SELECTION FOR DISEASE RESISTANCE IN DAIRY CATTLE

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SUMMARY

The Norwegian commercial breeding programme where resistance for mastitis and ketosis are included, has been described. Important genetic gain for resistance to these diseases has been obtained in this programme. The described genetic gain has been obtained in a conventional breeding programme with large emphasis on progeny testing. The genetic gain for milk yield has, because of this, been reduced by some 15%.

No efficient marker traits for disease resistance in dairy cattle has so far been established. It is further concluded that such markers, also in the future, more will be a supplement to conventional breeding programmes than the sole means of selecting for disease resistance.

INTRODUCTION

In his review, Emanuelson (1988) showed that the most important disease traits have low heritabilities and that they in general have small but antagonistic genetic correlations to milk yield. Østergaard et al. (1989) showed the economic importance of including these traits in breeding programmes. Shook (1989) reviewed different possibilities of selection for disease resistance. The present presentation will demonstrate how diseases are included in the commercial Norwegian dairy cattle breeding programme and the genetic gain obtained for disease resistance in this programme. This is a conventional breeding programme with main emphasis on progeny testing. The search for markers to assist in the selection of disease resistance is also described. The status of some of this research in Norway will be presented.

DISEASE RESISTANCE IN THE NORWEGIAN DAIRY CATTLE BREEDING PROGRAMME

The Norwegian dairy cattle population includes about 340 000 dairy cows of the same breed (Norwegian Cattle). AI is used to about 98% of all inseminations, and 83% of the cows are milk recorded.

Resistance to mastitis and ketosis have been included in the dairy cattle breeding programme since 1978, and reasonable genetic gain has been obtained for these traits. All AI bulls have since then been progeny tested for mastitis and ketosis. To be able to do this, a recording system for diseases is necessary. How this works in Norway is described by Solbu (1983).

Breeding programmes including traits with very low heritabilities (1-5%) need large progeny groups to be efficient. For this reason, all AI bulls in Norway have progeny groups of about 200 daughters. So even with the very low heritabilities, the accuracy of the estimated breeding values for the disease resistance traits for the AI bulls is reasonably high (50-70%), and genetic progress is possible. Disease resistance is only 2 out of 12 traits included in the selection criteria for AI bulls (see Fimland, (1984)). The total genetic gain for these traits is therefore limited. However, if these traits not were included, the genetic level for disease resistance would have decreased because of the antagonistic correlations to milk yield.

SELECTION RESPONCES OBTAINED FOR DISEASE RESISTANCE

Solbu and Steine (1989) described the response obtained for mastitis in the Norwegian situation. Without including mastitis in the selection criteria, a genetic increase in mastitis frequency of 1.3% units in a sire generation of 6 years would have been the result. With the inclusion of mastitis, the mastitis frequency is reduced by 0.9% units. The results for ketosis is similar, but a little less than for mastitis. This response might seem small, but the total genetic progress obtained in a sire generation is 2.2% units for mastitis (1.3% + 0.9%), and that is of great economic importance. (Mean frequency of mastitis is about 20%.)

The inclusion of disease traits (and other traits) in the selection criteria reduces the genetic gain for milk yield by about 15%. Even so, estimations have shown it economically right to include these traits on the cost of milk yield. It is also believed that ethical aspects of animal breeding will be more and more focused in the future. This will make it even more important to include health traits in the selection criteria. In Norway it has presently been decided to reduce the weight on milk yield and increase the weight on disease and health and fertility traits. A present yearly genetic progress for milk yield of about 70 kg per year will then be reduced to 50-55 kg. The selection effect on the health traits will increase by about 100% compared with today.

MARKER TRAITS

To improve the possibilities of genetic gain for disease traits, the use of marker traits has often been discussed. Numerous different markers have been proposed. Solbu and Steine (1990) have reviewed the possibility of using markers in sire selection. With present knowledge this is not too promising, but some effect of such selection might be expected in the future.

Solbu et al. (1987) reported results on some immunological traits studied in Norway that might be of interest as markers for infectious diseases (including mastitis). No strong correlations were found in that study, but complement and immune responses to different antigenes had some connections to mastitis. The same was also found for the bovine MHC-system (BoLA). More data have now been analysed regarding connections between BoLA and mastitis, and a possible connection among BoLA and ketosis has also been studied.

RESULTS OF A STUDY OF BoLA AS A MARKER TRAIT FOR MASTITIS AND KETOSIS

Materials and methods

The animals and the data of the present study have been described by Solbu et al. (1987).

The data presently analysed included altogether 328 AI bulls both BoLA typed and progeny tested for mastitis and ketosis. However, only 193 of these bulls were BoLA typed as heterozygous for the BoLA class I allele. The other 135 bulls might be either homogygous for this allele or they might have an allele not identified by our typing methods. Because of this, they were excluded from the material.

The data were analysed by the following gene substitution model:

$$Y = YEAR + b_1AL_1 + \text{----} + b_{13}AL_{13} + E$$

where

Y	=	The progeny test result for mastitis and ketosis for each bull, respectively (dependent variables)
YEAR	=	Year of progeny test (5 years: 1985-89)
AL	=	The different BoLA alleles (13 included in the analyses, see Table 1)
b	=	The regression coefficient of the different alleles on the dependent variables
E	=	Random error

A model including sire of the bull was initially regarded as a better model than the presented one. However, no solution was obtained by that model because sire and year are confounded. The model including year was regarded as the more appropriate one since progeny tests are done separately for each year. This might, however, overestimate the allele effects somewhat, since "other sire effects" might be mistaken as allele effects.

A separate BoLA allele frequency for the daughter group of each bull was estimated.

RESULTS

Table 1 shows the results of the analyses.

Four BoLA alleles have at least a tendency of a connection with mastitis and ketose, respectively. However, the different alleles' effect on disease frequency is not very high. For W11 for example, the difference in mastitis frequency between animals carrying this allele and animals not carrying it, is 3-5%-units (average mastitis frequency is around 20%). Consequently, even if this holds true in further investigations, this allele is not a very good indication of mastitis susceptibility. Selecting for single alleles might also be doubtful before it has been established that such a selection has no side effects. Another worrying fact is that different analyses of correlations between BoLA alleles and mastitis do not give the same results (see Solbu et al. 1982, also unpublished results).

Table 1 Interactions between different BoLA alleles and progeny test results for mastitis and ketosis

BoLA allele	Mastitis * significant level (p)	Ketosis * significant level (p)
W 2	0.02 (+)	0.20 (-)
W 5	n.s.	n.s.
W 6	n.s.	0.14 (-)
W 6.1	n.s.	n.s.
W 6.2	n.s.	n.s.
W 7	0.18 (-)	n.s.
W 8	n.s.	n.s.
W10	n.s.	n.s.
W11	<0.01 (-)	n.s.
W12	n.s.	n.s.
W13	0.15 (+)	<0.01 (+)
W16	n.s.	0.06 (-)
W20	n.s.	n.s.

* (+) and (-) indicates whether the presence of an allele is positive or negative (i.e. the presence of W2 gives less mastitis).

It is also difficult to explain that as many alleles of the BoLA system has some correlation to ketosis as to mastitis. The obvious conclusion of this must be that with present knowledge the BoLA system can not be used as a marker trait for resistance to mastitis in ketosis.

CONCLUSIONS

Effective selection for disease resistance can be performed by traditional breeding methods as long as proper recordings and large enough daughter groups are used for progeny testing.

Presently no acceptable marker traits for disease resistance in dairy cattle have been established. What might come in the future is difficult to say. But a selection programme for disease resistance solely based on marker-selection seems unlikely.

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