Measures of mastitis

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Introduction

In a time of crisis in the dairy industry, it's important to find new ways to decrease the costs associated with dairy production. Mastitis is the most costly disease in the cattle dairy industry (Blosser, 1979). In Belgium, the number of herds with less than 500,000 bulk tank milk cells/ml has increased by 18.3% between 1987 and 1993, but the frequency, of mastitis is still high (Booth, 1995). It seems that traditional methods of sanitary prevention (the so-called 'English' plan) are not successful in decreasing the incidence of environmental mastitis pathogens (Smith et al., 1985). It is therefore necessary to find new ways to decrease the frequency of mastitis, such as the genetic selection of animals more resistant to the disease. Selection to increase mastitis resistance is economically possible if the resistance is heritable, and if an accurate, inexpensive field measure. highly correlated to mastitis resistance can be found.

Before considering choices of measures of mastitis, we should keep in mind that the outcome of an infection is dependent upon the pathogenicity of the infectious microorganisms, the environment of the host and the genetic susceptibility of the host. Actually, if it is possible to correct for some non-genetic factors affecting the occurrence and severity of the disease, indicators used to estimate the genetic variation in mastitis resistance measure globally the genetic susceptibility of cows to any mastitis pathogens. However, it is well documented that animal resistant to one pathogen are often susceptible to others and that resistance to all diseases is unrealistic (Axford and Owen, 1991). In this paper, we will review the different measures of mastitis in order to find measures describing more completely the interaction between the host, the infectious pathogen, and the environment.

Measures of mastitis

Number of subclinical and clinical mastitis cases and individual milk somatic cells counts averaged over the whole lactation (somatic cell score or SCS) are dependent upon the specific infectious pathogens involved in the disease, upon the sanitary environment of the cow and upon individual non-genetic factors such as the stage of lactation, the age at calving, and the season (Table 1). Generally, coliforms cause clinical mastitis shortly after parturition. especially in the best producers while subclinical chronic infection tend to develop in cows infected with *Staphyllococcus aureus* (Jain, 1979). Because of the rather low heritability estimate (2-6%) of the number of clinical cases (Detilleux et al., 1995b; Weller et al., 1992), other indirect measures are necessary to select animals for their resistance to mastitis.

Cows infected with environmental mastitis pathogens show elevated SCS for a very short period of time, often in the beginning of lactation but cows infected with contagious pathogens show moderately elevated SCS, for a long period of time, and at any stage of lactation (Hill, 1981). In herds with low bulk tank cell counts and high milk production, the ability of SCS to predict an intramammary infection is reduced (Smith et al., 1985). This is probably because the prevalence of intramammary infections and of clinical mastitis due to environmental pathogens is increased (Kehrli and Schuster, 1994). The genetic correlation between clinical mastitis and lactation SCS is around 60-80%, which indicates also an imperfect genetic association

between the two traits. On the other hand, heritability for SCS is around 10% (Boettcher et al., 1192, Detilleux et al., 1995) which is higher than heritability obtained for direct measure of mastitis.

Certain aspects of udder and teat conformation have been used also to predict susceptiblity or resistance to mastitis. Several authors reported highest incidence of mastitis for cows with low rear udders, widely placed teats, and teats short and wide (Schutz et al., 1993,Thomas et al., 1984). In fact, those traits indicate the facility of entry of mastitis pathogens into the gland. Badly shaped udder which don't allow tight attachement of the milking machine facilitates the entry of contagious pathogens during milking. Widest teat orifice and teats closest to the ground facilitate teat lesions, exposure to manure, and the penetration of environmental pathogens between milking. Heritablilty estimates obtained in Belgium for 5 udder conformation traits on 69633 heifers are shown in Table 2 (Detilleux et al., 1996).

During mammary inflammation, more than 90% of milk cells are polymorphonuclear neutrophils (PMN) that migrate from blood. Speed of mobilization of the PMNs is crucial in the outcome of infection with *Escherichia coli*. A negative relationship exists between the pre-infection chemotactic response of blood PMNs and the severity of experimental *Escherichia coli* mastitis (Kremer et al., 1993) and between the preinfection ability of PMN to produce oxygen metabolites and the number of *Escherichia coli* in the mammary gland (Heyneman et al., 1990). Therefore, measures of PMNs functionality may enhance the precision in measuring the outcome of mammary infection. Heritability estimates for various functions of the PMNs were obtained on 137 periparturlent cows of the Iowa State University (Detilleux et al., 1994) and are shown in Table 3. The periparturent period was chosen because bovine neutrophil functions are usually depressed in periparturlent cows (Detilleux et al., 1995a: Kehrli et al., 1989) and because the susceptibility to mastitis and other diseases is increased around calving time (Grohn et al., 1989). Some susceptibility genetic variation was found in assays measuring oxygen-dependent killing ability (iodination, chemiluminescence, cytochrome C reduction). None or little variation was observed for PMN ingestion and migration. Additional studies are necessary to check whether cows selected for strong 'killer PMNs' are more resistant to subsequent challenge with specific mastitis phatogens. Among the molecular genetic markers, alleles at the bovine major histocompatibility complex (BoLA) were associated with different levels of resistance to clinical mastitis. There were more treatment for mastitis in milking cows with class I serotype w6 (Weigel et al., 1990) and allele BoLA-DQ IA (Lunden et al., 1990). However, there is some evidence that cows with particular BOLA haplotype are more resistant to infection but less able, once they are infected, to resist the pathogenic effects of the microorganisms.

Conclusion

After reviewing the traits available actually to measure mastitis, we may conclude that additional work is necessary to find new traits describing more completely the interrelation between environment, pathogen. and host. The lack of precise measurement of mastitis makes conclusive statement on genetic variability in mastitis resistance not repeatable across studies. decreases the efficiency of breeding selection programs, and could even lead to the selection of cows less resistant to mastitis.

References

Axford, R.F.E. and J.B. Owen. Strategies for disease control. 1991. In: Breeding for disease resistance in farm animals. pp 3-9.

Boettcher, P.J.. L.B. Hansen, P.M. VanRaden, and C.A. Ernst, 1992. Genetic evaluation of Holstein bulls for somatic cells in milk of daughters. J. Dairy Sci. 75: 1127.

Blosser, T.H., 1979. Economic losses from and the national research program on mastitis in the United States. J. Dairy Sci. 62 :119.

Booth, J.M., 1995, Progress in the control of

mastitis. Proceedings of the third IDF international mastitis seminar. Mastitis control systems. pp. 3-11

Detilleux, J.C., K.J. Koehler, A.E. Freeman, M.E. Kehrli, Jr., and D.H. Kelley. 1994. Immunological parameters of periparturient Holstein cattle: genetic variation. J. Dairy Sci. 77: 2640.

Detilleux, J.C., M.E. Kehrli, Jr., J.R. Stabel, A.E. Freeman, and D.H. Kelley. 1995a. Study of immunological dysfunction in periparturietit Holstein cattle selected for high and average milk production. Vet. Immunol. Immunopathol. 44: 251.

Detilleux, J.C., M.E. Kehrli, Jr.. A.E. Freeman, L.K. Fox, and D.H. Kelley, 1995b. Mastitis in periparturient Holstein cattle: A phenotypic and genetic study. J. Dairy Sci. 78: 2285.

Detilleux, J.C., and P.L. Leroy, 1996. Genetic evaluation of type traits in Belgium (in preparation).

Grohn,Y.T., H.N. Erb, C.E. McCulloch, and H.S. Salonleini. 1989. Epidemiology of metabolic disorders in dairy cattle: association among host characteristics, disease, and production. J. Dairy Sci.. 72: 1876.

Heyneman, R., Burvenich, C. and R. Vercauteren, 1990. Interaction between the respiratory burst activity of neutrophil leukocytes and experimentally induced Escherichia coli mastitis in cows. J. dairy Sci. 73: 985.

Hill, A.W. 198 1. Factors influencing the outcome of Escherichia coli mastitis in the dairy cow. Res. Vet. Sci., 31: 107.

Kehrli, M.E., Jr., B.J. Nonnecke, and J.A. Roth, 1989. Alterations in bovine neutrophil function during the periparturuient period. Am. J. vet. Res. 50:207.

Kehrli, M.E., Jr., D.E. Shuster, 1994. Factors affecting milk somatic cells and their role in health of the bovine mammary gland. J. Dairy Sci. 77: 619.

Jain, N.C., 1979. Conimon mammary pathogens and factors in infection and mastitis. J. Dairv Sci. 62:128.

Kremer, W.D.J., E.N. Noordhuizen-Stassen. F.J. Grommers, A.J.J.M Daemen, P.A.J. Henricks, A. Brand, and C. Burvenich. 1993. Preinfection chemotactic response of blood polymorphonuclear leukocytes to predict severity of Escherichia coli mastitis. 1993. J. Dairy Sci. 76: 1568.

Lunden, A.,S. Sigurdartottir, I. Edfors-Lilja, B. Danell. J. Rendel, and L. Andersson. 1990. The relationship between bovine major histocomptatibilit), complex class II polymorphism and diease studied by use of bull breeding values. Animal Genetics 21: 221.

Miller, R.H. Traits for sire selection related to udder health and management. 1984. J. Dairy Sci. 67:459.

Schutz,M.M., P.M. VanRaden, P.J. Boettcher, and L.B. Hansen. 1993. Relationship of somatic cell score and linear type trait evaluations of Holstein sires. J. Dairy Sci. 76:658.

Smith, K.L., D.A. Todhunter, and P.S. Schoenberger. 1985. Environmental mastitis: cause, prevalence, prevention. J. Dairy Sci. 68: 1531.

Thomas, C.L., W.E. Winson, R.E. Pearson, F.N. Dickinson, and L.P. Johnson. 1984. Relationship between linear type scores, objective the measures and indicators of mastitis. J. Dairy Sci. 67: 1281.

Weigel, K.A., M.E.Kehrli. Jr., A.E. Freeman, M.J. Stear, and D. Kelley, 1990. Association of class I bovine lymphocyte antigen complex alleles with health and production traits in dairy cattle. J. Dairy Sci., 73: 2538

Weller, J.I., A. Saran, and Y. Zeliger. 1992. Genetic and environmental relationships aniong somatic cell count, bacterial infect I ion. and clinical mastitis. J. Dairy Sci. 75: 2532- 2540.

Table 1. Some characteristics of udder infection by contagious or environmental pathogens

	Environmental pathogens	Contagious pathogens
Pathogens	Coliforms, S. uberis, S. dysgalactiae	S. aureus, S. agalactiae, S. dysgalactiae
Signs	clinical mastitis of short duration systemic signs	subclinical mastitis of long duration loss function of udder
Cow milk solmatic cell count	>2,000,000 cells/ml (PMN) short period (10 days) seasonal variation early in lactation	500,000 cells/ml long period any stage
Source of infection	manure, dirt, mud, standing water, bedding materials	infected mammary gland, replacement heifers

Table 2. Estimates of heritability (diagonal), genetic (above), and residual (below) correlations between udder conformation traits.

	RUD	RUH	RUW	SL	TP
RUD	.41	.34	.07	.33	.26
RUH	.13	.28	.69	.41	.24
RUW	.03	.29	.27	.42	.41
SL	.18	.20	.21	.21	.49
TP	.17	.12	.08	.23	.40

RUD = rear udder depth
RUH = rear udder height
RUW = rear udder width
SL = suspensory ligament
TP = teat placement

Table 3. Heritability estimates (and standard error) for 8 laboratory assays before, during, and after immunosupression (I.S.)

Laboratory assays	Before I.S.	During I.S.	After I.S.
Resting chemiluminescence	.53 (.36)	.19 (.35)	.0
Stimulated chemiluminescence	.0	.71 (.37)	.30 (.56)
Iodination	.51 (.38)	.25 (.20)	.0
Cytochrome C reduction	.22 (.31)	.88 (.35)	.99 (.22)
Direct migration	.03 (.37)	.23 (.24)	.0
Random migration	.30 (.30)	.0	.47 (.47)
Ingestion	.26 (.32)	.0	.27 (.42)