Clinical Ketosis: Phenotypic and Genetic Correlations Between Occurrences and with Milk Yield

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ABSTRACT

The repeatability and heritability of ketosis were estimated using data from 28,277 Finnish Ayrshire cows. A four-trait linear model including community-year, calving age and month, genetic group, and random sire effects was used to describe first and second lactation milk yields and veterinary diagnoses of ketosis. Variance components were estimated using REML. The disease traits were also analyzed with a categorical model including the same effects except that community and year were separate factors. Variance components were estimated with marginal maximum likelihood. Genetic relationships between 339 sires analyzed were included in models. The phenotypic correlation between the first and second lactation was defined as a repeatability of trait. The lactational incidence risk of ketosis was .05 in both the first and the second lactation. Average milk production was 4956 and 5547 kg in the first and second lactations, respectively. Estimates of heritabilities were .09 and .07 for ketosis and .23 and .19 for milk in the first and second lactations, respectively. Genetic correlations between first and second lactation recordings were .64 for ketosis and .93 for milk. Repeatabilities between subsequent lactations were .36 (.13 in linear analysis) for ketosis and .68 for milk. In the first lactation, genetic relationship between milk yield and ketosis was unfavorable, but in the second lactation ketosis and milk yield were genetically and phenotypically unrelated.

(Key words: repeatability, ketosis, Bayesian method)

INTRODUCTION

Milk production has in recent years become much more dependent on intensive methods of husbandry. Understanding the relationships among disease, production efficiency, and the genetic background of the individual cow is central to the dairy industry. Until recently, research on these questions has been limited because of a lack of comprehensive data, computer capacity, and statistical techniques for characteristics observed only in discrete categories. Evolving health data programs in Scandinavia, North America, and Israel have provided valuable data for epidemiologic research. These data also contribute health information for genetic selection programs. Many economically important diseases exhibit genetic variation (7), but the proportion of the total variation that is genetic (heritability) is small. Therefore, the evaluation of the animals has to be based on progeny testing and sophisticated statistical methods. Knowledge, not only of the heritabilities of the disease traits, but also of repeatabilities, is needed for these evaluation programs to use information from several lactations. In addition to the animal evaluation purpose, the knowledge of the repeatability of diseases is important for management decisions.
The crudest way to access the repeatability of all-or-none traits is a cross-tabulation of data with respect to the first and second occurrence of disease. From these tables, the risk for developing the disease later can be calculated conditionally on the previous history (29). However, such results are not corrected for other confounding factors. A more refined method is to use earlier disease as a predictor variable for the later disease (26). This method allows inclusion of confounding effects in the model but still limits the estimation to data sets in which all animals have two complete lactations. Analyzing only cows with both lactations may also introduce bias. If a disease increases the risk of culling, directly or indirectly, the subset of animals with both lactations is not a representative sample of the whole population, and, for instance, the observed incidence in the first lactation will be underestimated.

Substantial theoretical and methodological progress has been made in recent years in the analysis of qualitative data. Logistic regression and log-linear analysis have been made widely available to researchers via texts (17). These methods have been applied primarily to fixed effects models. In genetic and epidemiologic studies, the underlying models are frequently mixed; i.e., they include both fixed and random factors. New procedures for analysis of discrete outcomes with underlying mixed or Bayesian models have been introduced by several authors (9, 11, 15). Common to these methods is the assumption of an underlying continuous, normal or logistic, liability variable, which is used to model probabilities of discrete outcomes.

Foulley et al. (9) proposed a multivariate method that allowed joint analysis of three variables, two continuous and one binary. In such models, genetic correlations between normal variables, i.e., milk yield and dichotomous trait (i.e., disease occurrence), can be evaluated. Later, Foulley et al. (10) extended methodology to bivariate all-or-none cases, opening access to correlations, such as repeatability, between binary variables.

Because of computational demands, the bivariate or multivariate methods previously mentioned have not yet been used on practical field data. Even in the univariate analysis of categorical traits, the computational burden is enormous. The algorithms proposed (11, 15) require reformulation of the matrix of second derivatives, or their expectations, of posterior density within each round of iteration. Animal evaluations on practical breeding work might involve hundreds of thousands of equations; thus, the matrix of second derivatives is just too large for current computers.

For genetic evaluations, the work of Zhao (30) opened new possibilities. Her computational algorithm was based on expectation-maximization (3), thus avoiding the need for second derivatives at least in the case of binary traits. However, before any practical applications of threshold models are to be used, estimates of genetic parameters in underlying scale are needed. The usefulness of the variance and covariance component estimation methods based on a threshold model were recently studied on simulated data (20). The current study applies this technique on field data.

Our objective was to estimate the repeatability and heritability of clinical ketosis in the first and second lactation and the relationship between ketosis and milk yield. The repeatability was defined to be the phenotypic correlation between the observations of the trait in the subsequent lactations with no reference as to whether the trait could genetically be considered as a single trait. For the analysis of disease traits, a two-trait categorical model based on threshold concept (4, 10) was used. The model included several independent variables that were assumed to be confounders and allowed use of information on animals with second lactation observation missing. Results obtained with the categorical method were compared with REML estimates. These were calculated with a four-trait linear model in which the first and the second lactation milk yields were also included as dependent variables.

MATERIALS AND METHODS

Data

The data were obtained from the Finnish health data recording system, which has been described previously (12). The data were from Finnish Ayrshire cows that calved for the first time in 1983 through 1985. The data on 23
veterinary diagnoses were collected from 2 d before the first calving through the second lactation or removal from the herd. For disease follow-up period, at least 6 mo after calving were required. The data included only those diagnoses made according to ordinary clinical methods under field conditions by veterinarians during farm visits. Only the first diagnosis of each disease in each lactation was considered, and repeated treatments or treatments by telephone prescription (i.e., without the veterinarian seeing the cow) were excluded. Only ketosis diagnosis was considered in the current analysis. All cows were pedigreed, bred by artificial insemination, and in herds that recorded milk production. Measures of production were 305-d complete, uncorrected, milked twice daily, milk yields. The herds were from the 80 (out of 461) communities that were judged to have the best record keeping (12). From 78,017 cows in these communities, only cows with proper records, cows that had sires with at least 50 daughters, and cows that were born before their sires received their first progeny test evaluations were used. After these edits, the final data set included 28,277 daughters of 339 sires. Average milk production was 4956 kg ± 917 (6095 kg as 3.5% FCM) in the first lactation (n = 20,659) and 5547 kg ± 1092 (6805 kg as 3.5% FCM) in the second lactation (n = 10,648).

Statistical Analysis

Analyses were performed in two parts. First, a linear multivariate model was used to describe the first and the second lactation disease outcomes and milk yields. Then, a categorical model yielding marginal maximum likelihood estimators of variance components in underlying scale (10) was used to analyze two disease variables. The following linear model was used:

\[ Y_{i j k m n}^t = \alpha_{i} + \alpha_{j} + \alpha_{k} + \varepsilon_{i j k m n}^t \]

where \( Y_{i j k m n}^t \) was an observation on trait \( t \), \( \alpha_{i} \) a community-year, \( \alpha_{j} \) calving age, \( \alpha_{k} \) calving season, \( \varepsilon_{i j k m n}^t \) genetic group, \( u_{m(n)}^t \) a random sire, and \( e_{i j k m n}^t \) a random residual effect for trait \( t \) (\( t = 1, \ldots, 4 \)). For the disease traits, \( Y_{i j k m n}^t \) was either 0 or 1. Age, season, and genetic group had six classes each for every trait (see Table 1). Genetic group also had six classes and was defined to be the birth year of the sire of the cow. It was assumed that environmental effects were fixed and that the only random effects in the model were sire and residual. The distribution of sire vector \( u \) and residual vector \( e \) was assumed to be multivariate normal with zero means and with \( \text{var}(u) = A \otimes G_0 \), \( \text{var}(e) = I \otimes R_0 \), and \( \text{cov}(u,e) = 0 \), where \( A \) is a matrix of additive relationships among sires, \( G_0 \) and \( R_0 \) are the 4 by 4 (co)variance matrices for sire and residual effects, and \( \otimes \) denotes direct product.

(Co)variance matrices, \( G_0 \) and \( R_0 \), were estimated using REML. Solutions were iterated using an expectation-maximization algorithm as described by Mäntysaari and Van Vleck (22).

In the categorical analysis, the model was redefined to describe the underlying liability variables \( Y_{n1} \) and \( Y_{n2} \) for animal \( n \). The model equation was the same as Equation [1], except that community-year effect was separated into two factors, community and year. According to the threshold concept (4), all animals that had their underlying liability values below a certain threshold were observed as ketotic cases, and all the animals that had their liability values above threshold were observed healthy. In a case of two observations, all the animals could be categorized into six different categories with respect to diseases: \{(-1,-1), (-1,+1), (1,-1), (1,1), (-1,0), (+1,0)\} where, for example, the cows that were not ketotic in the first lactation but contracted ketosis in the second lactation belong to the category \((-1,+1)\); the last two categories correspond to the animals with only the first observation recorded. The probability of the response in a particular category is determined by the values of \( Y_{n1} \) and \( Y_{n2} \) jointly. For example, the probability of the category \((-1,+1)\) is

\[ \text{Prob}\{(-1,+1)|Y_{n1}, Y_{n2}\} = P\{Y_{n1} \geq x_1 \text{ and } Y_{n2} \leq x_2\} \]

where \( x_1 \) and \( x_2 \) are the threshold values.

Using the threshold concept, the joint posterior distribution of the parameters was formed (8). This was maximized with respect to the location parameters to give their
TABLE 1. Lactational incidence risk of ketosis in Finnish Ayrshire cows by month and age of calving and corresponding maximum likelihood (ML) solutions from multivariate threshold model.

<table>
<thead>
<tr>
<th>Calving month</th>
<th>First (n = 28,277)</th>
<th>Second (n = 16,789)</th>
<th>First SD1</th>
<th>Second SD1</th>
</tr>
</thead>
<tbody>
<tr>
<td>January-February</td>
<td>8.31</td>
<td>8.40</td>
<td>-0.78</td>
<td>-0.78</td>
</tr>
<tr>
<td>March-April</td>
<td>6.22</td>
<td>7.60</td>
<td>-0.63</td>
<td>-0.70</td>
</tr>
<tr>
<td>May-June</td>
<td>1.88</td>
<td>1.74</td>
<td>-0.08</td>
<td>0.07</td>
</tr>
<tr>
<td>July-August</td>
<td>1.54</td>
<td>1.22</td>
<td>0.0</td>
<td>0.16</td>
</tr>
<tr>
<td>September-October</td>
<td>3.81</td>
<td>3.62</td>
<td>-0.41</td>
<td>-0.32</td>
</tr>
<tr>
<td>November-December</td>
<td>6.87</td>
<td>7.13</td>
<td>-0.69</td>
<td>-0.66</td>
</tr>
<tr>
<td>Calving age, d</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>550-649</td>
<td>4.58</td>
<td>...</td>
<td>0.01</td>
<td>0.07</td>
</tr>
<tr>
<td>650-699</td>
<td>5.36</td>
<td>...</td>
<td>-0.09</td>
<td>0.06</td>
</tr>
<tr>
<td>700-749</td>
<td>4.80</td>
<td>...</td>
<td>-0.10</td>
<td>0.06</td>
</tr>
<tr>
<td>750-799</td>
<td>4.40</td>
<td>...</td>
<td>-0.11</td>
<td>0.07</td>
</tr>
<tr>
<td>800-849</td>
<td>3.41</td>
<td>...</td>
<td>0.0</td>
<td>...</td>
</tr>
<tr>
<td>850-1500</td>
<td>3.27</td>
<td>...</td>
<td>0.06</td>
<td>0.09</td>
</tr>
<tr>
<td>800-999</td>
<td>...</td>
<td>2.70</td>
<td>...</td>
<td>0.36</td>
</tr>
<tr>
<td>1000-1049</td>
<td>...</td>
<td>5.13</td>
<td>...</td>
<td>0.10</td>
</tr>
<tr>
<td>1050-1099</td>
<td>...</td>
<td>5.16</td>
<td>...</td>
<td>0.09</td>
</tr>
<tr>
<td>1100-1149</td>
<td>...</td>
<td>5.55</td>
<td>...</td>
<td>0.05</td>
</tr>
<tr>
<td>1150-1199</td>
<td>...</td>
<td>6.01</td>
<td>...</td>
<td>0.10</td>
</tr>
<tr>
<td>1200-2000</td>
<td>...</td>
<td>7.19</td>
<td>...</td>
<td>-0.09</td>
</tr>
</tbody>
</table>

1Square root of the corresponding diagonal element of inverse of the coefficient matrix.

RESULTS AND DISCUSSION

Occurrence

The risk of ketosis was approximately the same as in the previous Finnish studies (13, 14). Altogether, 30% (8483) of the first lactation cows and 28.6% (4802) of the second lactation cows were treated at least once by a veterinarian for some disease (Table 2). Reproductive disorders, mastitis, and ketosis were the most common veterinary treatments of the cows in both lactations. Cows treated for ketosis were 4.7 and 5.4% of the total first and second calvings. These cows made up 15.6 and

Figure 1. Predicted lactational incidence risks of ketosis by month of calving for first and second lactation Finnish Ayrshire cows. Predicted values are calculated from the threshold model as \( \Phi \) (maximum likelihood solution + constant) where constant is chosen such that the predicted and observed values are equal in the March to April class.
TABLE 2. Risks of cases treated by veterinarians for 28,277 Finnish Ayrshire cows that calved the first time in 1983 through 1985.

<table>
<thead>
<tr>
<th>Diagnosis</th>
<th>Lactational incidence risk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>First (n = 28,277)</td>
</tr>
<tr>
<td>Dystocia</td>
<td>1.4</td>
</tr>
<tr>
<td>Parturient paresis</td>
<td>0.01</td>
</tr>
<tr>
<td>Ketosis</td>
<td>4.7</td>
</tr>
<tr>
<td>Metritis</td>
<td>4.1</td>
</tr>
<tr>
<td>Acute mastitis</td>
<td>5.8</td>
</tr>
<tr>
<td>Ovulatory dysfunction(^1)</td>
<td>13.0</td>
</tr>
<tr>
<td>Reproductive disorders(^2)</td>
<td>16.1</td>
</tr>
<tr>
<td>All first treatments</td>
<td>30.0</td>
</tr>
</tbody>
</table>

\(^1\)Includes diagnoses for silent estrus (anestrus or subestrus), cyst, and other types of infertility (those which were not included in the former diagnoses).

\(^2\)Includes diagnoses for ovulatory dysfunction, metritis, and abortion.

That the community-year effect in the model did not remove as much of the environmental variation as the herd-year or herd-year-season factors used in other studies. Community-year effect was used because a large number of the herd-year subclasses would have had no reported cases of ketosis. In the categorical analysis, this would lead to the elimination of the observations in these herd-years.

Heritability estimates for ketosis by a linear model were similar to previous studies (7). Grün et al. (13) reported heritability for ketosis for both the first and the second lactation to be the same, 0.02. Their analysis was done with Finnish health recording data, but with Henderson's method III.

Genetic correlation between the first lactation ketosis and the milk production in both lactations were found to be moderate (+0.17). Second lactation ketosis seemed to be uncorrelated with the yields. These estimates were lower than usually reported for the yield and ketosis relationship (7, 13). The sampling variance of the genetic correlations is large when the heritability of one or both traits is low.

The phenotypic correlation between ketosis and milk yield in both lactations was close to zero. The phenotypic correlation between milk yield in the first and second lactation (0.68) was clearly higher than usually observed (19, 25). The explanation for the high repeatability estimate may be the inadequacy of the model to remove the herd variation, which entered the covariance between consecutive measures. The
TABLE 3. Restricted maximum likelihood estimates of phenotypic standard deviations, heritabilities,\(^1\) genetic and phenotypic correlations\(^2\) for occurrence of ketosis in the first (ketosis\(_1\)) and second (ketosis\(_2\)) lactations and milk yield in the first (milk yield\(_1\)) and second (milk yield\(_2\)) lactations for 28,277 Finnish Ayrshire cows that calved the first time in 1983 through 1985.

<table>
<thead>
<tr>
<th>Trait</th>
<th>SD</th>
<th>Ketosis(_1)</th>
<th>Milk yield(_1)</th>
<th>Ketosis(_2)</th>
<th>Milk yield(_2)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketosis(_1)</td>
<td>.209</td>
<td>.02</td>
<td>.17</td>
<td>.26</td>
<td>.17</td>
</tr>
<tr>
<td>Milk yield(_1)</td>
<td>839</td>
<td>.01</td>
<td>.23</td>
<td>-.03</td>
<td>.93</td>
</tr>
<tr>
<td>Ketosis(_2)</td>
<td>.218</td>
<td>.13</td>
<td>.02</td>
<td>.03</td>
<td>.00</td>
</tr>
<tr>
<td>Milk yield(_2)</td>
<td>1036</td>
<td>.01</td>
<td>.68</td>
<td>.01</td>
<td>.19</td>
</tr>
</tbody>
</table>

\(^1\)Heritabilities are on the diagonal.
\(^2\)Genetic correlations are above diagonal, phenotypic below diagonal.

genetic correlation between milk production in the first lactation and milk production in the second lactation was found to be high (.93).

The estimates from the categorical method are in Table 4. To make the estimation procedure numerically more stable, three communities that did not have reported cases of ketosis in the first lactation were deleted. Solutions were obtained after 65 rounds of iteration when the convergence criterion was fulfilled. Convergence was slow for sire variances and very slow for genetic covariance.

As expected, the categorical method gave greater heritability and correlation estimates than the linear model (Tables 3 and 4). The treatment occurrence of ketosis is a typical all-or-none trait, and the estimate in the observed scale is a function of the incidence (4). When a trait with an underlying heritability \(h^2\) is estimated from binary observations with an incidence \(p\), the observed \(h^2 = z^2 \times h^2/\pi(1 - p)\) where \(z\) is an ordinate of a standard normal distribution evaluated at cutoff point \(p\). This approximation held well for the observed first lactation ketosis, but if the second lactation underlying heritability estimate, .07, was transformed to observed, the value, .016, slightly underestimated the observed value, .03.

The heritability estimates (Table 4) were in good agreement with those corrected to the normal scale in the literature. Solbu (28) found a slightly lower value, .05, but the earlier estimates from the Finnish data (13, 14) were at the same level as ours. Dohoo et al. (6) reported a significantly higher estimate of .31.

Repeatability of ketosis from the first to the second lactation was relatively high, .36, when measured in the hypothetical underlying level

Figure 2. Predicted and observed lactational incidence risk of ketosis by age of calving for 28,277 Finnish Ayrshire cows in the first lactation. Predicted values are calculated from the threshold model as \(P(\text{solution} + \text{constant})\) where constant is chosen such that the predicted and observed values are equal in the class of 650 to 699 d.

Figure 3. Predicted and observed lactational incidence risk of ketosis by age of calving for 16,789 Finnish Ayrshire cows in the second lactation. Predicted values are calculated from the threshold model as \(P(\text{solution} + \text{constant})\) where constant is chosen such that the predicted and observed values are equal in the class of 1000 to 1049 d.
differences in resistance to diseases, no work is known in which both phenotypic and genetic components of repeatabilities, or relationships among lactations in disease susceptibility, have been quantified.

Although veterinarians usually do all medical treatments in Finland, it is likely that the observed risk of ketosis underestimates the true occurrence. If a farmer treats a mild case, or if subclinical cases are not recognized, those cows will be included within the nonketotic group. Analyzing blood acetoacetate concentration in early lactation, Kauppinen (16) found the prevalence of 13%, and Lindström et al. (18), using commercial milk ketone body test, reported 11.5% of prevalence for ketosis in Finnish dairy cows. The potential misclassification of ketosis leads to the underestimation of the calculated heritability and phenotypic and genetic correlations.

Observing Repeatability

The repeatability estimates can be used to calculate the risk of ketosis in the subsequent lactation. In the calculation, we can consider different incidence levels of ketosis. For instance, at the incidence level of 5% (Table 2), the cows treated for ketosis in the first lactation have a 17% chance of being treated for ketosis in the second lactation, whereas the cows not treated for ketosis in the first lactation have a 4.4% chance of being treated for ketosis in the second lactation. This gives an incidence rate ratio (17) of 3.9. Solbu (29) reported an empirical second lactation incidence of 36.6% for the cows with ketosis in the first lactation and 10.6% for the cows without the history of ketosis in the first lactation (incidence rate ratio = 3.5). Bendixen et al. (1) found an increased recurrence risk of ketosis from 4.4 to 12.3 depending on parity and breed. Markusfeld's (23) estimate for the recurrence of ketonuria was 2.0. His estimate was stratified by farm, parity, season, occurrence of metritis, retained placenta, and twinning. Because diseases related to ketonuria were included in the model, the estimate of recurrence risk was underestimated. The corresponding unstratified risk ratio was 3.1. Rowlands et al. (26) included previous lactation ketosis in a model for the next lactation. They reported probabilities of 7 versus 4%

### Table 4. Marginal maximum likelihood estimates of heritabilities and genetic and phenotypic correlations for occurrences of ketosis in the first (ketosis1) and second (ketosis2) lactations for 28,277 Finnish Ayrshire cows that calved the first time in 1983 through 1985.

<table>
<thead>
<tr>
<th>Trait</th>
<th>Ketosis1</th>
<th>Ketosis2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ketosis1</td>
<td>.09</td>
<td>.64</td>
</tr>
<tr>
<td>Ketosis2</td>
<td>.36</td>
<td>.07</td>
</tr>
</tbody>
</table>

1Heritabilities are in the diagonal.
2Genetic correlations are above diagonal, phenotypic below diagonal.

(4). The functional relationship between observed phenotypic correlation and its counterpart in the underlying level was described by Mäntysaari et al. (20). The high estimates may be explained by the fact that we were not able to remove the herd effect in the current analysis. Both milk yield and ketosis incidence are highly influenced by the herd (27).

There have been few studies of repeatability of diseases between lactations in dairy cattle. Using discriminant analysis, Dohoo and Martin (5) found evidence that metabolic diseases and udder diseases tend to recur in the same cow. Reproductive diseases also tend to recur but less frequently. Their study was based on 817 lactations in Canadian herds. Rowlands et al. (26) reported that cows with ketosis, hypocalcemia, mastitis, and certain lameness lesions in one lactation were twice as likely to have these disorders in the following lactation. They had 1215 pairs of consecutive lactations from the 849 cows available for analysis. Recently Markusfeld (23) used the Mantel and Haenszel technique (17) to assess the recurrence risk ratio of eight periparturient and reproductive traits. He had 8782 pairs of lactations of 5600 Israeli Holstein dairy cows. In all these studies, only phenotypic repeatability was investigated, although Markusfeld (23) evaluated the relative components of the intrinsic and extrinsic factors.

In epidemiologic studies, animal-based differences are typically considered as an explained residual variation. However, earlier studies with Finnish data (13, 14) have demonstrated a genetic component in the etiology of ketosis. Heritability was estimated to be 0.08. Emanuelson (7) reviewed a number of studies and proposed a heritability of 5 to 8%. Although studies have shown individual animal
developing ketosis with and without a history of ketosis in the previous lactation.

CONCLUSIONS

If a disease has a high lactation to lactation repeatability, the cows with a previous history of the disease have a greater risk of developing the same problem in a subsequent lactation. Because the probability of reoccurrence of clinical ketosis is 17%, the farmer should consider it in management decisions. By taking appropriate actions, it might be possible to prevent the occurrence of the disease, or it may be economically advantageous in certain situations to cull the high risk animal.

Occurrence of ketosis seemed to have a fairly high genetic correlation with milk yield in the first lactation. That the relationship is not clearly visible in the phenotypic level can be shown by calculating the genetic regression of production to ketosis. Using values in Table 3, we find that the 100-kg genetic improvement in milk yield increases by only .1% the risk of ketosis. The predicted increase in ketosis incidence is small because of a low heritability estimate of ketosis in the linear model. Phenotypic relationship between milk production and ketosis is difficult to estimate from the data with 305-d lactation records. Even though the high yield would be associated with an increased risk of ketosis, the observed correlation is confounded by the fact that decreased milk yield is one of the clinical symptoms of ketosis.

Heritability levels .07 to .09 can be considered high enough to justify the breeding efforts for lower ketosis incidence. In most countries with developed breeding programs, traits such as reproduction are considered in selection criterion, although their heritabilities are much lower than we found for ketosis (7). However, only a breeding program based on progeny testing can be considered effective. With the heritability of .09, the selection index based on 100 daughters will yield a correlation of .83 between index and the true breeding value. The standard deviation of sire breeding values in the underlying scale would then be .261. If we assume the threshold value of zero, the mean liability value in 3% incidence would be 1.65. This implies that 10% of the bulls should have progeny with mean liability values lower than 1.48 and the top 10% higher than 1.81. These underlying susceptibilities can be transformed to ketosis incidences of .069 and .035.

Results obtained do not give clear advice on whether sire evaluation should be based on first lactation ketosis, multitrait, or repeated record models. For binary trait, the single trait-single lactation evaluation would be most easily calculated; thus, it might be the most cost effective choice. Additional work in evaluation associated with repeatability model is not excessive, but the assumption of unity for genetic correlation between ketosis occurrence in subsequent lactations might be difficult to accept.

Several authors (2, 24, 30) have compared the effectiveness of threshold models with linear models in animal evaluation for categorical traits. These studies suggest that the nonlinear methodology is most advantageous when the heritability is high to moderate and when the data are unbalanced with respect to the factors with large effects on underlying liability. For ketosis, the underlying heritability is not large, but the data are highly unbalanced with respect to the herds and calving months, thus implying that sire rankings could be improved by using evaluations based on threshold model.

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REPEATABILITY OF KETOSIS


