55th Annual Meeting of the European Association for Animal Production,
5. September – 9. September 2004, Bled, Slovenia

Trajectories and variance component estimation for economically important disease categories

D. Hinrichs, W. Junge, E. Stamer and E. Kalm*

Institute of Animal Breeding and Husbandry, Christian-Albrechts-University of Kiel, D- 24098 Kiel

* *dhinrichs@tierzucht.uni-kiel.de*

Introduction

Diseases reduce animal welfare and result in economic losses for the farmer due to extra veterinary treatments, extra labour, decreasing milk production, discarded milk, and involuntary early culling (Nielsen et al., 1999). In addition, there is a growing interest from consumers regarding food production methods.

Most genetic analyses have been undertaken using lactation models, and therefore trajectories of diseases are not described (Lyons et al., 1991; Uribe et al., 1995). Furthermore it should be noted that most analyses of disease data apply linear models which assume a normal distribution of the data. Heritabilities estimated with these models are low, with most values in the interval of 0.00 to 0.10 (Lin et al., 1989; Lyons et al., 1991; Uribe et al., 1995). Threshold models are an alternative to the traditional linear models as they take the binary nature of the data into account (Gianola and Foulley, 1983). However, it should be noted that in most analyses disease information was considered as 'all or none' trait and that this definition does not utilise all information provided by the data, since some cows have more than one disease case per lactation. Therefore Rekaya et al. (1998) recommended the development of test day models for the analysis of longitudinal binary response such as disease field data. The use of longitudinal threshold models would improve the estimation of genetic parameters, because repeated disease cases are distinct observations in those models. Heritabilities were estimated with those models by Rekaya et al. (1998), Kadarmideen et al. (2000), and Heringstad et al. (2003). The resulting estimates for the heritabilities of mastitis ranged between 0.03 and 0.41. In addition, there is a lack of literature reports where other diseases than mastitis were analysed with longitudinal threshold models.

In the present study the disease categories all diseases, udder diseases, and metabolic diseases were analysed with longitudinal threshold models. Firstly the trajectories of the different diseases categories are described. Afterwards, variance components were estimated, where data recording comprises the first 50, 100, and 300 days of lactation.

This project was supported by Federal Ministry of Consumer Protection, Food and Agriculture and NOG Nord- Ost Genetic GmbH & Co. KG.

Material and Methods

Data recording took place from February 1998 to December 2002 on three commercial milk farms with an overall total herd size of 3200 German Holstein cows. Disease information was recorded by veterinarian or farm staff. During the first 300 days of lactation 75,595 disease treatments were recorded in total and thereof 39,934 come under udder disease treatments. The remaining treatments could be divided into treatments against fertility diseases (24,563), claw and leg diseases (5085), metabolic diseases (4132), and other diseases (1881).

Table 1: Number of observations, cows and sires for the different data sets

Data set	Disease category	Lactation length	Observations	Cows	Sires
1		50 days	839,473	10,071	767
2	Udder diseases	100 days	1,612,471	10,210	783
3		300 days	4,361,909	10,521	803
4		50 days	751,462	9968	763
5	Metabolic diseases	100 days	1,440,692	10,317	780
6		300 days	3,908,830	10,456	801
7		50 days	839,473	10,071	767
8	All diseases	100 days	1,612,471	10,210	783
9		300 days	4,361,909	10,521	803

In all data sets disease information was treated as an 'all or none' trait and the analysed data sets contained one observation per cow and day. Each observation received a disease code, '1' if a cow showed a disease and '0' if not.

In a first step all data sets were checked for extreme categories and they where discarded. In a further step the impact of systematic environmental effects was investigated by a generalised linear model with probit link function using the GENMOD procedure of the SAS package (SAS, 1999).

Posterior distributions of the permanent environment and additive genetic variance for the liability for mastitis were determined with the Gibbs sampling algorithm implemented in the program LMMG_TH, a threshold derivate of LMMG (Reinsch, 1996). For all models 100,000 cycles were generated. First 25,000 cycles were discarded (burn in plus a safety margin). The convergence detection was done by visual inspection and the results of the remaining 75,000 cycles were used to calculate the genetic parameters, using the MEANS procedure of the SAS package (SAS, 1999).

The following test day model was applied to all data sets:

$$E[\pi_{ijklmn}] = \Phi(B_i + K_j + A_k + f_{k(\text{days in milk})} + m_l + t_m),$$

where:

$E[\pi_{ijkl}]$	= expected percentage of animals with any disease	
Φ	= cumulative probability function of the standard normaldistribution	
B_i	= fixed effect of herd * week (of observation)	(i = 1,...,771)
K_j	= fixed effect of herd *year* season (of calving)	(j = 1,...,45)
A_k	= fixed effect of age of calving	(k = 1,...,8)
$f_{k(\text{days in milk})}$	= lactation curve nested into age of calving k	
m_l	= random permanent environment effect	(l=1,...,10,071)*
t_m	= random effect of the m th animal	(m=1,...,15,802)

* The number of permanent environment effects was 10,071 for data set 1and 7. For the reaming data sets the number of permanent environment effects are given in Table 1.

Results

Figure 1 shows the trajectories of the different disease categories during the first 50 days of lactation. As expected the incidence of all diseases was higher than the incidence of udder diseases and metabolic diseases. Furthermore, the trajectory of all diseases showed three distinct peaks during the first 50 days of lactation, whereas udder diseases and metabolic disease showed only one peak in this part of lactation. In addition, the peak of udder diseases becomes more apparent compared to the peak of the metabolic diseases.

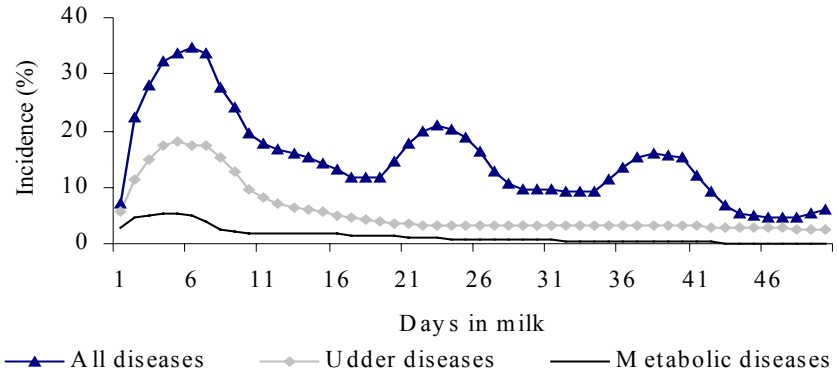

Figure 1: Trajectories of all diseases, udder diseases, and metabolic diseases during the first 50 days of lactation

The results of the variance component estimation are shown in Table 2. For all analysed disease categories the additive genetic variance, as well as the permanent environmental variance decreased with increasing lactation length. This was more distinct for udder diseases and metabolic diseases. In addition, a higher additive genetic variance and permanent environmental variance could be observed for udder diseases and metabolic diseases, compared to all diseases.

Table 2: Additive genetic and permanent environment variance, heritability and repeatability for different data sets

Data set	Disease Category	Lactation length	σ^2_a	σ^2_{pe}	h^2 ($\pm s_{h2}$)	r ($\pm s_w$)
1		50	0.58	1.80	0.17 (0.02)	0.70 (0.01)
2	Udder diseases	100	0.44	1.30	0.16 (0.02)	0.64 (0.01)
3		300	0.24	0.83	0.12 (0.01)	0.52 (0.01)
4		50	0.91	3.16	0.18 (0.03)	0.80 (0.01)
5	Metabolic diseases	100	0.78	2.84	0.17 (0.03)	0.78 (0.01)
6		300	0.43	2.12	0.12 (0.02)	0.72 (0.01)
7		50	0.07	0.60	0.04 (0.01)	0.40 (0.01)
8	All diseases	100	0.05	0.40	0.03 (0.01)	0.31 (0.01)
9		300	0.04	0.28	0.03 (0.01)	0.25 (0.01)

For udder diseases the estimated heritabilities ranged from 0.12 to 0.17, and they were between 0.12 and 0.18, and 0.03 and 0.04 for metabolic diseases and all diseases, respectively. The highest repeatabilities were estimated for metabolic diseases, followed by udder diseases and all diseases.

Discussion

In our analyses each disease category showed a typical trajectory during the first 50 days of lactation. The trajectory of udder diseases was in the line with the trajectory of mastitis reported by Schomaker et al. (2002) and Heringstad et al. (2003). No literature reports could be found where trajectories for metabolic diseases or all diseases were described. One of the reasons for this lack could be that most genetic analyses of disease data (Lin et al.,1989; Lyons et al., 1991; Simianer et al., 1991; Uribe et al., 1995; Nielsen et al., 1999) were done with lactation models. These models used blocked information and therefore the trajectory of a disease or disease category is not the main object of interest. If test day models are used the trajectory of the analysed trait has an important effect. Therefore, models should be fitted to the trajectory of the analysed disease category. For udder diseases and metabolic diseases this could be done by using the Ali Schaeffer function, but it should be noted that the possibilities of these function are also limited. Therefore we treated the first days of lactation as fixed effects. This was also done by Schomaker et al. (2002). Especially for the disease category all diseases alternative function should be tested, because the results of our study suggests that the use of the Ali Schaeffer function is not appropriate if a disease category showed distinct peaks.

For the estimation of variance components test day models should be used because repeated cases of diseases could be considered as distinct observations. The estimates of heritabilities for udder diseases in this study are higher than those found by Rekaya et al. (1998), Kadarmideen et al. (2000) and Heringstad et al. (2003) for mastitis if test day threshold models were used. The estimated heritabilities of metabolic diseases are in the upper range of literature reports (Simianer et al., 1991; Lyons et al., 1991; Uribe et al., 1995; Nielsen et al., 1999). It should be noted that in most cases cows are by one metabolic disease during lactation. Therefore , the improvement in genetic parameter estimation, caused by the use of test day threshold models is limited. This is not correct for udder diseases where repeated cases occured in a higher frequency, compared to metabolic diseases. If all diseases were analysed with test day threshold models the estimated heritabilities are similar to those estimated with a lactation threshold model (Simianer et al., 1991; Hinrichs et al., 2004). One possibility to avoid this could be to eliminate ovarian problems because they are the reason for the latter peaks of the trajectory of all diseases

All in all the results shows that test day threshold models are an good alternative for the genetic parameter estimation of udder diseases and metabolic diseases. In addition, it should be noted that further research had to be done for the development of such models. Especially alternatives to functions should be tested for the modelling of trajectories of different disease categories.

References

Gianola, D. and J.L. Foulley. 1983. Genet. Sel. Evol. 15: 201–224.

Hinrichs, D., E. Stamer, W. Junge, and E. Kalm. 2004. Submitted for publication.

Heringstad, B., Y.M. Chang, D. Gianola, and G. Klemetsdal. 2003. J. Dairy Sci. 86: 2676–2683

Kadarmideen, H.N., R. Rekaya, and D. Gianola. 2000. 51th Annual Meeting of EAAP, The Hague, The Netherlands.

Lin, H.K., P.A. Oltencau, L.D. van Vleck, H.N. Erb, and R.D. Smith. 1989. J. Dairy Sci. 72: 180-186.

Lyons, D.T., A.E. Freeman, and A.L. Kuck. 1991. J. Dairy Sci. 74:1092–1100.

Nielsen, U.S., G.A. Pedersen, J. Pedersen, and J. Jensen. 1999. Interbull Bull. 21:170–177.

Reinsch, N. 1996. Arch. Tierzucht 39: 203–209.

Rekaya, R., S.L. Rodriguez-Zas, D. Gianola, and G.E. Shook. 1998. 49th Annual Meeting of EAAP, Warsaw, Poland.

SAS Institute Inc. 1999. SAS/STAT® User's Guide Version 8, Cary NC, USA

Simianer, H., Solubu, H., Schaeffer, L. R. 1991. Journal of Dairy Science 74, 4359 – 4365.

Schomaker, T., W. Junge, E. Stamer, N. Reinsch, and E. Kalm. 2002. Züchtungskunde 74: 250 – 265.

Uribe, H.A., W. Kennedy, S.W. Martin, and D.F. Kelton. 1995. J. Dairy Sci. 78: 421-430.