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1 **Genetic parameters for clinical mastitis in the first three lactations of Dutch**
2 **Holstein cattle**

3

4

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19

19 **Abstract**

20

21 The first breeding value for udder health of a bull is based on the performance
22 of his daughters in their first lactation. However, clinical mastitis (CM) is not a
23 problem in first lactation only. Therefore, the objective of this study was to estimate
24 genetic parameters for CM and somatic cell count (SCC) for the first 3 lactations of
25 Dutch Holstein cattle. Data from 250 Dutch herds recording CM were used to
26 quantify the genetic variation of CM in parity 1, 2, and 3, respectively. The dataset
27 contained 35,379 lactations from 21,064 animals of different parities. Test-day SCC
28 was available from all lactations. Somatic cell counts were log-transformed to somatic
29 cell scores (SCS) and averaged over test-day records between 5 and 335, 5 and 150,
30 and 151 and 335 days in milk. Variance components for CM and SCS were estimated
31 using a sire-maternal grandsire model. The heritability for CM was approximately 3%
32 in all parities. Genetic correlations between CM in consecutive lactations were high
33 (0.9), but somewhat lower between parity 1 and 3 (0.6). All genetic correlations
34 between CM and SCS were positive, implying that genetic selection on lower SCC
35 will reduce CM-incidence. Estimated genetic correlations were stronger for SCS in
36 the first half of lactation than in the second half of lactation. Selection indices showed
37 that most progress could be achieved when treating CM in parity 1, 2, and 3 as
38 different traits and by including SCS between 5 and 150 days in the udder health
39 index.

40 **Keywords:** clinical mastitis, somatic cell count, genetic parameters

41

41 1. Introduction

42

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

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

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

65 SCC and CM is moderate to high, ranging from 0.2 to 0.7 (Rupp and Boichard, 1999;
66 Carlén et al., 2004; Koivula et al., 2004).

67 The first breeding value for udder health of a bull is based on the performance
68 of his daughters in their first lactation. However, CM is not a problem in first lactation
69 only (Carlén et al., 2004). Actually, both frequency of CM (Pösö and Mäntysaari,
70 1996), and level of SCC (Reents et al., 1995) increase with increasing parity. Genetic
71 correlations between CM in the first 3 parities, estimated using linear sire models,
72 vary from 0.67 to 0.92 (Pösö and Mäntysaari, 1996; Carlén et al., 2004). When using
73 multivariate threshold models, the range of estimates of genetic correlations between
74 parities is even broader, ranging from 0.42 to 0.91 (Heringstad et al., 2004; Zwald et
75 al., 2006). These studies have been performed in Scandinavia and the United States.

76 In the Netherlands, the breeding goal is to improve resistance against CM in
77 parity 1, 2, and 3. However, cases of CM are currently not routinely recorded in
78 management information systems (MIS) in the Netherlands. Therefore, selection is
79 based on an udder health index, which includes the following indicator traits: SCC,
80 udder depth, fore udder attachment, teat length, and milking speed (NRS, 2005). In
81 the current Dutch udder health index CM has been treated as the same trait in parities
82 1, 2 and 3. However, several authors suggest that CM in different parities is a
83 different trait (Pösö and Mäntysaari, 1996; Carlén et al., 2004; Heringstad et al.,
84 2004). So far, SCC averaged over 3 lactations has been used in the Dutch udder health
85 index, but several studies have shown that the incidence of CM is much higher in the
86 first half of lactation than in the second half (Emanuelson et al., 1988; Barkema et al.,
87 1998; De Haas et al., 2002). Therefore, the hypothesis is that SCC in the first half of
88 lactation is more informative as a mastitis-indicator than SCC in the second half of
89 lactation or SCC throughout the whole lactation.

90 The aims of this study were: (1) to estimate genetic parameters for CM for the
91 first three lactations of Dutch Holstein cattle; (2) to correlate CM with SCC, where
92 SCC is averaged over the first half, the second half, and the whole lactation; and (3) to
93 estimate accuracy of selection for different indices to improve resistance against CM
94 in parity 1, 2, and 3.

95

96

97 **2. Materials and Methods**

98

99 *2.1. Available data*

100

101 Records on CM treatments were available from MIS on 307 Dutch dairy
102 farms. Data recording was done on a voluntary basis and was performed by the
103 farmers. Data on CM were recorded from June 1998 until May 2006. All herds
104 participated in the national milk recording system. The NRS (Arnhem, The
105 Netherlands) provided the complete history of the milk production recordings (MPR)
106 from all animals that participated in the study. A record included an animal
107 identification number, herd number, date of calving, parity, date of MPR, MPR yields
108 (kilogram milk, fat percentage, and protein percentage) and SCC (in 1,000 cells per
109 ml). A pedigree file of all participating animals was available and contained their
110 ancestry of approximately 110,000 animals (about 88,400 animals and 12,600 bulls)
111 back to 1919. Record also included date of birth and breed of the animal. Breed was
112 subdivided into three main contributing breeds, with each having up to nine classes (0,
113 1/8, ..., 8/8) depending on the degree of contribution. The main breeds were Holstein-
114 Friesian, Dutch-Friesian, and Meuse-Rhine-Yssel.

115

116 *2.2. Data editing at farm level*

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

126

127 *2.3. Data editing at lactation level*

128

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

133 The data were edited using the following inclusion criteria: (1) age at calving
134 ≥ 640 days; (2) parity ≤ 3 ; and (3) observations on cell count available between 5 and
135 335 days in milk (DIM). All animals with less than 75% Holstein-Friesian were
136 excluded. Animals with unknown parents and daughters from sires with less than
137 three daughters in the dataset were also removed.

138 Somatic cell count was transformed to somatic cell score (SCS):

139 $1000+100*(^2\log(\text{SCC}/1000))$. Test-day SCS were averaged per lactation from 5 up to

140 335 DIM, from 5 up to 150 DIM, and from 151 up to 335 DIM (SCS5-335, SCS5-150
141 and SCS151-335, respectively). An average was calculated only if the animal had 2 or
142 more test-day records in the first or second half of the lactation, and 4 or more test-
143 day records for 335 DIM, otherwise a missing value was assigned.

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

149

150 2.4. Statistical analyses

151

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

156

$$157 Y = \mu + \text{fixed effects} + S_{\text{sire}} + \frac{1}{2} S_{\text{mgs}} + e$$

158

159 The random sire effect was identified by the subscripts for sire and MGS; S_{sire} and
160 S_{mgs} , respectively. The sire effects were linked using the relationship matrix, and were
161 assumed to be normally distributed with $\text{var}(S_{\text{sire or mgs}}) = \sigma_s^2$. For the CM-traits, fixed
162 effects included were an interaction between herd and year of calving (948 classes)
163 and month of calving (12 classes). Age at calving (in days) was included in the model

164 as a fixed continuous effect. For the SCS-traits, a repeatability model was used and
165 therefore parity (3 classes) was included in the fixed effects.

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

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

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

189 study. The genetic correlations with the other traits in the index were assumed to stay
190 the same as used in the current Dutch udder health index (Table 1).

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

199

200

201 **3. Results**

202

203 *3.1. Descriptive analyses*

204

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

211 The mean proportion of animals that had CM at least once during lactation
212 was 15.8%, and was lowest for heifers (13.4%) and highest for third parity cows
213 (19.6%). Of all second parity cows 16.1% experienced CM at least once during

214 lactation. The proportion of heifers with CM increased rapidly up to 50 DIM (Figure
215 1). Half of all first cases of CM in heifers occurred before 10 DIM, whereas half of
216 the total proportion of 2nd and 3rd parity cows with CM was approached around 70
217 DIM. Before 150 DIM, 75% of all first cases of CM had occurred.

218

219 *3.2. Genetic parameters for clinical mastitis*

220

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

224

225 *3.3. Genetic correlations between clinical mastitis and somatic cell count*

226

227 The genetic correlations between CM and lactation-average SCS were 0.8 (\pm
228 0.1) in parity 2 and 3 (Table 4), but somewhat lower in first parity (0.6 ± 0.1). Even
229 stronger genetic correlations were estimated for SCS in the first half of lactation,
230 whereas SCS in the second half of lactation showed weak correlations with CM
231 (Table 4).

232

233 *3.4. Selection index calculations*

234

235 Accuracy was 72% for young sires with 100 daughters using the current Dutch
236 udder health index (Table 5). However, when parity-specific CM was included in the
237 breeding goal, accuracy of selection increased to 75%. Even higher accuracy could be

238 achieved when selecting on SCC in first half of lactation (SCS5-150) (83%). For well
239 proven sires with 10,000 daughters accuracy increased to 90% (Table 5).

240

241

242 **4. Discussion**

243

244 *4.1. Representativeness of data*

245

246 The descriptive analyses showed that the data used to estimate genetic
247 parameters was representative of the Dutch dairy cattle population. The average milk
248 production of the animals in this study (26.8 kg) was equal to the average daily milk
249 production in the Netherlands (26.8 kg) (NRS, 2006). The average fat percentage
250 (4.51%) and protein percentage (3.55%) in this study (data not shown) were only
251 slightly higher than the national average fat percentage (4.36%) and the average
252 protein percentage (3.49%) (NRS, 2006).

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

260

261 *4.2. Genetic parameters of clinical mastitis*

262

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

269 The genetic correlations between CM in consecutive lactations (i.e. 1 and 2,
270 and 2 and 3) were higher than the genetic correlation between CM in non-adjacent
271 lactations (i.e. 1 and 3). This is in agreement with the results from previous studies
272 where also the highest genetic correlations were estimated between CM in second and
273 third parity and lowest between CM in first and third parity (Pösö and Mäntysaari,
274 1996; Carlén et al., 2004). This implies that CM in consecutive lactations is,
275 genetically, more the same trait, than CM in non-adjacent lactations. Rupp et al.
276 (2000) also showed that udder health in first and second parity was genetically
277 correlated. They concluded that animals with the lowest mean SCC in the first
278 lactation had the lowest risk for CM in the second lactation. The lower genetic
279 correlation estimated between first and third lactation (0.63 ± 0.22) could be due to
280 selection. Animals that suffered from mastitis might be removed from the herd and
281 were not fulfilling three lactations.

282 Parity-specific CM and lactation-average SCS over 3 parities showed a
283 moderate favorable genetic correlation in first parity (0.64), and strong favorable
284 genetic correlations in later parities (0.79). The favorable genetic correlations imply
285 that selection for lower lactation-average SCS will decrease the incidence of CM.
286 Similar parity-specific genetic correlations were estimated by Carlén et al. (2004),
287 who also observed the highest genetic correlation between CM in parity 3 and

288 lactation-average SCS. In general, the estimated genetic correlations are in line with
289 those reported in literature (see review Mrode and Swanson, 1996; Heringstad et al.,
290 2000).

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

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

309

310 *4.3. Selection against mastitis*

311

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

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

333 Average SCC values do not reflect dynamic variation in SCC. The most
334 desirable animal would respond very quickly to an infection, resulting in increased
335 SCC, followed by return to normal SCC levels. De Haas et al. (2003) showed that
336 patterns of peaks in SCC, which are based on deviations from the normal lactation

337 curve, are more effective in the selection against CM than lactation average SCC.
338 Further optimization of indices might therefore possibly be achieved by including
339 patterns of peaks in SCC in addition to lactation average SCC. This will be
340 investigated in future studies.

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

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

362 should focus on defining alternative SCC traits that provide additional information for
363 selection that aims to decrease genetic susceptibility to CM.

364

365

366 **5. Conclusions**

367

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

387

388

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390

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395 comments on the manuscript.

396

397

398 **Conflict of interest**

399

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

404

405

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407

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- 484

484 Table 1

485 Genetic parameters of clinical mastitis (CM) and the traits¹ in the current Dutch udder

486 health index, heritabilities on diagonal and genetic correlations below diagonal.

	CM	UD	FUA	TL	MS	SCSLAC1-3	b-values
CM	0.04						
UD	0.40	0.40					0.07
FUA	0.35	0.72	0.30				0.04
TL	-0.15	-0.26	-0.26	0.43			-0.09
MS	-0.30	0.00	0.00	-0.30	0.21		-0.12
SCSLAC1-3	0.70	0.35	0.30	0.05	-0.30	0.35	0.38

487 ¹ CM= Clinical mastitis, UD= Udder depth, FUA= Fore udder attachment, TL= Teat

488 length, MS= Milking speed, SCSLAC1-3= Somatic cell score averaged over 3

489 lactations

490 Table 2

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

	CM	Milk	Fat	Protein	SCC
Overall	0	26.7	4.52	3.55	132.7
	1	27.3	4.47	3.54	318.3
1 st parity	0	23.9	4.50	3.51	114.6
	1	23.9	4.47	3.52	263.8
2 nd parity	0	28.3	4.53	3.58	133.1
	1	28.5	4.46	3.57	314.6
3 rd parity	0	29.8	4.53	3.55	165.9
	1	29.8	4.47	3.54	383.5

494

495

495 Table 3

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

	CM1	CM2	CM3
CM1	0.03 _{0.01}	0.88 _{0.13}	0.63 _{0.22}
CM2	0.06 _{0.01}	0.03 _{0.01}	0.91 _{0.12}
CM3	0.05 _{0.02}	0.12 _{0.01}	0.04 _{0.01}

499

500

500 Table 4

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

	CM1	CM2	CM3
SCS5-335	0.64 _{0.12}	0.79 _{0.10}	0.79 _{0.10}
SCS5-150	0.65 _{0.11}	0.86 _{0.08}	0.88 _{0.09}
SCS151-335	0.50 _{0.15}	0.59 _{0.14}	0.61 _{0.14}

505

506

506 Table 5

507 Accuracies of udder health indices with regard to increased resistance to clinical
 508 mastitis (CM) in parity 1, 2, and 3 treated as the same trait (Current Dutch udder
 509 health index) and treated as different traits (Parity-specific CM). Including different
 510 somatic cell count traits¹ in addition to the indicator-traits udder depth, fore udder
 511 attachment, teat length, and milking speed, based on sires with 100 or 10,000
 512 daughters.

	100 daughters	10,000 daughters
Current Dutch udder health index	0.72	0.75
Parity-specific CM	0.77	0.81
Parity-specific CM and SCS5-335	0.80	0.84
Parity-specific CM and SCS5-150	0.83	0.90
Parity-specific CM and SCS151-335	0.67	0.70

513 ¹ somatic cell scores averaged from 5 to 335 days (SCS5-335), from 5 to 150 days
 514 (SCS5-150) and from 151 to 335 days (SCS151-335).

515

515 Table 6

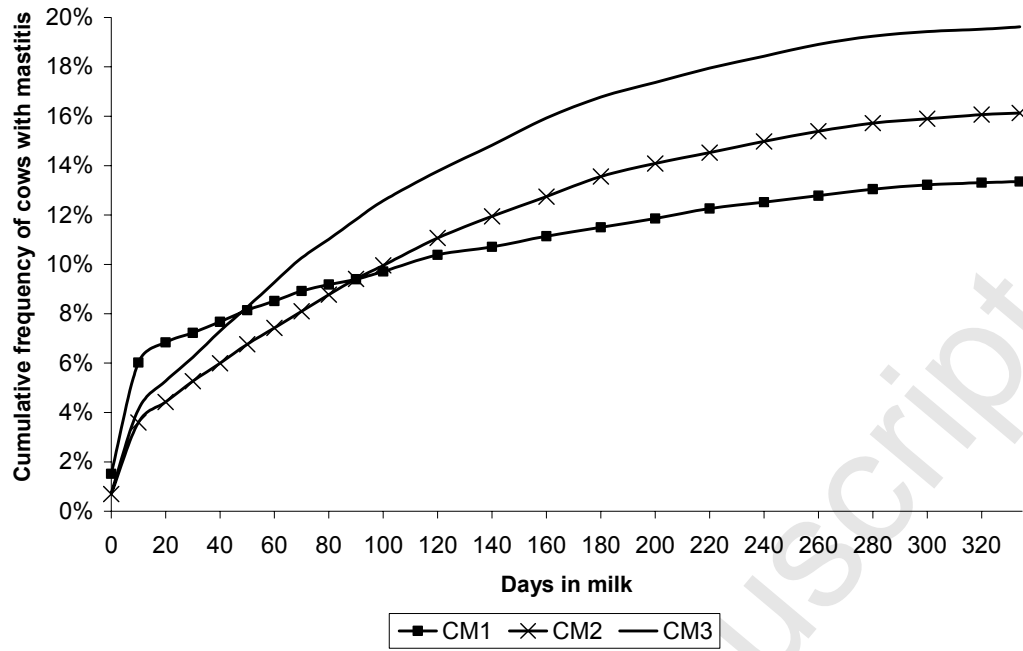
516 Accuracies of udder health indices with regard to increased resistance to clinical
 517 mastitis (CM) in parity 1, 2, and 3 treated as the same trait (Current Dutch udder
 518 health index) and treated as different traits (Parity-specific CM). Including direct
 519 clinical mastitis (CM) information in first lactation of 10 daughters, 25 daughters, 50
 520 daughters and 100 daughters, in addition to different somatic cell count traits¹ and the
 521 indicator-traits udder depth, fore udder attachment, teat length, and milking speed,
 522 based on young sires with 100 daughters.

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

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

525

526



526

527 Figure 1

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