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Conference paper

CLINICAL MASTITIS IN THE POPULATION: EPIDEMIOLOGY AND GENETICS

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Summary

Considerable progress in controlling mastitis has been accomplished by applying standard mastitis control programmes. The progress can easily be seen in the decrease in average bulk milk somatic cell count achieved in many countries during the last decade. Nevertheless, the rate of clinical mastitis continues to pose a problem, especially in some environments, and alternative mastitis control measures may therefore be warranted. Epidemiological techniques offer the possibility to identify herd risk factors and consequently aid in adopting current control programmes to a changing environment. Some of the recent results in this field are briefly reviewed.

Host-related factors, i.e. one of the corners of the epidemiological triangle of mastitis, deserve increased attention. An improved disease resistance of the cow is within reach, especially since an accumulating number of studies have unequivocally shown that there is a genetic component involved. Estimated heritabilities of resistance to clinical mastitis are typically low, but the genetic variation has been shown to be reasonably high. Recent results are reviewed and prospects of including mastitis resistance in dairy cattle breeding are discussed.

Keywords: Dairy cow; Mastitis control programmes; Mastitis resistance; Somatic cell count

Introduction

An effective programme for the control of clinical mastitis must involve all three corners of the epidemiological triangle: the pathogen, the environment, and the cow. Control programmes have also been devised accordingly, eg. the

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Five Point Plan (Natzke, 1981; Dodd, 1983), focusing mainly on milking practices, postmilking teat disinfection, and dry cow therapy. The application of such programmes has been successful, as can be seen in the decrease in average bulk milk somatic cell count (BMSCC) achieved in many countries. Thus, seven out of eight countries, providing data over the years 1985 to 1993, had reduced their mean BMSCC, and the average reduction was approximately 23% (Booth, 1995).

However, there is little evidence that this decrease in average BMSCC has been accompanied by a concurrent decrease in mastitis incidence. On the contrary, available data, although admittedly rather scarce, even suggest a possible deterioration with respect to subclinical and clinical mastitis during the same period (Booth, 1995). There are also results indicating that cows in herds with low BMSCC may be at higher risk for clinical mastitis (Hogan et al., 1989; Schukken et al., 1989; Miltenburg et al., 1996), although other results from Sweden and elsewhere (eg. Barkema et al., 1997) show no differences in average incidence between herds af varying BMSCC. Additionally, there has been a general change in pathogens involved in clinical mastitis (Myllys et al., 1994; Booth, 1995). More specifically, it has been shown that the pattern of pathogens is different in herds with different levels of BMSCC (Erskine et at., 1988; Schukken et al., 1989; Barkema et al., 1997).

These observations do not mean that the actions devised by traditional control programmes have been inadequate or unsuccessful. They do, however, possibly call for new approaches in the fight against mastitis in the future, and the purpose of this presentation is to review some aspects that can contribute in this process.

Epidemiology

A primary component in developing successful control programmes should be to identify pertinent factors that increase the risk for mastitis. Epidemiological methods provide tools that are useful in the process of studying mastitis in the population. Most facets of the epidemiological triangle of mastitis have been throughly investigated, and there is an abundance of published studies. It is obviously impossible to do justice to such numbers and this review does not attempt doing so. However, a few items of particular interest will be highlighted.

Postmilking teat disinfection has been regarded as an important item in current control programmes, but its role has recently been challenged e.g. by

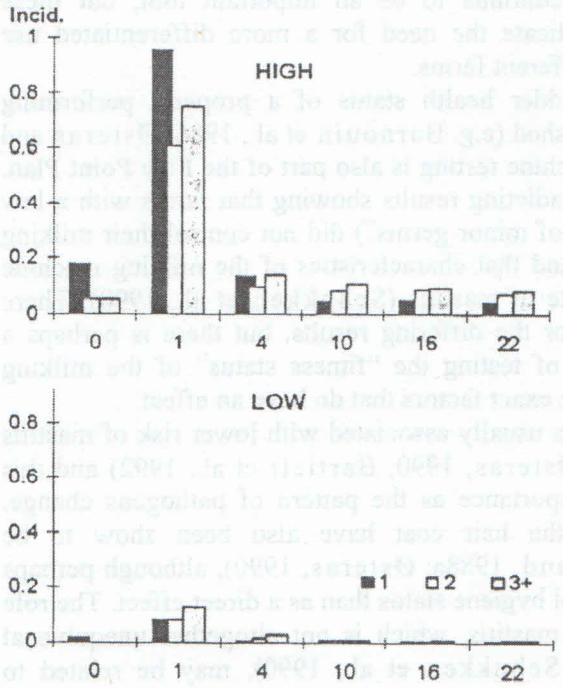
Schukken et al. (1990) and Faye et al. (1994). It can be argued that causal relationships can not be determined from such observational epidemiological studies. However, the change in bacteriological patterns with respect to environmental vs. contagious pathogens that we have seen, may influence the effect of teat disinfection and lead to the observed results. Postmilking teat disinfection will most likely continue to be an important tool, but these observations may possibly indicate the need for a more differentiated use according to the situation on different farms.

The importance for the udder health status of a properly performing milking machine is well established (e.g. Barnouin et al., 1986; Østeras and Lund, 1988b), and regular machine testing is also part of the Five Point Plan. However, there are again contradicting results showing that farms with a low rate of mastitis (but "high rate of minor germs") did not control their milking machine (Faye et al., 1994), and that characteristics of the milking machine had no association with the rate of mastitis (Schukken et al., 1990). There may be several explanations for the differing results, but there is perhaps a need for a more refined way of testing the "fitness status" of the milking machine in order to pinpoint the exact factors that do have an effect.

A clean cow and/or udder is usually associated with lower risk of mastitis (eg. Schukken et al., 1990; Østeras, 1990; Bartlett et al., 1992) and this factor may even expand in importance as the pattern of pathogens change. Hoof trimming and cutting the hair coat have also been show to be advantageous (Østeras and Lund, 1988a; Østeras, 1990), although perhaps more as indicators of the general hygiene status than as a direct effect. The role of rubber mats in the risk of mastitis, which is not altogether unequivocal (Østeras and Lund, 1988a; Schukken et al., 1990), may be related to effects on the hygiene and, most likely, estimated relationships express an interaction with housing and pathogens present in the environment.

In a recent observational study on herds with low and high incidence of clinical mastitis (Tivemo-Eftring, 1996) we found associations much in line with other published results. However, an interesting result was, however, that the temporal distribution of the cases of mastitis varied between the types of herds (Emanuelson and Hallen Sandgren, 1997). Thus, the incidence rate during the high risk period, i.e. around calving, was higher for first parity cows compared with older cows in herds with a high incidence of clinical mastitis, while the opposite (and more expected) was found in herds with low incidence (Fig. 1). The patterns found were in line with those of Myllys and Rautala (1995) and indicate that heifer management may be a risk factor for mastitis and related management practises may be an interesting topic for future epidemiological studies.

Figure 1. - PREDICTED INCIDENCE RATES (CASES/100 COW-DAYS AT RISK) OF CLINICAL MASTITIS (INCID.) IN HERDS WITH HIGH AND LOW INCIDENCES, WITH RESPECT TO PARITY (1, 2, 3+) AND STAGE OF LACTATION (0-22).



The many divergent results with respect to effects of management practises on mastitis, that are found in epidemiological studies in the published literature, should not be too surprising. Mastitis is recognised as a multifactorial disease, and the actual conditions at hand in a particular study do influence the results. This is most obvious as regards the presence of pathogens of a more environmental or contagious nature. Hopefully, we will see epidemiological studies in the future that capitalise more on the interactions between management and environmental conditions, in order to develop control programmes more adaptable to a changing environment.

Genetics

Traditional mastitis control programmes emphasise actions related to the environment and the pathogen and, when it comes to the third corner of the epidemiological triangle, i.e. the cow, they mainly focus on the use of antibiotics. There is, however, a growing concern over a too liberal use of antibiotics and ways to enhance the resistance of the cow would therefore be advantageous. Accordingly, vaccination strategies has been investigated, but another alternative would be to improve the innate resistance through genetic selection. The fact that resistance to diseases in domestic animals has a genetic component is by no means a new idea (Hutt, 1958) and several reviews in relation to mastitis have been published (Miller, 1984; Emanuelson, 1988; Shook, 1989; Leslie, 1995). This presentation will therefore mainly focus on the more recent results.

Clinical mastitis

A direct selection for mastitis resistance could either be performed on the basis of bacteriological test results or on recording of clinical cases. However, bacteriological testing is not feasible to perform on the large scale required for genetic evaluation, and clinical mastitis consequently remains the only option. Some of the recent estimates of the heritability of clinical mastitis are summarised in Table 1.

Table 1. - HERITABILITY OF CLINICAL MASTITIS

Source	Estimates
Lyons et al., 1991	0.13 - 0.23*
Simianer et al., 1991	0.06 - 0.09
Weller et al., 1992	0.01
Groen et al., 1994	0.06
Koenen et al., 1994	0.02*
Lund et al., 1994	0.02*
Uribe et al., 1995	0.00 - 0.15
Lund and Jensen, 1996	0.10 - 0.12
Nielsen et al., 1996	0.02 - 0.06*
Pösö and Mäntysaari, 1996	0.02 - 0.05*
Heringstad et al., 1997	0.08 - 0.09
Luttinen and Juga, 1997	0.01 - 0.02*
Pryce et al, 1997	0.05 - 0.07*

* Linear model on the observed scale

It should be noted that some of the estimates presented originate from analyses with traditional linear methods on the observable scale, and others from analyses with generalised linear models on the underlying liability scale. Still, the estimates are very much in line with earlier results, and show that there is definitely a genetic component in the susceptibility to mastitis.

The heritability of clinical mastitis is low due to a large environmental variation, possibly associated with inconsistent and incomplete recordings, but also with the large effects of management on mastitis. However, a low heritability should not mistakenly be regarded as synonymous with poor prospects for genetic progress. The genetic variation is more important, and it has been shown to be reasonably large as indicated by a wide difference between sire daughter groups (e. g. Philipsson et al., 1995; Heringstad et al., 1997). However, the low heritability means that mass selection is not

possible and that large groups of progeny is needed in order to achieve reasonably accurate genetic evaluations.

Somatic cell count

Recording of clinical cases of mastitis is not feasible in all countries and indirect criteria have to be used in genetic evaluations. The indirect measure receiving most attention is the milk somatic cell count (SCC) because it is readily available through most milk recording schemes at low additional costs and it is related to mastitis. Consequently, estimates of genetic parameters are becoming common, and a slightly more restricted selection of the available estimates than for clinical mastitis is given in Table 2.

Table 2. - HERITABILITY OF SOMATIC CELL COUNT

Source	Estimates
Lund et al., 1994	0.18
Schutz et al., 1994	0.07 - 0.11
Reents et al., 1995	0.09 - 0.11
Rogers et al., 1995	0.05 - 0.15
Lund and Jensen, 1996	0.17
Nielsen et al., 1996	0.09 - 0.15
Pösö and Mäntysaari, 1996	0.14 - 0.19
Boettcher et al., 1997	0.14 - 0.19
Boichard and Rupp, 1997	0.09 - 0.18
Luttinen and Juga, 1997	0.14 - 0.23
Pryce et al., 1997	0.15
Pösö et al., 1997	0.08 - 0.13
Weller and Ezra, 1997	0.16

There is a considerable variation in the definition of SCC in the cited references, but the heritability estimates are nevertheless remarkably similar. The estimates are higher than for clinical mastitis, but are low compared to what is usually seen for production traits. Hence, SCC could be a more promising selection criterion than clinical mastitis, but still require reasonably large progeny groups.

However, the usefulness of SCC as a selection criterion for increased mastitis resistance relies on its genetic correlation with the breeding objective. Estimates of the genetic correlation between clinical mastitis and SCC are not too common (Table 3).

Table 3. - GENETIC CORRELATION BETWEEN CLINICAL MASTITIS AND SOMATIC CELL COUNT

Source	Estimates
Weller et al., 1992	0.30
Lund et al., 1994	0.97
Philipsson et al., 1995	0.71 - 0.79
Lund and Jensen, 1996	0.80
Nielsen et al., 1996	0.00 - 0.71
Pösö and Mäntysaari, 1996	0.37 - 0.68
Luttinen and Juga, 1997	0.31 - 0.50
Pryce et al., 1997	0.65

The estimated correlations are generally moderately high to high, indicating that selecting on SCC would increase the mastitis resistance. On the other hand, most correlations are also far from unity, showing that SCC and records of clinical mastitis are not completely measuring the same trait, a fact that could perhaps be capitalised on.

Somatic cell counts are most frequently evaluated as a lactation mean SCC (after log transformation), although other ways are also used (e.g. Heuven, 1987; Reents et al., 1995; Lund and Jensen, 1996). An average does not give full justice to the dynamic variation in SCC. The most advantageous cow would respond very quickly to an infection and then return to normal levels. Such a picture is not necessarily reflected in an average. Ways to summarize SCC, in a more biologically informative way, should be investigated further. This becomes even more interesting if more frequent records than once a month are available in the future through the development of automated recording and milking.

One concern over using SCC in genetic selection has been that it would not only select against cases of clinical mastitis but also against the possibility for a cow to respond to an infection. Philipsson et al. (1995) showed, however, that the relationship between sires' breeding values for clinical mastitis and for SCC was completely linear, i.e. the lower the SCC the lower the incidence of clinical mastitis throughout the full range. It can be argued that these results are only valid under the current situation and that the relationship might change when the level of SCC of the population has been decreased. If SCC is used in genetic selection, this must of course be closely monitored. However, given that SCC is used in a total merit index with a relevant economic weight, a large shift in the level of SCC due to genetic selection is not likely to happen within a near future.

Conformation and markers

The second most investigated indirect selection criterion for mastitis resistance is probably conformation traits. In many countries dairy cows are evaluated for a number of traits reflecting body, locomotor, and udder characteristics. Several traits have also been studied in relation to mastitis (usually expressed in terms of SCC) and have given rise to varying genetic correlations. However, udder depth seems to be the trait most commonly associated with mastitis (most genetic correlations ranging from -0.2 to-0.5), and selection for higher udders will likely improve resistance to mastitis (Rogers et al., 1991; Lund et al., 1994; Rogers et al., 1995; Boettcher et al., 1997).

An alternative indirect selection method could be to use genetic markers. Interesting associations between marker genes in the major histocompatability complex (BoLA) and mastitis have also been found (e. g. Lunden et al., 1990; Schmutz et al., 1992; Schukken et al., 1994). However, selecting on one or a few marker genes might be hazardous, unless all associations really are determined, and a profile combining genetic markers with physiological measures might be a promising alternative (e. g. Almlid, 1981; Mallard et al., 1995).

Aspects of selection

It would seem obvious that it should be best to select sires based on clinical mastitis if clinical mastitis is the trait to be improved. However, this is not necessarily the case. The repeatability achieved with small sizes of daughter groups will be too low, due to the low heritability of clinical mastitis. Indirect selection based on SCC is actually more effective than selection based on clinical mastitis with a progeny group size of 50 daughters, and the two methods are more or less equal with 100 daughters (Philipsson et al., 1995; De Jong and Lansbergen, 1996). Selection based on both SCC and clinical mastitis has been shown to be the most efficient, even with progeny group sizes of 200 daughters (Sender et al., 1992; Philipsson et al., 1995; De Jong and Lansbergen, 1996), and this is what is applied e. g. in Sweden (Eriksson, 1991). Other studies have shown that, in a situation where information on clinical mastitis is not available, combining SCC with udder conformation and milkability improves the efficiency considerably (Rogers, 1993; De Jong and Lansbergen, 1996; Boettcher et al., 1997).

Effects of selection

There is an accumulating body of literature showing that there is a genetic antagonism between mastitis resistance and milk production (e. g. Madsen et al., 1987; Emanuelson et al., 1988; Banos and Shook, 1990; Lyons et al., 1991; Simianer et al., 1991; Weller et al., 1992; Groen et al., 1994; Uribe et al., 1995; Lund and Jensen, 1996; Nielsen et al., 1996; Pösö and Mäntysaari, 1996; Heringstad et al., 1997; Luttinen and Juga, 1997; Pryce et al., 1997; Weller and Ezra, 1997). This means that selection for increased milk yield will simultaneously decrease the genetic level of resistance to mastitis. The deterioration has been predicted by several authors (Emanuelson, 1987; Madsen et al., 1987; Strandberg and Shook, 1989; Rogers, 1993; Colleau and Le Bihan-Duval, 1995), and although not very large per year, a projection over a longer time span should give rise to considerable concern. Recently, Weller and Ezra (1997) presented a genetic trend for lactation average SCC in Israel and it did indeed show a continuous increase. The estimated trend was actually fourfold the predicted value, but the reason for his was not know.

However, the deterioration can be counteracted by applying selection on mastitis resistance in the breeding programme. The deterioration might not be reversed into an improvement, when actual economic weights are used, but it would at least be diminished (Emanuelson, 1987; Madsen et al., 1987; Strandberg and Shook, 1989; Rogers, 1993; Colleau and Le Bihan-Duval, 1995). An estimate of the genetic trend for mastitis resistance in Swedish dairy bulls showed that there had been no systematic change over the 10 years that were covered (Bratt, 1992). The favourable trend, i. e. not a deterioration, being a result of evaluating bulls for resistance to mastitis (in place since 1987) and for other traits, positively correlated with mastitis resistance, and including these traits in the total merit index (Eriksson, J.-A., 1991).

Conclusions

Compared to sanitation, management and therapeutic methods, genetic improvement certainly is not the primary method to control mastitis. However, genetic selection can provide for a cost-effective and permanent way to increase resistance to disease. Since genetic resistance to mastitis is slowly deteriorating in dairy cattle populations as a result of a selection emphasising increased production, genetic improvement strategies are especially important for controlling resistance to mastitis.

REFERENCES

1. Almlid, T. (1981): Indirect selection of bulls for improved resistance to diseases in dairy cattle. Livest. Prod. Sci., 8:321-330.
2. Banos, G., G. E. Shook (1990): Genotype by environment interaction and genetic correlations among parities for somatic cell count and milk yield. 1. Dairy Sci., 73: 2563-2573.
3. Barkema, H. W., Y. H. Schukken, T. J. G. M. Lam, M. L. Beiboer, H. Wilmink, G. Benedictus, A. Brand (1997): Incidence of clinical mastitis in dairy herds in three bulk milk somatic cell count cohorts. Epidemiol. Sante anim., 31-32: 05.15.1-05.15.3.
4. Barnouin, J., J. C. Fayet, M. Jay (1986): Enquete eco-pathologique continue: factuers de risque des mammites de la vache laitiere. I. Analyses multidimensionelles sur donnees d'elevage. Can. Vet. J., 27: 135-145.
5. Bartlett, P. C., G. Y. Miller, S. E., Lance, D. D. Hancock, L. E. Heider (1992): Managerial risk factor of intramammary infection with Streptococcus agalactiae in dairy herds in Ohio. Am. J. Vet. Res., 53: 1715-1721.
6. Boettcher, P. J., J. C. M. Dekkers, B. W. Kolstad (1997): Development of an udder health index for sire selection based on somatic cell score, udder conformation, and milking speed. J. Dairy Sci. (submitted).
7. Booth, J. M. (1995): Progress in the control of mastitis. Proc. 3rd International mastitis seminar, Tel Aviv, Israel, S4.3-S4.11.
8. Boichard, D., R. Rupp (1997): Genetic analysis and genetic evaluation for somatic cell score in French dairy cattle. Workshop on Genetic Improvement of Functional Traits in Cattle -Health Traits, Uppsala, Sweden.
9. Bratt, G. (1992): Experiences of breeding-evaluation for diseases and female fertility traits. International Bull Evaluation Service Bulletin No. 7: 8.1-8.12.
10. De Jong, G., L. Lansbergen (1996): Udder health index: selection for mastitis resistance. International Bull Evaluation Service Bulletin No. 12: 42-47.
11. Dodd, F. H. (1983): Mastitis - progress on control. J. Dairy Sci., 66: 1773-1780.
12. Colleau, J. J., E. Le Bihan-Duval (1995): A simulation study of selection methods to improve mastitis resistance of dairy cows. J. Dairy Sci., 78: 659-671.
13. Emanuelson, U. (1987): Genetic studies on the epidemiology of mastitis in dairy cattle. Report 73. Dept. Animal Breeding and Genetics, Swedish University Agric. Sci., Uppsala, Sweden.
14. Emanuelson, U. (1988): Recording of production diseases in cattle and possibilities for genetic improvements: A review. Livest. Prod. Sci., 20: 89-106.
15. Emanuelson, U., C. Hallen Sandgren (1997): A comparison of high yielding Swedish dairy herds with low somatic cell counts and with high or low incidence of clinical mastitis. Epidemiol. Sante anim., 31-32: 05.14.1-05.14.3.
16. Emanuelson, U., B. Danell, J. Philipsson (1988): Genetic parameters for clinical masitits, somatic cell counts, and milk production estimated by multiple-trait restricted maximum likelihood. J. Dairy Sci., 71: 467-476.
17. Eriksson, J. A. (1991): Mastitis in cattle. In: Owen, J. B. and Axford, R. F. E. (Eds.), Breeding for disease resistance in farm animals. Redwood Press Ltd., Melksham, UK, pp. 394-411.
18. Erskine, R. J., R. I. Eberhart, L. J. Hutchinson, S. B. Spencer, M. A. Campbell (1988): Incidence and types of clinical mastitis in dairy herds with high and low somatic cell counts. J. Am. Vet. Med. Assoc., 192: 761-76.

19. Faye, B., N. Dorr, F. Lescourret, J. Barnouin, M. Chassagne (1994): Farming practices associated with the 'udder infection' complex. Vet. Res., 25: 213-218.
20. Groen, A. F., I. Hellinga, J. K. Oldenbroek (1994): Genetic correlations of clinical mastitis and feet and legs problems with milk yield and type traits in Dutch Black and White dairy cattle. Neth. J. Agric. Sci., 42: 371-378.
21. Heringstad, B., A. Karlsen, G. Klemetsdal, J. Ruane (1997): Preliminary results from a genetic analysis of clinical mastitis data. Workshop on Genetic Improvement of Functional Traits in Cattle - Health Traits, Uppsala, Sweden.
22. Heuven, H. C. M. (1987): Diagnostic and genetic analysis of mastitis field data. Ph. D. Diss., Univ. Wisconsin, Madison, Wisconsin, U.S.A.
23. Hogan, J. S., K. L. Smith, K. H. Hoblet, P. S. Schoenberger, D. A. Todhunter, W. D. Hueston, D. E. Pritchard, G. L. Bownan, L. E. Heider, B. L. Brockett, H. R. Conrad (1989): Field survey of clinical mastitis in low somatic cell count herds. J. Dairy Sci., 72: 1547-1556.
24. Hutt, F. B. (1958): Genetic resistance to diseases in domestic animals. Cornell University Press, Ithaca, NY, U.S.A.
25. Koenen, E., B. Berglund, J. Philipsson, A. Groen (1994): Genetic parameters of fertility disorders and mastitis in the Swedish Friesian breed. Acta. Agric. Scand., 44: 202-207.
26. Leslie, K. E. (1995): Genetic selection for resistance to mastitis. Proc. 3rd International mastitis seminar, Tel Aviv, Israel, S8.2-S8.12.
27. Lund, M. S., J. Jensen (1996): Bayesian estimation of genetic and phenotypic parameters for clinical mastitis, somatic cell production deviance and protein yield in dairy cattle using Gibbs sampling. 47th Annual Meeting of the EAAP, Lillehammer, Norway.
28. Lund, T., F. Miglior, J. C. M. Dekkers and E. B. Burnside (1994): Genetic relationships between clinical mastitis, somatic cell count, and udder conformation in Danish Holsteins. Livest. Prod. Sci., 39: 243-251.
29. Lunden, A., S. Sigurdardottir, I. Edfors-Lilja, B. Danell, J. Rendel, L. Andersson (1990): The relationship between bovine major histocompatability complex class II polymorphism and disease studied by use of bull breeding values. Anim. Genet., 21: 221-232.
30. Luttinen, P., J. Juga (1997): Genetic relationships between milk yield, somatic cell count, mastitis, milkability and leakage in Finnish dairy cattle population. Workshop on Genetic Improvement of Functional Traits in Cattle - Health Traits, Uppsala, Sweden.
31. Lyons, D. T., A. E. Freeman, A. L. Kuck (1991): Genetics of health traits in Holstein cattle. J. Dairy Sci., 74: 1092-1100.
32. Madsen, P., S. M. Nielsen, M. Dam Rasmussen, O. Klastrup, N. E. Jensen, P. Thode Jensen, P. Scmidt Madsen, B. Larsen, J. Hyldegaard-Jensen (1987): Investigations on genetic resistance to bovine mastitis. Report 621. National Institute of Anim. Sci., Copenhagen, Denmark.
33. Mallard, B. A., S. Sharif, J. Sargeant, M. Scott, J. C. M. Dekkers, K. Leslie (1995): Genetic selection for enhanced immune response and disease resistance in Canadian Holstein cattle. Proc. 3rd International mastitis seminar, Tel Aviv, Israel, S8.13-S8.20.
34. Miller, R. H. (1984): Traits for sire selection related to udder health and management. J. Dairy Sci., 67: 459-471.
35. Miltenburg, J. D., D. de Lange, A. P. P. Crauwels, J. H. Bongers, M. J. M., Tielen, Y. H. Schukken, A. R. W. Elbers (1996): Incidence of clinical mastitis in random sample of dairy herds in the southern Netherlands. Vet. Rec., 139: 204-207.

36. Myllys, V., H. Rautala (1995): Characterization of clinical mastitis in primiparous heifers. J. Dairy Sci., 78: 538-545.

37. Myllys, V., T. Honkanen-Buzalski, P. Huovinen, M. Sandholm, E. Nurmi (1994): Association of changes in the bacterial ecology of bovine mastitis with changes in the use of milking machines and antibacterial drugs. Acta Vet. Scand., 35: 363-369.

38. Natzke, R. P. (1981): Elements of mastitis control. J. Dairy Sci., 64: 1431-1442.

39. Nielsen, U. S., G. A. Pedersen, J. Pedersen, J. Jensen (1996): Genetic parameters for mastitis, other diseases and somatic cell count in different parities in Danish dairy breeds. 47th Annual Meeting of the EAAP, Lillehammer, Norway.

40. Philipsson, J., G. Ral, B. Berglund (1995): Somatic cell count as a selection criterion for mastitis resistance in dairy cattle. Livest. Prod. Sci., 41: 195-200.

41. Pryce, J. E., R. F. Veerkamp, R. J. Esslemont, M. A. Kossaibati, G. Simm (1997): Genetic associations amongst health and fertility traits for two UK recording schemes. Workshop on Genetic Improvement of Functional Traits in Cattle -Health Traits, Uppsala, Sweden.

42. Pösö, J., E. A. Mäntysaari (1996): Relationships between clinical mastitis, somatic cell score, and production for the first three lactations of Finnish Ayrshire. J. Dairy Sci., 79: 1284-1291.

43. Pösö, J., E. A. Mäntysaari, A. Kettunen (1997): Estimates of genetic parameters for test day and lactation average SCS of Finnish Ayrshire. Workshop on Genetic Improvement of Functional Traits in Cattle - Health Traits, Uppsala, Sweden.

44. Reents, R., J. Jamrozik, L. R. Schaeffer, J. C. M. Dekkers (1995): Estimation of genetic parameters for test day records of somatic cell score. J. Dairy Sci., 78: 2847-2857.

45. Rogers, G. W. (1993): Index selection using milk yield, somatic cell score, udder depth, teat placement and foot angle. J. Dairy Sci., 76: 664-670.

46. Rogers, G. W, G. L. Hargrove, J. B. Cooper (1995): Correlations among somatic cell scores of milk within and across lactations and linear type traits of Jerseys. J. Dairy Sci., 78: 914-920.

47. Rogers, G. W., G. L. Hargrove, Jr. T. L. Lawlor, J. L. Ebersole (1991): Correlations among linear type traits and somatic cell counts. J. Dairy Sci., 74: 1087-1091.

48. Schmutz, S. M., T. G. Berryere, J. W. Robbins, T. D. Carruthers (1992): Resistance to Staphylococcus mastitis detected by a DNA marker. Proc. 315st Ann. Mtg. Natl. Mastitis Council, 124-133.

49. Schukken, Y. H., F. J. Grommers, D. Van de Geer, A. Brand (1989): Incidence of clinical mastitis on farms with low somatic cell counts in bulk milk. Vet. Rec., 125: 60-63.

50. Schukken, Y. H., F. J. Grommers, D. Van de Geer, H. N. Erb, A. Brand (1990): Risk factors for clinical mastitis in herds with a low bulk milk somatic cell count. I. Data and risk factors for all cases. J. Dairy Sci., 73: 3463-3471.

51. Schukken, Y. H., B. A. Mallard, J. C. M. Dekkers, K. E. Leslie, M. J. Stear (1994): Genetic impact on the risk of intramammary infection following Staphylococcus aureus challange. J. Dairy Sci., 77: 639-647.

52. Schutz, M. M., P. M. Vanraden, G. R. Wiggans (1994): Genetic variation in lactation means of somatic cell scores for six breeds of dairy cattle. J. Dairy Sci., 77: 284-293.

53. Sender, G., J. Juga, T. Hellman, H. Saloniemi (1992): Selection against mastitis and cell count in dairy cattle breeding programs. Acta Agric. Scand., Section A, Anim. Sci., 42: 205-210.

54. Shook, G. E. (1989): Selection for disease resistance. J. Dairy Sci., 72: 1349-1361.
55. Simianer, H., H. Solbu, L. R. Schaeffer (1991): Estimated genetic correlations between disease and yield traits in dairy cows. J. Dairy Sci., 74: 4358-4365.
56. Strandberg, E., G. E. Shook (1989): Genetic and economic responses to breeding programs that consider mastitis. J. Dairy Sci., 72: 2136-2142.
57. Tivemo-Eftring, M. (1996): Besattningar med lag cellhalt och hog produktion med antingen lag eller hog mastitfrekvens. Djurhalso- och utfodringskonferens 1996 (Animal Health Conference 1996). Swedish Association for Livestock Breeding and Production, S-631 84 Eskilstuna, Sweden.
58. Uribe, H. A.,B. W. Kennedy, S. W., Martin, D. F. Kelton (1995): Genetic parameters for common health disorders of Holstein cows. J. Dairy Sci., 78: 421-430.
59. Weller, J. I., E. Ezra (1997): Genetic analysis of somatic cell score and female fertility of Israeli Holsteins with an individual animal model. J. Dairy Sci., 80: 586-593.
60. Weller, J. I.,A. Saran, Y. Zeliger (1992): Genetic and environmental relationships among somatic cell count, bacterial infection, and clinical mastitis. J. Dairy Sci.; 75: 2532-2540.
61. Østeras, O. (1990): Udder health and environmental factors. Seminar - Machine milking and mastitis. Koldkaergaard, Aarhus, Denmark.
62. Østeras, O., A. Lund (1988a): Epidemiological analyses of the associations between bovine udder health and housing. Prev. Vet. Med., 6: 79-90.
63. Østeras, O., A. Lund (1988b): Epidemiological analyses of the associations between bovine udder health and milking machine and milking management. Prev. Vet. Med., 6: 91-108.

KLINIČKI MASTITIS U POPULACIJI: EPIDEMIOLOGIJA I GENETIKA

Sažetak

Znatan je napredak postignut u suzbijanju mastitisa primjenom standardnih programa u suzbijanju mastitisa. Napredak se lako može vidjeti u smanjenju prosječnog broja somatskih stanica u mliječnoj masi postignutom u mnogim zemljama tijekom zadnjeg desetljeća. Pa ipak, stopa kliničkog mastitisa i dalje predstavlja problem, osobito u nekim sredinama te bi stoga mogle biti opravdane alternativne mjere suzbijanja mastitisa. Epidemiološki postupci pružaju mogućnost pravovremenog prepoznavanja čimbenika rizika u stadu te tako pomažu u prilagođivanju sadašnjih programa suzbijanja okolini koja se mijenja. Ukratko su izneseni neki od najnovijih rezultata u tom području.

Čimbenici u vezi s domaćinom, tj. jedan od uglova epidemiološkog trokuta mastitisa zaslužuju povećanu pozornost. Na dohvatu je poboljšana otpornost krava na bolest, osobito otkad je sve veći broj radova jasno pokazao da je uključena genetska komponenta. Procijenjeni heritabiliteti otpornosti na klinički mastitis značajno su niski, ali se pokazalo da je genetska varijacija ne pretjerano visoka. Dat je pregled najnovijih rezultata te se raspravlja o izgledima uključivanja otpornosti na mastitis u uzgoju goveda.

Ključne riječi: mliječna krava, programi za suzbijanje mastitisa, broj somatskih stanica.

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