

GENETIC SELECTION PROGRAMS FOR RESISTANCE TO MASTITIS

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INTRODUCTION

Interactions. Occurrence of disease results from an interaction of host, environment, and agent (Martin *et al.*, 1994). An increase in the incidence of disease occurs around parturition (van Dorp *et al.*, 1999) during which the immune system is impaired (Preisler *et al.*, 2000). Recording of visible disease of different etiologies, i.e., environmental, contagious, metabolic, and trauma are usually grouped under one main heading, such as clinical mastitis (CM).

Pathogens. Mastitis causing pathogens are categorized into minor and major pathogens. Minor pathogens are to a certain extent associated with a decreased incidence of CM caused by major pathogens (Lam *et al.*, 1997). Pathogens are also divided depending on their etiology into environmental (ENV) and contagious (CONT) pathogens.

Management. Several studies have identified risk factors associated with the incidences of CM (Schukken *et al.*, 1991 ; Lam *et al.*, 1997). Genetic selection for resistance might be beneficial for those pathogens that are difficult to control and eradicate such as, *Streptococcus aureus* and coliform species.

Genetic selection. In Canada, genetic selection programs select for resistance to mastitis indirectly on the basis of somatic cell score (SCS), milking time (MT), and udder depth (UD). The ultimate goal is to decrease SCS, incidence of CM, and MT (Boettcher and van Doormaal, 1999). An experimental challenge study found that extremely low quarter somatic cell count (SCC) was associated with an increased incidence of CM (Kehrli and Shuster, 1994). However, most other studies identified associations of decreased SCC with a reduced incidence of CM (Nash *et al.*, 2000). Low bulk milk SCC (BMSCC) herds ($\leq 150,000$ cells/ml) were identified with a larger variance for the incidence of CM than herds with higher BMSCC ($>150,000$ cells/ml) (Barkema *et al.*, 1998).

Conformation. The shape of the udder is related to the incidence of CM, where cows with less desirably shaped udders are more susceptible to infection by a mastitis causing pathogen (Nash *et al.*, 1999). MT is partially related to the integrity of the teat sphincter, and selection for improved MT is beneficial only up to a certain threshold (Schukken *et al.*, 1991 ; Manninen, 1995).

Variance. Selection causes a reduction of variances of the trait of interest (mastitis), and depends on its correlation with the indirect traits on which indirect selection was based. If selection for improved udder health were successful, variances of mastitis caused by all major pathogens compared to major ENV and CONT, separately, would show a similar reduction in their variance.

Data collection. Data necessary to investigate the effect of current genetic selection programs on disease resistance include records of CM cases and bacteriological culture results from milk samples (MS) collected from mastitic cows as well as apparently healthy individuals. These MS could be obtained during a milk test or when the producer identified a clinical case. The most appealing manner to collect these data is to initiate a study where a large number of herds employing a variety of management practices are followed for several years and contribute data to a multi-disciplinary database. Additional data such as hormone status, electro conductivity, automatic feeding, and general disease information could be collected and would be an asset in assessing disease resistance.

Objective. For this study, data were collected for several disciplines, i.e. epidemiology, immunology, and genetics. Objectives of this study were to estimate variances of herd, cow, and residual for subclinical mastitis (SCM) caused by either major ENV or CONT (ALL) and for SCM caused by major ENV and CONT, separately.

MATERIALS AND METHODS

Data. Data were collected from 60 dairy herds in Ontario, Canada. These herds were visited 2 to 6 times from July 1997 to February 1999. During a herd visit composite MS were taken aseptically. "Composite" refers to the collection of milk from all four quarters into a single reservoir. MS were frozen and bacteriology was performed at the Ontario Veterinary College at the University of Guelph, Guelph, Canada.

A cow was determined to have SCM if the composite milk culture yielded a bacterial pathogen in the absence of overt clinical signs of mastitis. In the final dataset, cows were classified on the basis of their SCM status into three main categories; ALL, ENV, CONT. These dependent variables were coded as binary variables. Presence of SCM was coded as 1, while MS identified as having no bacterial growth or no bacterial pathogens were coded as 0. The final dataset contained 5,174 cows with 13,512 observations. The number of observations identified with SCM caused by ALL, ENV, and CONT were 1,571, 766, and 805, respectively.

Statistical analysis. A generalized linear mixed model with a log link was used to analyze and account for the binary nature of the data. Variance components were estimated using GLIMMIX (SAS) by the following model:

$$y_{ijklm} = \mu + \text{time}_i + \text{lact}_j + \beta_k \text{dim} + \text{herd}_l + \text{cow}_m + \varepsilon_{ijklm}$$

Where : Y is a binary trait of SCM caused by ALL, ENV, and CONT herd l ; μ is the intercept ; time_i is the fixed effect of period of sampling (13 categories, reference = August 1997) ; lact_j is the fixed effect of lactation number (1, 2, 3 and 4, ≥ 5) ; β_k is the regression coefficient of y

on days in milk ; dim is the covariate of days in milk ; $herd_i$ is the random effect of herd ; cow_m is the random effect of cow nested within herd ; ε_{ijklm} is the random residual.

The relative importance among the variance components contributed by herd (σ^2_{herd}), cow (σ^2_{cow}), and residual ($\sigma^2_{residual}$) were estimated with the intraclass correlation (ρ) (Shoukri and Pause, 1999) :

$$\rho_{herd} = \frac{\sigma^2_{herd}}{\sigma^2_{herd} + \sigma^2_{cow} + \sigma^2_{residual}} \quad \text{and} \quad \rho_{cow} = \frac{\sigma^2_{cow}}{\sigma^2_{herd} + \sigma^2_{cow} + \sigma^2_{residual}}$$

RESULTS AND DISCUSSION

Phenotypic variance. Variance components of herd, cow, and residual are provided in table 1. Total variance was the largest for SCM caused by CONT. Most of the variability and incidence of SCM was dependent on the predisposition of the cow and the identification of SCM caused by CONT (table 1). The larger herd and cow variance was caused by the tendency of these CONT to cluster within a cow and within a herd. This spread of CONT within a herd is facilitated by improper milking management techniques and practices (Manninen, 1995). ENV tend not to cluster within a herd and its occurrence is more evenly spread among herds than CONT. ENV are always present in the surroundings of the cow and usually cause mastitis when the immune system is impaired, especially the peri-parturient period. Barkema *et al.* (1998) found that CM caused by ENV was more associated with herds with low BMSCC. High BMSCC herds were likely to develop CM caused by CONT (Barkema *et al.*, 1998).

The variance of SCM caused by ALL was expected to be greater than the variance of ENV and CONT (table 1). However, the opposite was found. The variance of SCM caused by ALL was smaller compared to SCM caused by ENV and CONT, separately. This lower phenotypic variance might point towards a number of different possibilities. The first was a possible data issue, but then all three observations would have been subjected to the same dilemma and results would closely resemble the expectations. Second option was the possible existence of a negative covariance between the effects of ENV and CONT. This negative covariance was not present, when two-trait genetic analysis was performed on ENV and CONT. Clustering might increase the variance but this change was not expected (table 1). The results might also be an indication of not achieving a decrease in the incidence of CM. Because, if selection for decreased SCS and reduced incidence of CM was successful the variances of ENV and CONT would have been lower than ALL.

Intracluster correlation. Intracluster correlation provided information about the relative importance of a variance component compared to the other variance components in the model (table 1). The intra cow correlation indicated that SCM caused by ALL (0.86) was situated between SCM caused by ENV (0.94) and CONT (0.82) (table 1). SCM caused by CONT was more correlated with herd than SCM caused by ALL, and ENV (table 1). This spread of SCM caused by CONT is facilitated by the milking process (Pyörälä, 1995). Whereas, the residual correlation was the highest for SCM caused by ALL.

Table 1. Variance components and the intracluster correlation of the random effects of herd, cow, and residual of SCM of ALL, ENV, and CONT

	Variance components			Intracluster correlation		
	ALL	ENV	CONT	ALL	ENV	CONT
Herd	0.52	0.24	1.75	0.09	0.04	0.16
Cow	4.90	6.05	8.81	0.86	0.94	0.82
Residual	0.30	0.17	0.12			
Total	5.73	6.47	10.68			

CONCLUSION

On the basis of this study, effects of genetic selection programs on the incidence of SCM and CM need to be assessed. Because the size of the variances of ALL, ENV, and CONT might indicate a negative effect of selection for resistance to CM on basis of SCS, MT, and UD, then an increase in the incidence of CM could be expected.

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