PRODUCTION OF CLEAN MILK THROUGH GENETIC SELECTION FOR MASTITIS RESISTANCE

Muhammad Sajjad Khan
Department of Animal Breeding and Genetics,
University of Agriculture, Faisalabad-38040, Pakistan

INTRODUCTION

Dairy producers in the developed world have passed through many stages of defining quality milk. Quality now includes protein alone or in some cases, in combination with fat as the farmers are paid premiums for these constituents. Milk hygiene has also become the norm and milk is required to be clean from pathogens. Payment systems penalize the farmers for having less hygienic milk and restrictions are likely to become tighter in the years to come (Allore *et al.*, 1995). The criteria used for determining whether milk is acceptable for processing (for consumption) is the level of somatic cells in the milk.

Somatic or body cells in milk are of two types, sloughed epithelial cells from the udder and leukocytes from the blood. The epithelial cells are present in normal milk as a normal breakdown and repair while leukocytes enter milk from blood, being attracted by chemical substances released from injured mammary tissue. Most somatic cells are primarily leukocytes, which include macrophages, lymphocytes and neutrophils. Studies identifying the cell types in milk have shown that epithelial cells usually range from 0 to 7% of the cell population. During inflammation, however, major increase in somatic cell count (SCC) is because of the influx of neutrophils into the milk (Miller and Paape, 1985). Because the lactating mammary gland is a very active metabolic organ, it is logical to expect variation in cell counts from day to day or milking to milking.

Testing of milk for somatic cells have gone through many phases of development and with the advent of electronic somatic cell counting, SCC has become one of the most popular and most important management aids to most dairymen. Millions of cows, throughout the world are thus tested every month. About four million cows are tested in the United States alone. Genetic basis of mastitis resistance has led to the inclusion of mastitis in the sire summaries and sire and dams are being selected to reduce the rate of increase in mastitis susceptibility (Shook and Schutz, 1994).

Situation in Pakistan is quite different from the developed world both in terms of quantity of milk produced on per animal basis as well as the quality in terms of constituents and hygiene. Dairy farmers have long been trying or at least wishing to improve the performance of their dairy animals. Selling one's animal's milk was not a socially acceptable thing, especially in the villages, few decade ago. Commercialization made it less shameful to sell surplus milk, and with establishment of milk plants, emphasis shifted to quality. Fat percentage thus became important. Payments to the milk sellers, especially those selling to the milk plants is made for quantity of milk and a differential is paid for fat percentage. We are still far away to check milk protein at farm level and include it in the definition of quality. Lack of appreciation for cheese as an important dairy product and perhaps the lack of taste for this product is the major reason for this inadequacy.

Similarly, milk is not checked for its health and hygiene. Majority of dairy animals suffer from some form of mastitis (Allore, 1993; Fazal-ur-Rehman, 1995), yet issue is quite neglected. Discussion with some of the mastitis researchers on possibility of routine milk testing for any mastitis indicator and lack of appreciation for genetic basis of disease resistance prompted this paper.

WHY WORRY ABOUT MASTITIS ?

a) Mastitis incidence and economic losses

Mastitis is considered as the most costly disease of dairy animals around the world. It is the second most important factor (after milk yield) to determine profit from dairying (Andrus and McGilliard, 1975). Estimates of economic loss generally ranged from $100 to $200 per lactation in the US. Jasper *et al.* (1982) estimated an annual cost of $2.5 billion to the US dairy industry. Shook (1989) reviewed various studies form US, Canada, and Europe and found that treatments for mammary reasons were the highest of all the categories of treatments. Shanks *et al.* (1982) also reported that the mammary category was the largest and accounted for about half the total health cost. In the five studies reviewed by Shook (1989), mastitis ranked third, after low production and reproduction, as the most frequent reasons for disposal of dairy cows. In Finland, percentage of cows culled due to udder problems were 35% (cited from Poso and Mantysaari, 1996).

Most of estimates available for mastitis losses in

Pakistan are based on opinions rather than actual data. The few surveys conducted for this purpose have been inadequate both in terms of criteria for detecting various mastitis forms, sampling, as well as the statistical techniques applied, it is difficult to access to losses. Still, in terms of economic losses, Khan *et al.* (1991) placed mastitis third to foot-and-mouth and parturient prolapse. Allore (1993) reviewed mastitis prevalence in cattle and buffaloes in India and Pakistan and reported that quarter wise prevalence of subclinical mastitis were 17-93% in cows and 4-48% in buffaloes.

b) Mastitis and milk yield

Relationship between SCC and milk production has been well documented. The observed negative relationship between milk yield and SCC partly reflects both the true biological effects of udder inflammation and a dilution effect. About half of the decrease in average bulk milk SCC over the years could be attributed to the increase in milk yield (Emanuelson and Funke, 1991). Raubertas and Shook (1982) reported that linear regression of 305-day lactation milk yield on average $\log_e$ SCC of lactation was -135 kg for first parity and on the average -270 kg for later parities. A unit increase in the lactation average $\log_e$ SCC was associated with a loss of approximately 296 kg of milk per lactation in the study of Fetrow *et al.* (1991). Among the six management traits to explain variation in herd average production (Appleman *et al.*, 1985), 16% of the variation was explained by SCC alone, SCC being the most important management trait in large herds (>80 cows) and low producing herds (<6311 kg).

Similar association between cell counts and test-day milk yield has been reported to exist. Eberhart et al. (1982) reported that bulk tank SCC accounted for 26% of the variation in average daily milk yield of the cows. Cell counts were negatively correlated with milk yield and accounted 23% of the variation in milk yield in the studies of Sender *et al.* (1992). Quarter foremilk SCC was reported to be slightly more useful in predicting milk yield than bucket SCC in the study of Miller *et al.* (1993). Within-cow regression of milk yield on (SCS) ranged from -5.2 to -6.3 kg. Reduction in milk yield per day at various levels of SCC is presented in Table 1. The effect on daily milk yield of Holsteins is presented in Fig. 1.

c) Other losses

Expenses and revenue losses arisen from mastitis, other than the realized (clinical) and unrealized (subclinical) reduced milk production are:

i. Loss of antibiotic-contaminated milk or milk of abnormal composition,
ii. Drug and veterinary costs,
iii. Cost of labor to care for mastitic cows,
iv. Decreased sale value of cows sold for dairy purposes
v. Increased herd replacement costs, and
vi. Loss of genetic material from the herd.

Table 1: Reduction in milk yield per day at various levels of SCC[1]

Daily SCC (Cells per ml)	Reduction in daily milk yield (kg)
50,000	0
100,000	1.0
200,000	1.9
400,000	2.8
800,000	3.8
1600,000	4.7

[1](Shook and Bringe, 1987)

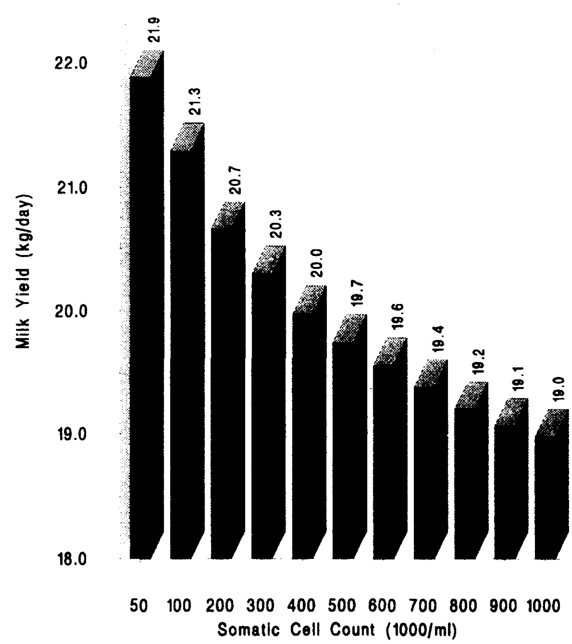

Fig. 1. Average daily milk yield for various levels of SCC (adapted from Jones *et al.*, 1984).

d) Factors affecting somatic cell count

The major factor affecting SCC is the infection of mammary gland. SCC from normal (i.e., uninfected) quarters are generally below 200,000 but may be below 100,000 during first lactations of cows. About 50% of uninfected cows have SCC under 100,000/ml, and 80% have under 200,000 (Eberhart *et al.*, 1979). The pathogens causing the greatest increase in SCC include *Staphylococus aureus*, *Streptococus agalactiae*, *colifoms*, and other Streptococus species. (Sheldrake *et al.*, 1983). Most studies however, indicate that the use of SCC alone to classify quarters as infected or uninfected results in error false positive and false negatives (Dohoo and Meek, 1982; Reneau, 1986; Fazal-ur-Rehman, 1995). The magnitude of response to major pathogens also varies among cows. Minimum level of SCC as an indicator of mastitis has thus been quite variable with species and breeds within species, as well as populations within breeds.

SCC generally increases with advancing age and stage of lactation. Second and third parities have been reported to be more similar for these trends as compared with first parity (Banos and Shook, 1990). Somatic cells are usually high during early stages of lactation, reach a minimum around peak, and rise gradually throughout the rest of the lactation.

Stress and season of calving have also been reported to influence somatic cell counts in milk. Schultz (1977) reviewed earlier studies on the effect of season on cell counts and reported that, although, cell counts were elevated by hot weather, evidence was not convincing for field conditions. Richardson and Owen (1982) found lowest values for spring and highest values for fall and winter months. SCC in the milk samples were lowest in winter and highest in summer in the study of Dohoo and Meek (1982). Different trends of lactation average somatic cells for different months of calving from five dairy record processing centers were reported by Boettcher *et al.* (1992).

The patterns of change in cell counts coincide with the incidence pattern of clinical mastitis. Stress of severe temperature and humidity has been reported to increase both the susceptibility to infection as well as increase in the number of pathogens to which cow is exposed (Smith *et al.*, 1985). A significant increase in SCC from heat stressed cows was shown by Elvinger *et al.* (1991). Decrease in milk yield due to seasonal differences is thus attributed partly to the decrease in milk yield and partly to the stress of the climate.

e) Relationship between Somatic Cell Count (SCC) and Somatic Cell Score (SCS)

Measurement of SCC is less expensive and more consistent than is the assessment of clinical mastitis. Log_2-transformed SCC, often called as linear somatic cell score (SCS) is the preferred trait for selection for mastitis resistance as it has a higher genetic control than the clinical mastitis. Advantages over SCC are a linear relationship between SCS and milk yield, its statistical properties, such as normal distribution, mean is near the median (i.e. 50th percentile), and standard deviation of SCS is homogeneous among herds or sires and thus mean alone characterizes the group. The mathematical relationship between SCS and SCC is:

$$SCS = Log_e(SCC/100) \div Log_e(2) + 3$$

For example, assume SCC = 400 cells/μl

$$SCS = Log_e(4) \div Log_e(2) + 3$$
$$= 1.3863 \div 0.69315 + 3 = 2 + 3 = 5$$

The SCS for various ranges of SCC are given in Table 2.

Table 2. Relationship between SCC and SCS.

	SCC (cells/μl)	
SCS	Mid-point	Range
0	12.5	0-17
1	25	18-34
2	50	35-70
3	100	71-140
4	200	141-282
5	400	283-565
6	800	566-1130
7	1600	1131-2262
8	3200	2263-4525
9	6400	4526-

GENETIC ASPECTS

Mastitis is very unique trait. It is a disease trait instead of a production trait like milk yield. Its measurement is more subjective and is not similar to measurements like liters or days. Use of SCS as a marker for mastitis has however, solved some of these difficulties. Dairy producers, mastitis researchers and veterinarians should thus be aware of the prospects of selection for this trait if progress is required in overall genetic merit of the dairy animals. Basic concepts of selection for mastitis resistance are presented here.

a) Degree of genetic control

The extent to which genetics influences a trait is called heritability. It is a population parameter and is not estimated for any individual animal. In statistical terms, it is a ratio of additive genetic variance (variance among breeding values of individuals) to phenotypic (phenotypes are what is observed or measured about a particular trait) variance. As heritability is a ratio, its value varies between 0 and 1.0. In percentage, it means 0 to 100% genetic control. This extent of genetic control is different for each trait. Approximate heritabilities for some of the common traits of dairy animals are given in Table 3. The higher the heritability, the greater is the genetic control on the trait and selection is likely to achieve rapid genetic progress. Generally, yield and type traits are moderately heritable; fat and protein percentages and stature, and size have higher heritability, while reproductive efficiency traits are lowly heritable.

Mastitis resistance has a heritability of about 0.10 i.e., genetics accounts for 10% of the variation in the capacity of cows to resist mastitis infection, while environment accounts for the remaining 90% of the variation. Heritability estimates vary for different populations; SCS has heritability between 12-15%. Some of the studies are presented in Table 4.

Table 3: Heritabilities of common traits in dairy cattle and buffaloes.

Species	Trait	h^2 (%)
Dairy cattle*		
	Milk yield	25
	Fat yield	25
	Protein yield	30
	Fat percentage	50
	Protein percentage	50
	Stature	50
	Body weight	50
	Overall type	20
	Reproductive efficiency	5
	Mastitis resistance	10
Buffaloes		
	Milk yield**	20
	Fat yield***	40

*Wilcox (1992)
**Khan *et al.* (1996)
***Iqbal (1996)

Table 4. Heritability of SCS by lactation.

Lactation					
1	2	3	4	5	Reference
9	8	10	10	14	Monardes and Hayes (1985)
13	12	10	-	-	Banos and Shook (1990)
10	-	-	-	-	Boettcher *et al.* (1992)
19	-	-	-	-	Weller *et al.* (1992)
9	9	11	-	-	Reents *et al.* (1995)
16	18	17	-	-	Poso and Mantysaari(1996)

b) Genetic relationship with milk yield

Relationship between breeding values (explained next) for two traits is called the genetic correlation. It indicates the extent to which two traits are influenced by the same genes. For example, the genetic correlation between milk and protein yield is high (0.9). This means that many of the same genes that influence the milk yield also influence the protein yield, and a bull with daughters that have a high mean for milk yield almost always sire daughters that have a high mean for protein yield. Similarly, genetic correlation of milk yield with fat percentage is negative (-0.3), meaning that bull with daughters that have high milk yield often have daughters with low fat percentage.

The genetic correlation of mastitis and milk yield have been reported previously. Shook (1989) reviewed many studies and reported that mean genetic correlation of clinical mastitis and milk yield was 0.20. Weller *et al.* (1992) found the correlation of bacterial infection status with milk yield to be 0.22. A higher correlation (0.51) between mastitis and milk yield was also reported by Simianer *et al.* (1991). Thus a slow but steady increase in mastitis incidence is expected to accompany genetic gain for milk yield.

c) Breeding Value

The value for a particular trait of an animal in a breeding program is called the breeding value. It is estimated to be twice the expected performance of its progeny relative to a population mean when mated at random. The reason for doubling the progeny performance is that only one half of the genes are transmitted to any offspring (and the other half comes from the other parent). As the true breeding value of any individual for any trait is never known due to sampling nature of inheritance of all polygenic traits and the role of environment, estimates are based on animal's own performance and performance of the other relatives. Dividing such estimated breeding values by 2 is used to predict the performance of future offsprings, relative to the population mean and is usually called as Predicted Transmitting Ability (PTA). A daughter of the same sire may thus have varying performance but daughters of a bull with a PTA of 100 kg for milk yield, for example, would be expected to produce 200 kg more milk per lactation from the daughters of a bull with PTA of 800 kg. PTA's for the six dairy breeds of US differed among breeds, their ranges are presented in Table 5. PTA of individual sires help to identify those transmitting poor mastitis resistance and deter their use as sires of future A.I. bulls.

Table 5: Range of bulls' PTA somatic scores for different breeds[1].

Breed	Minimum	Maximum
Ayrshire	2.82	3.48
Brown Swiss	2.52	3.41
Guernsey	2.88	3.89
Holstein	2.94	4.02
Jersey	3.14	3.93
Milking Shorthorn	2.61	3.10

[1]USDA Animal Improvement Programs Laboratory (Franck, 1994)

d) Degree of confidence

The measure of accuracy or degree of confidence in PTA's is called reliability (REL). For any trait, the heritability of the trait and the amount of information available determine the degree of confidence. The information may come from the animal's own performance, from the performance of the offsprings, and from information on parents or other relatives. As the heritability and the amount of information increases, REL also increases. Thus an animal has higher REL for milk yield than for reproductive efficiency (even if the same number of records are available from the animal and its relatives) because milk yield is under greater genetic control. Also, a bull with many daughters has a more reliable PTA for any trait than a bull with few daughters. Maximum value for REL is 100.

The heritability of mean lactation SCS is about twice of the current estimate of heritability (20-25%) of milk yield. Consequently, reliabilities of genetic evaluation are lower for SCS than for milk from same group of daughters. Hansen (1993) reported that progeny test programs designated to produce reliabilities of 75 to 80% for milk yield would produce reliabilities of 50 to 60% for SCS which is in a range of reliabilities for production traits in buffalo breeding programs (Khan, 1997).

e) Relative emphasis on SCS

It may be pointed out the selection against high SCS would be possible alongwith the other traits, but relative emphasis would depend on its relative economic value. The simulated study of Strandberg and Shook (1989) suggested that optimal selection policy would not stop increase in, or reduce incidence of, mastitis but would slow the rate of increase by 20 to 25%. They showed that overzealous use of SCS evaluations could be detrimental to economic gain for a breeding program. However, ignoring mastitis in breeding programs would accumulate undesirable long-term genetic consequences that might be difficult to overcome by environmental change alone. If the desired genotype for total merit include milk yield, clinical mastitis, milking labor and laminitis (Rogers, 1993), selection for five traits (Table 6) would improve total merit by 1 to 4% more than selection on milk yield alone. An example of weights for a typical combination of realistic assumptions is shown in Table 6 (Rogers, 1993). Possibility of modifications in type-production index (TPI), milk , fat, protein dollars (MFP$) index and other such indexes have been discussed by Cassell (1994). Net merit index, now part of the sire summaries from US is a combination of productive life, somatic cell score and production (milk, fat and protein) and feed cost information (Franck, 1994).

Table 6. Relative emphasis to SCS for selection[1]

Trait	Relative emphasis
Milk Yield	1.00
Udder depth	0.12
Teat placement	0.04
Foot angle	0.07
SCS	-0.05

[1](Rogers, 1993)

f) Other issues

We in Pakistan are faced with a dilemma of short term planning. As animal improvement is a long term endeavor, benefits of which are harvested for ever, most current projects focus on short term benefits. Current genetic improvement projects although, few, focus on milk yield only. Fat or protein or yield of total solids are traits to be incorporated in the future. Beauty and/or the beast is another important aspect to be incorporated in the selection goals. But as we plan animal production and disease reduction in the future, milk hygiene would also be an important aspect in the light of the above discussion. Policy makers should thus consider these aspects simultaneously instead of singling out any one.

CONCLUSIONS

Mastitis is an expensive, management-intensive problem, the treatment of which raises public health questions. Proper management practices are the first approach to mastitis prevention. Yield of milk and its constituents will remain the more important traits of economic importance but ignoring mastitis in breeding programs would accumulate undesirable long-term genetic consequences that might be difficult to handle by changes in the environment alone. Bulls can be selected so that their daughters have lesser incidence of mastitis. Overemphasizing the selection against mastitis may however, be counterproductive as it will affect the selection for other economic traits. Nevertheless, genetic evaluations for traits like SCS can be another tool available to the producers that can minimize the use of antibiotic therapy in the long run. Reduction in the risk of dairy product contamination and healthy cows and buffaloes are additional benefits. We as planners and scientists have to play our part so that milk continues to enjoy its reputation as "nature's most nearly perfect food".

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