GENETICS AND BREEDING

Genetic Parameters for Common Health Disorders of Holstein Cows

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ABSTRACT

Observations on 7416 Canadian Holstein cows were examined to estimate genetic parameters for the most common diseases of dairy cows. Mastitis, ovarian cyst, ketosis, milk fever, abomasal displacement, and culling that is due to reproductive failure or leg problems were analyzed as binomial traits, assuming an underlying threshold model that included fixed and random effects. Sire and residual components of variance were estimated by REML to provide heritability estimates from paternal half-sibs. A multiple-trait mixed model was also used to estimate genetic and environmental correlations between production and disease traits. Heritabilities of disease traits were relatively low and ranged from 0 to .15, except for displaced abomasum ($h^2 = .28$). Evidence of genetic antagonism existed between incidence of mastitis and milk production. Incidence of milk fever was genetically associated with cows of lower genetic potential for production. Genetic associations between displaced abomasum and production traits were small, and estimates of genetic correlations between ovarian cyst and milk production were inconsistent across lactations. Ketosis was antagonistically associated genetically with production of milk and fat but was favorably associated with production of protein. The long-term cumulative effect of genetic selection against diseases might be useful to diminish their incidence.

(Key words: heritability, genetic correlation, milk production, disease incidence)

INTRODUCTION

Milk production has increased dramatically over the last two decades. However, this improvement has not necessarily resulted in proportional increases of profits to dairy farmers. Cows with high milk production have relatively greater demands for labor (11) and increased frequency of health disorders (11, 25). As a result, some North American (26) and Scandinavian (28) dairy researchers have questioned the selection of cows solely on milk production traits and have suggested that some emphasis be placed on other traits, such as health, that contribute to profitability. Inclusion of health traits in selection programs has been limited because of a lack of reliable data concerning health disorders. In addition, the discrete nature of most disease observations makes their statistical analysis and interpretation more difficult (16).

Antagonistic genetic correlations between milk production and disease traits indicate that disease incidence increases as a consequence of genetic improvement of milk production (27). The argument that losses from increased disease incidence can be compensated by additional revenue from higher production has not been completely researched (26). Even if losses that were due to disease can be offset by additional production (11, 25, 34), ethical considerations might not allow researchers or producers to ignore the impact of selection for increased production on the health status and general welfare of cows (26, 29).

Some attempts have been made, principally by Scandinavian researchers (9, 20, 28, 30), to examine genetic aspects of disease traits from large data files recorded in the field. In Canada, the genetic components of diseases in dairy cows, except for SCC, have not been widely investigated.

The intent of this work was to estimate heritabilities of some common clinical diseases of Holstein dairy cows and to measure genetic and environmental correlations between disease and production traits.

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MATERIALS AND METHODS

Health data were provided by the Department of Population Medicine, University of Guelph, with cooperation from the Ontario DHI Corporation and the Ontario Milk Marketing Board. The information was recorded between 1989 and 1991 from herds in southern Ontario enrolled in a research project known as the Ontario Dairy Monitoring and Analysis Program. Its objective was to study relationships among measures for herds of disease, management, production, reproductive performance, and profitability in Ontario. The data were information on 7416 cows in 98 herds.

A cow was considered to be diseased at the first report of a particular disease by the veterinarian or the farmer. The same disease shown more than once during a lactation was considered to be one incidence for analysis. Health traits studied were mastitis, ovarian cyst, ketosis, milk fever, displaced abomasum, and culling for reproductive failure or leg problems.

The production data used for analyses corresponded to the same lactation in which the cow was recorded as having or not having the disease during a given lactation. Production traits were 305-d production of milk, fat, and protein and percentages of fat and protein. Production data were from Agriculture Canada from the data used for evaluation of bulls and cows in Canada and were adjusted for age and month of calving.

Clinical presence or absence of a specific disease generates discrete data with a binomial distribution. Disease observations may be grouped in two ordered categories (having or not having the disease), and susceptibility of cows to a certain disease follows an underlying continuous normal distribution that is not observed. Only cows that exceed some unknown threshold of susceptibility show clinical signs of the disease (6, 23). Gianola and Foulley (8) and Harville and Mee (12) proposed a nonlinear set of equations that are solved iteratively for the values of the thresholds and effects in the model for analysis of categorical data.

A FORTRAN program GFCATI (L. R. Schaeffer, 1992, personal communication) was used to compute the elements and to solve the nonlinear equations (8). Estimation of variance components was by REML (21).

A sire model was used to estimate heritability of each disease trait as follows:

\[ Y_{ijklm} = \mu + H_i + C_j + Y_k + S_l + e_{ijklm} \]

where

\[ Y_{ijklm} = \text{value of observation } ijkml, \]
\[ \mu = \text{population mean}, \]
\[ H_i = \text{fixed effect of herd } i, \]
\[ C_j = \text{fixed effect of calving season } j, \]
\[ Y_k = \text{fixed effect of year } k, \]
\[ S_l = \text{random effect of sire } l \sim N(0, \sigma^2_s), \]
\[ e_{ijklm} = \text{residual error } \sim N(0, \sigma^2_e). \]

Because of the low incidence of diseases, inclusion of herd-year interaction in the model would have generated empty cells, and subclasses containing empty cells must be eliminated from the analysis (12).

First lactation cows differ from multiparous cows; they produce less milk and have different rates of incidence for many diseases (19). Therefore, a separate analysis was performed for primiparous cows if \( \geq 5\% \) of primiparous cows were recorded with the disease. When the frequency of disease was \(< 5\% \) during first lactation, data for primiparous and multiparous cows were pooled to avoid loss of information by empty cells. For the combined analysis of all cows, the model also included the fixed effect of parity. Cows were only represented once; i.e., cows did not have records repeated over lactations. Only sires having at least five daughters with records in at least two herds were considered for analysis. Relationships among sires were ignored. Heritability was calculated as the ratio of four times the sire component of the variance to one plus the sire variance (1).

To assess the genetic relationship between each health trait and the production of milk, fat, and protein and percentages of fat and protein, the data were analyzed using a two-trait mixed sire model for one continuous and one discrete trait using the method of Janss and Foulley (13). The method allowed joint analysis of the continuous and the discrete traits with unequal design matrices and could accommodate missing data on some traits. Estimation of genetic effects was based on
threshold model methodology (12, 13). The extension of the threshold model to include discrete and continuous variables was examined by Foulley et al. (7).

The computations were performed using a FORTRAN program BIVTHM (L. Janss, 1993, personal communication). The statistical model used for the discrete traits was as indicated previously for estimation of heritability. The model used for the continuous traits was as follows:

\[ y_{ijkl} = \mu + HY_i + C_j + S_k + e_{ijkl} \]  

where

- \( y_{ijkl} \) = production record (production of milk, fat, or protein or percentages of fat or protein) from the same lactation as the measure of disease occurrence,
- \( \mu \) = overall mean,
- \( HY_i \) = fixed effect of the herd-year of calving \( i \),
- \( C_j \) = fixed effect of the calving season \( j \) \( (j = 1, 2, 3) \),
- \( S_k \) = random effect of the sire \( k \) ~ N(0, \( \sigma^2_s \)), and
- \( e_{ijkl} \) = random error associated with observation \( ijk \) ~ N(0, \( \sigma^2_e \)).

When data were analyzed across lactations, the fixed effect of parity was included. Sires having records on at least five daughters in two or more herds were used for analysis. Relationships among sires were ignored.

Estimates of genetic correlations were computed from sire (co)variances. Environmental (co)variance was calculated as the residual (co)variance minus three times the sire (co)variance. Environmental correlations between health and production traits were computed from environmental (co)variances.

**RESULTS AND DISCUSSION**

Table 1 shows the total number of original observations and the number actually used for each analysis. One of the operational problems in the analysis of these data was that, for some herds, all of the observations were in one category (no disease), which necessitated removal of records for those herds. Some observations were also lost because of the restriction that sires have at least five daughters in two herds. When sires with fewer records were removed, additional empty cells were generated.

**Clinical Mastitis**

Analyses were based on 81.0 and 95.8% of the available information for first and all lactations, respectively. Heritability of clinical mastitis for first lactation cows was .15, which was similar to the estimates of Lin et al. (14) for Holstein cows and those of Philipsson et al. (22) for Swedish Red and White cows. Heritabilities for this trait were lower for Swedish (4) and Norwegian cattle (27).

<table>
<thead>
<tr>
<th>Health trait</th>
<th>Lactation</th>
<th>Incidence rate (%)</th>
<th>Observations</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(no.)</td>
<td>Total</td>
<td>Used</td>
</tr>
<tr>
<td>Mastitis</td>
<td>1</td>
<td>12.5</td>
<td>2639</td>
</tr>
<tr>
<td>All</td>
<td>All</td>
<td>16.7</td>
<td>5229</td>
</tr>
<tr>
<td>Ovarian cyst</td>
<td>1</td>
<td>7.4</td>
<td>2639</td>
</tr>
<tr>
<td>All</td>
<td>All</td>
<td>9.1</td>
<td>5217</td>
</tr>
<tr>
<td>Ketosis</td>
<td>All</td>
<td>4.1</td>
<td>5181</td>
</tr>
<tr>
<td>Milk fever</td>
<td>All</td>
<td>6.8</td>
<td>5217</td>
</tr>
<tr>
<td>Displaced abomasum</td>
<td>All</td>
<td>2.8</td>
<td>5181</td>
</tr>
<tr>
<td>Culling for leg problems</td>
<td>All</td>
<td>3.5</td>
<td>5217</td>
</tr>
<tr>
<td>Culling for reproduction</td>
<td>All</td>
<td>6.0</td>
<td>5217</td>
</tr>
</tbody>
</table>

\( ^1 \)Sire variance.
TABLE 2. Genetic and environmental correlations between health and production traits.

<table>
<thead>
<tr>
<th>Health trait</th>
<th>Lactation</th>
<th>Milk</th>
<th>Fat</th>
<th>Protein</th>
<th>Fat</th>
<th>Protein</th>
</tr>
</thead>
<tbody>
<tr>
<td>Genetic correlations</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Mastitis</td>
<td>1</td>
<td>.37</td>
<td>.39</td>
<td>.20</td>
<td>.21</td>
<td>-.12</td>
</tr>
<tr>
<td>Ovarian cyst</td>
<td>1</td>
<td>-.14</td>
<td>.50</td>
<td>.11</td>
<td>.76</td>
<td>.57</td>
</tr>
<tr>
<td>Ketonis</td>
<td>All</td>
<td>-.06</td>
<td>-.12</td>
<td>-.43</td>
<td>-.13</td>
<td>-.56</td>
</tr>
<tr>
<td>Milk fever</td>
<td>All</td>
<td>.77</td>
<td>.41</td>
<td>-.30</td>
<td>.21</td>
<td>-.79</td>
</tr>
<tr>
<td>Displaced abomasum</td>
<td>All</td>
<td>-.04</td>
<td>.18</td>
<td>.09</td>
<td>.24</td>
<td>.12</td>
</tr>
<tr>
<td>Culling for leg problems</td>
<td>All</td>
<td>.27</td>
<td>.20</td>
<td>.21</td>
<td>.20</td>
<td>.07</td>
</tr>
<tr>
<td>Environmental correlation</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mastitis</td>
<td>1</td>
<td>-.01</td>
<td>.03</td>
<td>.02</td>
<td>.03</td>
<td>.07</td>
</tr>
<tr>
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<td>.27</td>
<td>.00</td>
<td>.20</td>
<td>-.37</td>
<td>-.23</td>
</tr>
<tr>
<td>Ketonis</td>
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<td>.20</td>
<td>.19</td>
<td>.25</td>
<td>0</td>
<td>.15</td>
</tr>
<tr>
<td>Milk fever</td>
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<td>.02</td>
<td>-.02</td>
<td>.09</td>
<td>-.12</td>
<td>.08</td>
</tr>
<tr>
<td>Displaced abomasum</td>
<td>All</td>
<td>.15</td>
<td>.09</td>
<td>.16</td>
<td>-.08</td>
<td>.04</td>
</tr>
<tr>
<td>Culling for leg problems</td>
<td>All</td>
<td>-.10</td>
<td>-.07</td>
<td>-.17</td>
<td>.05</td>
<td>-.12</td>
</tr>
</tbody>
</table>

1First lactation.
2All lactations.

Heritability of clinical mastitis for all cows was 0, which is in agreement with results of Dohoo et al. (3), but contrary to those of Lin et al. (14), who obtained a heritability estimate of .18 for cows after their second lactation. Heritability estimates of clinical mastitis across lactations of Holstein cows have ranged from low, .03, to moderate, .14 (15, 31).

Genetic and environmental correlations between clinical mastitis during first lactation and production traits are presented in Table 2. Production of milk, fat, and protein was positively correlated with incidence of mastitis for first lactation cows. The genetic correlation between mastitis and milk production of .37 for first lactation cows was similar to the .38 found by Syväjärvi et al. (30) for first lactation Finnish cows. Higher estimates have been reported: Simianer et al. (27) found a correlation of .51 for primiparous Norwegian cows, and Syväjärvi et al. (30) found a correlation of .57 across all lactations for Finnish Ayrshire cows. In contrast, Lyons et al. (15) estimated the genetic correlation between mastitis and milk production to be .18 across lactations, Thompson (31) found a correlation of .26 for Holstein cows across lactations, and Bunch et al. (2) reported a correlation of .21 for first lactation cows.

The estimated genetic correlation between fat production and mastitis for first lactation cows was .39, which is in contrast to results of Lyons et al. (15), who found a genetic correlation of 0. Genetic correlation between protein production and mastitis during first lactation in this study was .20 (Table 2).

Genetic correlation between fat percentage and clinical mastitis for first lactation cows was .21, which is contrary to the genetic correlation of -.15 reported by Simianer et al. (27) for first lactation Norwegian cows. In contrast to fat percentage, the genetic correlation between protein percentage and mastitis incidence was negative, -.12.

Environmental correlations (Table 2) between mastitis and production traits during first lactation were all low. Environmental correlations between mastitis and percentages of fat and protein were low for first lactation Norwegian cows (27).

Heritabilities estimated from the multivariate analyses for first lactation mastitis and production traits are given in Table 3. Estimates of heritability of mastitis during first lactation were similar for each of the five analyses with production traits and were very similar to the .15 obtained from the single-trait analysis (Table 1).
The heritability estimates of .15 for clinical mastitis during first lactation suggest considerable genetic variability. Also, clinical mastitis for first lactation cows was positively correlated with the three production traits, which supported the concern of other studies that continued selection for increased production, ignoring information on diseases, might be detrimental to long-term cow health. Despite the small antagonism between production and mastitis, its cumulative effect over several generations might be important.

**Ovarian Cyst**

For number of herds, no cases of ovarian cysts were reported; when ovarian cysts were reported, they occurred in the daughter of a sire with <5 daughters. Thus, the analyses used only 55.1 and 88.9% of the available information for first and all lactations. Sire components of estimates of variance and heritability for cystic ovarian disease are in Table 1.

Heritability of ovarian cyst was .13 for first lactation cows and .08 over all lactations. These estimates were very similar to respective estimates of .12 and .07 reported by Lin et al. (14) for US Holstein cows. The heritability estimate computed herein for ovarian cyst for first lactation cows is larger than that of .05 found by Erb et al. (5) and that of .01 found for ovulatory disorders of primiparous Finnish cows (17). However, in the latter study, the trait also included other infertility problems. Dohoo et al. (3) indicated that heritability of ovarian cyst for Holstein cows was 0. Lyons et al. (15) found a heritability of .05 for ovarian cysts across lactations of Holstein cows.

Genetic and environmental correlations between cystic ovarian disease and production traits are given in Table 2. For first lactation cows, the estimated genetic correlations between ovarian cyst and production of milk, fat, and protein were -.14, .50, and .11, respectively. For all lactations, respective genetic correlation estimates were -.06, -.12, and -.43. Lyons et al. (15) estimated genetic correlations between cystic ovaries and production of milk and fat of -.01 and .24, respectively. Mäntysaari et al. (17) found genetic correlations between milk production and ovulatory disorders of .65 and .02 for Finnish cows during their first and second lactations, respectively. Genetic correlations (Table 2) between ovarian cyst and percentages of fat and protein for first lactation cows were high and positive, .76 and .57, but were negative over all lactations, -.13 and -.56.

Environmental correlations during first lactation were .27, 0, and .20 between cystic ovaries and production of milk, fat, and protein, respectively, and were in a similar range over all lactations (Table 2). Environmental correlations between ovarian cysts and percentages of fat and protein were moderate and negative during first lactation, -.37 and -.23, respectively, and close to 0 over all lactations, 0 and .15, respectively.

Heritability estimates from the multtrait analysis for ovarian cyst and production traits are given in Table 3. Estimates ranged from .12 to .22. Mean heritability of the five estimates was .17. Heritability of ovarian cyst was similar to that for mastitis (.13). Genetic variation of cystic ovaries might be higher than previously thought for other traits related to fertility.

Genetic correlation estimates between cystic ovaries and production traits were inconsistent except for those with milk production. The small and negative correlation with milk production (Table 2) indicated that selection for high volume of milk was not antagonistic or predisposing to the disease.

**Ketosis**

The frequency of clinical ketosis was very low for first lactation cows. Loss of informa-
tion because of empty cells provided too few data for first lactation cows for meaningful analyses. Therefore, sire components of variance and the heritability of clinical ketosis are across all lactations in Table 1. Nevertheless, only 38.3% of the 5181 observations available were used for analysis.

Heritability of clinical ketosis was .09, which is in agreement with results of Philipsson et al. (22), who found heritabilities of .06 and .09, respectively, for first lactation Swedish Red and White and Swedish Friesian, and those of Lyons et al. (15), who reported a heritability for ketosis of .08 for US Holsteins. Heritabilities across lactations were similar for Finnish Ayrshires (9) and first lactation Norwegian cows (27). Estimates by Solbu (28) for Norwegian cows were smaller than that of the present study and averaged .02 over subsets of data corresponding to different years. Heritabilities were smaller, .02, for Finnish Ayrshires (10) than those estimated in the present study. Recently, heritabilities for ketosis were estimated to be .02 and .03 for Finnish cows during their first and second lactations, respectively (18). Heritability for ketosis estimated in the present study, however, was much lower than estimates of .30 and .31 by Van Vleck (33) and Dohoo et al. (3), respectively.

Genetic and environmental correlations between ketosis and production traits across lactations are in Table 2. The highest genetic correlation, .77, was between ketosis and milk production. This correlation was higher than the estimates of Gröhn et al. (9) for first lactation Finnish Ayrshire cows, .30, Lyons et al. (15) for US Holsteins, .26, and Mäntysaari et al. (16) for primiparous Finnish cows, .17. However, this estimate was close to the .65 of Simianer et al. (27) for first lactation Norwegian cows. Genetic correlation between fat production and incidence of ketosis was .41. Protein production was moderately and negatively correlated, -.30, with the disease.

Ketosis was moderately and positively correlated, .21, with fat percentage (Table 2). The genetic correlation between protein percentage and ketosis was high and negative, -.79. Simianer et al. (27) reported genetic correlations between ketosis and percentages of fat and protein of -.38 and -.65, respectively. Environmental correlations between ketosis and the production traits were all small and ranged from -.02 to .09 (Table 2).

Heritability estimates from the multiple-trait analyses are in Table 3. Heritability of clinical ketosis was the same for four of the five analyses, .09, and was very similar to that found by the single-trait analysis. However, heritability from the multiple-trait analyses involving protein percentage was .16.

The 2% incidence of this metabolic disease was much lower than that in the literature. The genetic component of variation for ketosis across lactations was low (h² = .09). This estimate is consistent with most of the literature and indicates that environmental factors, particularly nutritional management, are more important in the short term for control of ketosis than is genetics. Nonetheless, genetic variability exists for ketosis.

Increased production of milk and fat were highly positively correlated with incidence of ketosis. This finding indicates that, even if heritability of ketosis is low, selection schemes based solely on production of milk and fat should result in a long-term increase in the frequency of clinical ketosis. However, ketosis and protein production showed a moderately negative genetic correlation, indicating that selection to favor protein production reduces incidence of ketosis.

**Milk Fever**

Because of its low frequency, the trait for milk fever was also analyzed only across lactations. Information from 154 sires with records in 82 herds were used for analysis, which represented 90% of the available information (Table 1). Incidence of milk fever in the final data was 319, which represented a rate of 6.1%. The sire variance component was .023, and heritability was .09.

Milk fever is a syndrome produced by a sudden decrease in concentration of Ca in plasma at calving. Heritability for milk fever across lactations in this study was smaller than the estimate of Tveit et al. (32) based on postparturient concentration of Ca in plasma of Norwegian cows, although the traits are not exactly the same. Lyons et al. (15) found a much higher heritability estimate for milk fever across lactations, as did Lin et al. (14), who found heritabilities for Holstein cows from parities ≥2 of .30 and .42, respectively. In contrast, Philipsson et al. (22) found no genetic
variation for incidence of milk fever for two breeds of Swedish cows.

Genetic and environmental correlations between milk fever and production traits are given in Table 2. The three production traits were negatively associated genetically with milk fever. Milk production and milk fever were highly negatively correlated, \(-.67\). Gröhn et al. (9) also found negative genetic correlation between parturient paresis and milk production, but their estimate was smaller, \(-.09\). However, Tveit et al. (32) found an undesirable negative correlation, \(-.5\), between postpartum concentration of Ca in blood and milk production. The genetic association between production of fat and protein and milk fever in this study were moderately to largely negative, \(-.21\) and \(-.66\), respectively, and larger than that found by Lyons et al. (15), \(-.01\). These results suggest that cows with superior genetic potential for production of milk and protein might be more resistant to the disease. Genetic correlations between milk fever and fat and protein percentage were \(.29\) and \(-.24\), respectively.

Pooled heritability of milk fever across the five multiple test analyses was \(.10\) (Table 3) and was similar to that from the single-trait analysis (\(.09\)) (Table 1). All multiple-trait estimates were similar: \(.08\) to \(.11\).

**Displaced Abomasum**

Heritability of displaced abomasum across lactations was \(.28\) (Table 1), which was considerably higher than the \(.09\) estimated by Lyons et al. (15). Genetic and environmental correlations between displaced abomasum and production traits are in Table 2. The genetic correlation between displaced abomasum and milk production in the present study was low and negative: \(-.04\). Lyons et al. (15) also found a negative but larger genetic correlation, \(-.15\), between these traits across lactations for US Holsteins. The genetic correlation between fat production and displaced abomasum was \(.18\), which is in disagreement with results of Lyons et al. (15), who found a negative association between these traits, \(-.28\). Protein production and displaced abomasum were positively associated, but the correlation was small, \(.09\). Genetic correlations between displaced abomasum and fat and protein percentage were moderate, \(.24\), and small, \(.12\), respectively. Similar to the analysis of heritability of displaced abomasum using the single-trait analysis (Table 1), multiple-trait estimates of heritabilities were high and consistent for all five analyses, \(.30\) to \(.31\) (Table 3).

Displaced abomasum has not been extensively studied genetically. Its heritability estimated from almost 3000 records was moderate \((.28)\) and indicates that, if records were available, genetic selection could reduce the incidence of the disease. Long-term selection for fat production might tend to increase the frequency of displaced abomasum because of the small positive correlation, \(.18\). Genetic correlations between displaced abomasum and production of milk and protein were very small, and the traits should be independent for selection.

**Culling for Leg Problems**

Culling for leg problems had a heritability of \(.15\) across lactations (Table 1). No other references of heritability of this trait were found in the literature. Lyons et al. (15) estimated heritabilities for leg and foot problems at \(.08\) and \(.11\), respectively. Genetic correlations between culling for leg problems and production of milk, fat, and protein were positive and moderate; environmental correlations were negative (Table 2). Genetic correlations between culling for leg problems and percentages of fat and protein were moderate, \(.20\), and low, \(.07\), respectively; corresponding environmental correlations were negative.

Culling for leg problems would normally occur near the end of a lactation, and a negative correlation might be expected unless leg problems were very severe, causing cows to be culled regardless of milk production. Genetic correlations between culling for leg problems and production of milk, fat, and protein were positive (Table 2), indicating that long-term selection for these traits might increase culling for impaired legs.

**Culling for Reproductive Failure**

Cows were culled from the herd for reduced reproductive performance. As with ketosis and milk fever, the incidence of this disorder was very low for first lactation cows; therefore, the analysis was only performed across lactations.
No genetic variation was found for this trait (Table 1). Although the literature does not contain many reports on this trait, heritabilities for individual reproductive traits were low (15, 17, 18, 33).

Because this trait showed no genetic variability, selection would not have an important role in improvement of general fertility of dairy cattle. This conclusion is consistent with the literature, which indicates very low heritability of reproductive disturbances (15, 17).

Production Traits

Multiple-trait heritabilities of production traits are in Table 3. Estimates were similar to those of some previous reports (24, 27). The estimate for milk production tended to be low. Considerable variability occurred among analyses for estimates of heritability. Estimates were obtained using a sire model and ignored relationships among sires.

General Discussion

As with many of the other studies of health disorders, in this work the sample size used was small for determination of genetic relationships. As with most other genetic studies related to disease, other shortcomings were that relationships among sires were ignored in the analyses, and standard errors of the estimates could not be computed. Ideally, these data should have been analyzed considering relationships among animals; unfortunately, computing packages to analyze binary data under an animal model were not available at the time of the study.

Also, data on multiparous cows are potentially subject to selection bias. Only cows with acceptable milk production and health status survive for later lactations, and such selection can influence estimates of genetic parameters. Unfortunately, it is difficult to correct for this selection when data are limited to a relatively short period and historical records on all cows are not available.

Direct comparison of results across studies is difficult with this type of data because the recording systems vary across studies and different traits are often recorded. The diagnosis of a particular disease is somewhat subjective. The categorization of a cow as diseased depends on the ability and experience of the veterinarian, the farmer, or both in recognizing the disorder. Also disease recording was on the basis of treatment, particularly for mastitis, milk fever, and ketosis; i.e., treatments for disease were recorded, but the diagnosis of disease was not necessarily recorded. An over recording of milk fever may be implied; some cows in some herds are treated prophylactically with Ca whether or not they show signs of hypocalcemia. However, mastitic cows that were considered by the owner to have mild clinical mastitis and were untreated were not necessarily recorded on the data sheet. This procedure undoubtedly resulted in an underestimate of clinical mastitis incidence in some herds. An underlying feature of the present study is that all farms were serviced by veterinarians who were enrolled in the same continuing education program given by the Ontario Veterinary College.

A number of antagonistic correlations were suggested between disease traits and measures of milk production. Measurement of the true production potential of cows is also difficult, particularly if the diseases of concern are likely to influence production. In our study, milk fever, ketosis, and displaced abomasum occurred early in the lactation and could have had considerable impact on milk production during the rest of the lactation. This impact would be particularly important for cows with displaced abomasum because they would undergo a surgical procedure and the essential postsurgical treatments.

In general, most of the parameters estimated herein are within the range of those indicated in previous studies. The heritability estimate was highest, .28, for displaced abomasum. Although conclusions vary, genetic variation for most of the diseases was sufficient to be potentially useful in programs for genetic selection. With few exceptions, however, heritabilities were small; therefore, short-term management practices focused to provide an adequate environment for the cows should continue to be the most important and logical means to prevent these diseases. However, the long-term cumulative effect of genetic selection could significantly affect the permanent improvement of disease resistance of dairy cows.

Some diseases are negatively correlated with production traits, indicating that selection schemes might have to trade some genetic
improvement for production to gain health status. Because of the relatively low heritability of diseases, the current North American progeny-testing schemes, in which relatively few daughters of each sire are tested, do not allow for much effective emphasis on health-related traits for selection.

Feasibility of including diseases in indexes for dairy selection depends on the economic importance of a specific disease. Economic considerations might differ over countries or regions. Also, ethical considerations could require the dairy industry to improve animal welfare by alleviation of health disturbances, although this might not be the best economic option.

Recently, statistical approaches to analyze discrete data have been developed to the extent that they can be applied to field data for animal breeding, and enhanced computing technology allows the application of these methods. However, in North America, programs to collect data pertaining to diseases have not been as comprehensive as in Scandinavian countries.

CONCLUSIONS

Genetic variability, although relatively small, was sufficient for most diseases studied to be potentially useful in breeding programs. Genetic antagonisms between some diseases and production traits suggest the need to consider disease implications in long-term improvement programs and possibly disease measures for selection.

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