

# Economic value and genetic prediction of clinical mastitis in South African Holstein cattle

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By

Edson Man'ombe

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*Faculty of AgriSciences*



Department of Animal Sciences

Supervisor: Professor K Dzama

Co-supervisor: Dr C Banga

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Date: April 2014

## **ABSTRACT**

<b>Candidate:</b>	<b>Edson Man'ombe</b>
<b>Study leader:</b>	<b>Professor K Dzama</b>
<b>Co-study leader:</b>	<b>Dr C Banga</b>
<b>Department:</b>	<b>Animal Sciences</b>
<b>Faculty:</b>	<b>AgriSciences</b>
<b>Degree:</b>	<b>MSc Agric</b>

Mastitis is the most prevalent and costly production disease of dairy cattle; hence mastitis incidence is a distinctly important trait in dairy cattle. The primary objective of the study was to determine the economic value, and develop a model for genetic prediction of clinical mastitis in South African Holstein cattle. These procedures are a prerequisite to including this trait in the breeding objective. The cost of clinical mastitis per incident was calculated as the sum of revenue loss due to discarded milk during the infection period and the associated treatment costs. Economic value (ZAR/incident) was calculated as the change in profit (increase in costs) resulting from a simulated marginal increase in mastitis incidence in an average herd. Average economic losses due to clinical mastitis were estimated at ZAR919.96/cow/year and the average incidence was 0.9cases/cow/year. The economic value of clinical mastitis was -ZAR1079.51/incident. A model for predicting estimated breeding values (EBVs) for clinical mastitis using somatic cell score (SCS), fore teat length (FTL), udder depth (UD) and rear udder height (RUH) was developed, using genetic (co)variances among these traits. Since EBVs for SCS, FTL, UD and RUH are routinely estimated under the national genetic evaluation programme, EBVs for clinical mastitis can

be predicted from the model developed in the current study. Thus, the results of the study provide the basis for including clinical mastitis in the breeding objective for South African Holstein cattle.

## Opsomming

<b>Kandidaat:</b>	<b>Edson Man'ombe</b>
<b>Studieleier:</b>	<b>Prof K Dzama</b>
<b>Mede- Studieleier:</b>	<b>Dr C Banga</b>
<b>Departement:</b>	<b>VeekundigeWetenskappe</b>
<b>Fakulteit:</b>	<b>LandbouWetenskappe</b>
<b>Graad:</b>	<b>M.Sc.Landbou</b>

Mastitis is die mees algemeenste en duursteproduksie siekte wat voorkom by melkbeeste, daarom is die voorkoms van mastitis 'n belangrike eienskap in melkbeeste. Die primêre doel van die studie was om die ekonomiese waarde te bepaal, asook die ontwikkeling van 'n model vir genetiese voorspelling van kliniese mastitis in Suid-Afrikaanse Holstein beeste. Hierdie prosedures is 'n voorvereiste vir insluiting van hierdie eienskap as 'n teeldeelwit in seleksie programme. Die koste van kliniese mastitis per voorval is bereken as die som van die inkomste verlies weens melk weggegooi tydens die infeksie periode en die gepaardgaande koste vir die behandeling. Ekonomiese waarde (ZAR / voorval) is bereken as die verandering in wins (toename in koste) wat voortspruit uit 'n gesimuleerde marginale toename in mastitis voorkoms in 'n gemiddelde kudde. Gemiddelde ekonomiese verliese as gevolg van kliniese mastitis was beraam op ZAR919.96/koei/jaar en die gemiddelde voorkoms was 0.9gevalle/koei/jaar. Die ekonomiese waarde van kliniese mastitis was - ZAR1079.51/geval. 'n Model vir die voorspelling van beraamde teelwaardes (EBV's) vir kliniese mastitis is ontwikkel deur gebruik te maak van die ko-variensies tussen die onderskeie eienskappe: somatiese sel telling (SST), voorspeen lengte (VSL), uier diepte (UD) en agter uier hoogte (AUH). Aangesien teelwaardes vir SST, VSL, UD en AUH gereeld beraam word onder die Nasionale genetiese evaluasie program, kan teelwaardes vir kliniese mastitis voorspel word vanuit die model wat ontwikkel is in die huidige studie. Dus verskaf die resultate van hierdie studie 'n basis vir die insluiting van kliniese mastitis as 'n teeldeelwit in seleksie programme van die Suid-Afrikaanse Holstein beeste.

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## Table of Contents

DECLARATION .....	ii
ABSTRACT.....	iii
ACKNOWLEDGEMENTS.....	v
List of figures and tables .....	ix
Chapter 1.....	1
General Introduction.....	1
1.1 Justification .....	4
1.2 Objectives.....	5
1.3 References .....	6
Chapter 2.....	9
Review of Literature.....	9
2.1 Introduction .....	9
2.2 Clinical mastitis in dairy cows .....	10
2.3 Genetic selection for mastitis resistance .....	12
2.4 Inclusion of clinical mastitis in the breeding objective .....	17
2.5 References .....	28
Chapter 3.....	34
Materials and Methods.....	34
3.1 Data .....	34
3.2 Definition of traits.....	34
3.3 Calculation of costs of Clinical Mastitis.....	38
3.4 Calculation of economic value .....	40
3.5 General prediction equation.....	41
3.6 Sensitivity analyses .....	42
Results.....	43
4.1 Genetic predictions.....	43
4.3 Financial losses due to Clinical Mastitis .....	46
Chapter 5.....	50
Discussion.....	50
5.3 References .....	54
Chapter 6.....	55

Conclusions and Recommendations ..... 55



**List of figures and tables**

Figure 2.1: Estimates of genetic parameters among mastitis, SCC and milk production traits ..... 14

Table 2.1: Breeding goal traits and potential index traits for indirect selection..... 19

Table 2.2: Relative emphasis on udder health in national selection indices..... 19

Table 2.3: Milk losses and treatment costs as a proportion of total losses per case of clinical mastitis in various countries ..... 27

Table 3.1: Genetic standard deviations ( $\sigma_a$ ) of CM and its predictor traits ..... 36

Table 3.2: Genetic correlations between CM and the predictor traits ..... 37

Table 3.3: Drug costs..... 39

Table 3.4: ADMY and milk price..... 39

Table 4.1: Genetic (co) variances among indicator traits (from ARC) ..... 43

Table 4.2: Genetic (co)variances between CM and measured traits ..... 44

Table 4.3: Indicators of financial losses due to CM ..... 46

Table 4.4: Variables in the model for calculating financial losses due to clinical mastitis (Base Herd) ..... 47

Table 4.5: Variables in the model for calculating financial losses due to clinical mastitis (Alternative Herd) ..... 48

Table 4.6: Sensitivity analyses ..... 49

Table 5.1 Proportions of discarded milk and treatment costs to economic value in various countries ..... 51

Table 5.2: Economic values of different countries ..... 52

## Chapter 1

### General Introduction

Clinical and subclinical mastitis are a major concern within the dairy cattle sector due to huge economic losses, public health and animal welfare concerns. Mastitis is the most frequent and costly production disease in the dairy cow population and selection for resistance to mastitis is highly desirable despite the difficulty of direct selection (Gengler & Groen, 1997). Mastitis incidence has been associated with the intense selection for milk production, which characterized the dairy industry for the past three decades (Banga, 2009). The incidence of mastitis associated with increased production has also been observed in several other dairy cattle populations around the world (e.g. Carlen, 2008). Heringstad *et al.* (1994) reported an increase in mastitis incidence from 14 % in 1978 to 28 % in 1994 in the Nordic dairy population. In the last decades, there has been, on average, 12 to 40% clinical mastitis incidence, depending on population and lactation average (Zwald *et al.*, 2004; Wolfova *et al.*, 2006). The major concern for the dairy industry is, therefore, reduced profit margins due to increased costs associated with udder health problems.

Udder health problems have negative impacts on the economics of dairy herds because of the direct and indirect costs incurred. The direct costs include medicinal treatments by veterinarians and farmers, reduced milk yield and compromised milk composition, discarded milk and penalties due to contamination, as well as extra labour. Indirect costs are due to reduced cow life-time milk production, reduced consumer confidence in milk and its

products and involuntary culling (Osteras, 2000). Costs reported in literature range from 43 to 189€ per clinical case (equivalent to 145 to 325€ per cow and year) depending on monetary unit price and country, severity level, age of cow, and on factors considered on calculation (Osteras, 2000). The high frequency and costs of clinical mastitis, its antagonistic relationship with milk production, the palliative reduction by use of antibiotics and vaccines, and additional economic gains realised due to reduced production costs, makes use of permanent and cumulative breeding principles a valid consideration.

Direct selection against clinical mastitis is challenging, mainly because, in most countries, including South Africa, clinical mastitis cases are not widely and routinely recorded. Scandinavian countries are the pioneers in using clinical mastitis incidence as a trait of economic importance (Heringstad *et al.*, 2000). The heritability of clinical mastitis has been estimated to be around 2-4% on the observed scale (Heringstad *et al.*, 2000; Hansen *et al.*, 2002; Interbull, 2008) and between 6-12% on the underlying scale (Heringstad *et al.*, 2000; Zwald *et al.*, 2004; Heinrichs *et al.*, 2005). This low heritability can be due to its binary nature, which may reduce accuracy of selection. Selecting for indicator traits of mastitis, which are routinely recorded, hence have more records, may increase selection accuracy. This entails the prediction of clinical mastitis resistance from traits genetically correlated with it, as an alternative to direct selection (Collau & Bihan-Duval, 1995).

The indirect traits for mastitis commonly available are somatic cell count (SCC) and udder morphological traits. Somatic cell count is an easily, widely and inexpensively measured trait in most national dairy recording schemes, including South Africa. It has a medium to high genetic correlation (0.50 to 0.70) with clinical mastitis (Mrode & Swanson, 1996; Koivula *et*

*al.*, 2005). The heritability of SCC is higher than that of clinical mastitis (Mrode & Swanson, 1996). However, use of SCC as the sole indicator trait can only achieve low genetic progress in resistance to clinical mastitis. Furthermore, although selection against high SCC should reduce mastitis incidence, the question remains whether SCC should be decreased to the lowest possible or should not be lower than a critical threshold (Rupp & Boichard., 2003). Therefore, SCC and mastitis incidence can be reduced substantially if udder type traits are also considered in selection programs.

Udder morphological traits are routinely measured in most dairy cattle recording schemes and are more heritable than clinical mastitis and SCC (Rupp & Boichard, 1999; De Groot *et al.*, 2000; Marie-Etancelin *et al.*, 2008). Dube *et al.* (2008) found favourably high genetic correlations among udder conformation traits; hence the possibility of optimization of the number of traits in the selection criteria for mastitis resistance. Heritability estimates for most udder type traits are low to medium (around 0.30), implying that slight genetic change can be achieved when selection for improved udder health is applied on these traits alone (Dube *et al.*, 2008). Scandinavian countries have been including data on udder health in their national recording schemes and they are the pioneers in selecting for udder health (Heringstad *et al.*, 2000). Since there are different traits associated with mastitis, these traits can be used together to improve udder health.

Traits can be selected for simultaneously using a selection index, where each trait is weighted. Despite mastitis realizing some extra total economic merit in the breeding objective (Kadarmideen & Pryce, 2001; Heringstad *et al.*, 2003), mastitis can be further improved by selecting for its indicator traits. Thus, SCC and udder type traits can be

combined in an udder health index to calculate genetic predictions for resistance to clinical mastitis (Dube *et al.*, 2008). Rogers *et al.* (1991) indicated that the inclusion of udder type traits and somatic cell score (SCS) in an udder health index is a gain in breeding for robustness in dairy cattle. Furthermore, de Jong & Lansbergen (1996) postulated that combining SCC, udder type traits and milking speed can give a higher response to selection for udder health compared to using SCC as the only indicator trait.

To construct the udder health index, the economic value of clinical mastitis, the breeding values of the indicator traits and the genetic co variances between clinical mastitis and indicator traits should be estimated. The genetic co variances for clinical mastitis with SCC and udder type traits are available in literature, while the breeding values for SCC and type traits are routinely estimated in South Africa (SA). However, the economic value of clinical mastitis has not been estimated in SA. The udder health index will enable the estimation of the breeding objective for mastitis in SA dairy cattle.

### **1.1 Justification**

Selection for higher milk yield has resulted in a considerable udder health strain on the modern dairy cow. The continuing high incidence of mastitis suggests that practical husbandry methods alone do not give adequate control, and that additional benefit could be obtained from increasing the resistance and reducing the susceptibility of cows to udder infections. The development and implementation of breeding principles that have long-lasting, cumulative and permanent effects can complement udder health care. Selection in South African dairy herds in the past has been for milk yield, which had some deleterious genetic effects on udder health (Dube *et al.*, 2008). The selection for milk yield may have

changed the frequency of alleles that confer resistance to mastitis as a correlated response to selection on milk yield. The astronomical costs of mastitis to the dairy farmer have hampered profitability. Modern day profitability depends on reducing costs more than increasing income (by improving production) and selection focusing on fitness traits, such as, clinical mastitis, fertility and lameness (Philipsson & Lindhe, 2003; Stott *et al.*, 2005). Somatic cell count is already included in the dairy cattle breeding objective (as an udder health trait) because it is an economically relevant trait in its own right. However, clinical mastitis has not been included in the breeding objective of SA dairy cattle and its economic value has not been estimated. Therefore, this study forms the basis for the inclusion of clinical mastitis in the South African national dairy breeding objective to set up a platform for genetic improvement of resistance to mastitis.

## **1.2 Objectives**

The aim of the study was to develop genetic predictions and economic weight for clinical mastitis for inclusion in dairy cattle breeding objectives under South African farm production systems. The specific objectives were to:

- (i) Determine the economic value of clinical mastitis in South African Holstein cows;  
and
- (ii) Develop an equation for the genetic prediction of clinical mastitis from correlated udder health traits - somatic cell score (SCS), udder depth (UD), fore teat length (FTL) and rear udder height (RUH) in South African Holstein cows.

### 1.3 References

- Carlen, E., 2008. Genetic evaluation of clinical mastitis in dairy cattle. PhD Thesis. Swedish University of Agricultural Science, Uppsala, Sweden.
- Carlen E., Strandberg, E. & Roth A., 2004. Genetic parameters for clinical mastitis, somatic cell score, and production in the first three lactations of Swedish Holstein cows. *J. Dairy Sci* 87, 3062-3070.
- Colleau, J.J. & Le Bihan-Duval, E., 1995. A simulation study of selection methods to improve mastitis resistance in dairy cows. *J. Dairy Sci.* 78, 659-671.
- DeGroot, B.J., Keown, J.F., Van Vleck, L.D. & Marotz, E.L., 2002. Genetic parameters and responses of linear type, yield traits, and somatic cell score to divergent selection for predicted transmitting ability for type in Holsteins. *J. Dairy Sci.* 85, 1578-1585.
- de Jong, G. & Lansbergen, L., 1996. Udder health index: selection for mastitis resistance. In: *Proc. International workshop on genetic improvement of functional traits in cattle.* Gembloux, Belgium, January 1996. *Interbull Bulletin* 12, 42-47.
- Dube, B., Banga, C. B., Dzama, K. & Norris., 2009. Genetic analysis of somatic cell score and linear type traits in South African Holstein cattle. *S. Afr. J. Anim. Sci.* 8 (1), 292-232.
- Gengler, N. & Groen, A.F., 1997. Potential benefits from multitrait evaluation – an example in selection for mastitis resistance based on somatic cell score and udder conformation. A simulation study. *Interbull Bulletin* No. 5. pp.106-112.
- Heringstad, B., Klemetsdal, G. & Ruane, J., 2000. Selection for mastitis resistance in dairy cattle: a review with focus on the situation in the Nordic countries. *Livest. Prod. Sci.* 64, 95-106.

- Heringstad B., Klemetsdal G., & Ruane J., 1999. Clinical mastitis in Norwegian cattle: frequency, variance components, and genetic correlation with protein yield. *J. Dairy Sci.* 82, 1325-1330.
- Koivula, M., Mantysaari, E.A., Negussie, E. & Serenius, T., 2005. Genetic and phenotypic relationships among milk yield and somatic cell count before and after clinical mastitis. *J. Dairy Sci.* 88, 827–833.
- Marie-Etancelin, C., Astruc, J.M., Porte, D., Larroque, H. & Robert-Granie, C., 2005. Multiple-trait genetic parameters and genetic evaluation of udder-type traits in Lacauneewes. *Livest. Prod. Sci.* 97, 211-218.
- Mrode, R.A. & Swanson, G.J.T., 1996. Genetic and statistical properties of somatic cell count and its suitability as an indirect means of reducing the incidence of mastitis in dairy cattle. *Anim. Breed. Abstr.* 64, 847-857.
- Østeras O., 2000. The cost of mastitis - an opportunity to gain more money. Institute for Animal Health/Milk Development Council. Proc. British Mastitis Conf, Shepton Mallet, pp. 67-77.
- Philipson J., & Lindhe B., 2003. Experiences of including reproduction and health traits in Scandinavian dairy cattle breeding programmes. *Livest. Prod. Sci.* 83, 99-112.
- Rogers, G.W., Hargrove, G.L., Lawlor, T.J. & Ebersole, J.L., 1991. Correlations among linear type traits and somatic cell counts. *J. Dairy Sci.* 74, 1087-1091.
- Rupp, R. & Boichard, D., 2003. Genetics of resistance to mastitis in dairy cattle. *Vet. Res.* 34, 671-688.
- Rupp, R. & Boichard, D., 1999. Genetic parameters for clinical mastitis, somatic cell score, production, udder type traits, and milking ease in first lactation Holsteins. *J. Dairy Sci.* 82, 2198-2204.



Stott A.W., Coffey M.P. & Brotherstone S., 2005. Including lameness and mastitis in a profit index for dairy cattle. *Anim. Sci.* 80, 41-52.

Wolfova, M., Stipkova M., & Wolf, J., 2006. Incidence and economics of clinical mastitis in five Holstein herds in the Czech Republic. *Prev. Vet. Med.* 77, 48-64.

Zwald N.R., Weigel K.A., Chang Y.M., Welper R.D. & Clay J.S. 2006., Genetic analysis of clinical mastitis data from on-farm management software using threshold models. *J.Dairy Sci.* 89, 330-336.

Zwald, N.R., Weigel, K.A., Chang, Y.M., Welper, R.D. & Clay, J.S., 2004. Genetic selection for health traits using producer-recorded data. I. Incidence rates, heritability estimates, and sire breeding values. *J.Dairy Sci.* 87, 4287-4294.

## Chapter 2

### Review of Literature

#### 2.1 Introduction

The application of animal breeding principles in mastitis control is a crucial development in combining production and functional traits in the breeding objective of the dairy industry as experienced by Scandinavian countries and simulation models the world over (Kadarmideen & Pryce, 2001; Heringstad *et al.*, 2003). Mastitis has been recognised as the most costly and common production disease in the dairy farming enterprise (Odegard *et al.*, 2003; Mostert *et al.*, 2004). A compromised udder health causes significant losses through reduced milk production, lower milk quality and treatment costs.

The last decade has seen a continuous and unfavourable trend for mastitis incidence and challenged the developed world's dairy industry to update their national breeding objectives by incorporating fitness traits such as clinical mastitis (Rupp & Boichard, 2003). There has been a serious concern on the continued decline in udder health traits in South African dairy cows (Banga, 2002; Dube *et al.*, 2008).

Genetic selection for increased resistance to mastitis can be performed by direct selection using clinical mastitis records; by indirect selection using traits genetically correlated to clinical mastitis; and/or by a combination of both. For clinical mastitis to be included in the South African dairy breeding objective, there is need to determine its economic weight and genetic parameters among clinical mastitis and relevant indicator traits. This chapter

reviews these genetic predictions and aspects of costs and economic weight associated with clinical mastitis in the South African dairy industry.

## **2.2 Clinical mastitis in dairy cows**

Clinical mastitis is the inflammation of udder tissue which is usually characterised by fever, pain, redness, swelling of the udder and change in composition and appearance of milk as well as decreased milk production (Bishop *et al.*, 2010). It is a highly complex disease which is mainly caused by bacteria (like *Staphylococcus* species) and some other pathogens (infectious) and non-infectious means. The non-infectious ways include chemical, thermal and mechanical injuries, which are equally damaging. The development of mastitis is influenced by host resistance (in which several genes are involved) and many environmental and microbial factors acting in singularity or combination – multifactorial background. In subclinical mastitis, there are no visible signs of infection. There is, however, reduced milk yield and a change in milk composition with an increased concentration of somatic cells (white blood cells and epithelial cells) and bacteria or other pathogens present in the milk.

### **2.2.1 Factors affecting incidence of mastitis**

The rate at which new cases of mastitis occur in a herd depends on the cow's exposure to causative pathogens in the environment as well as the innate immunity of the cow. There are management and non-management risk factors that are associated with the occurrence of mastitis. The management practices include housing (bedding), feed and water hygiene, milking equipment and technique, preventive health measures and environmental stress. Non-management factors, such as, season, parity, lactation stage, breed, udder conformation, milk production, milking speed and reproductive disorders are also

associated with the occurrence of mastitis (Hagnestam *et al.*, 2007; Nyman, 2007). The incidence of clinical mastitis has been reported to increase with increasing parity and that it is highest in early lactation, especially first parity cows (Zwald *et al.*, 2006).

There are marked differences between and within breeds. Dairy cattle breeds with roots in eastern France (Montbeliarde, Abondance) or central Europe (Simmental and Brown Swiss) have lower somatic cell counts and clinical mastitis frequency than the Holstein (Rupp & Boichard, 2003). They also reported that within breed, the genetic standard deviation of clinical mastitis frequency is up to 5%. In New Zealand, the breed comparison done revealed that Jersey cows had 2.9% less incidence of clinical mastitis than Holstein-Friesian cows and heterosis in crossbred cows had 13.4% less than the average of the parental breeds (Jury, 2011). The genetic constitution and innate immune defence of a cow plays a vital role in determining the resistance to mastitis of an individual cow.

### ***2.2.2 Impact of reducing mastitis incidence***

The occurrence of mastitis is associated with huge economic losses to the dairy farmer. These are related to a reduction in milk production, discarded milk due to contamination and veterinary care (Heringstad *et al.*, 2003). There is also a high risk of involuntary culling, penalties of milk price linked to abnormally high SCC in the bulk tank as well as an increased disease risk in the future of affected and previously unaffected cows. Udder health disorders are implicated in impaired animal welfare as well as consumer and ethical concerns. Milk is ideally expected to come from a healthy animal and to be of high quality. The extensive use of antibiotics as a remedy for clinical mastitis implies a greater risk of antibiotic residues in milk; thus increasing chances of generating antibiotic resistance in consumers, which is of

utmost public health significance. The uncontrolled and non-specific use of antibiotics to treat bacterial mastitis is often unsuccessful, causing relapses; hence extra costs to the farmer.

### **2.3 Genetic selection for mastitis resistance**

There has been a remarkable advancement in animal breeding and genetics pertaining to selection for disease resistance to aid in animal disease control. The observed animal performance (for example, disease state) is the outcome of the interaction between the animal's genetic makeup and the specific environment it was exposed (Berry *et al.*, 2011). Selection for more robust animals has the potential to complement current and future approaches to disease control.

Mastitis control programs previously focused on environmental measures (improved management) as a way of reducing the incidence of mastitis. This has been an efficient and primordial way to control mastitis, which, however, remains a frequent and erratic costly disease (Yancey, 1999). Clinical mastitis has a low heritability, which has been often misinterpreted as a limitation to improve the innate resistance of dairy cattle through genetic selection (Carlen, 2008). However, it has been claimed that the low heritability is mainly due to large environmental variation, which is difficult to control by any means, and also that there are considerable differences that exist between bulls (Philipsson *et al.*, 1995; Rupp & Boichard, 2003; Zwald, 2004). Improvement in mastitis control programs through genetic selection is beneficial as genetic gain is cumulative, permanent and potentially has a high investment return (Kadarmideen & Pryce, 2001).

### **2.3.1 Genetic parameters**

Genetic parameters of a trait are essential and are calculated from variances and (co)variances obtained in statistical analyses of phenotypic records. The validity of these parameters is only for a certain population and can change with time. Frequent evaluation is, therefore, needed.

There are positive and, hence, unfavourable genetic correlations for milk yield with clinical mastitis and SCC. This emphasises the need to include resistance to mastitis in the breeding goal to curtail a decreased genetic level of resistance to mastitis as a consequence of solely selecting for milk yield (Kadarmideen & Pryce, 2001). Genetic selection aligned to both milk yield and mastitis-related traits counteract the susceptibility and also to increase the economic response when compared to selection for milk yield only (Strandberg & Shook, 1989; Colleau & Le Bihan-Duval, 1995).

A summary of the heritabilities for udder health and production traits and the genetic correlations between traits across lactations for the first three lactations of Swedish Holstein cows are shown Figure 2.1 (Carlen, 2008).

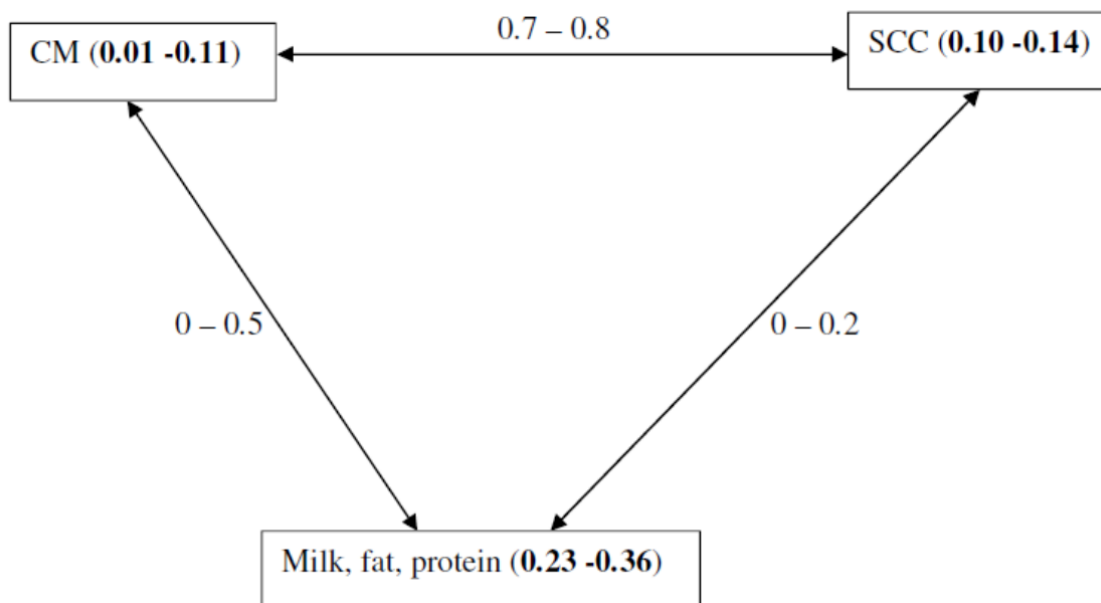


Figure 2.1: Estimates of genetic parameters among clinical mastitis (CM), somatic cell count (SCC) and milk production traits. Figures in bold show heritability estimate.

### 2.3.2 Clinical mastitis as a trait – Direct selection

In direct selection, the particular trait of interest is measured and selected for. In the case of resistance to mastitis, direct selection could be based on clinical mastitis cases recorded or bacteriological test results (Carlen, 2008). One advantage of using bacterial infection is that it additionally gives an indication of both subclinical and clinical cases (Weller *et al.*, 1992), and some studies have recently been performed considering pathogen-specific mastitis (de Haas, 2003; Holmberg, 2007). Bacteriological testing is, however, not practical on a large scale, and therefore the most common option is to use clinical records (Emanuelson, 1997). Currently, only some Scandinavian countries (Sweden, Denmark, Finland and Norway) have well-established national health-recording systems and include clinical mastitis directly in their national breeding programs. In all these countries, data from the health-recording system is combined with data from milk recording and artificial insemination (AI) records to create a single database to be used for both management and selection purposes

(Heringstad *et al.*, 2000). This practice by the Nordic and other various simulation studies have supported that including clinical mastitis incidence in the aggregate breeding value increases the genetic gain for resistance to mastitis (Kadarmideen & Pryce, 2001). In many other countries, clinical mastitis records are available on a limited scale (i.e. from research herds or selected commercial herds) (Zwald *et al.*, 2004). South Africa has not incorporated udder health in her national breeding objective (Miglior *et al.*, 2005).

### **2.3.2 Indicator traits – Indirect selection**

A good indicator trait is one with a higher heritability than, and is highly correlated with the goal trait; ideally data on it ought to be easy to measure and collect (Mrode & Swanson, 1996). The use of indicator traits to aid in genetic improvement of resistance to mastitis is commonly practised in countries where there are limited clinical mastitis records.

#### 2.3.2.1 Somatic cell count (SCC)

There are many desirable attributes of SCC as an indicator trait for clinical mastitis; hence its use for this purpose is widespread (Interbull, 2008). Estimates of the heritability for lactation-average SCC are higher than those for clinical mastitis and usually within the range of 0.1 and 0.2. The genetic correlation between SCC and clinical mastitis is moderate to high (often between 0.6 and 0.8), suggesting that genes predisposing cows to a low SCC also result in a lower rate of clinical mastitis (Rupp & Boichard, 2000; Heinrichs *et al.*, 2005; Negussie *et al.*, 2006). The use of SCC is more advantageous in that SCC data are easily and objectively measured on a continuous scale and tend to be normally distributed when transformed to a logarithmic scale – somatic cell score (SCS). Somatic cell count data is also readily available at a low additional cost in most milk recording schemes, and they reflect



both clinical and subclinical mastitis (Philipsson *et al.*, 1995; Mrode & Swanson, 1996; Heringstad *et al.*, 2000). The major concern about genetic selection for reduced SCC has been that it might reduce not only susceptibility to mastitis infection but also the cow's ability to respond to other infections (Kehrli & Schuster, 1994; Schukken *et al.*, 1997). Hence, SCC should be decreased to the lowest possible value at least within the range covered by the population mean and the genetic variance (Emanuelson, 1997; Veerkamp & de Haas, 2005).

#### 2.3.2.2 Udder conformation

Udder conformation is the second most common indirect trait for mastitis resistance that is currently being used. The relationship between various udder type traits and clinical mastitis or SCC has been investigated, but genetic correlations are generally low and results are inconsistent. Udder depth and fore udder attachment seem to be most frequently associated with resistance to mastitis (Mrode & Swanson, 1996; Rupp & Boichard, 1999; Nash *et al.*, 2000; Rupp & Boichard, 2003). Selection for a higher and more tightly attached udder improves resistance. In the South African Holstein populations, Dube *et al.* (2008) concluded that low, shallow udders with narrowly placed teats are linked to low SCC whereas in the South African Jersey population, cows with tightly attached udders and narrowly placed rear teats have low SCC.

Other traits that are associated either with clinical mastitis or with SCC are milking speed, electrical conductivity in milk and markers of immune response (Norberg, 2004). Faster milking is genetically associated with increased SCC (Luttinen & Juga, 1997; Boettcher *et al.*, 1998). On the contrary, other studies have indicated an opposite relationship between

milking speed and clinical mastitis, suggesting slower milking increases clinical mastitis (Rupp & Boichard, 2003).

### **2.3.3 Combination of direct and indirect traits**

Resistance to mastitis is a complex aspect, and a combination of direct and indirect traits converging different aspects of udder health in an udder health index will probably represent the best approach to genetic selection (Schukken *et al.*, 1997). Some authors have advised that when there is limited data on clinical mastitis, the efficiency of selection can be improved by combining SCC, udder type traits and milking speed (de Jong & Lansbergen, 1996; Boettcher *et al.*, 1998).

## **2.4 Inclusion of clinical mastitis in the breeding objective**

A breeding goal is the overall goal of the genetic improvement scheme and the main goal can be to maximise profit or maximise economic efficiency or to minimise economic risk (Gibson, 2005). The setting up of breeding objective traits in dairy cattle production forms the baseline for the development of national and/or international breeding goals (Groen *et al.*, 1997). The incorporation of animal breeding into the long-term strategic plan for animal production is envisaged to modify the genetic merit of future animal generations (Heringstad *et al.*, 2003). The aim is to produce desired products efficiently and in a more sustainable manner under future socio-economic and natural circumstances.

Economic efficiency is the ratio of production income to production costs (Gibson, 2005). There are several traits that influence the income and costs of dairy cattle production. Production and functional traits are the major influential traits in the dairy cattle

improvement programs. All these traits need a simultaneous consideration in a sound breeding objective (Miglior *et al.*, 2005). Functional traits considered, among others, include disease resistance (for example udder health), fertility, feed utilisation efficiency and milkability. There has been an endangerment of functional traits over the past years because of exhaustive disproportionate selection on increased production (Rauw *et al.*, 1998). Functional or fitness traits represent those characters of an animal which increase efficiency not by higher output of products but by reduced costs of input. To reduce production costs, a profit-oriented goal need to have economically relevant traits (ERT) being considered for inclusion in the selection objective (Banga, 2009). The curbing of genetic deterioration of production traits whilst improving functional traits can have positive economic and social impacts.

Different functional or fitness traits in a breeding objective can have different indicator traits (that are regularly measured) forming an index as shown in Table 2.1.

Table 2.1: Breeding goal traits and potential index traits for indirect selection

Breeding goal traits		Potential index traits for indirect selection
<b>Production traits</b>		
Milk	Milk yield, fat %, protein %	
<b>Functional traits</b>		
	Mastitis	SCS, udder depth, fore udder attachment, teat placement/length, milking speed
Health	Feet and legs	Rear legs, claw diagonal, mobility score
	General resistance	Longevity, persistency
Fertility	Showing heat	Calving to 1 <sup>st</sup> heat, calving to 1 <sup>st</sup> insemination
	Pregnancy rate	Non-return, 1 <sup>st</sup> insemination to pregnancy
Calving ease	Maternal effects	Rump angle
Milkability	Milking speed, behaviour	

(Groen *et al.*, 1997)

The developed world countries have formulated national genetic udder health indices with different relative emphasis on udder health as shown in Table 2.2.

Table 2.2: Relative emphasis on udder health in national selection indices

Country	Index	Udder health
Australia	APR	5.2
Canada	LPI	5.0
Switzerland	ISEL	1.0
Germany	RZG	5.0
Denmark	S-Index	14.0
Spain	ICO	3.0
France	ISU	12.5
Great Britain	PLI	5.0
Great Britain	TOP	8.0
Ireland	EBI	-
Israel	PD01	11.0
Italy	PFT	1.0
Japan	NTP	-
Netherlands	DPS	4.0
New Zealand	BW	1.0
United States	Net Merit	7.0
United States	TPI	-

(Miglior *et al.*, 2005)

### **2.4.1 Aggregate genotype**

An aggregate genotype is a mathematically-derived function of genetically-controlled traits that, when they are maximised, they will achieve the breeding objective set (Gibson, 2005). A defined breeding objective entails the necessity to define the relative importance (emphasis) of the traits to be improved that will contribute to the overall breeding objective. The identification of the traits to be genetically improved is identified and then follows the determination of the economic weights of improving each of those traits. For a given animal that is a candidate for selection, the sum of its additive genetic values multiplied by the economic weight for each trait is the aggregate genotype.

$$H = v_1g_1 + v_2g_2 + \dots + v_n g_n$$

Whereby H: aggregate (economic) genotype

$v_i$ : economic weight of the trait

$g_i$ : additive genetic values (Gibson, 2005)

The purpose of the aggregate genotype is to describe the genetic variation of the breeding objective in terms of biological traits, determination of the criteria for deciding which traits to include in the breeding objective. The traits to be included in the breeding objective must have a direct contribution to that specific objective (breeding). The indicator traits, those that have an indirect impact on the objective, do not belong to the aggregate genotype, but do belong to the index. Those traits with little or no genetic variation do not need to be included, although low heritability (like clinical mastitis) does not necessarily give an implication of low genetic variation. Breeders are, however, warned not to ignore traits such

as fertility and udder health in the aggregate genotype as this could lead to suboptimal decisions.

#### **2.4.2 Economic weights of clinical mastitis**

The potential increase in efficiency of dairy cattle production through selection is determined, in part, by the relative emphasis that is put on traits in the breeding goal. The economic value (weight) of a trait is the effect on efficiency of production of a marginal unit increase in genetic merit of the trait, independent of changes in other traits included in the linear breeding goal (Groen *et al.*, 1997). The relative weights that lead to maximum change in efficiency are quantified by the economic weight of a trait. Hazel (1943) revealed that the aggregate genotype is used to represent the genetic merit of an animal – the sum of its genotypes for several traits (assuming a distinct genotype for each economic trait). Each genotype is weighted by their predicted contribution to the increase in the overall breeding objective.

A balanced integration of functional traits in dairy cattle breeding goals with a correct weighting relative to milk production requires the economic values of these traits. To calculate accurately the economic revenues of breeding programs, which are required to optimise the structure of breeding programs, the absolute economic values are needed. The determination of the economic value for each trait in the breeding goal is the key to the installation of a total merit index (TMI) (Samore *et al.*, 2006). Market conditions influence economic values and these vary among different environments; hence economic values need to be estimated for each environment.

#### 2.4.1.1 Methods for estimating economic weights

The 'best' methodology in deriving economic values is heavily dependent on the traits and production circumstances considered (Groen *et al.*, 1997). More so, theoretically superior methods are not always the ones to easily implement practically. However, the derivation of economic values requires one to be conscious that genetic improvement is a technological development; aspects involved should be considered in deriving economic values. This awareness should help in making appropriate choices of a method for deriving economic weights. There is need to differentiate objective and non-objective methods.

##### **a) Objective methods**

The cornerstone in deriving economic values objectively is use of modelling or 'systems analysis' (Groen *et al.*, 1997). A model is an equation or a set of equations that describes the behaviour of a given system (France & Thornely, 1984).

##### **(i) Accounting method**

The economic value ( $v_i$ ) in this method is calculated by deducting costs ( $c_i$ ) from returns ( $r_i$ ).

$$v_i = r_i - c_i \quad (\text{Gibson, 2005})$$

In this scenario,  $r_i$  is the extra return received from a one unit increase in the mean for trait  $i$ , and then  $c_i$  is the extra cost associated with a one unit increase in the mean for trait  $i$ . There is a call to avoid double counting when using the accounting procedure. An exemplary case is one formulated by Sadeghi-Sefidmazgi *et al.* (2011), when they estimated the economic values for clinical mastitis and somatic cell score in the Iranian Holstein population. To avoid double counting when deriving economic values for clinical mastitis,



they excluded costs incurred by reduced milk price due to high somatic cell count (SCC), costs of increased replacement rate due to culling and suboptimal milk yield post mastitis occurrence. The main reason was that, the economic effect of these parameters is already in the breeding goal as traits in their own right.

In addition to double counting, it is important to note that  $r_i$  and  $c_i$  are marginal rather than average returns and costs; hence must be evaluated on the basis of a marginal increase of the trait value above its current value.

(ii) Profit function

A profit function is a single equation that describes the change in net economic returns as a function of a series of physical, biological and economic parameters (Banga, 2009). The economic value of a trait can be obtained as the first partial derivative of the profit function evaluated at the current population mean for all the traits (Banga, 2009). The advantage of the profit function method is that it avoids double counting as it uses partial derivatives.

(iii) Bio-economic model

A bio-economic model is a multi-equation simulation model (Groen, 1988), whereby the relevant biological and economic aspects of the production system are described as a system of equations (Gibson, 2005). The derivation of economic values using the bio-economic model has been applied in the dairy cattle production system (Koenn *et al.*, 2000; Veerkamp *et al.*, 2002; Shook, 2006). Bio-economic models are precise (Bourdon, 1998) and they describe the life cycle of an animal, including inputs and outputs, as a function of biological and economic parameters (Gibson, 2005). Groen (1997) highlighted that when

using simulated systems, the economic values are derived by assessing their reaction to a change of the endogenous element representing the genetic merit of the animal for a particular trait and the other traits remain unchanged. The shortfall of the bio-economic model lies in its complexity; hence errors are inevitable, making this model costly and tedious to develop. In the evaluation of the economic consequences of mastitis in Swedish herds, Nielsen *et al.*(2010) used 'The SimHerd Model', which is a dynamic bio-economic model with stochastic elements and the individual animal as the simulation unit.

#### **b) Non-objective methods**

These methods assign economic values in order to archive a desired or restricted amount of genetic gain for some traits (Groen, 1997). The economic values are not derived by direct calculation of influences of improvement of a trait on the increase in efficiency of the production system. Banga (2009) observed that the complexity of objective methods and the unpredictable future trends and parameters required for simulation modelling makes the non-objective approach the often preferred option. There is also lack of data to include all relevant aspects in the equations. In commercial porcine and poultry breeding, non-objective methods can be applicable as economic weights and can be calculated according to the performance of their stock relative to those of competitors (Groen, 1997).

#### 2.4.1.2 Economic values of clinical mastitis

There is an on-going effort to quantify the economic consequences of clinical mastitis. The main reasons for the concern are to develop the breeding objective and/or to quantify all the costs incurred due to cases.

The economic value of mastitis in the United Kingdom was estimated at US\$1.35 per percent incidence, giving an index weight for SCC predicated transmitting ability (PTA) of US\$0.33 (Stott *et al.*, 2005). Wolfova *et al.* (2006) found that the economic value of clinical mastitis in the Czech Republic dairy cattle population was -US\$91.40 per clinical mastitis case per year. In the Swedish dairy herds, Nielsen *et al.* (2010) estimated the cost per case of clinical mastitis to be €278. The annual avoidable cost of mastitis in a Swedish 150-cow dairy herd with an initial incidence of 32 clinical and 32 subclinical cases per 100 cow-years was estimated at €8235. In Hungarian Holstein-Friesian cows, functional traits (clinical mastitis incidence, calving difficulty score, total conception rate of heifers and calf mortality) reached a relative economic importance between 0.5 and 2.0% (Komlosi *et al.*, 2009).

In the South African dairy cattle population, emphasis on fitness traits is still at infancy. In the South African Holstein population, Dube *et al.* (2008) created a benchmark in the incorporation of both SCS and udder type traits in evaluating genetic predictions for resistance to mastitis by formulating an udder health index. They recommended further research on the determination of traits of importance in such an index as well as the relative economic emphasis of each trait considered. Banga (personal communication) derived the economic value of the South African Holstein and Jersey cattle. They communicated that a rise in SCS by 1 score lead to reduction in profit averaging ZAR1143.53 (ranging from ZAR491.48 to ZAR1795.57) per cow per year. This varied according to the breed (economic value was nearly double in the Holstein compared to the Jersey), production system (economic value nearly double in the concentrate-fed system relative to the pasture-based system) and payment system. Somatic cell score was among the most important traits in the

breeding objective, its value ranging from 26 to 118% compared to the most important trait, protein.

#### 2.4.2 Indices

Table 2.3 shows the proportion of major influencing factors in clinical mastitis costs for different countries and the reason for deriving such costs.

Table 2.3: Milk losses and treatment costs as a proportion of total losses per case of clinical mastitis in various countries

Country	Author(s)	Year	Proportions (%) of		Notes (reason)
			Milk losses	Treatment costs	
Denmark	Nielsen	1994	38	46	Breeding goal development
England	Kossaibati & Esslemont	1997	60	34	Quantifying all costs
India	Sasidhar <i>et al.</i>	2002	38	46	Quantifying all costs
Czech Republic	Wolfova <i>et al.</i>	2006	58 – 68	12 -25	Breeding goal development
Netherlands	Huijps <i>et al.</i>	2008	10	14	Quantifying all costs
Sweden	Svensson & Hultgren	2008	21	23	Quantifying all costs
USA	Bar <i>et al.</i>	2008	68	28	Quantifying all costs
Spain	Perez-Cabal <i>et al.</i>	2009	57	16	Quantifying all costs
Iran	Sadeghi-Sefidmazgi <i>et al.</i>	2011	68 – 78	19 – 27	Breeding goal development

(Sadeghi-Sefidmazgi *et al.*, 2011)

## Summary

The economic value evaluation and genetic prediction of clinical mastitis is a pre-requisite for its inclusion in the national dairy breeding objective. Clinical mastitis cases records are scarce hence need to use correlated udder health traits to formulate a model to predict its estimated breeding value. The inclusion of clinical mastitis in the national breeding objective by some Scandinavian countries has shown an increased genetic gain in resistance to mastitis.

## 2.5 References

- Banga, C. B., 2009. The development of breeding objectives for Holstein and Jersey cattle in South Africa. PhD Thesis. University of Free State, Bloemfontein, South Africa.
- Banga, C. B., 2004. Relationship between somatic cell count and milk production in South African Jersey cattle. Proc. S. Afri. Soc. Anim. Sci. Confr., Goudini, 28 June – 1 July 2004.
- Banga, C. B., Theron, H. E., Mostert, B. E. & Jordaan, F., 2002. Analysis of longevity in South African Holstein cattle. Proc. South African Society of Animal Science Congress, Christiana, 11-15 May 2002.
- Boettcher, P. J., Hansen, L.B., Van Raden, P.M & Ernst, C. A., 1992. Genetic evaluation of Holstein bulls for somatic cells in milk of daughters. J.Dairy Sci. 75,1127 – 1137.
- Boettcher, P. J., Dekkers, J. C. M. & Kolstad, B. W., 1998. Development of an udder health index for sire selection based on somatic cell score, udder conformation, and milking speed. J.Dairy Sci. 81, 1157-1168.
- de Haas, Y., 2003. Somatic cell count patterns – Improvement of udder health by genetics and management. Doctoral Thesis. Wageningen University/Animal Sciences Group Lelystad, The Netherlands.

- de Jong, G. & Lansbergen, L., 1996. Udder health index: selection for mastitis resistance. In: Proc. International workshop on genetic improvement of functional traits in cattle. Gembloux, Belgium, January 1996, Interbull Bulletin 12, 42-47.
- Dube, B., Banga, C. B. & Dzama, K., 2008. Genetic analysis of somatic cell score and linear type traits in South African Holstein cattle. *S. Afr. J. Anim. Sci.* 8 (1), 292-232.
- Dube, B., Banga, C.B., Dzama, K. & Norris, D., 2009. An analysis of the genetic relationship between udder health and udder conformation traits in South African Jersey cows. *Animal* 3(4), 494 – 500.
- Emanuelson, U., 1997. Clinical mastitis in the population: Epidemiology and genetics. 48<sup>th</sup> Annual Meeting of the EAAP. Vienna, Austria, Aug 25-28 1997.
- Emanuelson, U., Danell, B. & Philipsson, J., 1988. Genetic parameters for clinical mastitis, somatic cell counts, and milk production estimated by multiple-trait restricted maximum likelihood. *J. Dairy Sci.* 71, 467-476.
- Gianola, D., 1982. Theory and analysis of threshold characters. *J. Anim. Sci.* 54, 1079-1096.
- Gibson, J. P., 1995. An Introduction to the Design and Economics of Animal Breeding Strategies. Course Notes. Prague-Uhrineves, 7-16 September.
- Hagnestam, C., Emanuelson, U. & Berglund, B., 2007. Yield losses associated with clinical mastitis occurring in different weeks of lactation. *J. Dairy Sci.* 90, 2260-2270.
- Hansen, M., Lund, M.S., Sørensen, M.K. & Christensen, L.G., 2002. Genetic parameters of dairy character, protein yield, clinical mastitis, and other diseases in the Danish Holstein cattle. *J. Dairy Sci.* 85, 445-452.
- Heinrichs, D., Stamer, E., Junge, W. & Kalm, E., 2005. Genetic analyses of mastitis data using animal threshold models and genetic correlation with production traits. *J. Dairy Sci.* 88, 2260-2268.

- Heringstad, B., Klemestad, G. & Steine, T., 2003. Selection responses for clinical mastitis and protein yield in two Norwegian dairy cattle selection experiments. *J. Dairy Sci.* 86, 2990 – 2999.
- Heringstad, B., Klemetsdal, G. & Ruane, J., 2000. Selection for mastitis resistance in dairy cattle: a review with focus on the situation in the Nordic countries. *Livest. Prod. Sci.* 64, 95-106.
- Holmberg, M., 2007. Genetic dissection of functional traits in dairy cattle. Doctoral Thesis. Swedish University of Agricultural Sciences. Uppsala, Sweden. Electronic version available at <http://epsilon.slu.se/eng>
- Ingvartsen, K. L., Dewhurst, R. J & Friggens, N. C., 2003. On the relationship between lactational performance and health: is it yield or metabolic imbalance that causes diseases in dairy cattle? A position paper. *Livest. Prod. Sci.* 83, 277 - 308
- Interbull., 2008. Description of national genetic evaluation systems for dairy cattle traits as applied in different Interbull member countries. Retrieved September 4, 2008 from [http://www-interbull.slu.se/national\\_ges\\_info2/framesida-ges.htm](http://www-interbull.slu.se/national_ges_info2/framesida-ges.htm)
- Jury, K., 2011. Genetic analysis of incidence of clinical mastitis in New Zealand dairy cattle. PhD Thesis. Massey University, Palmerston North, New Zealand.
- Kadarmideen, H. N & Pryce, J. E., 2001. Genetic and economic relationships between somatic cell count and clinical mastitis and their use in selection for mastitis resistance in dairy cattle. *Anim. Sci.* 73, 19–28.
- Kehrli, M.E. & Shuster, D.E., 1994. Factors affecting milk somatic cells and their role in health of the bovine mammary gland. *J. Dairy Sci.* 77, 619-627.
- Luttinen, A. & Juga, J., 1997. Genetic correlations between milk yield, somatic cell count, mastitis, milkability and leakage in Finnish dairy cattle population. In: *Proc.*

- International workshop on genetic improvement of functional traits in cattle; health. Uppsala, Sweden, June 1997, Interbull Bulletin 15, 78-83.
- Miglior F, Muir B.L & Van Doormal B.J., 2005. Selection indices in Holstein cattle of various countries. *J. Dairy Sci.* 88, 1255–1263.
- Mrode, R.A. & Swanson, G.J.T., 1996. Genetic and statistical properties of somatic cell count and its suitability as an indirect means of reducing the incidence of mastitis in dairy cattle. *Anim. Breed. Abstr.* 64, 847-857
- Nash, D.L., Rogers, G.W., Cooper, J.B., Hargrove, G.L., Keown, J.F. & Hansen, L.B., 2000. Heritability of clinical mastitis incidence and relationships with sire transmitting abilities for somatic cell score, udder type traits, productive life, and protein yield. *J. Dairy Sci.* 83, 2350-2360.
- Negussie, E., Koivula, M. & Mäntysaari, E. A., 2006. Genetic parameters and single versus multi-trait evaluation of udder health traits. *Acta Agriculturae Scandinavica, Section A - Animal Sciences* 56, 73-82.
- Norberg, E., 2004. Electrical conductivity of milk as a phenotypic and genetic indicator of bovine mastitis. *Doctoral Thesis. The Royal Veterinary and Agricultural University, Fredriksberg/Danish Institute of Agricultural Sciences, Tjele, Denmark.*
- Nyman, A-K., 2007. Epidemiological studies of risk factors for bovine mastitis. *Doctoral Thesis. Swedish University of Agricultural Sciences. Uppsala, Sweden.*  
<http://epsilon.slu.se/eng>
- Rupp, R. & Boichard, D., 2003. Genetics of resistance to mastitis in dairy cattle. *Vet. Res.* 34, 671-688.



- Rupp, R. & Boichard, D., 1999. Genetic parameters for clinical mastitis, somatic cell score, production, udder type traits, and milking ease in first lactation Holsteins. *J. Dairy Sci.* 82, 2198-2204.
- Sadeghi – Sefidmazgi, A., Moradi – Shahrabak, M., Nejati – Javaremi, A., Miraei – Ashtiani, S. R. & Amer, P. R., 2011. Estimation of economic values and financial losses associated with clinical mastitis and somatic cell score in Holstein dairy cattle. *Anim.* 5 (1), 33 – 42.
- Samore, A. B. & Groen, A. F., 2006. Proposal of an udder health genetic index for the Italian Holstein Friesian based on first lactation date. *Ital. J. Anim. Sci.* 5, 359 – 370.
- Schukken, Y.H., Lam, T. J. G. M. & Barkema, H. W., 1997. Biological basis for selection on udder health traits. In: Proc. International workshop on genetic improvement of functional traits in cattle; health. Uppsala, Sweden, June 1997, *Interbull Bulletin* 15, 27-33.
- Shook, G.E., 1989. Selection for disease resistance. *J.Dairy Sci.* 72, 1349-1362.
- Strandberg, E. & Shook, G.E., 1989. Genetic and economic responses to breeding programs that consider mastitis. *J.Dairy Sci.* 72, 2136-2142.
- Veerkamp, R.F. & de Haas, Y., 2005. Genetic improvement in mastitis control programmes. In: Hogeveen, H. (Ed). *Mastitis in dairy production*. pp115-122. Wageningen Academic Publishers.
- Weller, J.I., Saran, A. & Zeliger, Y., 1992. Genetic and environmental relationships among somatic cell count, bacterial infection, and clinical mastitis. *J. Dairy Sci.* 75, 2532-2540.

Winkelman, A. M., Harris, B. L., Montgomerie, W. A & Pryce J. E., 2003. Calculation of economic weights for somatic cell count for inclusion in the New Zealand dairy cattle breeding objective. *Interbull Bulletin*. No. 31, 84 - 87

Zwald N.R., Weigel K.A., Chang Y.M., Welper R.D. & Clay J.S., 2006. Genetic analysis of clinical mastitis data from on-farm management software using threshold models. *J.Dairy Sci.* 89, 330-336.

## Chapter 3

### Materials and Methods

#### 3.1 Data

Data for calculating the economic value of clinical mastitis was obtained from two dairy herds with sound record keeping of clinical mastitis cases and the treatment thereof. Three additional herds were used in the determination of average incidence of clinical mastitis. The information needed was extracted using a questionnaire (Appendix 1). Production parameters considered were: production system, average daily milk yield and average milk price. For each case of clinical mastitis, the following information was recorded: starting and ending date of treatment, drugs administered (despite number of quarters affected), veterinary service, lactation number, herdsman's labour service and whether the cow recovered, died or was culled.

Estimates of genetic (co) variances among somatic cell score, fore teat length, rear udder height and udder depth were obtained from the national genetic evaluation program of the Agricultural Research Council (ARC).

#### 3.2 Definition of traits

3.2.1 Unmeasured trait – the predicted trait in the breeding objective

**Clinical Mastitis (CM)** - inflammatory disease of the mammary gland with visible udder ailments and abnormal milk composition.

### 3.2.2 Measured traits – predictor traits

**Somatic Cell Score (SCS)** – log transformed somatic cell count (SCC) – mammary epithelial cells and leucocytes.

**Fore Teat Length (FTL)** – important for machine milking.

**Rear Udder Depth (RUD)** – measures the height of the udder parenchyma. It is important for udder capacity.

**Udder Depth (UD)** – udder depth of the udder floor. The trait is important for longevity and udder health problems.

Schneeberger *et al.* (1992) formulated that the breeding value of an unmeasured breeding objective trait can be estimated from the breeding values of correlated traits that are routinely measured (selection criteria). To predict the breeding value for the trait in the objective, that is clinical mastitis ( $\hat{g}_i$ ), the following equation was used (Schneeberger *et al.*, 1992):

$$\hat{g}_i = G_{21}G_{11}^{-1}\hat{u}_i \quad (i)$$

Where:

$G_{21}$  = genetic (co)variance matrix among the selection criteria (SCS, UD, FTL, RUH) and trait in the objective (CM)

$\hat{u}_i$  = vector of predicted breeding values for selection criteria

$G_{11}$  = the genetic variance-covariance matrix among selection criteria

### 3.2.3 Calculation of genetic (co)variance between unmeasured trait and measured traits

There were no local estimates of genetic (co)variances among CM and the predictor traits available, therefore estimates from literature were used. Table 3.1 shows the genetic standard deviations from literature of the traits considered in the study.

Table 3.1: Genetic standard deviations ( $\sigma_a$ ) of CM and its predictor traits

Trait	$\sigma_a$
Clinical	
Mastitis	0.41
Somatic	
Cell Score	0.15
Udder	
Depth	1.14
Fore Teat	
Length	1.58
Rear	
Udder	
Height	1.39

(Lund *et al.*, 1999)

Table 3.2 shows the genetic correlations between clinical mastitis and the predictor traits and these were used to calculate the genetic variance as shown further below.

Table 3.2: Genetic correlations between CM and the predictor traits

	CM
Somatic	
Cell Score	0.7
Udder	
Depth	0.11
Fore Teat	
Length	-0.13
Rear	
Udder	
Height	-0.14

(Lund *et al.*, 1999)

The following equation (ii) was used to derive the genetic variance among the measured traits and the unmeasured traits using the genetic standard deviations and genetic correlations of the traits in the study from literature.

$$r_{xy} = \frac{\sigma_{xy}}{\sigma_x \sigma_y} \quad (\text{ii})$$

Where,

$r_{xy}$  - genetic correlation between two random variables

$\sigma_{xy}$  - co variance between two random variables

$\sigma_x$  - standard deviation of a random variable

$\sigma_y$  - standard deviation of a random variable

### **3.3 Calculation of costs of Clinical Mastitis**

The herds with well recorded and available records from which data were collected were from different production systems (concentrate and pasture fed) and the cows were at different lactation stages. The recording of cases (number of cases per year) was done for a one year cycle (1 January to 31 December 2011). All the herds were milked twice a day. Antibiotic therapy was done to all cows at dry-off time. The milk collected during the infection period of a cow was considered unmarketable to avoid antibiotic residues in milk for human consumption. It was assumed that such milk was not fed to calves because of risk of re-infection and antibiotic residues potentially leading to antibiotic resistance. The withdrawal period was also recorded observed and if more than one drug was used for treatment, the drug with longer withdrawal period was considered. The prices used for milk sales and treatment costs were as current as possible (2011) because prices change due to a variety of factors. The prices of the drugs used are shown in Table 3.3.

Table 3.3: Drug prices

<b>Drug</b>	<b>Price(ZAR)</b>
Curaclox LC 24	265.25
Tylo 200 (100ml)	125.40
Pen-strep (100ml)	153.23
Ketofen 10% (100ml)	320.65
Spectrazol MC	606.32

Table 3.4 shows the average daily milk yield (ADMY) and average milk price used in the calculation of costs incurred.

Table 3.4: ADMY and milk price

<b>ADMY (kg)</b>	<b>Milk price (ZAR/L)</b>
19.44	3.5

The costs of mastitis (ZAR/cow/year) for each herd were calculated as follows:

Mastitis costs = treatment costs + revenue loss due to discarded milk

where,

- Treatment costs = drug unit costs  $\times$  daily drug units  $\times$  infection period  $\times$  mastitis incidence
  - Revenue loss due to discarded milk = discarded milk (kg)  $\times$  milk price (ZAR/kg)
- \*Amount of discarded milk = average daily milk yield (ADMY)  $\times$  (infection period + withdrawal period)  $\times$  mastitis incidence



$$\text{Clinical mastitis incidence (CMI)} = \frac{\text{cows in a herd with at least one case per year}}{\text{Total herd size in a year}}$$

**Note:** To avoid double counting, the following were not included when clinical mastitis costs were calculated:

- (i) replacement costs due involuntary culling
- (ii) income reduction due to reduced lifetime milk yield post udder tissue infection
- (iii) lost income due to penalties when SCC is high

Replacement costs and lifetime production loss due to udder damage were included in the economic value of longevity. Somatic cell count is already a trait in the breeding objective. Veterinary costs were not included because of incomplete information and the need for a more extensive time frame of information recording.

### 3.4 Calculation of economic value

The economic value (v) was derived as the difference between baseline herd clinical mastitis costs and clinical mastitis costs in an alternative herd when incidence increased by one case per cow per year. This represents loss in profit that the farmer incurs when there is an increase in the baseline incidence of clinical mastitis of one case per cow per year.

The profit equation can be expressed as follows:

$$P = P(x,m) \tag{iii}$$

Where, P is farm profit, x is a vector of mean genetic values of the herd, and m is a vector of variables controlled by management. The calculation of the economic value can be denoted as the partial derivative of profit with respect to clinical mastitis incidence, at the current mean incidence rate, as follows:

$$v = \frac{\partial P}{\partial x}(\bar{x}, m_o) \quad (\text{iv})$$

All other management variables were kept constant, as denoted in equation (v)

$$\text{Where: } \frac{\partial P}{\partial m}(\bar{x}, m_o) = 0 \quad (\text{v})$$

### 3.5 General prediction equation

The EBV for CM can be calculated as an index of the predictor traits, using the following equation (Dube *et al.*, 2008):

$$EBV_{CM} = b_1 EBV_{SCS} + b_2 EBV_{UD} + b_3 EBV_{FTL} + b_4 EBV_{RUH} \quad (\text{vi})$$

Where,

$b_i$  = index weight for the measured trait i, computed as

$$\mathbf{b} = \mathbf{G}_{11}^{-1} \mathbf{G}_{12} \mathbf{v} \quad (\text{vii})$$

Where,

$\mathbf{G}_{11}$  is the genetic variance-covariance matrix among selection criteria (measured traits- SCS, UD, RUH and FTL)

$\mathbf{G}_{12}$  is the genetic variance-covariance matrix between selection criteria and clinical mastitis (unmeasured trait)

$v$  is the economic value of clinical mastitis

### **3.6 Sensitivity analyses**

Production changes and fluctuations in commodity markets can affect the costs incurred per case of clinical mastitis and the economic value of clinical mastitis. A sensitivity analysis of the model was carried whereby average daily milk yield, milk price and drug costs were varied by  $\pm 20\%$ .

## Chapter 4

### Results

The genetic predictions for the indicator traits and clinical mastitis are presented in this chapter. The calculated economic value was further used to develop a prediction model for estimating the breeding value of the unmeasured trait - clinical mastitis.

#### 4.1 Genetic predictions

Matrices  $G_{11}$  and  $G_{12}$ , co variances among indicator and co variances between indicator traits and clinical mastitis respectively were computed.

The genetic co variances among the indicator traits were obtained from on-going genetic evaluations by the Agricultural Research Council and are shown in Table 4.1

Table 4.1: Genetic (co) variances among indicator traits( $G_{11}$ )

	SCS	UD	FTL	RUH
SCS	0.0215	-3.6076	0.08	0.4094
UD	-3.6076	95.0664	1.0404	9.3139
FTL	-0.08	1.404	0.1024	0.3319
RUH	0.4094	9.3139	0.3319	7.4491

Table 4.2 shows the calculated genetic (co) variances between clinical mastitis and the measured traits (SCS, UD, FTL and RUH).

Table 4.2: Genetic (co)variances between CM and measured traits

	Clinical
	Mastitis
<hr/>	
Somatic	
Cell	
Score	0.0421
Udder	
Depth	0.0514
Fore	
Teat	
Length	-0.0839
Rear	
Udder	
Height	-0.0798
<hr/>	

$$G_{11}^{-1} = \begin{bmatrix} -4.227 & -0.1761 & -2.7516 & 0.5751 \\ -0.1761 & 0.0065 & -0.2709 & 0.0136 \\ -2.7516 & -0.2709 & 11.3863 & -0.0174 \\ 0.5751 & 0.0136 & -0.0174 & 0.0864 \end{bmatrix}$$

Above is the inverse matrix of  $G_{11}$  used to derive the  $b$  values.

$$G_{11}^{-1}G_{12} = \begin{bmatrix} 0.0020 \\ 0.0146 \\ 1.0837 \\ 0.0195 \end{bmatrix}$$

Above is the product matrix to be multiplied by the economic value to obtain the  $b$  values.

#### 4.3 Financial losses due to Clinical Mastitis

Table 4.3 shows the results of parameters recorded from the questionnaire

Table 4.3: Indicators of financial losses due to CM

	Herds	
	1	2
Herd Size (n)	66	188
Average daily milk yield (kg)	18.9	20
Clinical Mastitis Cases (n)	13	300
Clinical Mastitis Incidence (%)	0.2	1.6
Average Infection Period (days)	9.5	9.5
Discarded Milk (kg)	35.9	304

Tables 4.4 and 4.5 show the variables that were used to calculate the costs incurred due to clinical mastitis cases.

Table 4.4: Variables in the model for calculating financial losses due to clinical mastitis (Base Herd)

Variables	Farms		Mean
	1	2	
Revenue loss due to discarded milk (ZAR/cow/year)	143.49	912	527.74
Veterinary and drug costs(ZAR/cow/year)	87.16	697.26	392.21
Mastitis costs (ZAR/cow/year)	230.65	1609.26	919.96

Revenue losses due to discarded milk during infection period and treatment costs were higher in farm 1 than farm 2 due to the higher incidence of clinical mastitis in farm 2 as a result of a higher recurrence rate. The average mastitis incidence was 0.9 and ranged from 0.2 to 1.6. The duration of treatment period plus withdrawal time ranged from 2 days to 21 days with a mean of 9.5 days. It was estimated that during this infection period, 19.44kg of milk per cow per year was discarded at an average cost of ZAR527.74 per cow per year. In farm 1, costs of discarded milk contributed for 63% of mastitis costs and treatment costs accounted for 37% of total costs due to clinical mastitis. In farm 2, the two parameters were not much varied, 57% of total costs due to clinical mastitis was due to discarded and 43% due to treatment costs.



Table 4.5: Variables in the model for calculating financial losses due to clinical mastitis (Alternative Herd)

Variables	Farms		
	1	2	Mean
Revenue loss due to discarded milk (ZAR/cow/year)	860.93	1482	1171.45
Veterinary and drug costs(ZAR/cow/year)	522.95	1133.05	828.00
Mastitis costs (ZAR/cow/year)	1383.88	2615.05	1999.47
Economic value of CM ( $v$ )	1153.23	1005.79	1079.51

The economic values ( $v$ ) after increasing the average incidence of clinical mastitis by one case per cow per year had a mean of ZAR1079.51 and ranged from ZAR1005.79 to ZAR1153.23 per cow per year.

$$b = \begin{bmatrix} -2.2 \\ 15.7 \\ -1169 \\ 21.0 \end{bmatrix}$$

Hence, the prediction becomes:

$$EBV_{CM} = 15.71EBV_{UD} - 2.19EBV_{SCS} - 1169.3EBV_{FTL} + 21.02EBV_{RUH}$$

The estimated changes in the main contributing factors to costs and economic value of clinical mastitis are shown in Table 4.6.

Table 4.6: Sensitivity analyses

<b>Variables</b>	<b>% change (<math>\pm 20</math>)</b>	<b>Mastitis costs</b> <b>(ZAR/cow/year)</b>	<b>Economic value</b> <b>(ZAR/case/cow/year)</b>
<b>Base level</b>	0	919.96	1079.51
<b>Drug costs</b>	+20	1025.52	1208.33
	-20	814.54	951.45
<b>ADMY</b>	+20	998.40	1166.67
	-20	841.51	992.35
<b>Milk price</b>	+20	1025.50	1208.25
	-20	814.41	950.77

---

ADMY: Average Daily Milk Yield

## Chapter 5

### Discussion

The estimated breeding value and economic weight for clinical mastitis are a pre-requisite for clinical mastitis to be included in the national breeding objective as a trait on its own. The models used in this research work are applicable in situations where data is scarce due to poor recording of clinical mastitis cases.

The production losses due to milk discarded during the treatment period and observation of the withdrawal period accounted for the highest costs and varied from 57 to 63% of the economic costs due to clinical mastitis. These findings concur with other studies as indicated in Table 5.1. The second contributing factor to the losses was the expenditure due to treatment costs, which ranged from 37 to 43% of the total mastitis costs. This is similar to findings in Czech Republic, USA, Spain and Iran as show in Table 5.1. However, expenditure due to treatment costs was higher than revenue losses due to discarded milk in countries like the Netherlands and Sweden. The contrast can be due to a number of factors including whether the research was done to quantify costs or to develop a breeding objective (Sadeghi-Sefidmazgi *et al.*, 2010). The other factors are different drug and veterinary costs per country, different milk prices, farm production systems and methodology used in estimating costs.

Table 5.1: Proportions of discarded milk and treatment costs to economic value in various countries

Country	Author(s)	Year	Proportions (%) of	
			Discarded milk losses	Treatment costs
Czech Republic	Wolfova <i>et al.</i>	2006	63	19
USA	Bar <i>et al.</i>	2008	60	34
Netherlands	Huijps <i>et al.</i>	2008	10	14
Sweden	Svensson & Hultgren	2008	21	23
Spain	Perez-Cabal <i>et al.</i>	2009	57	16
Iran	Sadeghi-Sefidmazgi <i>et al.</i>	2010	73	23

The total cost per mastitic cow per year in the South African dairy farms in this study was estimated to be an average of ZAR919.96 at an average incidence of 0.9. Literature reports a variety of mastitis costs depending on methodology used on calculations. Wolfova *et al.* (2006) estimated financial losses ranging from 43.63 to 84.84€ in Czech Republic. The concept included costs from discarded milk, drugs, veterinary service, extra labour time, antibiotics for drying of cows and an extra milking machine. For a population from United States, Shim *et al.* (2004) reported 134€ as losses from milk (unproduced and unmarketable) and treatment. Mastitis was more expensive in multiparous than first-lactation cows because of higher milk yield. In an average Spanish herd of 110 cows, costs due to mastitis were estimated to be 3,190€ per year, only counting for treatment and discarded milk, and it can increase as milk price increases (Perez-Cabal *et al.*, 2009).

The economic value of increasing the average incidence of clinical mastitis by one case per cow per year had a mean of ZAR1079.51 and ranged from ZAR1005.79 to ZAR1153.23 per cow per year. Economic weights differ due to a various factors like currency used, inflation, method of estimation and others. Sadeghi-Sefidmazgi *et al.* (2010) estimated the economic value of clinical mastitis in Iran to be an average of US\$80.09 per cow per year. The economic value of clinical mastitis in Czech Republic was calculated to average US\$90.40 (Wolfova *et al.*, 2006). Economic values indicate the direction and emphasis of selection for a trait and these have been estimated in different countries as shown in Table 5.2.

Table 5.2: Economic values of different countries

<b>Country</b>	<b>Costs (per cow per year)</b>
Spain	<b>-€117</b> (Perez-Cabal <i>et al.</i> , 2009)
Sweden	<b>-€54</b> (Svensson & Hultgren, 2008)
USA	<b>-\$71</b> ( Bar <i>et al.</i> , 2008)
Iran	<b>-\$80</b> (Sadeghi-Sefidmazgi <i>et al.</i> , 2010)
Czech Republic	<b>-\$90.40</b> (Wolfova <i>et al.</i> , 2006)

Banga (2011) derived the economic value of the South African Holstein and Jersey cattle. They communicated that a rise in SCS by 1 score lead to reduction in profit averaging ZAR1143.53 (ranging from ZAR491.48 to ZAR1795.57) per cow per year. This varies according to the breed (economic value was nearly double in the Holstein compared to the Jersey), production system (economic value nearly double in the concentrate-fed system relative to the pasture-based system) and payment system.

Expectations are that broadening the breeding goal to encompass as many traits affecting profitability as possible will lead to an increase in profit by increasing output as well as by reducing health costs associated with production. Reducing health costs will improve the welfare of the cow and so a broader breeding goal should increase the welfare of the cow as well as farmer profit (Stott *et al.*, 2005).

### 5.3 References

- Bar, D., Tauer, L. W., Bennett, G., González, R. N., Hertl, J. A., Schukken, Y. H., Schulte, H. F., Welcome, F. L & Grohn Y. T., 2008. The cost of generic clinical mastitis in dairy cows as estimated by using dynamic programming. *J. Dairy Sci.* 91,2205–2214.
- Lund, M. S., Jensen, J. & Petersen, P. H., 1999. Estimation of genetic and phenotypic parameters for clinical mastitis, somatic cell production deviance, and protein yield in dairy cattle using Gibbs sampling. *J. Dairy Sci.* 82, 1045-1051.
- Lund, T., Miglior F., Dekkers, J.C.M. & Burnside, E.B., 1994. Genetic relationships between clinical mastitis, somatic cell count, and udder conformation in Danish Holsteins. *Livest. Prod. Sci.* 39(3), 243-251.
- Pérez-Cabal, M. A., Yaici, A.S. & Alenda, R., 2008. Clinical mastitis in Spanish dairy cows: incidence and costs. *Span. J. Agric. Res.* 6(4), 615-622.
- Sadeghi – Sefidmazgi, A., Moradi – Shahrabak, M., Nejati – Javaremi, A., Miraei – Ashtiani, S. R. & Amer, P. R., 2011. Estimation of economic values and financial losses associated with clinical mastitis and somatic cell score in Holstein dairy cattle. *Anim.* 5 (1), 33 – 42
- Stott, A. W., Coffey, M. P. & Brotherstone, S. 2005., Including lameness and mastitis in a profit index for dairy cattle. *J. Anim. Sci.* 80, 41–52.
- Svensson, C. & Hultgren J., 2008. Associations between housing, management, and morbidity during rearing and subsequent first-lactation milk production of dairy cows in southwest Sweden. *J. Dairy Sci* 91, 1510–1518.
- Wolfava, M., Stipkova, M. & Wolf J., 2006. Incidence and economics of clinical mastitis in five Holstein herds in the Czech Republic. *Prev. Vet. Med.* 77, 48-64.

## Chapter 6

### Conclusions and Recommendations

The genetic predictions for clinical mastitis derived from indicator traits and economic value for clinical mastitis estimated in this study form the baseline for the inclusion of clinical mastitis as a trait on its own in the South African Holstein dairy cows' breeding objective. The economic value calculated from simulating percentage increase of clinical mastitis incidence between the base herd and alternative herd depends on various factors amongst different countries. These factors can be data availability, methods of estimation applied, currency used and the general economy of the country among others. In the South African context, properly recorded data on clinical mastitis is scarce.

The cost of discarding contaminated milk during clinical mastitis infection period accounts for higher losses. This can be attributed to selection for increased milk yield; hence the volume of milk discarded per day is huge. The expenditure incurred to treat a clinical mastitis case per year is also high due to increased incidence of udder ailments as well as fluctuating and not-so-responsive treatment protocols. This depicts a picture whereby the milk producer has increased income from genetically superior cows in terms of milk volume, but incurs higher expenditure due to clinical mastitis in genetically inferior cows in terms of udder health.

Predicting breeding values for clinical mastitis from measured traits somatic cell score, fore teat length, udder depth and rear udder height gives an indication of the direction for udder health in which the dairy population is moving under genetic selection. A preliminary study



of this kind justifies the inclusion of clinical mastitis in the national breeding objective since it is an economically relevant trait. This also forms the building blocks for the formulation of a national udder health genetic index. More so, clinical mastitis is an important trait that needs to be well-recorded, as other traits like somatic cell count are being recorded. Deriving genetic predictions from properly recorded clinical mastitis data will give better estimated breeding values and up-to-date financial losses due to clinical mastitis. The basis has been developed to incorporate clinical mastitis in the breeding objective for South African Holstein cattle.

Appendix 1



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## QUESTIONNAIRE

### A Survey on Costs Incurred on a Clinical Mastitis Case

#### 1. Dairy Herd Details

- (i) Herd name .....
- (ii) Province .....
- (iii) Breed .....
- (iv) Size of milking herd (lactating & dry cows) .....
- (v) Production system .....

- (vi) Number of milking times/ day .....
- (vii) Average milk yield (kg/cow/day) .....
- (viii) Average milk price (R/kg) .....

**2. Clinical Mastitis Case**

- (i) Starting date of clinical mastitis treatment ..... / ..... / .....
- (ii) Ending date of clinical mastitis treatment ..... / ..... / .....
- (iii) Drugs administered .....
- .....
- .....
- .....
- (iv) Veterinary service (R/hr) .....
- (v) Lactation number .....
- (vi) Herdsman's labour service (R/hr) .....
- (vii) Recovered/died/culled .....