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Exploration of Relationships Between Claw Disorders and Milk Yield in Holstein Cows via Recursive Linear and Threshold Models

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ABSTRACT

Relationships between claw disorders and test-day milk yield recorded in 2005 on 5,360 Holstein cows, kept on 11 large-scale dairy farms in eastern Germany, were analyzed in a Bayesian framework with standard linear and threshold models and recursive linear and threshold models. Four different claw disorders, digital dermatitis (DD), sole ulcer (SU), wall disorder (WD), and interdigital hyperplasia (IH), were scored as binary traits within 200 d after calving and analyzed separately. Incidences of disorders were 13.7% for DD, 16.5% for SU, 9.8% for WD, and 6.7% for IH. Heritabilities of disorders were greater when applying threshold or recursive threshold models than with linear or linear recursive models. Posterior means of genetic correlations between test-day milk production and claw disorders ranged from 0.17 to 0.44, suggesting that breeding strategies focusing on increased milk yield will increase incidences of disorders as a correlated response. A progressive path of lagged relationships was postulated for recursive models describing first the influence of test-day milk yield (MY1) on claw disorders and second, the effect of the disorder on milk production level at the following test day (MY2). In recursive models, structural coefficients describe recursive relationships at the phenotypic level. The structural coefficient λ_{21} was the gradient of disease (trait 2) with respect to MY1 (trait 1) for a model with a recursive effect of trait 1 on trait 2. The increase of disease incidence of the 4 different disorders per 1-kg increase of MY1 ranged from λ_{21}= 0.006 to λ_{21}= 0.024 on the visible scale when applying recursive linear models, and from λ_{21}= 0.003 to λ_{21}= 0.016 on the underlying liability scale for recursive threshold models. The rate of change in MY2 (trait 3) with respect to the previous claw disorder is given by λ_{32} for a model with a recursive effect from trait 2 to trait 3. Structural coefficients λ_{32} ranged from –0.12 to –0.68 predicting that a 1-unit increase in the incidence of any disorder reduces milk yield at the following test day by up to 0.67 kg. Rank correlations between sire posterior means for the same claw disorders among different models were >0.84, but some changes in rank of sires in distinct top-10 lists were observed. Structural equation models are of increasing importance in genetic evaluations, and this study showed the possible application of recursive systems, even for categorical data.
Key words: claw disorder, milk yield, recursive threshold model, Bayesian method

INTRODUCTION

In most dairy cattle breeding programs, selection has focused mainly on increasing milk production traits. Miglior et al. (2005) compared national selection indices used in 15 countries and found that the average relative emphases on production, durability-health, and reproduction were 59.5, 28, and 12.5%, respectively. In the last decade, there has been growing interest in including functional or health traits in total net merit indexes. In theory, the evolution of disease incidences in dairy cattle depends on the sign and magnitude of genetic correlations between susceptibility to diseases and milk yield. Reliable estimates of phenotypic and genetic correlations between disorders and other traits of economic importance are required for defining an aggregate breeding value in dairy cattle in the near future, as has been the case for decades for several health traits in Nordic dairy cattle populations (Heringstad et al., 2000).

Due to their economic impact (e.g., Enting et al., 1997; Kossaibati and Esslemont, 2000), claw disorders in German Holsteins are receiving as much attention as fertility or mastitis. To cope with this problem, the German association for claw hygiene and trimming developed a computer-supported documentation and analysis system, as described by Landmann et al. (2006). Data from this recording system was used recently for estimating

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heritability of various claw disorders via logistic models (König et al., 2005a). Results agreed with those from other similar studies applying threshold animal models (Swalve et al., 2005) or threshold sire models (Van der Waaij et al., 2005). However, genetic correlations between claw disorders and production traits have varied markedly among studies, because of different definitions of production traits; for example, average of single test-day production (König et al. 2005a) vs. whole-lactation milk yield (Swalve et al., 2005). Difficulties in evaluating diseases and their correlations with other traits of interest that arise from the discrete nature of observations have been overcome by applying generalized linear mixed models (Wolfinger and O'Connell, 1993).

Recently, Gianola and Sorensen (2004) proposed an extension of the multivariate mixed linear model to account for possible feedback and recursiveness among response variables assuming an infinitesimal, additive model of inheritance. These feedback models for biological systems were discussed by Haldane and Priestley (1905), Turner and Stevens (1959), and Wright (1960) and have a long tradition in econometrics (Haavelmo, 1943). In dairy cattle and goats, de los Campos et al. (2006a,b) found an increased risk of infection in the udder with increasing milk yield, with the latter acting, probably, as a stress factor. On the other hand, an increase of infection or SCC could affect milk yield adversely, which defines a feedback situation. These simultaneous and recursive relationships cannot be modeled in standard linear models, at least explicitly.

Applications of recursive models in the context of animal breeding have been limited. In a recursive relationship, one variable affects another, but without a reciprocal effect. Sorensen and Varona (2006) used data from 2 breeds of pigs to investigate the influence of litter size on birth weight of piglets. Their specification defines a recursive 2-trait system, in which litter size is modeled to account for its effect on birth weight, but birth weight does not affect litter size. Legarra and Robert-Granié (2006) conducted a simulation study to analyze the impact of recursiveness of phenotypes for fertility and milk yield on estimates of genetic correlations between these traits. López de Maturana et al. (2007) investigated relationships between calving ease and fertility in Holsteins, accommodating censored and discrete outcomes. The relationship between calving ease and fertility traits is a recursive one, because calving ease may affect subsequent reproductive performance but not vice versa.

In the case of claw disorders and milk production in dairy cows, it seems sensible to postulate a lagged progressive path involving 3 traits (Figure 1). One path would describe the influence that test-day milk yield has on claw disorders, and the second path would pertain to the effect of the disorder on milk production level at the following test date. The main objective of this study was to apply recursive linear and threshold models using our own computer algorithms (Wu, 2007) to investigate relationships between different claw disorders and test-day milk yield and to infer the respective model parameters. The application of recursive systems for categorical health data or even the availability of computer algorithms for such cases is relatively new and limited in the field of animal breeding.

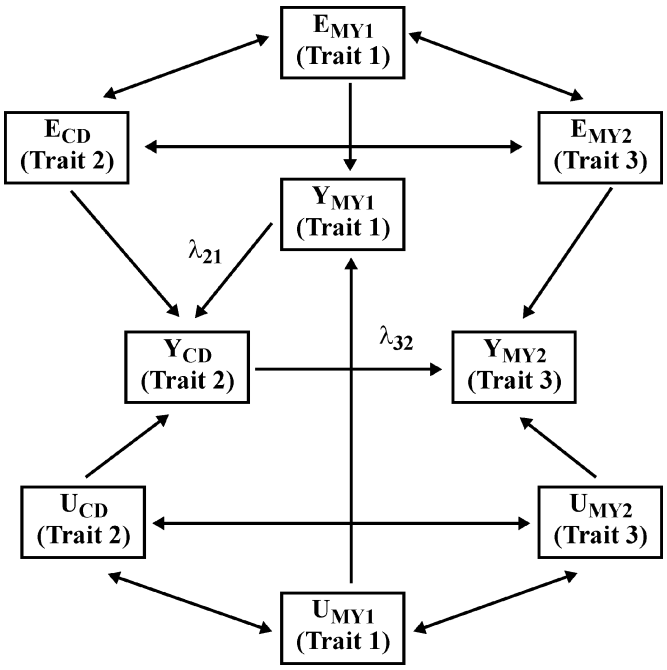

Figure 1. Recursive model for the 3 traits: trait 1 = test-day milk yield before diagnosis of a claw disorder (MY1), trait 2 = claw disorder (CD), trait 3 = test-day milk yield after diagnosis of claw disorder (MY2). Y indicates phenotypic values for test-day milk yield and claw disorders; E indicates residual effects; U indicates additive-genetic effects; a single-headed arrow indicates that variable *A* affects variable *B*, and trait 2 affects trait 3; a double-headed arrow indicates correlations between pairs of variables; and λ_{ij} indicates the rate of change of variable *i* with respect to variable *j*.

MATERIALS AND METHODS

Data

Data were from a new electronic recording system for claw disorders as described by Landmann et al. (2006) and collected by 5 different claw trimmers. The guideline for classification of individual claw disorders was developed by the German Agricultural Society, and all trimmers were trained for uniform identification of traits. The electronic recording system allows combination with data from herd management programs and with information on test-day records. The data set used

Table 1. Incidences of dermatitis digitalis (DD), sole ulcer (SU), wall disorders (WD), and interdigital hyperplasia (IH) within 200 d after calving, and average test-day milk yield before (MY1) and after (MY2) diagnosis of the disorder[1]

Disorder	Cows[2] (%; total n = 5,360)	Mean day of diagnosis[3]	MY1 (kg)	MY2 (kg)	MY2 – MY1 (in % of MY1)
Diagnosed for DD	13.67	95	32.61	30.14	–7.57
Undiagnosed for DD	86.33	100	31.31	30.01	–4.15
Diagnosed for SU	16.51	88	32.94	29.97	–9.02
Undiagnosed for SU	83.49	100	31.73	29.54	–6.90
Diagnosed for WD	9.78	86	33.18	30.04	–9.45
Undiagnosed for WD	90.22	100	32.21	30.09	–6.58
Diagnosed for IH	6.72	101	32.30	30.08	–9.52
Undiagnosed for IH	93.28	100	31.18	29.41	–5.68
Healthy	74.40	100	31.10	30.03	–3.44

[1]Test-day milk yields of undiagnosed cows for the respective disorder are presented by disorder. Cows without any diagnosis are given in the last row.

[2]A single cow may show several disorders; therefore, the percentages for diagnosed cows and the healthy cows sum up to >100%.

[3]The nearest test-day observation for healthy cows before a general dummy date of d 100 was defined as MY1 and the nearest test-day observation after d 100 was MY2.

here comprised test-day production records and claw and foot disorders collected in 2005 from 5,360 Holstein cows kept on 11 large-scale dairy farms within a single region in eastern Germany. These farms participated as cooperator herds in a progeny test program as described by Gernand et al. (2007). Hence, such a system ensured a reasonable number of progeny per bull for the present analysis and reliable genetic connectedness across herds. The average number of daughters for the 511 sires was 10.5, and 79 sires had more than 30 daughters scored for claw disorders.

Cows of all parities were included. Claw and foot disorders were divided into 4 conditions: digital dermatitis (**DD**), sole ulcer (**SU**), wall disorder (**WD**), and interdigital hyperplasia (**IH**), and scored separately as "all or none" traits. A detailed description of the individual disorders is given by König et al. (2005a). The period of observation spanned 200 d, starting at calving. If a cow had the condition within this period in one or both rear legs for the respective disorder, she was given a score of 1; otherwise she was given a score of 0. For each cow having a disorder, the nearest test-day observation before and after the occurrence of the specific disease was identified. This definition involved 3 different traits: test-day milk yield before occurrence of the disorder (**MY1** = trait 1); the disorder itself (trait 2), and test-day milk yield after occurrence of the disorder (**MY2** = trait 3). Repeated episodes of a disease were not taken into account. If a cow had several entries of the same disorder within the 200-d period, the first observation with complete information (i.e., a test-day record before the occurrence date of the specific disease) was stored. Cows without disorders were assigned a value of 0 for trait 2 at a general dummy date of d 100 within their lactation. The nearest test-day observation for healthy cows before d 100 was defined as MY1 and the nearest test-day observation after d 100 was MY2. Table 1 gives mean incidences of claw and foot disorders within the respective period and the average milk yield of cows before and after the occurrence of each specific disease.

Statistical Methods

Four different sire models were used. Model M1 was a standard 3-trait linear mixed model, and model M2 was a threshold-linear model treating the claw disorder (i.e., the second trait) as a binary trait. In the threshold-liability model (Gianola, 1982; Gianola and Foulley, 1983), it is assumed that an underlying continuous variable, liability (l_{i2}), exists such that the observed binary variable y_{i2} takes a value of 1 if l_{i2} is larger than a fixed threshold $\kappa = 0$.

Model M3 was a recursive model assuming a multivariate Gaussian distribution for the 3 traits, and model M4 was a recursive threshold-linear model with 2 Gaussian traits and 1 binary trait. In recursive models M3 and M4, the structural coefficient λ_{21} was the gradient of disease with respect to MY1 for a model with a recursive effect of trait 1 on trait 2. The rate of change in production level in MY2 with respect to the previous claw disorder was given by λ_{32} for a model with a recursive effect from trait 2 to trait 3 (Figure 1). The recursive models can be written as follows:

$$\begin{pmatrix} \Lambda\mathbf{y}_1 \\ \Lambda\mathbf{y}_2 \\ \cdots \\ \Lambda\mathbf{y}_n \end{pmatrix} = \begin{pmatrix} \mathbf{X}_1 \\ \mathbf{X}_2 \\ \cdots \\ \mathbf{X}_n \end{pmatrix} \beta + \begin{pmatrix} \mathbf{Z}_1 \\ \mathbf{Z}_2 \\ \cdots \\ \mathbf{Z}_n \end{pmatrix} \mathbf{u} + \begin{pmatrix} \mathbf{e}_1 \\ \mathbf{e}_2 \\ \cdots \\ \mathbf{e}_n \end{pmatrix} = \mathbf{X}\beta + \mathbf{Z}\mathbf{u} + \mathbf{e}$$

with $i = 1,2,...,n$ indexing the animal, each measured for the 3 traits. Above, $\mathbf{y}_i = (y_{i1} \quad y_{i2} \quad y_{i3})'$ in model M3 and $\mathbf{y}_i = (y_{i1} \quad y_{i2} \quad y_{i3})'$ in model M4; β is a vector of "fixed" effects (in a Bayesian context, these are location parameters with vague prior information) of order $f^* = \sum_{j=1}^{3} f_j$ and f_j is the number of fixed effects affecting trait j (j = 1, 2, 3). Fixed factors included the effects of herd (11 levels), calving season (January–March, April–June, July–September, October–December) and of parity of the cow (3 levels: 1, 2, and >2). Further, $\mathbf{X}_i$ is a $3 \times f^*$ known incidence matrix linking phenotypic measurements and liabilities in $\mathbf{y}_i$ (or a rotation thereof, via the matrix Λ explained later) to the fixed effects. Vector $\mathbf{u}$, of order $q^* = 3 \times q$, represents sire effects, where q is the number of sires. $\mathbf{Z}_i$ is a $3 \times q^*$ incidence matrix linking $\mathbf{y}_i$ or $\Lambda\mathbf{y}_i$ to $\mathbf{u}$, and $\mathbf{e}_i$ is a vector of residual effects of order 3. It is assumed that $\mathbf{u} \mid \mathbf{G}_0 \sim N(\mathbf{0},\mathbf{A} \otimes \mathbf{G}_0)$ and $\mathbf{e} \mid \mathbf{R}_0 \sim N(\mathbf{0},\mathbf{I} \otimes \mathbf{R}_0)$, where $\mathbf{G}_0$ is a genetic covariance matrix, $\mathbf{R}_0$ is a residual covariance matrix, $\mathbf{A}$ is an additive relationship matrix, and $\otimes$ indicates the Kronecker product. It is also assumed that $\mathbf{u}$ and $\mathbf{e}$ are mutually independent.

A remarkable difference between the recursive model and the standard mixed model is that in the former, each observation vector y_i is premultiplied by an unknown 3×3 matrix Λ, whose elements need to be estimated. This matrix Λ contains the structural coefficients λ'_{ij} describing the rate of change of trait i with respect to trait j' (Gianola and Sorensen, 2004). The form of Λ in this study was

$$\Lambda = \begin{bmatrix} 1 & 0 & 0 \\ -\lambda_{21} & 1 & 0 \\ 0 & -\lambda_{32} & 1 \end{bmatrix}.$$

In standard linear (M1) and threshold (M2) models, Λ is an identity matrix, because the traits do not affect each other.

The conditional distribution of all observed data for the linear-linear model M1 was:

$$p(\mathbf{y} \mid \boldsymbol{\lambda}, \beta, \mathbf{u}, \mathbf{R}_0, \mathbf{H}) \propto \prod_{i=1}^{n} \frac{1}{|\mathbf{R}_0|^{\frac{1}{2}}} \times \exp\left\{-\frac{1}{2}(\mathbf{y}_i - \mathbf{X}_i\beta - \mathbf{Z}_i\mathbf{u})'\mathbf{R}_0^{-1}(\mathbf{y}_i - \mathbf{X}_i\beta - \mathbf{Z}_i\mathbf{u})\right\} \quad [1]$$

and of the data and liabilities jointly for the linear-threshold model M2:

$$p(\mathbf{w}, \mathbf{y} \mid \boldsymbol{\lambda}, \beta, \mathbf{u}, \mathbf{R}_0, \mathbf{H}) \propto \prod_{i=1}^{n} I(y^b{}_i \le \kappa)I(w_i = 0) + I(y^b{}_i > \kappa)I(w_i = 1) \times \prod_{i=1}^{n} \frac{1}{|\mathbf{R}_0|^{\frac{1}{2}}} \times \exp\left\{-\frac{1}{2}(\mathbf{y}_i - \mathbf{X}_i\beta - \mathbf{Z}_i\mathbf{u})'\mathbf{R}_0^{-1}(\mathbf{y}_i - \mathbf{X}_i\beta - \mathbf{Z}_i\mathbf{u})\right\} \quad [2]$$

For the 2 recursive models, conditional distributions of the pseudo-data $\Lambda\mathbf{y}$ (M3), or of the pseudo-data and liabilities jointly (M4), given the unknown parameters, were:

M3 (recursive linear mixed model):

$$p(\Lambda\mathbf{y} \mid \boldsymbol{\lambda}, \beta, \mathbf{u}, \mathbf{R}_0, \mathbf{H}) \propto \prod_{i=1}^{n} \frac{1}{|\mathbf{R}_0|^{\frac{1}{2}}} \times \exp\left\{-\frac{1}{2}(\Lambda\mathbf{y}_i - \mathbf{X}_i\beta - \mathbf{Z}_i\mathbf{u})'\mathbf{R}_0^{-1}(\Lambda\mathbf{y}_i - \mathbf{X}_i\beta - \mathbf{Z}_i\mathbf{u})\right\} \quad [3]$$

M4 (recursive threshold-linear model):

$$p(\mathbf{w}, \Lambda\mathbf{y} \mid \boldsymbol{\lambda}, \beta, \mathbf{u}, \mathbf{R}_0, \mathbf{H}) \propto \prod_{i=1}^{n} I(y^b{}_i \le \kappa)I(w_i = 0) + I(y^b{}_i > \kappa)I(w_i = 1) \times \prod_{i=1}^{n} \frac{1}{|\mathbf{R}_0|^{\frac{1}{2}}} \times \exp\left\{-\frac{1}{2}(\Lambda\mathbf{y}_i - \mathbf{X}_i\beta - \mathbf{Z}_i\mathbf{u})'\mathbf{R}_0^{-1}(\Lambda\mathbf{y}_i - \mathbf{X}_i\beta - \mathbf{Z}_i\mathbf{u})\right\} \quad [4]$$

where w and y^b are vectors containing observable binary data and underlying liabilities of all animals, H represents the collection of all known hyper-parameters, and I (A) is an indicator function of value 1 if condition A is true, and 0 otherwise. Letting $\Lambda = \mathbf{I}$, [3] and [4] reduce to the corresponding densities for a standard linear model [1] and a standard linear-threshold model [2], respectively.

Bayesian inference via Markov chain Monte Carlo (**MCMC**) implementation was used to infer unknown parameters of interest. Bayesian analysis of linear model M1 was conducted as described by Sorensen and Gianola (2002), with location parameters sampled from a multivariate normal distribution, and covariance matrices $\mathbf{G}_0$ and $\mathbf{R}_0$ (the 3×3 covariance matrices between sire and residual effects, respectively) sampled from inverse Wishart distributions. When extending a standard mixed model to include 1 binary trait in a threshold-linear model, such as M2 and M4, one needs to

sample the residual covariance matrix from a conditional inverse Wishart distribution, given that the variance of liability is fixed to 1 (Korsgaard et al., 2003). Further, in the recursive models M3 and M4, structural coefficients ($\boldsymbol{\lambda}$) were sampled using a Gibbs sampler (Gianola and Sorensen, 2004). Bayesian modeling and MCMC sampling procedures for simultaneous and recursive (**SIR**) models (e.g., M3 and M4) are described in details in the users' manual of the SIR-BAYES software package (Wu, 2007).

The MCMC sampling procedure consists of successive iterative updating of each parameter or group of parameters. Length of burn-in and of the sampling period were assessed by the method of Raftery and Lewis (1992), as implemented in the BOA software package (Smith, 2005), and using the first 10,000 iterations of a Gibbs chain of coefficients λ_{ij}. The structural coefficients mix more slowly than other parameters, so this assessment was deemed conservative. Based on the diagnostics and visual inspections of trace plots, chain lengths of between 180,000 and 230,000 iterations were run for different models and trait combinations; the burn-in period was 10,000 rounds for all models.

Parameters from recursive models (M3 and M4) differ from those obtained using a standard mixed model and should be viewed as system parameters. Gianola and Sorensen (2004) described how parameters of a recursive model can be transformed into parameters of a standard mixed model. Estimates of genetic, residual, and phenotypic covariance matrices were obtained by applying the following matrix operations to the posterior samples of system parameters:

$$\mathbf{G}_0^* = \boldsymbol{\Lambda}^{-1}\mathbf{G}_0\boldsymbol{\Lambda}'^{-1}$$

$$\mathbf{R}_0^* = \boldsymbol{\Lambda}^{-1}\mathbf{R}_0\boldsymbol{\Lambda}'^{-1}$$

$$\mathbf{P}_0^* = \mathbf{G}_0^* + \mathbf{R}_0^*,$$

where $\mathbf{G}_0$ and $\mathbf{R}_0$ are the system covariance matrices for the sire and residual effects, and $\mathbf{G}_0^*$, $\mathbf{R}_0^*$, and $\mathbf{P}_0^*$ are the sire, residual, and phenotypic variance-covariance matrices, respectively. Also, sire effects were estimated as $\mathbf{S}^*_i = \boldsymbol{\Lambda}^{-1}\mathbf{S}_i$, where $\mathbf{S}_i$ is the vector of system sire effects.

Structural equation coefficients obtained from model M4 were estimated on the liability scale and, therefore, the impact on the outward scale was not obvious. An approach similar to that described by López de Maturana et al. (2007) was used for assessing the impact of different levels of MY1 on the incidence of disorders; the method used liabilities for each cow from model M4. Six classes of MY1 for the range between 28 and 34 kg in increments of 1 kg were created. Differences (Δ) on the outward scale for claw disorders between adjacent classes of MY1 were calculated as illustrated for MY1-class 1 (28–29 kg) and MY1-class 2 (29–30 kg):

$$\Delta \text{ [MY1-class 2 with respect to MY1-class 1]} =$$

$$\Phi \text{ [}\mu + \text{effect (MY1-class 2)}, \sigma_e^2\text{]}$$

$$- \Phi \text{ [}\mu + \text{effect (MY1-class 1)}, \sigma_e^2\text{]}$$

where effect indicates the effect estimated for the different MY1-classes on the underlying liability scale, $\Phi(x,\sigma_e^2)$ is the cumulative normal distribution function with mean x and residual variance σ_e^2, and μ refers to probit corresponding to the mean incidences of disorders reported in Table 1.

RESULTS AND DISCUSSION

Mean Incidences and Milk Yield

Mean incidences of observed claw disorders in the first 200 d of lactation (Table 1) were in the range reported by König et al. (2005a) when considering the entire lactation of a cow, and nearly identical to those found in other studies (Somers et al., 2003; Van der Waaij et al., 2005). In this study, the mean incidences of DD, SU, WD, and IH were 0.137, 0.165, 0.098, and 0.067, respectively, and there were substantial differences between herds (Figure 2).

On the phenotypic scale, cows affected by any of the claw disorders had a larger decrease in test-day milk yield (MY2 – MY1) compared with undiagnosed cows for the respective disorders (Table 1). Hence, following treatment after infection does not increase milk yield up to the level of undiagnosed or healthy cows. The average day of occurrence of disorders was close to d 100, which was the fixed dummy day used for undiagnosed cows to define the "before" and "after" test days. Claw disorders appear to occur in clusters; that is, a cow showing one disease has an increased genetic risk to show another claw disease. A relatively high percentage (74.40%) of all cows were completely healthy, indicating a remarkable intersection of cows affected within the 200-d period by several disorders simultaneously. Healthy cows without any of these 4 claw disorders had the lowest level of MY1, but also showed the smallest decrease in test-day milk yield when comparing MY1 and MY2. Collard et al. (1999) found that high milk yield within the first third of lactation increases a cow's risk to experience health problems. An explanation could be that potential resource intake is insufficient to express further production potential. Additional resources of energy are drawn away from fitness traits such as fertility and health (Van der Waaij, 2004).

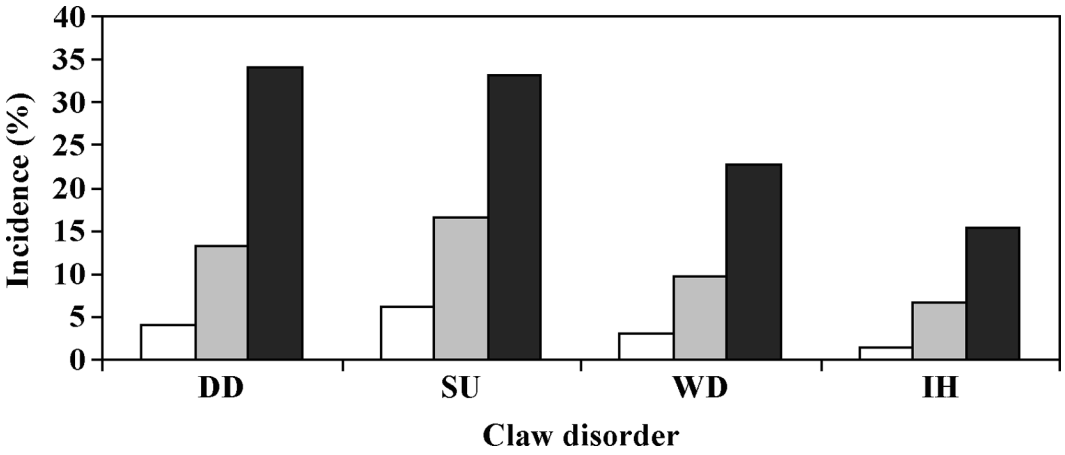

Figure 2. Incidences of dermatitis digitalis (DD), sole ulcer (SU), wall disorders (WD), and interdigital hyperplasia (IH) in the best herd (white bars), the worst herd (black bars), and on average over all herds (gray bars).

Genetic Parameters

Posterior means of selected parameters from the standard linear mixed model (M1, $\lambda = 0$), standard threshold-linear mixed model (M2, $\lambda = 0$), recursive mixed linear model (M3), and the recursive mixed threshold model (M4) are shown in Tables 2 (DD,) 3 (SU), 4 (WD), and 5 (IH). Heritability estimates of DD were in the range from 0.049 to 0.089; for SU between 0.100 and 0.136; for WD between 0.086 and 0.135, and for IH from 0.111 to 0.188. Threshold models (M2 and M4) lead to generally higher heritabilities on the liability scale than linear models (M1 and M3). For all disorders, the largest point estimates of heritability were from the threshold model (M2). Varona et al. (1999) analyzed calving ease and birth weight applying linear-linear and linear-threshold models. They found that threshold-linear models accounting for the probabilistic structure of the binary trait (i.e., calving ease) were better than linear models; also, heritability of the binary trait was larger from threshold-linear models. This is what theory for analysis of categorical traits would lead one to expect (Dempster and Lerner, 1950), and is in agreement with studies analyzing categorical data with different models (e.g., Weller and Ron, 1992; Andersen-Ranberg et al., 2005). For traits or disorders characterized by low incidences (e.g., IH), differences in heritabilities between linear-linear and threshold-linear models were substantial. However, Huang and Shanks (1995) estimated heritabilities of SU and IH applying threshold and linear models, and results were very similar. Freund and Walpole (1980) argued that estimates of parameters for categorical traits when assuming an underlying Gaussian would be unbiased when $n\pi$ is >5, with π being the incidence of a disorder, and n the size of the smallest subclass in the statistical model.

Table 2. Posterior means and standard deviations (in parentheses) of heritabilities (h^2), genetic correlations (r_g), and phenotypic correlations (r_p) for dermatitis digitalis (DD) and test-day milk yield before (MY1) and after (MY2) after diagnosis of disorder applying 4 different models[1]

	Model[1]			
Item	M1	M2	M3	M4
h^2_{MY1}	0.163 (0.05)	0.171 (0.05)	0.155 (0.05)	0.149 (0.05)
h^2_{DD}	0.072 (0.05)	0.089 (0.06)	0.049 (0.03)	0.053 (0.05)
h^2_{MY2}	0.177 (0.06)	0.181 (0.07)	0.162 (0.06)	0.157 (0.06)
$r_{g(MY1:DD)}$	0.355 (0.06)	0.330 (0.06)	0.269 (0.07)	0.283 (0.08)
$r_{g(MY1:MY2)}$	0.888 (0.07)	0.912 (0.06)	0.895 (0.05)	0.907 (0.05)
$r_{g(DD:MY2)}$	0.170 (0.06)	0.165 (0.07)	0.077 (0.07)	0.091 (0.07)
$r_{p(MY1:DD)}$	0.222 (0.10)	0.198 (0.09)	0.081 (0.10)	0.090 (0.10)
$r_{p(MY1:MY2)}$	0.766 (0.17)	0.700 (0.16)	0.773 (0.18)	0.810 (0.18)
$r_{p(DD:MY2)}$	0.193 (0.10)	0.192 (0.11)	0.156 (0.11)	0.145 (0.09)

[1]M1 = standard linear mixed model, M2 = threshold mixed model, M3 = recursive linear mixed model, M4 = recursive threshold mixed model.

Table 3. Posterior means and standard deviations (in parentheses) of heritabilities (h^2), genetic correlations (r_g), and phenotypic correlations (r_p) for sole ulcer (SU) and test-day milk yield before (MY1) and after (MY2) after diagnosis of disorder applying 4 different models[1]

	Model[1]			
Item	M1	M2	M3	M4
h^2_{MY1}	0.157 (0.06)	0.160 (0.05)	0.155 (0.04)	0.158 (0.05)
h^2_{SU}	0.103 (0.07)	0.136 (0.07)	0.100 (0.07)	0.129 (0.06)
h^2_{MY2}	0.165 (0.08)	0.168 (0.07)	0.154 (0.08)	0.164 (0.08)
$r_{g(MY1:SU)}$	0.409 (0.18)	0.443 (0.16)	0.365 (0.16)	0.317 (0.17)
$r_{g(MY1:MY2)}$	0.926 (0.11)	0.902 (0.10)	0.914 (0.11)	0.896 (0.10)
$r_{g(SU:MY2)}$	0.252 (0.11)	0.269 (0.12)	0.197 (0.10)	0.216 (0.11)
$r_{p(MY1:SU)}$	0.187 (0.13)	0.200 (0.13)	0.123 (0.14)	0.165 (0.14)
$r_{p(MY1:MY2)}$	0.803 (0.18)	0.810 (0.17)	0.692 (0.18)	0.720 (0.18)
$r_{p(SU:MY2)}$	0.233 (0.14)	0.254 (0.13)	0.169 (0.12)	0.186 (0.12)

[1]M1 = standard linear mixed model, M2 = threshold mixed model, M3 = recursive linear mixed model, M4 = recursive threshold mixed model.

The estimated genetic correlation between MY1 and any of the claw disorders was typically positive, in the range from 0.16 to 0.44 over models (Tables 2 to 5). The positive genetic correlations indicate that selection for increased milk yield at the early stage of lactation increases the susceptibility to claw disorders. In a previous study using logistic models, König et al. (2005a) averaged the amount of milk yield of the first 2 test days after calving and correlated this value with the estimated probability that a claw disorder occurred at any point of time in the same lactation. Despite differences in the definition of milk yield and in the observed time span, their results were nearly identical to those in the present study. König et al. (2006) showed that the market price of Holstein heifers sold at auction is mainly determined by their production measured at their first test day. In view of the estimates of genetic and of phenotypic correlations between MY1 and claw disorders, the most expensive heifers would have the greatest risk of being affected by any claw disorder.

Vinson and Kliewer (1976) compared linear and threshold models, and showed that genetic correlations from these models (at least for a simple specification) are expected to be the same. Genetic correlations from recursive models M3 and M4 were generally smaller than those estimated from models M1 and M2. Sorensen and Varona (2006) compared genetic correlations between litter size and litter weight using a standard mixed model and a recursive mixed model in 2 swine breeds. In Yorkshires, they found a sizable influence (λ coefficient) of litter size on birth weight; the genetic correlation in the recursive model was near zero, but it was –0.25 in the standard model. In the Landrace breed, the structural coefficient λ was negligible, and estimates of genetic correlations from the standard mixed model and the recursive mixed model were nearly the same. In our study, estimates for the posterior distribution of λ were different from zero (Table 6), which could explain differences in genetic correlations from standard linear or standard threshold models and

Table 4. Posterior means and standard deviations (in parentheses) of heritabilities (h^2), genetic correlations (r_g), and phenotypic correlations (r_p) for wall disorders (WD) and test-day milk yield before (MY1) and after (MY2) after diagnosis of disorder applying 4 different models[1]

	Model[1]			
Item	M1	M2	M3	M4
h^2_{MY1}	0.156 (0.05)	0.158 (0.05)	0.158 (0.05)	0.159 (0.04)
h^2_{WD}	0.103 (0.05)	0.135 (0.05)	0.086 (0.05)	0.129 (0.06)
h^2_{MY2}	0.178 (0.07)	0.187 (0.06)	0.175 (0.04)	0.181 (0.05)
$r_{g(MY1:WD)}$	0.417 (0.07)	0.436 (0.06)	0.311 (0.07)	0.303 (0.07)
$r_{g(MY1:MY2)}$	0.884 (0.06)	0.895 (0.05)	0.870 (0.05)	0.886 (0.05)
$r_{g(WD:MY2)}$	0.258 (0.08)	0.280 (0.09)	0.167 (0.10)	0.165 (0.09)
$r_{p(MY1:WD)}$	0.121 (0.08)	0.122 (0.07)	0.073 (0.11)	0.067 (0.11)
$r_{p(MY1:MY2)}$	0.720 (0.14)	0.738 (0.15)	0.711 (0.16)	0.692 (0.16)
$r_{p(WD:MY2)}$	0.193 (0.15)	0.199 (0.13)	0.099 (0.13)	0.090 (0.13)

[1]M1 = standard linear mixed model, M2 = threshold mixed model, M3 = recursive linear mixed model, M4 = recursive threshold mixed model.

Table 5. Posterior means and standard deviations (in brackets) of heritabilities (h^2), genetic correlations (r_g), and phenotypic correlations (r_p) for interdigital hyperplasia (IH) and test-day milk yield before (MY1) and after (MY2) after diagnosis of disorder applying 4 different models[1]

	Model[1]			
Item	M1	M2	M3	M4
h^2_{MY1}	0.158 (0.05)	0.162 (0.05)	0.157 (0.04)	0.148 (0.05)
h^2_{IH}	0.111 (0.05)	0.188 (0.05)	0.122 (0.06)	0.156 (0.06)
h^2_{MY2}	0.161 (0.06)	0.167 (0.06)	0.165 (0.09)	0.172 (0.09)
$r_{g(MY1:IH)}$	0.394 (0.11)	0.384 (0.12)	0.171 (0.11)	0.203 (0.12)
$r_{g(MY1:MY2)}$	0.831 (0.08)	0.902 (0.05)	0.896 (0.05)	0.899 (0.05)
$r_{g(IH:MY2)}$	0.350 (0.05)	0.346 (0.07)	0.155 (0.11)	0.178 (0.10)
$r_{p(MY1:IH)}$	0.121 (0.12)	0.195 (0.14)	0.062 (0.12)	0.077 (0.13)
$r_{p(MY1:MY2)}$	0.740 (0.16)	0.845 (0.17)	0.691 (0.17)	0.766 (0.18)
$r_{p(IH:MY2)}$	0.285 (0.13)	0.213 (0.12)	0.118 (0.12)	0.110 (0.13)

[1]M1 = standard linear mixed model, M2 = threshold mixed model, M3 = recursive linear mixed model, M4 = recursive threshold mixed model.

recursive models. In a simulation study, Legarra and Robert-Granié (2006) concluded that ignoring a recursive relationship leads to an overestimation of the genetic correlation. On the other hand, the genetic correlation would be underestimated when fitting a recursive model, if recursiveness does not exist. Wu et al. (2007) applied simultaneous and recursive models to infer relationships between milk yield and SCS of Norwegian Red cows. Heritability estimates from SIR models were similar to those from the mixed models, but some genetic correlations differed considerably among models.

Genetic correlations between all claw disorders investigated and MY2 were positive in a range from 0.077 to 0.170 for DD (Table 2), 0.317 to 0.443 for SU (Table 3), 0.165 to 0.280 for WD (Table 4), and 0.171 to 0.394 for IH (Table 5) for the various models. As shown in Tables 2 through 5, estimates of phenotypic correlations between MY1 (or MY2) and claw disorders were also positive, but generally lower than genetic correlations. The incidence of any disorder was associated with a substantial decrease of test-day milk yield on the phenotypic scale (Table 1); however, affected cows still produced more milk at the following test day than did healthy cows. Higher susceptibility to disorders was also associated with greater production at the genetic level. Sizable positive genetic and phenotypic correlations between MY1 and MY2 were found, which is consistent with estimates from several studies dealing with test-day models (e.g., Jamrozik and Schaeffer, 1997). This means that genetically superior cows for milk production at an early stage of lactation are also superior at a later stage but, nevertheless, these cows have a greater risk of being affected by any claw disorder. Heritability estimates of MY1 and MY2 at individual test days were identical to values found by König et al. (2005b) when analyzing genetic parameters of individual test-day production in large-scale dairy farms in eastern Germany.

Structural Equation Coefficients

The analysis of milk yield at test days before and after the occurrence of a disorder, plus the application of recursive models produce a clearer picture of the interplay between production and disorders than that attained in previous studies (König et al., 2005a; Swalve et al., 2005). Structural coefficients λ measure recur-

Table 6. Posterior means and standard deviations (in parentheses) of structural coefficients λ for 4 claw disorders and milk yield applying recursive linear mixed model (M3) or recursive threshold mixed model (M4)

	Dermatitis digitalis		Sole ulcer		Wall disorder		Interdigital hyperplasia	
Parameter[1]	M3	M4	M3	M4	M3	M4	M3	M4
λ_{21}	0.0244 (0.0019)	0.0158 (0.0018)	0.0060 (0.0018)	0.0039 (0.0045)	0.0044 (0.0073)	0.0031 (0.0059)	0.0189 (0.0018)	0.0031 (0.0013)
λ_{32}	–0.6771 (0.1931)	–0.4418 (0.2158)	–0.4515 (0.1570)	–0.3410 (0.1555)	–0.1233 (0.1299)	–0.1172 (0.1221)	–0.5602 (0.1880)	–0.4568 (0.1443)

[1]λ_{21} = change in incidence of a particular claw disorder (trait 2) per 1-kg increase of test-day milk yield (trait 1), λ_{32} = the change of milk yield (trait 3) at the following test day per 1-unit increase in the incidence of a particular claw disorder.

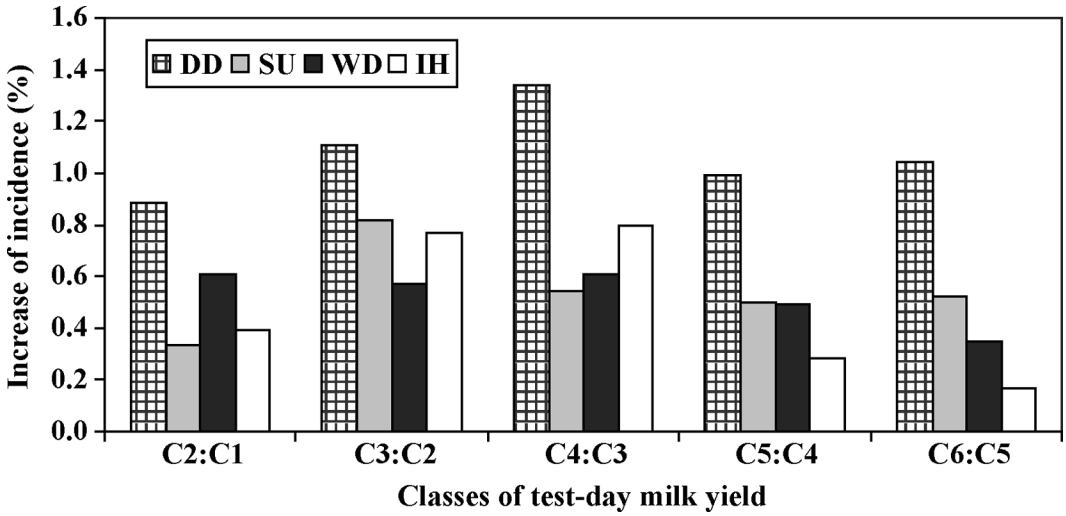

Figure 3. Posterior means for the effects of test-day milk yield (MY1 = trait 1) on the incidences of digital dermatitis (DD), sole ulcer (SU), wall disorders (WD), and interdigital hyperplasia (IH) obtained from the recursive threshold mixed model (M4). Six classes of test-day milk yield (C1, C2, ..., C6) were created within the range from 28 to 34 kg in increments of 1-kg. The effect of adjacent classes (C2:C1, C3:C2, etc.) on the incidence of claw disorders is depicted.

siveness at the phenotypic level (Gianola and Sorensen, 2004), and λ_{21} values describing the effect of MY1 on claw disorders were in the range from 0.006 to 0.024 when applying model M3, and between 0.003 and 0.016 when applying model M4 (Table 6). Under model M4, the structural coefficient λ_{21} is the gradient of the liability of the respective disease with respect to MY1; for model M3, the gradient is on the observed scale. For instance, a structural coefficient λ_{21} of 0.024 for DD in model M3 leads to the prediction that a 1-kg increase in MY1 results in an increase of incidence of DD of 2.4%. Structural coefficients for MY1 and the other 3 disorders were below 1% when applying model M3. When comparing structural coefficients λ_{21} for different claw disorders, the largest effect was found for DD (Table 6). A 1-kg increase in milk yield (MY1) increased the incidence of DD by nearly 2.5%. In contrast to the other disorders, DD is caused by a specific bacteria and it is expected that a high level in milk yield is associated with a low defense mechanism against the pathogen. Among all disorders, DD is of most concern when comparing mean incidences in recent years. For instance, Somers et al. (2003) studied Holstein cows in the Netherlands, and all herds investigated had cows infected by DD, resulting in an average cow level prevalence of 30%.

Structural equation coefficients λ_{21} on the underlying liability scale obtained from model M4 were in fair agreement with those estimated on the visible scale with model M3. To illustrate the impact of incidences of claw disorders on MY1 when applying a recursive threshold model, results using the approach of López de Maturana et al. (2007) are shown in Figure 3. For all classes of MY1 defined, an increase of test-day milk yield elevated the incidence of any claw disorder, with the largest effects for DD. The rate of change in incidences for adjacent classes was, on average, 1.14% for DD, 0.50% for SU, 0.44% for WD, and 0.38% for IH. A structural coefficient λ_{32} of –0.68 (Table 6) for DD and MY2 obtained from model M3 predicted that a 1% increase in the incidence of DD on the visible scale results in a reduction of 0.68 kg in MY2. A similar impact of claw disorders was observed for SU, WD, and IH. Coefficients λ_{32} from the recursive threshold model M4 were also negative (i.e., –0.442 for DD, –0.341 for SU, –0.117 for WD, and –0.457 for IH), but indicating the association between milk yield and the increase of the disorder by 1 unit on the underlying liability scale. Associations of the same magnitude compared with milk yield and claw disorders were reported by Wu et al. (2007) when inferring relationships between milk yield and SCS in SIR models. In their study, direct effects from SCS to milk yield were strong and negative, but varied with different production levels.

Selection Response and Sire Effects

The practical impact of different models was assessed via selection response, correlations between posterior sire effects, and the rank of sires in distinct top-10 lists. The choice of the model had consequences on prediction of response to selection. For direct comparison of results, heritabilities on the liability scale obtained from models M2 and M4 were transformed to the observed

Table 7. Rank correlation between sire posterior means estimated with 4 different models for 4 claw disorders[1]

Model[2]	Dermatitis digitalis	Sole ulcer	Wall disorder	Interdigital hyperplasia
M1:M2	0.93	0.92	0.92	0.93
M1:M3	0.91	0.90	0.88	0.92
M1:M4	0.86	0.86	0.88	0.85
M2:M3	0.85	0.86	0.87	0.84
M2:M4	0.93	0.92	0.93	0.95
M3:M4	0.95	0.97	0.98	0.98

[1]Only estimates from 511 sires with recorded daughters for claw disorders were considered.

[2]M1 = standard linear mixed model, M2 = threshold mixed model, M3 = recursive linear mixed model, M4 = recursive threshold mixed model.

scale using the formula of Robertson and Lerner (1949). As an illustration, consider selection on MY1 and the correlated selection response in IH for models M1 and M4. Applying model M1, the predicted direct response per generation for MY1 is $\Delta G_{(MY1)} = ih_{(MY1)}\sigma_{A(MY1)} = i(0.106)$ and the predicted correlated response for IH when selecting on MY1 is $\Delta G_{(IH,MY1)} = ih^2_{(MY1)}\sigma_{A(MY1)}r_{g(IH,\ MY1)} = i(0.0037)$. In comparison, for the recursive linear-threshold model (M4), the correlated selection response in IH would be equal to i(0.0016). For the same selection intensity, model M1 leads to an overstatement of correlated selection response of 1.1% per generation in incidence of IH, compared with that expected from model M4. This simple scenario illustrates the impact of model assessment and selection on estimates of genetic parameters, and on predicted selection response.

Rank correlations (Table 7) between sire posterior means within disorders applying different models M1, M2, M3, and M4 were >0.84 for all pairs of models, but there were substantial changes in rank for top-ranked sires (Table 8). For example, for IH, the best sire when applying model M1 was ranked as number 15 when using model M4. The rank correlations between sire posterior means were greatest between recursive models M3 and M4, and lowest between the standard linear model M1 and the recursive threshold model M4 (Table 7). In the global market of Holstein dairy cattle breeding, only the top-ranked sires are competitive, even if differences in predicted breeding values with lower-ranked bulls are minor (Dekkers et al., 1996). Again, this underlines the importance of model choice in breeding value estimation, and ongoing studies should address this topic.

The question of how the impact of mutual effects between distinct traits on prediction of breeding values should be handled is of increasing interest in animal breeding. In the case of production and fertility, several authors (e.g., Olori et al., 1997; Bormann et al., 2002) observed a significant impact of pregnancy status on test-day milk yield, depending on lactation stage. An analysis of Bohmanova et al. (2006) considered the effect of days open as well as state of pregnancy (i.e., traits describing fertility) on the prediction of breeding values for milk yield with a random regression test-day model. Recursive models constitute an appealing alternative for dealing with this problem. If needed, the model can be expanded into one with simultaneous or feedback (even time-lagged) relationships.

CONCLUSIONS

Claw disorders are of increasing concern within the German Holstein dairy cattle population and suitable data recording systems are just becoming available. Therefore, appropriate models for estimation of genetic parameters should be developed to make selection as effective as possible. In the case of disorders and produc-

Table 8. The top-10 sires based on sire posterior mean for 4 claw disorders estimated with model M1 and their ranking applying models M2, M3, and M4[1,2]

Dermatitis digitalis				Sole ulcer				Wall disorder				Interdigital hyperplasia			
M1	M2	M3	M4	M1	M2	M3	M4	M1	M2	M3	M4	M1	M2	M3	M4
1	3	9	11	1	5	8	10	1	9	1	11	1	4	10	15
2	5	5	3	2	4	2	8	2	7	13	13	2	9	2	11
3	11	10	14	3	9	6	11	3	10	9	5	3	1	11	14
4	6	1	7	4	16	13	16	4	2	18	20	4	10	16	1
5	16	24	12	5	15	10	17	5	11	3	9	5	11	19	20
6	8	11	13	6	8	3	13	6	12	2	10	6	17	4	24
7	2	8	10	7	1	12	9	7	8	12	14	7	2	12	13
8	1	14	5	8	10	5	1	8	15	20	1	8	8	20	4
9	19	7	15	9	3	18	5	9	1	7	5	9	3	19	6
10	10	20	6	10	11	24	20	10	18	14	21	10	26	3	19

[1]Only estimates from 79 sires with >30 recorded daughters for claw disorders were considered.

[2]M1 = standard linear mixed model, M2 = threshold mixed model, M3 = recursive linear mixed model, M4 = recursive threshold mixed model.

tion traits, recursiveness between traits as discussed by Gianola and Sorensen (2004) should be investigated further. The present study showed that ignoring recursive relationships between traits can lead to overestimation of the genetic correlation between claw disorders and test-day milk yield. Differences in values of genetic parameters among different models have consequences on predicted responses to selection, as illustrated for interdigital hyperplasia, and on the ranking of top sires based on predicted breeding values. As shown in this study, the methodology, although relatively new in the field of dairy cattle breeding, can also be extended for categorical traits. Recursive threshold models in a Bayesian framework are useful for investigating similar questions in animal breeding. Dairy cattle breeding schemes are moving toward the use of more complex breeding goals involving a multiplicity of binary-coded health-related traits. Hence, evaluation of recursive or even recursive threshold models for non-Gaussian traits is an important area of future research. A recursive threshold model seems to provide an appealing statistical specification for genetic evaluation of traits that are affected in a manner similar to that shown in Figure 1.

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