industry standards for classifying cows with mastitis (Dairy Australia, 2017). For the udder conformation traits: udder depth (UdDep) and texture (Udtex), fore attachment (ForeA), rear udder attachment width (RearAW) and height (RearAH), centre ligament (CentL), teat placement front (TeatPF) and rear (TeatPR), teat length (TeatL), mammary composite (Mamm), and other predictors included milking speed (Mspeed). The number of records for the predictor traits ranged from 6,744 to 26,937 for SCC traits and from 2,583 to 8,209 for

udder traits.

Models

Bivariate and Multi-variate analyses were conducted using a linear mixed animal model using ASReml Version 4 (Gilmour *et al*., 2015).

$$= \mu + HYS + MOC + \beta_1 Agecalving + \beta_2 Agecalving^2 + A + e,$$

where = clinical mastitis (binary trait 0 or 1), and predictor traits (SCC traits, udder traits, milking speed), μ = trait mean, HYS = herd-year-season, MOC = month of calving 1 to 12, Agecalving = age at calving (18 to 39 months 1st parity) covariate and 2nd order polynomial, A = random term for the pedigree structure, or genomic analysis incorporating genomic relationship matrix, and *e* = random error term.

Different multi-trait models were tested but for this paper we only selected the most important multi-trait models to report. Table 1 shows the combination of traits that were analysed.

Table 1. Different multi-trait models that were fitted using both pedigree and genomic data.

	Multi-trait models		
1.	MAST	4.	MAST + AvallSCC + Mamm
2	MAST + AvallSCC	5.	MAST + AvallSCC + Mspeed
2[1]	AvallSCC	6.	MAST + AvallSCC + RearAW
3.	MAST + AvallSCC + UDdep	7.	MAST + AvallSCC + UdDep + RearAW

[1] Model only with Average 305-day SCC for industry comparison

From each model EBVs and DGVs for MAST were calculated and reliabilities were calculated as follows:

$$\text{Reliability (R)} = 1 - \frac{PEV}{\sigma_g^2},$$

where, the prediction error variance (PEV) = squared standard error of the EBV or direct genetic value (DGV) for each animal in the dataset, and σ_g^2 is the additive genetic variance for mastitis, obtained from the REML estimate of each model. It is expressed as a percentage. Models were compared using reliabilities for EBVs and DGVs.

Results and Discussion

Average whole 305, early lactation (0-150 days), infection and severity traits had the highest genetic correlation with clinical mastitis out of the other SCC traits (Figure 1). Udder texture, rear udder attachment height and width, central ligament, mammary composite, and udder depth (negative but favourable; shallower udders, less mastitis cases) had the highest genetic correlations with clinical mastitis (Figure 1).

Figure 1. Genetic correlation between clinical mastitis and predictor traits; somatic cell count traits, udder type traits and milking speed. Standard errors ranged from 0.13-0.25. SCCSdLate was excluded due to having a large standard error.

Pedigree and genomic models had similar trends in reliabilities for each of the models, with genomic models having higher reliabilities, particularly for bulls (Table 2). Cows generally had lower reliabilities than bulls, this is expected as cows only had first parity records while bulls had greater than 10 daughters.

Table 2: Reliabilities (R %) for pedigree and genomic models for 1st parity Holstein cows and bulls with > 10 daughters and the difference in reliabilities (Diff %) compared to model 1.

	Pedigree				Genomics			
Models	R cows	Diff %	R bulls	Diff %	R cows	Diff %	R bulls	Diff %
1	9		11		-2		6	
2	18	+9	28	+17	20	+22	37	+31
2[1]	13	+4	22	+11	19	+21	35	+29
3	20	+11	30	+19	30	+32	48	+42
4	14	+5	25	+14	20	+22	37	+31
5	18	+9	27	+16	17	+19	35	+29
6	21	+12	32	+21	25	+27	44	+38
7	21	+12	32	+21				

When the trait of interest is clinical mastitis, the accuracy of the evaluation is always limited, even when clinical events are available for all animals. As a result, there is much to gain by adding indirect predictors (Rupp & Boichard, 2003). The reliability increases when average 305 lactation SCC is added to the mastitis model, reliabilities did not improve when average SCC was substituted with early lactation. Furthermore, including severity or the infection trait did not increase reliability. When there are no mastitis records available, average 305 SCC is still a good predictor of mastitis (Figure 1; Table 2), which is what the industry has been using to date, selecting indirectly on SCC only.

Cows that have better attached udders and shallow udders, tend to have fewer cases of mastitis (Haile-Mariam *et al*., 2001; Rupp & Boichard, 2003), this is evident from our results (Table 2) with udder depth and rear udder attachment width being the best two predictors of mastitis. When the mammary composite trait was added, reliabilities decreased by 3% for cows and 4 % for bulls, as a result of the different weights given to the traits in the composite being more of a driver.

Adding milking speed led to a 1 to 2% decrease in the reliability (Table 2) which may be because of the low genetic correlation (Figure 1). An earlier Australian study (Haile-Mariam *et al*., 2001) also found low associations between clinical mastitis and milk speed and concluded that selecting for low milking speed, to improve mastitis resistance, is not recommended. Accordingly, we find that the best multi-trait model with the highest reliability includes, clinical mastitis, average whole 305 lactation SCC, udder depth and rear udder attachment width.

This study demonstrated the value of combining the best indicator traits in an optimal way to increase the reliability of mastitis prediction. As the Australian dairy industry currently uses average SCC as a proxy breeding value for mastitis resistance, a further development could be adding actual mastitis data and udder traits, which opens up opportunities for greater responses to selection in mastitis resistance.

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