Selection Responses for Disease Resistance in Two Selection Experiments with Norwegian Red Cows

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ABSTRACT

Genetic trends for clinical mastitis (CM), ketosis (KET), retained placenta (RP), and 305-d protein yield (PY305) were calculated for 2 Norwegian dairy cattle selection experiments. The first experiment, accomplished from 1978 to 1989, included groups selected for high (HMP) and low milk production (LMP). The second experiment started in 1989 and included selection for high protein yield (HPY) and low mastitis frequency (LCM). In both experiments proven sires from the active breeding program of Norwegian Red were used as sires. To take into account that selection of sires was external to the experiment, all available data from the Norwegian Red population, including disease records for 2.7 million first-lactation cows, were analyzed with a multivariate animal model. Estimated breeding values for cows in the experiments were extracted from this analysis to calculate genetic trends in the selection groups. Genetic trends for PY305 were, as expected, positive for the HMP and HPY groups, and negative for LMP and LCM. The HMP group showed increasing genetic trends for all 3 diseases, arguably a correlated response after selection for increased milk production, whereas the LCM group showed decreasing genetic trends for CM, KET, and RP. The genetic trends for KET and RP in the LCM group are most likely correlated responses after selection against CM. After 5 cow-generations the genetic difference between HPY and LCM was 10 percentage units CM, 1.5 percentage units KET, and 0.5 percentage units RP.

Key words: clinical mastitis, correlated selection response, dairy cattle, disease resistance

INTRODUCTION

It is well known that the genetic relationship between milk yield and many health traits in dairy cows is antagonistic. Selection only or mainly for increased milk production is therefore expected to result in a genetic

deterioration of disease resistance. Clinical mastitis (CM), milk fever (MF), ketosis (KET), and retained placenta (RP) are among the most frequent diseases affecting dairy cows. In Norway, 67% of all veterinary treatments in dairy cows in 2005 were due to these 4 diseases (TINE, 2006). Unfavorable genetic correlations have been estimated between milk production and CM (Heringstad et al., 2005a), KET (Mäntysaari et al., 1991), MF (Pryce et al., 1997), and RP (Schnitzenlehner et al., 1998).

Heringstad et al. (2005b) obtained low or moderate positive genetic correlations between CM, MF, KET, and RP within and between lactations. For first lactation, the genetic correlations between CM and MF, KET, and RP, respectively, were 0.12, 0.26, and 0.16. Positive genetic correlations between CM and other diseases imply that selection against CM is expected to result in some genetic improvement of resistance to the other diseases as a correlated response.

Data from 2 selection experiments with Norwegian Red have enabled us to estimate correlated responses in disease resistance resulting both from selection for increased milk yield and from selection against mastitis. In the first experiment, between 1978 and 1989, selection was for high and low milk production. The second experiment, which started in 1989 and continues today, includes 2 groups selected for high protein yield and low CM frequency, respectively. In the second experiment Heringstad et al. (2003) reported a genetic difference of 8.6 percentage units of CM and 15 kg of 305-d protein yield (PY305) between the 2 groups, using data from 1978 to 1998 including 3 cow generations. Additional data are now available, and our objectives were to estimate updated genetic trends for CM in the second experiment, and to estimate selection responses for KET and RP in both selection experiments. Ketosis and RP were chosen because after CM they are the most frequent diseases in first-lactation cows.

MATERIALS AND METHODS

Selection Experiments

The same design was used in the 2 selection experiments (Heringstad et al., 2003). Eight herds were in-

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cluded, each with 2 selection groups, and approximately the same number of cows in each group. Experiment 1 was accomplished from 1978 to 1989. Two selection groups were established based on the Norwegian Red cows present in the herds when the experiment started: one group selected for high milk production (**HMP**) and one control group with low milk production (**LMP**). Proven Norwegian Red sires from the active breeding program were used as sires. Cows in the HMP group were mated to the highest ranking proven sires for milk production each year. For the LMP group, 21 sires with milk production index ≈ 90 (relative index with mean = 100, SD = 6) in the 1978–1979 progeny testing were selected and used as sires during the entire experiment. Experiment 2 began in 1989. Based mainly on HMP cows from the first experiment, 2 selection groups were created: one high protein yield (**HPY**) group and one low frequency of clinical mastitis (**LCM**) group. The best proven sires for the given trait (protein yield or mastitis) were selected each year from among the 120 to 130 progeny-tested Norwegian Red bulls and used as sires in the experiment. Cows in HPY were mated to the 3 to 4 highest-ranking proven sires for protein yield; cows in LCM were mated to the 2 to 4 best proven sires for mastitis resistance. This implies that the experiments were based on single-trait selection of sires preselected for Norwegian Red breeding objective. To take into account that sires were selected externally to the experiments, all available data from the total Norwegian Red population, from 1978 onwards, were analyzed with an animal model. Estimated breeding values for cows in the experiments were extracted from this analysis and used for calculation of genetic trends in the selection groups.

Cow-Generation. For these analyses, all cows present in the herds when the selection experiment started, that were mated with one of the defined sires, and produced a daughter that made a first-lactation record were defined as belonging to cow-generation 0. Their daughters were defined as cow-generation 1, and so on. Cows born in the same year could, therefore, belong to different cow-generations.

Data

Norwegian Red Population. Data were from the Norwegian Dairy Herd Recording System and included 2,719,353 first-lactation Norwegian Red cows. The data file had information on CM, KET, and RP for cows with first calving between September 1, 1978, and January 1, 2005, that had age at first calving between 20 and 40 mo. For each cow, absence or presence of each of the 3 diseases was scored as “0” or “1”, respectively, based on whether the cow had at least one veterinary treat-

Table 1. Summary statistics of the data sets used for prediction of breeding values for clinical mastitis (CM), ketosis (KET), retained placenta (RP), and 305-d protein yield (PY305)

Item	
Cows with health data, no.	2,719,353
Mean CM frequency, ¹ %	13.5
Mean KET frequency, ¹ %	7.0
Mean RP frequency, ¹ %	2.2
Cows with PY305 records, no.	2,283,059
Mean PY305, kg	202
Animals in the pedigree file, no.	3,264,928

¹Absence or presence of each of the 3 diseases was scored as 0 or 1, respectively, based on veterinary treatments within the interval from 15 d before to 120 d after first calving for CM and KET, and within the first 5 d for RP.

ment recorded within the interval from 15 d before to 120 d after calving for CM and KET, and within the first 5 d after calving for RP. For CM, the interval from 15 d before to 120 d after calving was chosen because this was the interval used in the national genetic evaluation of CM in Norway. The overall frequency was 13.5% CM, 7.0% KET, and 2.2% RP (Table 1).

A total of 2,283,059 (84%) of the cows had PY305 data (Table 1). Only cows that had at least 2 test-day records of protein yield had PY305. For cows with incomplete lactations, PY305 was predicted using the same procedure as used in genetic evaluation for Norwegian Red.

The pedigree file was built by tracing the pedigree of cows with data back as far as possible and included a total of 3.3 million animals (Table 1).

Selection Experiment 1. A total of 2,385 cows from experiment 1 had first-lactation health records, of which 1,369 were HMP cows and 1,016 were LMP cows (Table 2). The mean frequency of CM, KET, and RP in first lactation was 17.5, 11.5, and 2.6% for HMP cows

Table 2. Summary statistics for groups of cows selected for high milk production (HMP) and low milk production (LMP) in selection experiment 1, and high protein yield (HPY) and low clinical mastitis (LCM) in selection experiment 2

Item	Selection group			
	HMP	LMP	HPY	LCM
Cows with health data, no.	1,369	1,016	1,429	1,302
Mean CM frequency, ¹ %	17.5	12.8	14.1	10.6
Mean KET frequency, ¹ %	11.5	9.2	2.4	2.5
Mean RP frequency, ¹ %	2.6	2.4	2.6	2.2
Cows with PY305 records, no.	1,209	854	1,249	1,130
Mean PY305, kg	214	188	222	206
Cows with EBV for diseases, no.	1,559	1,137	1,475	1,351
Sires, no.	49	21	45	38

¹Absence or presence of each of the 3 diseases was scored as 0 or 1, respectively, based on veterinary treatments within the interval from 15 d before to 120 d after first calving for clinical mastitis (CM) and ketosis (KET), and within the first 5 d for retained placenta (RP). PY305 = 305-d protein yield.

Table 3. Number of cows with EBV for disease traits per selection group (HMP = high milk production, LMP = low milk production), cow-generation,¹ and herd in selection experiment 1

Herd	Selection group		Cow-generation ¹							Total no. of cows
	HMP	LMP	0	1	2	3	4	5	6	
Kalnes	331	297	132	228	149	91	26	2	0	628
Hvam	113	95	43	71	48	29	14	3	0	208
Jønsberg	123	132	46	87	62	44	13	3	0	255
Melsom	163	115	61	97	68	39	12	1	0	278
Åna	385	240	119	195	144	110	47	10	0	625
Øksnevad	129	84	51	86	49	21	4	1	1	213
Gjermundnes	89	46	25	39	34	27	9	1	0	135
Mære	226	128	68	100	86	66	29	5	0	354
Total no. of cows	1,559	1,137	545	903	640	427	154	26	1	2,696
Selection group	HMP		287	466	366	296	122	21	1	
	LMP		258	437	274	131	32	5	0	

¹Cow-generation = all cows present in the herd when the selection experiment started, that were mated with one of the defined sires and produced a daughter that made a first-lactation mastitis record were defined as belonging to cow-generation 0; all their daughters were defined as cow-generation 1; and so on.

and 12.8, 9.2, and 2.4% for LMP cows, respectively (Table 2). The distribution of the 2,696 cows from experiment 1 with EBV for disease traits over selection groups, herds, and cow-generations are given in Table 3.

Selection Experiment 2. A total of 2,731 cows from experiment 2, of which 1,429 were HPY and 1,302 were LCM cows, had first-lactation health data (Table 2). The mean frequency of CM, KET, and RP was 14.1, 2.4, and 2.6% for HPY cows and 10.6, 2.5, and 2.2% for LCM cows (Table 2). The number of cows with EBV for disease traits per selection group, herd, and cow-generation are given in Table 4.

Model

Breeding values for CM, KET, RP, and PY305 were calculated in a multivariate animal model using DMU

(Madsen and Jensen, 2005). The model for each trait had fixed effects of age at first calving (21 classes), month-year of first calving (316 classes), and herd-year of first calving (507,379 classes), and random effects of cow and residual.

Variance Components. Variance components were estimated in DMU (Madsen and Jensen, 2005) using a multivariate linear sire model with the same fixed effects as described above and data restricted to 873,318 first-batch daughters of the 3,150 Norwegian Red sires progeny-tested in the years from 1980 to 2004. The resulting (co)variances were used to calculate variance components relevant for the animal model.

Genetic Differences

Genetic Trends. Estimated breeding values for cows in the selection experiments were extracted from the

Table 4. Number of cows with EBV for disease traits per selection group (HPY = high protein yield, LCM = low clinical mastitis), cow-generation,¹ and herd in selection experiment 2

Herd	Selection group		Cow-generation ¹							Total no. of cows
	HPY	LCM	0	1	2	3	4	5	6	
Kalnes	377	321	121	228	138	133	65	13	0	698
Jønsberg	78	103	40	78	23	22	15	2	1	181
Valle	190	134	66	115	63	54	23	3	0	324
Buskerud	108	95	38	70	42	34	16	3	0	203
Åna	321	372	100	166	176	149	86	15	1	693
Øksnevad	150	93	58	93	40	32	17	3	0	243
Gjermundnes	67	88	20	38	33	34	22	8	0	155
Mære	184	145	57	105	70	56	31	9	1	329
Total no. of cows	1,475	1,351	500	893	585	514	275	56	3	2,826
Selection group	HPY		265	483	310	260	127	29	1	
	LCM		235	410	275	254	148	27	2	

¹Cow-generation = all cows present in the herd when the selection experiment started, that were mated with one of the defined sires and produced a daughter that made a first-lactation mastitis record were defined as belonging to cow-generation 0; all their daughters were defined as cow-generation 1; and so on.

Figure 1. Mean frequency of clinical mastitis (CM) and ketosis (KET) per cow-generation for groups of cows selected for high milk production (HMP) and low milk production (LMP) in experiment 1, and groups selected for high protein yield (HPY) and low clinical mastitis (LCM) in experiment 2.

multivariate animal model analysis and used to calculate genetic trends in the selection groups. The EBV for CM, KET, and RP were multiplied by 100 (equivalent to % disease). Genetic trends were estimated by linear regression of EBV on cow generation and on cows' birth-year.

Phenotypic Differences Between Selection Groups. The difference between phenotypic values corrected for fixed effects provide a measure of genetic change, independent of information on relatives as well as of prior values for genetic parameters. For each cow and trait, corrected phenotypic values ($y - \hat{y}$) were calculated based on solution for fixed effects of age, month-year, and herd-year of calving from a fixed effect model, similar to the model used for prediction of EBV but without the random effect of animal in the model. Mean difference between selection groups was calculated per cow-generation, in phenotypic standard deviation (SD) units. The phenotypic SD were 82.61 kg for PY305, 34% for CM, 15% for RP, and 26% for KET. After correcting for fixed effects, the difference in mean phenotypic values between selection groups is due to genetic differences and noise.

RESULTS AND DISCUSSION

Figure 1 shows mean frequency of first-lactation CM and KET per cow-generation in the 2 selection experiments. Cows in the LMP group showed a relatively flat

phenotypic trend for CM, whereas CM frequency in the HMP group increased with cow-generation. In selection experiment 2, CM frequency decreased in the LCM group and increased for the HPY group. Mean frequency of KET decreased with cow-generation in all 4 selection groups. Mean frequency of RP per cow generation varied between 0 and 6.3%, with no obvious trend or differences between selection groups.

The estimated heritabilities and genetic correlations are given in Table 5. The heritability of PY305 was in agreement with previous Norwegian studies (e.g., Heringstad et al., 2005a). The heritability estimates for diseases were, as expected, low: 0.03 for CM and KET and 0.01 for RP, and in agreement with previous estimates from linear models (Mäntysaari et al., 1991; van Dorp et al., 1998; Heringstad et al., 2003). Antagonistic genetic correlations of 0.31, 0.31, and 0.05 were found between PY305 and CM, KET, and RP, respectively.

Table 5. Estimates¹ of heritability (on the diagonal) and genetic correlations (above the diagonal) for clinical mastitis (CM), ketosis (KET), retained placenta (RP), and 305-d protein yield (PY305)

	CM	KET	RP	PY305
CM	0.03	0.26	0.23	0.31
KET		0.03	0.07	0.31
RP			0.01	0.05
PY305				0.22

¹Standard errors of genetic correlations varied between 0.02 (PY305 – KET) and 0.04 (KET – RP).

Figure 2. Mean EBV for clinical mastitis (CM) per cow-generation for groups of cows selected for high protein yield (HPY) and low clinical mastitis (LCM) in experiment 2.

The genetic correlations between diseases were all positive, which is in agreement with Heringstad et al. (2005b).

Figure 2 shows that the genetic trends for CM in experiment 2 reported by Heringstad et al. (2003) continue, with a strongly decreasing trend for LCM and a slowly increasing trend for HPY cows. The genetic difference of 10 percentage units CM after 5 cow-generations is mainly a result of direct selection against CM in the LCM group. The genetic improvement for LCM was equivalent to a reduction of 1.9 percentage units CM per cow-generation (Table 6).

The genetic trend for CM in the HPY group is, as discussed by Heringstad et al. (2003), not representative for what would happen in a population selecting for increased milk yield only. Here, sires were selected from the Norwegian Red population and were thus pre-selected for the breeding objective of Norwegian Red, which has a strong emphasis on mastitis. The HPY group shows a slowly increasing genetic trend for CM over the same time period (after 1990) where there has been a decreasing genetic trend for the Norwegian Red population (Figure 3). The trajectory of genetic trends for CM, KET, and RP in Norwegian Red in Figure 3 were in agreement with Heringstad et al. (2005b) who

Table 6. Regression coefficients from linear regression analysis of EBV for clinical mastitis (CM), ketosis (KET), retained placenta (RP), and 305-d protein yield (PY305), respectively, on cow-generation¹ for groups of cows selected for high milk production (HMP) and low milk production (LMP) in experiment 1, and for high protein yield (HPY) and low clinical mastitis (LCM) in experiment 2

Trait	Group	Regression coefficient	P-value ²
CM, %	HMP	0.91	<0.0001
	LMP	-0.04	0.52
	HPY	0.42	<0.0001
	LCM	-1.91	<0.0001
KET, %	HMP	0.41	<0.0001
	LMP	-0.42	<0.0001
	HPY	-0.04	0.18
	LCM	-0.44	<0.0001
RP, %	HMP	0.14	<0.0001
	LMP	0.09	<0.0001
	HPY	0.03	0.04
	LCM	-0.08	<0.0001
PY305, kg	HMP	4.46	<0.0001
	LMP	-3.00	<0.0001
	HPY	4.89	<0.0001
	LCM	-1.22	<0.0001

¹Cow-generation = all cows present in the herd when the selection experiment started, that were mated with one of the defined sires and produced a daughter that made a first lactation mastitis record were defined as belonging to cow-generation 0; all their daughters were defined as cow-generation 1; and so on. Regressions were based on cow-generations 0 to 4 for LMP and 0 to 5 for HMP, HPY, and LCM groups.

²For hypothesis, regression coefficient = 0.

estimated genetic change for disease resistance using a multivariate threshold model.

Mean EBV for KET in the 2 selection experiments are given in Figure 4. Figure 4 indicates genetic differences,

Figure 3. Mean EBV for clinical mastitis (CM), ketosis (KET), and retained placenta (RP) by birth-year for the Norwegian Red population.

Figure 4. Mean EBV for ketosis (KET) per cow-generation for groups of cows selected for high milk production (HMP) and low milk production (LMP) in experiment 1, and for high protein yield (HPY) and low clinical mastitis (LCM) groups in experiment 2.

after 4 and 5 cow-generations, respectively, of more than 3 percentage units KET between HMP and LMP cows in experiment 1, and 1.5 percentage units KET between HPY and LCM cows in experiment 2. The genetic correlation was unfavorable between PY305 and KET (0.31) and positive between KET and CM (0.26; Table 2). The increasing trend for KET in HMP is probably a correlated response to selection for increased milk production, whereas the decreasing trend for LCM is most likely a correlated response to selection against CM. In the Norwegian Red population there has been a decreasing genetic trend for KET over time (Figure 3), which may explain the flat trend for HPY from experiment 2.

Mean EBV for RP in the 2 selection experiments are given in Figure 5. After 4 cow-generations, the genetic difference between HMP and LMP cows was equivalent to 0.2 percentage units RP. Further, Figure 5 indicates a genetic difference of 0.5 percentage units RP between HPY and LCM after 5 cow-generations. These small genetic differences between selection groups were probably mainly due to low RP frequency. However, the difference between HMP and LMP cows harmonizes with the genetic correlation between RP and PY305 being close to zero (0.05; Table 5), whereas the positive genetic correlation (0.23) found between RP and CM (Table 5) indicates that the observed genetic improvement in LCM may be a correlated response after selection against CM. There has been little or no genetic

change for RP in the Norwegian Red population (Figure 3).

The HMP and HPY groups showed, as expected, positive genetic trends for PY305 (Figure 6), whereas a negative genetic trend was found for LMP cows. For the LCM group, mean EBV for PY305 decreased from cow-generation 0 to 2, but after cow-generation 2 there has been little or no genetic change for PY305 in this group. Again, this can probably be explained by sires coming from the Norwegian Red population. If there were a true single-trait selection against CM, a steadily decreasing trend for PY305 would be expected as a result of the antagonistic genetic correlation between PY305 and CM.

Regression coefficients from the linear regression analyses of EBV on cow-generation were significantly different from zero ($P < 0.0001$) for all 4 traits in all selection groups, except CM in the LMP group and KET and RP in HPY (Table 6).

Genetic differences between selection groups given as mean differences between corrected phenotypic values per cow-generation are given in Figures 7 and 8. Figure 7 indicates increasing differences between HMP and LMP for PY305 and CM, and only small differences for RP and KET. In cow-generation 0, the difference between the 2 groups was 0.056 kg of PY; in cow-generation 4 the difference between groups had increased to 0.963 kg of PY. Over 4 cow-generations, the mean difference between the selection groups increased with 0.91,

Figure 5. Mean EBV for retained placenta (RP) per cow-generation for groups selected for high milk production (HMP) and low milk production (LMP) in experiment 1, and for high protein yield (HPY) and low clinical mastitis (LCM) groups in experiment 2.

0.25, 0.07, and 0.06 phenotypic SD for PY305, CM, RP, and KET, respectively. Figure 8 shows that the mean difference between HPY and LCM increases with cow-generation for all 4 traits. Over 5 cow-generations, the mean difference between selection groups increased by 0.52, 0.64, 0.42, and 0.06 phenotypic SD for PY305, CM, RP and KET, respectively. The difference between selection groups in mean phenotypic values corrected

for fixed effects provides a measure of genetic change that affirm the genetic trends presented in Figures 2, 4, 5, and 6.

Figure 6. Genetic trends for 305-d protein yield (PY305) per cow-generation for groups of cows selected for high milk production (HMP) and low milk production (LMP) in experiment 1, and for high protein yield (HPY) and low clinical mastitis (LCM) in experiment 2.

Figure 7. Mean difference in corrected phenotypic values per cow-generation for clinical mastitis (CM), ketosis (KET), retained placenta (RP), and 305-d protein yield (PY305) between groups selected for high milk production and low milk production in experiment 1, given in phenotypic standard deviation (SD) units.

Figure 8. Mean difference in corrected phenotypic values per cow-generation for clinical mastitis (CM), ketosis (KET), retained placenta (RP), and 305-d protein yield (PY305) between the groups selected for high protein yield and low clinical mastitis in experiment 2, given in phenotypic standard deviation (SD) units.

Increased health costs as a correlated response to selection for increased milk production has been found in several studies (e.g., Short et al., 1990; Dunklee et al., 1994; Jones et al., 1994). Our study is, however, the first to report genetic improvement in resistance to other diseases (KET and RP) as a correlated response to selection against CM.

CONCLUSIONS

The results from the 2 selection experiments clearly demonstrate the effect of direct and indirect selection on CM, as well as correlated selection responses in KET and RP. Selection for increased milk production will result in unfavorable correlated selection responses for disease resistance. Considerable genetic improvement can be achieved for mastitis resistance, and selection against CM results in favorable correlated selection responses for KET and RP.

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