J. Dairy Sci. 88:3273–3281
© American Dairy Science Association, 2005.

Genetic Analysis of Clinical Mastitis, Milk Fever, Ketosis, and Retained Placenta in Three Lactations of Norwegian Red Cows

B. Heringstad,[1,2] Y. M. Chang,[3] D. Gianola,[1,3] and G. Klemetsdal[1]
[1]Department of Animal and Aquacultural Sciences, Norwegian University of Life Sciences, N-1432 Ås, Norway
[2]Geno Breeding and AI Association, N-1432 Ås, Norway
[3]Department of Dairy Science, University of Wisconsin, Madison 53706

ABSTRACT

The objectives were to infer heritability and genetic correlations between clinical mastitis (CM), milk fever (MF), ketosis (KET), and retained placenta (RP) within and between the first 3 lactations and to estimate genetic change over time for these traits. Records of 372,227 daughters of 2411 Norwegian Red (NRF) sires were analyzed with a 12-variate (4 diseases × 3 lactations) threshold model. Within each lactation, absence or presence of each of the 4 diseases was scored based on the cow's health recordings. Each disease was assumed to be a different trait in each of the 3 lactations. The model for liability had trait-specific effects of year-season of calving and age of calving (first lactation) or month-year of calving and calving interval (second and third lactations), herd-5-yr, sire of the cow, and a residual. Posterior means of heritability of liability in first, second, and third lactations were 0.08, 0.07, and 0.07, respectively, for CM; 0.09, 0.11, and 0.13 for MF; 0.14, 0.16, and 0.15 for KET, and 0.08 in all 3 lactations for RP. Posterior means of genetic correlations between liability to CM, MF, KET, and RP, within disease between lactations, ranged from 0.19 to 0.86, and were highest between KET in different lactations. Correlations involving first lactation MF were low and had higher standard deviations. Genetic correlations between diseases were low or moderate (from –0.10 to 0.40), within as well as between lactations; the largest estimates were for MF and KET, and the lowest involved MF or KET and RP. Positive genetic correlations between diseases suggest that some general disease resistance factor with a genetic component exists. Trends of average sire posterior means by birth-year of daughters were used to assess genetic change, and the results indicated genetic improvement of resistance to CM and KET and no genetic change for MF and RP in the NRF population.
(**Key words:** dairy cattle, disease, genetic correlation, threshold model)

Abbreviation key: CM = clinical mastitis, **KET** = ketosis, **MF** = milk fever, **NRF** = Norwegian Red, **RP** = retained placenta.

INTRODUCTION

Clinical mastitis (**CM**), ketosis (**KET**), milk fever (**MF**), and retained placenta (**RP**) are among the most frequent diseases affecting dairy cattle. In Norway, 45% of all veterinary treatments in dairy cows in 2002 were for CM, 9% for KET, 8% for MF, and 5% for RP (TINE, 2003).

Most genetic studies on diseases other than mastitis have been based on relatively small datasets and have used methods that do not take into account that disease is often scored as a categorical trait (e.g., absent or present). Several studies have reported heritability estimates for one or more of the previously mentioned disease traits (Solbu, 1984; Lyons et al., 1991; Mäntysaari et al., 1991; Simianer et al., 1991; Uribe et al., 1995; Pryce et al., 1997; Schnitzenlehner et al., 1998; van Dorp et al., 1998; Wassmuth et al., 2000; Zwald et al., 2004a, b), but there are very few estimates of genetic correlations between these diseases. Exceptions are Pryce et al. (1997) and Zwald et al. (2004b), who reported genetic correlations between MF and mastitis (0.64; SE = 0.11) and between KET and mastitis (0.17; SD = 0.21), respectively.

Frequencies of all 4 diseases are higher in later lactations. A question of importance is, therefore, whether or not the traits can be considered to be the same across lactations. Pösö and Mäntysaari (1996) and Nielsen et al. (1997) found genetic correlations ranging from 0.65 to 1 between mastitis in the first 3 lactations. Heringstad et al. (2004) estimated genetic correlations between liability to CM in different intervals of the first 3 lactations of Norwegian Red (**NRF**), and their estimates ranged between 0.24 and 0.73. Mäntysaari et al. (1991) reported a genetic correlation of 0.68 between KET in

Received March 8, 2005.
Accepted May 9, 2005.
Corresponding author: B. Heringstad; e-mail: bjorg.heringstad@umb.no.

Table 1. Mean frequency of clinical mastitis (CM), milk fever (MF), ketosis (KET), and retained placenta (RP) by lactation.

	First lactation	Second lactation	Third lactation
Cows, no.	372,227	247,692	147,051
Mean CM frequency,[1] %	15.8	19.8	24.2
Mean MF frequency, %	0.1	1.9	7.9
Mean KET frequency, %	7.5	13.0	17.2
Mean RP frequency, %	2.6	3.4	4.3

[1]Percentage of cows with at least one case of CM in the period from 15 d prior to calving to 120 d after calving.

the first and second lactations, and Schnitzenlehner et al. (1998) found a genetic correlation of 0.79 between RP in the first and second lactations.

In Norway, all veterinary treatments have been recorded on an individual cow basis since 1978 (Heringstad et al., 2000). Thus, NRF represents one of few dairy populations where assessment of genetic change for several diseases is possible. The objectives were to infer heritability of and genetic correlations between CM, KET, MF, and RP within and between the first 3 lactations and to estimate genetic change for these traits in the NRF population. A Bayesian multivariate threshold model was fitted to NRF field data for this purpose.

MATERIALS AND METHODS

Data

Data on all cows included in the study by Heringstad et al. (2004) were used. Records were extracted from a dataset with 1.6 million cows, such that only first-crop daughters (i.e., when the difference between the birth year of a daughter and the birth year of her sire was <6 yr) of the 2411 NRF sires that were progeny tested from 1978 through 1998 were included. The data were further restricted to include only cows that had their first calving in a herd-5-yr class with at least 10 first lactation cows in the dataset. The edited dataset had 372,227 first lactation cows, of which 247,692 had a second lactation and 147,051 had a third lactation.

For each cow, all cases of veterinary-treated CM, MF, KET, and RP in the first 3 lactations were included in the dataset. Within each of the 3 lactations, absence or presence of each of the 4 diseases was scored as "0" or "1", respectively, based on whether or not the cow had at least one veterinary treatment recorded within the interval from 15 d before to 120 d after calving for CM and KET, from 15 d before to 30 d after calving for MF, and within the first 5 d after calving for RP. For CM, the interval from 15 d before to 120 d after calving was chosen because this is the interval used in the national genetic evaluation of CM in Norway. Mean disease frequencies, by lactation, are given in Table 1. For all 4 diseases, frequency was higher in second and third lactations. The mean frequency of CM was 15.8, 19.8, and 24.2% in first, second, and third lactations, respectively. The frequency of MF was very low in first lactation cows (0.1%) and increased to 1.9 and 7.9% for cows in second and third lactations, respectively. The mean frequency of KET ranged from 7.5% in first lactation to 17.2% in third lactation cows; similarly, RP had an incidence of 2.6%, increasing to 4.3% in third lactation. In Norway, the incidence of KET has decreased gradually since the mid 1980s. For first lactation cows, the mean frequency of KET decreased from 10.6% in 1987 to 4.3% in 1998. The frequency of MF and RP varies somewhat between years without following any obvious phenotypic trend. The frequency of CM increased from 1978 to 1995 and decreased thereafter, as shown by Heringstad et al. (2004).

The sire pedigree file had 2726 males, including the 2411 sires with daughter records in the dataset.

Model

A 12-variate threshold-liability model (Gianola and Foulley, 1983; Foulley et al., 1987) was used, assuming that the 4 diseases were different traits in each of the lactations. Similar models have been used previously for analyzing CM in different time intervals within and between lactations (Chang et al., 2002, 2004; Heringstad et al., 2004). The threshold model postulates an underlying continuous variable, liability (λ), such that the observed binary variable takes value 1 if λ is larger than a fixed threshold and 0 otherwise. With binary data, the threshold and the residual variance are not identifiable; therefore, these parameters were set to 0 and 1, respectively.

In matrix notation, the model fitted can be written as

$$\boldsymbol{\lambda} = \mathbf{X}\boldsymbol{\beta} + \mathbf{Z_h h} + \mathbf{Z_s s} + \mathbf{e}$$

where λ is a vector of unobserved liabilities for the 12 "traits" (disease × lactation combinations); β is a vector of systematic effects; **h** is a vector of herd-5-yr effects; **s** is a vector of sire-transmitting abilities; **e** is the vector of residual effects; and $\mathbf{X}$, $\mathbf{Z_h}$, and $\mathbf{Z_s}$ are the corresponding incidence matrices. The vector β included effects of age at calving and year × season of calving for first lactation traits and preceding calving interval and month × year of calving for second and third lactation traits. Age of calving had 15 levels, and year × season of calving had 80 levels (each year was divided into 4 seasons: January through March, April through June, July through September, and October through December). Calving interval in previous lactations were in 9 classes, whereas month × year of calving had 229 and

Download English Version:

https://daneshyari.com/en/article/2441428

Download Persian Version:

https://daneshyari.com/article/2441428

Daneshyari.com