Genetic parameters for clinical mastitis in the first three lactations of Dutch Holstein cattle
Saskia Bloemhof, Gerben De Jong, Yvette De Haas

To cite this version:
Saskia Bloemhof, Gerben De Jong, Yvette De Haas. Genetic parameters for clinical mastitis in the first three lactations of Dutch Holstein cattle. Veterinary Microbiology, Elsevier, 2009, 134 (1-2), pp.165. <10.1016/j.vetmic.2008.09.024>. <hal-00532484>

HAL Id: hal-00532484
https://hal.archives-ouvertes.fr/hal-00532484
Submitted on 4 Nov 2010

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L’archive ouverte pluridisciplinaire HAL, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d’enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.
Accepted Manuscript

Title: Genetic parameters for clinical mastitis in the first three lactations of Dutch Holstein cattle

Authors: Saskia Bloemhof, Gerben de Jong, Yvette de Haas

PII: S0378-1135(08)00379-9
Reference: VETMIC 4164

To appear in: VETMIC

Please cite this article as: Bloemhof, S., de Jong, G., de Haas, Y., Genetic parameters for clinical mastitis in the first three lactations of Dutch Holstein cattle, Veterinary Microbiology (2008), doi:10.1016/j.vetmic.2008.09.024

This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.
Genetic parameters for clinical mastitis in the first three lactations of Dutch
Holstein cattle

Saskia Bloemhof¹,², *, Gerben de Jong¹, Yvette de Haas¹

¹ NRS, Animal Evaluation Unit, P.O. Box 454, 6800 AL Arnhem, The Netherlands
² Animal Breeding and Genomics Centre, Wageningen University, P.O. Box 338, 6700 AH Wageningen, The Netherlands

† Present address:
IPG, Institute for Pig Genetics B.V., P.O. Box 43, 6440 AA Beuningen, The Netherlands

* Corresponding author:
E-mail address: Saskia.Bloemhof@ipg.nl (S. Bloemhof)
Tel.: +31-24-677 9999
Fax: +31-24-677 9800
Abstract

The first breeding value for udder health of a bull is based on the performance of his daughters in their first lactation. However, clinical mastitis (CM) is not a problem in first lactation only. Therefore, the objective of this study was to estimate genetic parameters for CM and somatic cell count (SCC) for the first 3 lactations of Dutch Holstein cattle. Data from 250 Dutch herds recording CM were used to quantify the genetic variation of CM in parity 1, 2, and 3, respectively. The dataset contained 35,379 lactations from 21,064 animals of different parities. Test-day SCC was available from all lactations. Somatic cell counts were log-transformed to somatic cell scores (SCS) and averaged over test-day records between 5 and 335, 5 and 150, and 151 and 335 days in milk. Variance components for CM and SCS were estimated using a sire-maternal grandsire model. The heritability for CM was approximately 3% in all parities. Genetic correlations between CM in consecutive lactations were high (0.9), but somewhat lower between parity 1 and 3 (0.6). All genetic correlations between CM and SCS were positive, implying that genetic selection on lower SCC will reduce CM-incidence. Estimated genetic correlations were stronger for SCS in the first half of lactation than in the second half of lactation. Selection indices showed that most progress could be achieved when treating CM in parity 1, 2, and 3 as different traits and by including SCS between 5 and 150 days in the udder health index.

Keywords: clinical mastitis, somatic cell count, genetic parameters
1. Introduction

Clinical mastitis (CM) is one of the major diseases in Dutch dairy herds. It is characterized by visible signs such as clots and flakes in the milk (Hamann, 2005), and possibly decreased milk production, swelling of the udder, pain of the quarter, and an increased body temperature (Smith and Hogan, 1993; Harmon, 1994).

Risk of CM is affected by a number of factors such as pathogenicity of microorganisms, management factors like treatment and prevention strategies (Schukken et al., 1997), conformation and immunological performance of the cow, and genetic predisposition of the cow. This makes genetic selection a strategy to reduce the incidence of CM. The advantage of reducing CM by breeding is that it results in a permanent change in the genetic composition of the dairy herd (Shook, 1989).

Breeding for increased resistance to CM can be performed in three ways, i.e. by direct selection against CM, by indirect selection using indicator traits, or by a combination of both. For direct selection, CM needs to be measured on the cow or her relatives. Heritability of CM is generally below 0.05 when estimated with linear models (Mrode and Swanson, 1996; Heringstad et al., 2000). Direct selection to reduce the incidence of CM is used in Scandinavia (Norway, Sweden, Finland, and Denmark). In most other countries, cases of CM are not recorded. Therefore, indirect selection needs to be performed using traits that are genetically correlated to CM. Somatic cell count (SCC) is the trait that is most commonly used for indirect selection (Rupp and Boichard, 1999; Heringstad et al., 2000; Carlén et al., 2004). Heritability estimates of SCC or especially of log transformed SCC are around 0.10 (De Haas et al., 2003; Carlén et al., 2004; Ødegård et al, 2004). The genetic correlation between...
SCC and CM is moderate to high, ranging from 0.2 to 0.7 (Rupp and Boichard, 1999; Carlén et al., 2004; Koivula et al., 2004).

The first breeding value for udder health of a bull is based on the performance of his daughters in their first lactation. However, CM is not a problem in first lactation only (Carlén et al., 2004). Actually, both frequency of CM (Pösö and Mäntysaari, 1996), and level of SCC (Reents et al., 1995) increase with increasing parity. Genetic correlations between CM in the first 3 parities, estimated using linear sire models, vary from 0.67 to 0.92 (Pösö and Mäntysaari, 1996; Carlén et al., 2004). When using multivariate threshold models, the range of estimates of genetic correlations between parities is even broader, ranging from 0.42 to 0.91 (Heringstad et al., 2004; Zwald et al., 2006). These studies have been performed in Scandinavia and the United States.

In the Netherlands, the breeding goal is to improve resistance against CM in parity 1, 2, and 3. However, cases of CM are currently not routinely recorded in management information systems (MIS) in the Netherlands. Therefore, selection is based on an udder health index, which includes the following indicator traits: SCC, udder depth, fore udder attachment, teat length, and milking speed (NRS, 2005). In the current Dutch udder health index CM has been treated as the same trait in parities 1, 2 and 3. However, several authors suggest that CM in different parities is a different trait (Pösö and Mäntysaari, 1996; Carlén et al., 2004; Heringstad et al., 2004). So far, SCC averaged over 3 lactations has been used in the Dutch udder health index, but several studies have shown that the incidence of CM is much higher in the first half of lactation than in the second half (Emanuelson et al., 1988; Barkema et al., 1998; De Haas et al., 2002). Therefore, the hypothesis is that SCC in the first half of lactation is more informative as a mastitis-indicator than SCC in the second half of lactation or SCC throughout the whole lactation.
The aims of this study were: (1) to estimate genetic parameters for CM for the first three lactations of Dutch Holstein cattle; (2) to correlate CM with SCC, where SCC is averaged over the first half, the second half, and the whole lactation; and (3) to estimate accuracy of selection for different indices to improve resistance against CM in parity 1, 2, and 3.

2. Materials and Methods

2.1. Available data

Records on CM treatments were available from MIS on 307 Dutch dairy farms. Data recording was done on a voluntary basis and was performed by the farmers. Data on CM were recorded from June 1998 until May 2006. All herds participated in the national milk recording system. The NRS (Arnhem, The Netherlands) provided the complete history of the milk production recordings (MPR) from all animals that participated in the study. A record included an animal identification number, herd number, date of calving, parity, date of MPR, MPR yields (kilogram milk, fat percentage, and protein percentage) and SCC (in 1,000 cells per ml). A pedigree file of all participating animals was available and contained their ancestry of approximately 110,000 animals (about 88,400 animals and 12,600 bulls) back to 1919. Record also included date of birth and breed of the animal. Breed was subdivided into three main contributing breeds, with each having up to nine classes (0, 1/8, ..., 8/8) depending on the degree of contribution. The main breeds were Holstein-Friesian, Dutch-Friesian, and Meuse-Rhine-Yssel.
2.2. Data editing at farm level

Observations from farms without any case of CM were removed. It was assumed that when no cases of CM were recorded, the farmer was not using the MIS for registration of CM. For each farm, data were included from animals that had calved at least half a year after the first case of CM was registered on that farm. It was assumed that a farmer needed this period of six months to learn to use the MIS consistently. This was confirmed by incidence profiles, which showed that the incidence of CM per farm was very unstable in the first six months after installing the MIS. After that period, the incidence of CM stabilized. All test-days that were recorded later than 28 days after the last recorded case of CM on a farm were removed.

2.3. Data editing at lactation level

Clinical mastitis was defined on a lactation basis (between 1 and 335 DIM) as an all-or-none trait; either an animal had CM in a certain lactation (1) or she did not (0). Therefore, only the first cases of CM per lactation were included in the dataset (CM1, CM2 and CM3 for clinical mastitis during parity 1, 2, and 3, respectively).

The data were edited using the following inclusion criteria: (1) age at calving $\geq 640$ days; (2) parity $\leq 3$; and (3) observations on cell count available between 5 and 335 days in milk (DIM). All animals with less than 75% Holstein-Friesian were excluded. Animals with unknown parents and daughters from sires with less than three daughters in the dataset were also removed.

Somatic cell count was transformed to somatic cell score (SCS): $1000+100*(3\log(SCC/1000))$. Test-day SCS were averaged per lactation from 5 up to
335 DIM, from 5 up to 150 DIM, and from 151 up to 335 DIM (SCS5-335, SCS5-150 and SCS151-335, respectively). An average was calculated only if the animal had 2 or more test-day records in the first or second half of the lactation, and 4 or more test-day records for 335 DIM, otherwise a missing value was assigned.

The final dataset consisted of 35,379 lactations from 21,064 animals on 250 farms, with a total of 7,266 cases of CM from 6,426 animals. A pedigree file was constructed based on sires and maternal grand sires (MGS) of animals in the data. This file contained 3,855 AI bulls of which 1,629 were sires and 2,226 were MGS of the animals in the dataset. The 3,855 AI bulls came from 766 sires and 2,874 dams.

2.4. Statistical analyses

ASREML (Gilmour et al., 2002) was used to estimate variance components. Heritabilities were estimated in univariate analyses using a linear mixed model for CM1, CM2, CM3, SCS5-335, SCS5-150, and SCS151-335. The model included random effects for sire and MGS. The model used was:

\[ Y = \mu + \text{fixed effects} + S_{\text{sire}} + \frac{1}{2} S_{\text{mgs}} + e \]

The random sire effect was identified by the subscripts for sire and MGS; \( S_{\text{sire}} \) and \( S_{\text{mgs}} \), respectively. The sire effects were linked using the relationship matrix, and were assumed to be normally distributed with \( \text{var}(S_{\text{sire or mgs}}) = \sigma^2_s \). For the CM-trait, fixed effects included were an interaction between herd and year of calving (948 classes) and month of calving (12 classes). Age at calving (in days) was included in the model.
as a fixed continuous effect. For the SCS-traits, a repeatability model was used and therefore parity (3 classes) was included in the fixed effects.

Bivariate analyses were carried out to estimate correlations between CM and SCS-traits. Fixed effects included the interaction of herd and year of calving (948 classes) and month of calving (12 classes). Age at calving (in days) was included in the model as a fixed continuous effect. For the SCS-traits, parity (with 3 classes) was added as fixed effect. Animals with missing values for one of the two traits were still included in the bivariate analyses.

Genetic parameters were calculated from the estimated variance components. The additive genetic variance was calculated by multiplying the sire variance by 4. The phenotypic variance ($\sigma^2_p$) was the sum of the sire variance multiplied by 1.25, where 1.25 was included because MGS was fitted in the model separately, plus the residual variances. Division of the additive genetic variance by the phenotypic variance resulted in the heritability. Genetic, phenotypic, and error correlations were estimated using the corresponding variances and covariances.

The breeding goal in the Netherlands is to select for an improved resistance against CM in parity 1, 2, and 3. Accuracies of several selection indices were calculated for this breeding goal (Hazel, 1943; Van der Werf, 2006). The selection indices consisted of the same traits as the current Dutch udder health index (i.e. SCS averaged over first 3 lactations, udder depth, fore udder attachment, teat length, and milking speed). Table 1 lists the genetic parameters that are currently used. In the Dutch udder health index, CM in parity 1, 2, and 3 has been considered as the same trait (Table 1). To test if including parity-specific CM in the breeding goal increases accuracy of selection, overall CM (Table 1) was replaced by CM1, CM2, and CM3. The genetic correlations used between the 3 CM-traits were those estimated in our
study. The genetic correlations with the other traits in the index were assumed to stay the same as used in the current Dutch udder health index (Table 1).

To test if the accuracy of selection could be improved and to test which SCS trait would be genetically most informative as mastitis-indicator, the cell count trait (Table 1) was replaced by SCS5-335, SCS5-150 or SCS151-335. The genetic correlations between cell count traits and parity-specific CM traits were those estimated in our study. Genetic correlations with the other traits in the index were assumed to stay the same as used in the current Dutch udder health index (Table 1). Comparisons were made between sires with 100 or 10,000 daughters, i.e. young versus proven bulls.

3. Results

3.1. Descriptive analyses

Across parities, milk production of mastitic animals was 0.6 kilograms higher than the milk production of the healthy animals, and fat percentage for mastitic animals was lower for healthy animals (Table 2). The differences in protein percentage between mastitic animals and healthy animals were small. Somatic cell count was highest for mastitic animals. The largest difference between the SCC of mastitic compared to healthy animals was seen in parity 3 (Table 2).

The mean proportion of animals that had CM at least once during lactation was 15.8%, and was lowest for heifers (13.4%) and highest for third parity cows (19.6%). Of all second parity cows 16.1% experienced CM at least once during
lactation. The proportion of heifers with CM increased rapidly up to 50 DIM (Figure 1). Half of all first cases of CM in heifers occurred before 10 DIM, whereas half of the total proportion of 2nd and 3rd parity cows with CM was approached around 70 DIM. Before 150 DIM, 75% of all first cases of CM had occurred.

3.2. Genetic parameters for clinical mastitis

The heritabilities for CM in parity 1, 2, and 3 were all around 3% (Table 3). The genetic correlations between CM in consecutive parities were high (≈ 0.9 ± 0.1), but lower between parity 1 and 3 (0.6 ± 0.2).

3.3. Genetic correlations between clinical mastitis and somatic cell count

The genetic correlations between CM and lactation-average SCS were 0.8 (± 0.1) in parity 2 and 3 (Table 4), but somewhat lower in first parity (0.6 ± 0.1). Even stronger genetic correlations were estimated for SCS in the first half of lactation, whereas SCS in the second half of lactation showed weak correlations with CM (Table 4).

3.4. Selection index calculations

Accuracy was 72% for young sires with 100 daughters using the current Dutch udder health index (Table 5). However, when parity-specific CM was included in the breeding goal, accuracy of selection increased to 75%. Even higher accuracy could be
achieved when selecting on SCC in first half of lactation (SCS5-150) (83%). For well
proven sires with 10,000 daughters accuracy increased to 90% (Table 5).

4. Discussion

4.1. Representativeness of data

The descriptive analyses showed that the data used to estimate genetic parameters was representative of the Dutch dairy cattle population. The average milk production of the animals in this study (26.8 kg) was equal to the average daily milk production in the Netherlands (26.8 kg) (NRS, 2006). The average fat percentage (4.51%) and protein percentage (3.55%) in this study (data not shown) were only slightly higher than the national average fat percentage (4.36%) and the average protein percentage (3.49%) (NRS, 2006).

The mean proportion of animals that had CM at least once during lactation in this study was 15.8%. The Dutch udder health centre reported a frequency of 25% for the Dutch situation based on a study of Barkema et al. (1999). This higher frequency refers to all cases of CM during the entire lactation, for all parities, whereas the frequency in our study only includes the first cases of CM per lactation in the first three lactations. Incidence of CM increases with increasing parity (Pösö and Mäntysaari, 1996).

4.2. Genetic parameters of clinical mastitis
Heritability estimates of parity-specific CM were low (Table 4) but in the
range of 0.01 to 0.06 reported in previous studies using linear models (Pösö and
Mäntysaari, 1996; Heringstad et al., 2000; Carlén et al., 2004). However, CM is an
all-or-none trait and heritability estimates on the linear scale are therefore frequency
dependent. Heritability estimates of different studies are therefore not easily
comparable (Heringstad et al., 2000).

The genetic correlations between CM in consecutive lactations (i.e. 1 and 2,
and 2 and 3) were higher than the genetic correlation between CM in non-adjacent
lactations (i.e. 1 and 3). This is in agreement with the results from previous studies
where also the highest genetic correlations were estimated between CM in second and
third parity and lowest between CM in first and third parity (Pösö and Mäntysaari,
1996; Carlén et al., 2004). This implies that CM in consecutive lactations is,
genetically, more the same trait, than CM in non-adjacent lactations. Rupp et al.
(2000) also showed that udder health in first and second parity was genetically
correlated. They concluded that animals with the lowest mean SCC in the first
lactation had the lowest risk for CM in the second lactation. The lower genetic
correlation estimated between first and third lactation (0.63 ± 0.22) could be due to
selection. Animals that suffered from mastitis might be removed from the herd and
were not fulfilling three lactations.

Parity-specific CM and lactation-average SCS over 3 parities showed a
moderate favorable genetic correlation in first parity (0.64), and strong favorable
genetic correlations in later parities (0.79). The favorable genetic correlations imply
that selection for lower lactation-average SCS will decrease the incidence of CM.
Similar parity-specific genetic correlations were estimated by Carlén et al. (2004),
who also observed the highest genetic correlation between CM in parity 3 and
lactation-average SCS. In general, the estimated genetic correlations are in line with those reported in literature (see review Mrode and Swanson, 1996; Heringstad et al., 2000).

Lower genetic correlations were estimated between CM and average SCS in the latter half of the lactation, than between CM and average SCS in the first half of the lactation. Standard errors were quite large, and correlations should be interpreted with caution. Even so, the estimated genetic correlations implied that selection for lower SCS, especially during early lactation, decreases the incidence of CM. Emanuelson et al. (1988) reported a similar trend in the estimated genetic correlations in three separate Swedish datasets. The genetic correlations estimated in our study between CM and average SCS in the first half of the lactation were stronger than the genetic correlations with lactation-average SCS in the entire lactation. This implied that a real difference exists in the genetic resistance to CM between different parts of the lactations, which is consistent with earlier findings (Emanuelson et al., 1988; De Haas et al., 2002).

Heritabilities and correlations in this study were estimated using linear mixed sire maternal grandsire models. These models assume normality of residuals. However, CM is a binary trait and residuals will therefore not be normal distributed. Non-linear threshold models have been shown to be theoretically better for analyses of binary traits (Kadarmideen et al., 2000; Heringstad et al., 2004). This is something that will be taken into account in future studies when optimizing the analyses further.

4.3. Selection against mastitis
The estimated heritabilities of CM in consecutive lactations were around 3%. This is very low when compared with production traits such as milk production which has a heritability of around 30% (Pösö and Mäntysaari, 1996; Carlén et al., 2004). Effectiveness of selection for traits with low heritability could be improved by testing more daughters per progeny-test sire. This is very costly for breeding organizations (Veerkamp and De Haas, 2005).

Another option is to combine indicator traits in an index. An “index method” combines information into a single figure that gives an optimal selection criterion. Optimal is defined as ‘most accurate’ or ‘giving the highest selection response when selecting for it’. In the Netherlands, the breeding goal is to improve resistance against CM in parity 1, 2, and 3. Therefore indices were developed to optimally predict the animal’s susceptibility for cases of CM. In the udder health index currently used in the Netherlands CM in parity 1, 2, and 3 was not treated as different traits and the SCC trait included was SCS averaged over three lactations, this resulted in accuracy for young sires of 72%. When parity-specific CM (CM1, CM2, and CM3) was included in the breeding goal the accuracy increased to 77% for young sires. SCC in the first half of lactation (SCS5-150) was the most informative indicator trait. When including SCS5-150, accuracy of selection increased to 83% for young sires, which is an improvement of 6 percentage points. Similarly, for well-proven sires with 10,000 daughters, selection against lower average SCC in the first half of lactation (SCS5-150) was more effective than selection on other SCC measures explored in our study.

Average SCC values do not reflect dynamic variation in SCC. The most desirable animal would respond very quickly to an infection, resulting in increased SCC, followed by return to normal SCC levels. De Haas et al. (2003) showed that patterns of peaks in SCC, which are based on deviations from the normal lactation
curve, are more effective in the selection against CM than lactation average SCC. Further optimization of indices might therefore possibly be achieved by including patterns of peaks in SCC in addition to lactation average SCC. This will be investigated in future studies.

Cases of CM are not routinely recorded in the Dutch breeding evaluation program, but many commercial dairy farmers record disease treatments in a MIS (Zwald et al., 2006). If these farmers give permission to upload the CM treatment information to the Dutch breeding evaluation program, direct information on CM could be included in the Dutch udder health index.

To investigate if combining direct and indirect information in an index increased accuracy of selection, CM1 information of 10, 25, 50, and 100 daughters was added to the index for young sires with 100 daughters (Table 6). Without optimizing the SCC trait, the accuracy of the current udder health index could increase with 2 to 10 percentage points. Combining the most informative SCC trait (SCS5-150) and direct CM1 information on 100% of the daughters resulted in an even higher accuracy of selection (89%). This is an improvement of 12 percentage points. However, at this moment it is not feasible to have CM information of the complete population. It was shown that including CM1 information of 10% of the daughters in the index already resulted in an increased accuracy of selection of 86% (Table 6). This is 9 percentage points higher than the accuracy currently achieved for young bulls with the Dutch udder health index. Higher accuracies result in more consistent estimated breeding values of young bulls, and a better distinction will be given between the best and worst sires, with regard to udder health. Therefore it is worthwhile for the Dutch dairy industry to develop a national recording system for CM. Until direct CM information is available in a continuous data flow, future studies
should focus on defining alternative SCC traits that provide additional information for selection that aims to decrease genetic susceptibility to CM.

5. Conclusions

The occurrence of CM increases with increasing parity, from 13% to 19%. The estimated heritabilities of CM in parity 1, 2, and 3 were all low, around 3%. Genetic correlations between CM in consecutive parities were high, but somewhat lower between CM in non-adjacent parities. This implies that CM in consecutive lactations is, genetically, more the same trait, than CM in non-adjacent lactations. Strong genetic correlations were estimated between CM and all SCC traits, implying that selecting on lower SCC leads to an increased genetic resistance against CM. Genetic correlations between parity-specific CM and SCC traits increase with increasing parity. Somatic cell score across the first half of lactation is more strongly correlated with CM than SCS across the whole lactation or the second half of the lactation; however this should be interpreted with caution because of high standard errors. Dutch dairy bulls are selected for udder health based on an udder health index. This index currently treats CM as the same trait in parity 1, 2, and 3 and comprises udder conformation traits, milking speed and SCC averaged over 3 lactations. However, higher accuracies can be achieved when parity-specific CM is considered in the breeding goal and when SCC averaged over first 150 DIM is included in the index, instead of the currently used 335d SCC. Heifer mastitis is genetically a different trait than mastitis in older cows. Therefore breeding programs should consider CM in different parities as different traits.
Acknowledgements

The farmers are greatly acknowledged for providing information on occurrence of cases of clinical mastitis. This study is part of the five-year mastitis program of the Dutch Udder Health Centre and was financially supported by the Dutch Dairy Board. The authors thank Henk Bovenhuis for his suggestions and useful comments on the manuscript.

Conflict of interest

None of the authors (S. Bloemhof, G. de Jong, Y. de Haas) has a financial or personal relationship with other people or organizations that could inappropriately influence or bias the paper entitled “Genetic parameters for clinical mastitis in the first three lactations of Dutch Holstein cattle”.

References


Table 1

Genetic parameters of clinical mastitis (CM) and the traits\(^1\) in the current Dutch udder health index, heritabilities on diagonal and genetic correlations below diagonal.

<table>
<thead>
<tr>
<th></th>
<th>CM</th>
<th>UD</th>
<th>FUA</th>
<th>TL</th>
<th>MS</th>
<th>SCSLAC1-3</th>
<th>b-values</th>
</tr>
</thead>
<tbody>
<tr>
<td>CM</td>
<td>0.04</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>UD</td>
<td>0.40</td>
<td>0.40</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.07</td>
</tr>
<tr>
<td>FUA</td>
<td>0.35</td>
<td>0.72</td>
<td>0.30</td>
<td></td>
<td></td>
<td></td>
<td>0.04</td>
</tr>
<tr>
<td>TL</td>
<td>-0.15</td>
<td>-0.26</td>
<td>-0.26</td>
<td>0.43</td>
<td></td>
<td></td>
<td>-0.09</td>
</tr>
<tr>
<td>MS</td>
<td>-0.30</td>
<td>0.00</td>
<td>0.00</td>
<td>-0.30</td>
<td>0.21</td>
<td></td>
<td>-0.12</td>
</tr>
<tr>
<td>SCSLAC1-3</td>
<td>0.70</td>
<td>0.35</td>
<td>0.30</td>
<td>0.05</td>
<td>-0.30</td>
<td>0.35</td>
<td>0.38</td>
</tr>
</tbody>
</table>

\(^1\) CM = Clinical mastitis, UD = Udder depth, FUA = Fore udder attachment, TL = Teat length, MS = Milking speed, SCSLAC1-3 = Somatic cell score averaged over 3 lactations
Table 2

Average daily milk production (in kg), fat percentage, protein percentage, and somatic cell count (SCC) (*1,000 cells/ml) for healthy (0) and mastitic (1) animals, over all parities, and divided per parity

<table>
<thead>
<tr>
<th>CM</th>
<th>Milk</th>
<th>Fat</th>
<th>Protein</th>
<th>SCC</th>
</tr>
</thead>
<tbody>
<tr>
<td>Overall</td>
<td>0</td>
<td>26.7</td>
<td>4.52</td>
<td>3.55</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>27.3</td>
<td>4.47</td>
<td>3.54</td>
</tr>
<tr>
<td>1st parity</td>
<td>0</td>
<td>23.9</td>
<td>4.50</td>
<td>3.51</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>23.9</td>
<td>4.47</td>
<td>3.52</td>
</tr>
<tr>
<td>2nd parity</td>
<td>0</td>
<td>28.3</td>
<td>4.53</td>
<td>3.58</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>28.5</td>
<td>4.46</td>
<td>3.57</td>
</tr>
<tr>
<td>3rd parity</td>
<td>0</td>
<td>29.8</td>
<td>4.53</td>
<td>3.55</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>29.8</td>
<td>4.47</td>
<td>3.54</td>
</tr>
</tbody>
</table>
Table 3

Estimated parameters for clinical mastitis in parity 1, 2, and 3 (CM1, CM2, and CM3, respectively). Heritabilities on diagonal, phenotypic correlations below diagonal and genetic correlations above diagonal, with standard errors as subscripts.

<table>
<thead>
<tr>
<th></th>
<th>CM1</th>
<th>CM2</th>
<th>CM3</th>
</tr>
</thead>
<tbody>
<tr>
<td>CM1</td>
<td>\textbf{0.03} \text{0.01}</td>
<td>0.88 \text{0.13}</td>
<td>0.63 \text{0.22}</td>
</tr>
<tr>
<td>CM2</td>
<td>0.06 \text{0.01}</td>
<td>\textbf{0.03} \text{0.01}</td>
<td>0.91 \text{0.12}</td>
</tr>
<tr>
<td>CM3</td>
<td>0.05 \text{0.02}</td>
<td>0.12 \text{0.01}</td>
<td>\textbf{0.04} \text{0.01}</td>
</tr>
</tbody>
</table>
Table 4

Genetic correlations between clinical mastitis in parity 1, 2 and 3 (CM1, CM2, and CM3, respectively), and somatic cell scores averaged from 5 to 335 days (SCS5-335), from 5 to 150 days (SCS5-150), and from 151 to 335 days (SCS151-335), with standard errors as subscripts

<table>
<thead>
<tr>
<th></th>
<th>CM1</th>
<th>CM2</th>
<th>CM3</th>
</tr>
</thead>
<tbody>
<tr>
<td>SCS5-335</td>
<td>0.64 ± 0.12</td>
<td>0.79 ± 0.10</td>
<td>0.79 ± 0.10</td>
</tr>
<tr>
<td>SCS5-150</td>
<td>0.65 ± 0.11</td>
<td>0.86 ± 0.08</td>
<td>0.88 ± 0.09</td>
</tr>
<tr>
<td>SCS151-335</td>
<td>0.50 ± 0.15</td>
<td>0.59 ± 0.14</td>
<td>0.61 ± 0.14</td>
</tr>
</tbody>
</table>
Table 5

Accuracies of udder health indices with regard to increased resistance to clinical mastitis (CM) in parity 1, 2, and 3 treated as the same trait (Current Dutch udder health index) and treated as different traits (Parity-specific CM). Including different somatic cell count traits\(^1\) in addition to the indicator-traits udder depth, fore udder attachment, teat length, and milking speed, based on sires with 100 or 10,000 daughters.

<table>
<thead>
<tr>
<th></th>
<th>100 daughters</th>
<th>10,000 daughters</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Dutch udder health index</td>
<td>0.72</td>
<td>0.75</td>
</tr>
<tr>
<td>Parity-specific CM</td>
<td>0.77</td>
<td>0.81</td>
</tr>
<tr>
<td>Parity-specific CM and SCS5-335</td>
<td>0.80</td>
<td>0.84</td>
</tr>
<tr>
<td>Parity-specific CM and SCS5-150</td>
<td>0.83</td>
<td>0.90</td>
</tr>
<tr>
<td>Parity-specific CM and SCS151-335</td>
<td>0.67</td>
<td>0.70</td>
</tr>
</tbody>
</table>

\(^1\) somatic cell scores averaged from 5 to 335 days (SCS5-335), from 5 to 150 days (SCS5-150) and from 151 to 335 days (SCS151-335).
Table 6

Accuracies of udder health indices with regard to increased resistance to clinical mastitis (CM) in parity 1, 2, and 3 treated as the same trait (Current Dutch udder health index) and treated as different traits (Parity-specific CM). Including direct clinical mastitis (CM) information in first lactation of 10 daughters, 25 daughters, 50 daughters and 100 daughters, in addition to different somatic cell count traits\(^1\) and the indicator-traits udder depth, fore udder attachment, teat length, and milking speed, based on young sires with 100 daughters.

<table>
<thead>
<tr>
<th># daughters with direct CM information</th>
<th>10</th>
<th>25</th>
<th>50</th>
<th>100</th>
</tr>
</thead>
<tbody>
<tr>
<td>Current Dutch udder health index</td>
<td>0.74</td>
<td>0.76</td>
<td>0.79</td>
<td>0.82</td>
</tr>
<tr>
<td>Parity-specific CM</td>
<td>0.78</td>
<td>0.79</td>
<td>0.80</td>
<td>0.81</td>
</tr>
<tr>
<td>Parity-specific CM and SCS5-335</td>
<td>0.82</td>
<td>0.82</td>
<td>0.83</td>
<td>0.85</td>
</tr>
<tr>
<td>Parity-specific CM and SCS5-150</td>
<td>0.86</td>
<td>0.87</td>
<td>0.88</td>
<td>0.89</td>
</tr>
<tr>
<td>Parity-specific CM and SCS151-335</td>
<td>0.69</td>
<td>0.71</td>
<td>0.73</td>
<td>0.76</td>
</tr>
</tbody>
</table>

\(^1\)Somatic cell scores averaged from 5 to 335 days (SCS5-335), from 5 to 150 days (SCS5-150) and from 151 to 335 days (SCS151-335).
Figure 1

The cumulative frequency of animals with at least one case of clinical mastitis during parity 1, 2, or 3 (CM1, CM2, and CM3, respectively) per day in milk.