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MANAGEMENT AND ECONOMICS OF DAIRY COW MASTITIS IN THE URBAN AND PERI-URBAN AREAS OF ADDIS ABABA (ADDIS ABABA MILKSHED)

Erick Ouma Mungube

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FREIE UNIVERSITÄT BERLIN - ADDIS ABABA UNIVERSITY

MANAGEMENT AND ECONOMICS OF DAIRY COW MASTITIS IN THE URBAN AND PERI-URBAN AREAS OF ADDIS ABABA (ADDIS ABABA MILKSHED)

A thesis submitted in partial fulfiment for the degree of Master of Science in Tropical Veterinary Epidemiology at the Freie Universität Berlin and Addis Ababa University.

by

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Erick Ouma Mungube

Board of Examiners

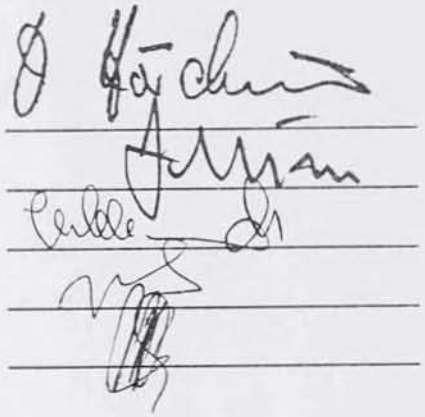
Professor Dr. F. Hörchner

Professor Dr. K.-H. Zessin

Professor Dr. G. Hildebrandt

Dr. Getachew Tilahun

Dr. Bayleyegn Molla

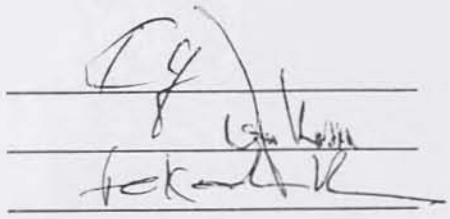


Academic Advisors

Dr. Tenhagen

Dr. Tesfu Kassa

Dr. Fekadu Regassa



SUPERVISORS:

Dr Bernd-Alois Tenhagen

Freie Universität Berlin
Fachbereich Veterinämedizin
Tierklinik für Fortpflanzung
AG Bestandsbetreuungund
Qualitätsmanagement

Dr Tesfu Kassa

Institute of Pathobiology
Addis Ababa University

Dr Fekadu Regassa

Faculty of Veterinary Medicine
Addis Ababa University

Dedication

Dedicated to my mother, Leunita Ajiambo and my father Ouma Walwe Kidwoli, brothers, the late Benedict Ouma Kidwoli, Moses Musebe Ouma and Josephat Roji Ouma and sisters Christine and Sylvia. Above all, glory to the Almighty God for this wonderful opportunity and good health.

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List of Abbreviations

Abbreviation	Full expression
BCR	Benefit cost ratio
CI	Confidence interval
CM	Clinical mastitis
CMT	California Mastitis Test
CSA	Central Statistics Authority
DDE	Dairy development Enterprise
DHIST	Dairy herds in secondary towns
EARO	Ethiopian Agricultural research organization
Ha	Hectare
IAR	Institute of Agricultural Research
ILCA	International Livestock Center for Africa
ILRI	International Livestock Research Institute
IMI	Intramammary infection
LF	Left front
LR	Left rear
MOA	Ministry of Agriculture
NMC	National mastitis control
OR	Odds ratios
Pexp	Expected prevalence
RF	Right front
RR	Right rear
SCC	Somatic cell count
SCM	Sub-clinical mastitis
TB	Total benefits
TC	Total costs

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Abstract

A study on management and economics of dairy cow mastitis was undertaken between February and September 2001 in the urban and peri-urban areas of Addis Ababa milk shed, Ethiopia. The study had the following main objectives: prevalence estimation, risk factor assessment and economic impact of mastitis. Questionnaire survey was used to gather various risk factors, while clinical examination and California Mastitis Test (CMT) screening were used to determine mastitis prevalence. Economic impact of mastitis was assessed using 51 dairy herds randomly selected for the study.

A questionnaire survey on 51 stratified sampled farms was administered to each herd owner/manager. The response rate of 90% was recorded. Dry cow therapy, post milking teat dipping, and antiseptic facilitated udder cleaning were not routinely done on the study farms. Zero grazing was the main method of animal husbandry.

Three hundred and sixty three (363) cows were randomly selected to participate in the mastitis prevalence study. Overall, the prevalence of sub-clinical mastitis was 46.6% (95% confidence interval (CI): 41.3-51.8%) and 27.8% (95% CI: 25.4-30.2%) at cow and quarter levels, respectively. Sub-clinical mastitis prevalence compared among farm sizes was higher on small than large-scale farms although this difference was not significant ($P > 0.05$) at both cow and quarter levels. Sub-clinical mastitis prevalence was higher in the urban than in the other production systems with no significant ($P= 0.403$) difference at cow level but significantly ($P = 0.006$) different at quarter level.

Overall clinical mastitis prevalence was 6.6% (95% CI: 4.3-9.7%) at and 2.8% (95% CI: 2-3.8%) at cow and quarter levels, respectively. Clinical mastitis prevalence was higher in large-scale farms than in small-scale ones but not significantly ($P > 0.05$) different at cow level and quarter levels, respectively. Clinical mastitis prevalence on the production sub-system was higher in the peri-urban system than in DHIST and urban. These differences were significant at cow and quarter levels ($P < 0.05$).

Cows aged at least 8 years, with poor body conditions, with at least 8 parities and in at least 8th month of lactation had a significantly higher risk for sub-clinical mastitis ($P<0.05$ and 95% CI's

of odds ratios (ORs) >1). Cows aged at least 4 years, with at least 8 parities, cows in at least 4 month of lactation, poor body conditions, leaking milk and with previous udder infections had a significantly higher risk of clinical mastitis (P<0.05, 95% CI* of OR >1).

A split udder investigation on 30 crossbred cows with sub-clinical mastitis showed an average quarter production of 0.82 ± 0.40 kg per milking. This was significantly (P=0.008) associated with CMT score. Reduced milk production due to sub-clinical mastitis were 1.2%, 6.3%, and 33% in quarters with scores 1+, 2+, and 3+, respectively. With the given distribution of CMT scores in the study population, a quarter with sub-clinical mastitis lost 17.1% of its milk production.

Mastitis losses amounted to 76513.1664 Birr in a single lactation or 210.8 Birr per cow per lactation. Milk production losses due to culling, treatment and withdrawal contributed 38.4%, 49%, 9.3% and 3.3%, respectively. A partial budget analysis indicated a benefit cost ratio (BCR) of 4.06 and net benefits of 29559 Birr per lactation with treatment as the only control strategy. Sensitivity analysis indicated a decrease in both net benefits and BCR when the efficacy of treatment is less than 100%. BCR was only done for clinical mastitis.

Mastitis was a major problem and further prospective investigations addressing infective agents, their antibiotic sensitivity profiles and the effect of the agent on the economic losses are recommended.

Key words: Management, economics, dairy cow, mastitis, urban, peri-urban, Addis Ababa

1 INTRODUCTION AND OBJECTIVES

1.1 Introduction

Ethiopia has the largest cattle population in Africa, with an estimated 30 million heads, accounting for 17% of the estimated total cattle population in Africa (FAO, 1996). The huge cattle resource plays an important role in terms of food security, cash income, capital assets social livelihood. However, the contribution of cattle towards food supply and other needs is unable to meet the demands of the growing population as local cows produce on average 230kg/lactation (FAO, 1996). The upgraded exotic cows in contrast yield 8.8 kg per cow per day over a 328 day-lactation period (Mekonnen *et al.*, 1985). Therefore, Ethiopia is one of the 7 largest importers of milk and milk by-products among Sub-Saharan countries (Von Massow, 1989). Nevertheless, the consumption of animal protein of 10g/head/day is low (FAO, 1996).

The Ethiopian dairy industry has evolved through the assistance of various institutes and organizations, for example, the Institute for Agricultural Research (IAR), Arsi Regional Development program (ARDP), Teaching institutions, Ministry of Agriculture (MOA) and Ministry of State Farms Development (Kebede, 1985). However, the impact of these programs has achieved minimal results due to various reasons such as low genetic potential of the indigenous breeds and diseases of various kinds.

Among the challenges of dairy development in the tropics such as breed improvement, nutrition management, control of infectious, tick-borne, blood and internal parasitic diseases, inadequate livestock infrastructure, mastitis is well recognized as being a major problem as it causes a serious wastage and undesirable milk quality (Thirapatsakun, 1989). When modern dairy farming in the tropics was first adopted, mastitis was foreshadowed to be an important disease in dairy cattle. Today, most tropical dairy farmers have experienced disaster caused by this disease, and can no longer afford its costs in addition to their other burdens (Thirapatsakun, 1989).

Mastitis as a disease has received little or no attention in Ethiopia especially the sub-clinical type (Hussein et al., 1999). Many efforts have been focused on treatment of clinical cases rather than tackling this devastating disease from the control point of view. Most dairy farmers normally ignore sub-clinical mastitis, which incidentally occurs at a much higher frequency than clinical

mastitis, yet it is the worst in terms of lowered productivity. Microbiological causes of the mastitis are many and multiple factors involved in the management, housing and milking are of importance when trying to understand the control of this disease on dairy farms. The economics of mastitis control requires a mixture of both epidemiological and economic data (Omore *et al.*, 1996). Unfortunately, these 2 major constraints have been considered independently by researchers in the past. It is important to assess them together to determine their relationship and their simultaneous effect on production (Omore *et al.*, 1996). The institution of a programme to either control or eradicate a disease in an animal population must be based on knowledge of the amount of the disease in that population, factors associated with its occurrence, the facilities required to control the disease, and the costs and benefits involved (Thrusfield, 1995). Therefore, it is important that the control of mastitis requires a clear understanding of its causes and management techniques which limit the spread of infection (Thirapatsakun, 1989).

Economic analysis is required in order to balance the cost of controlling mastitis against economic loss attributable to this disease. This is an essential part of most modern planned animal health programs (exceptions are found on diseases of companion and sacred animals) (Thrusfield, 1995). Although it may be economic to reduce a high level of a disease in a herd/flock, it may be uneconomic to reduce even further the level of a disease that is present at only a very low level. When the prevalence of mastitis in a herd is 15% for example, productivity would be severely affected and a control programme would be likely to yield financial benefit. On the contrary, if only 1 % of the cows in the herd are affected, the cost of further reduction of the disease might not result in a sufficient increase in productivity to pay for the control programme.

Mastitis is economically the single most important disease of the dairy cattle. It reduces milk yields, profits and the quality of milk and milk products in all dairy-producing countries of the world (Bulletin of International Dairy Federation, 1999). Previous studies on the mastitis complex in the Addis Ababa milk shed have not addressed the economic implications of mastitis to the dairy farmers, hence this study was specifically designed to address economics together with management aspects that are appropriately related to mastitis in the central Ethiopian highlands.

1.2 Objectives

- Estimate prevalence of mastitis in the Addis Ababa milk shed based on CMT and clinical examination.
- Assess the effect of management risk factors on the occurrence of mastitis in the Addis Ababa milk shed.
- Quantify economic impact of mastitis in the respective production sub-systems of the Addis Ababa milk shed

1.3 Specific objectives

- Estimate prevalence of sub-clinical and clinical mastitis at farm level, herd size level, production sub-system level.
- Summarize management variables in the study farms in a categorical order.
- Establish association of management related factors with the occurrence of mastitis
- Identify the factors that determine mastitis losses and estimate these losses.
- Evaluate the success of a mastitis control strategy in use

2 LITERATURE REVIEW

2.1 General

Mastitis, inflammation of the mammary gland, can be caused by physical or chemical agents but the majority of cases are infectious and are caused by a variety of microorganisms, mostly bacteria, which gain access to the interior of the mammary glands through the teat canal (Quinn *et al.*, 1994). Initially, the small numbers of somatic cells in the milk that are normally present attempt to resolve this intramammary infection immediately. Both bacteria and leukocytes in the infected quarters release products, many of which are chemoattractants for the leukocytes. In response, neutrophils move rapidly from the blood stream into milk in order to fight the infection. This constitutes the inflammatory response, which may go unnoticed in the form of sub-clinical mastitis, or it may be severe enough to be classified as clinical mastitis (Suriyasathaporn *et al.*, 2000). If the bacteria are contained/destroyed, recruitment of neutrophils into the gland cases and only a mild inflammatory episode will be required to restore health in the gland. Occasionally, the innate defense mechanisms of the infected mammary gland lose the battle with bacteria and bacteria multiply. This leads to a prolonged immune response within the mammary gland. Various cell types in the udder produce abundant soluble factors, such as cytokines, which eventually cause the clinical signs of mastitis characterized by physical, chemical, and usually bacteriological changes in the milk and by pathological changes in the mammary tissue (Suriyasathaporn *et al.*, 2000). Hence, the udder inflammatory responses to intramammary infection can result in an absence or a presence of clinical signs. Additionally, there may be clinical cases of mastitis in which no pathogens can be detected, usually defined as bacteriologically negative or aseptic mastitis (Radostitis *et al.*, 2000). Radostitis *et al.*, (2000) have classified clinical forms of mastitis according to severity and duration.

Severity is characterized as:

- Peracute: severe inflammation with swelling, heat and pain of the quarter, with a marked systemic reaction, which may be fatal
- Acute: severe inflammation without a marked systemic reaction
- Subacute: mild inflammation with persistent abnormality of the milk

- Sub-clinical when there is evidence of inflammation, e.g. high somatic cell counts (SCC) in the milk without any visible abnormality of the milk or udder.

Duration is characterized as:

- Short-term clinical or sub-clinical (as in coliform)
- Recurrent clinical (as in *Staphylococcus aureus* and *Streptococcus dysagalactiae*)
- Persistent clinical or sub-clinical (as in *Strep. agalactiae*)

Clinical manifestations include abnormalities of secretion, size, consistency and increased temperature of the mammary glands and, frequently, a systemic involvement (Radostitis *et al.*, 2000).

2.2 Etiology

2.2.1 Mastitis causing pathogens

Reports indicate that approximately 137 microbes are incriminated as etiological agents (subspecies and serovars) of mastitis and have been isolated from bovine udders (Watts, 1988). The most common isolates from bovine and other species' mastitic milk are *Staphylococcus aureus*, streptococci and members of the enterobacteriaceae (Quinn *et al.*, 1994). The pathogens have been classified etiologically into two groups; namely, contagious and environmental pathogens based on their distinct characteristics of distribution and interaction with the teat and teat duct (Calvinho *et al.*, 1998). Within the two groups, there are 2 other sub-divisions as major and minor pathogens.

2.2.1.1 Major pathogens

Major pathogens mostly cause clinical mastitis. Under major mastitis pathogens there are 2 groups, the contagious and environmental pathogens. The contagious pathogens include *Streptococcus agalactiae*, *Staphylococcus aureus*, and *Mycoplasma bovis*. Contagious mastitis pathogens live and multiply on and in the cow's mammary gland and are spread from animal to animal primarily during milking (Calvinho *et al.*, 1998). Infections due to contagious major

pathogens tend to be chronic and sub-clinical but with periodic clinical episodes (Fox and Gay, 1993). The environmental pathogens classified as major agents consist of environmentals *streptococci, coliforms and enterococci*. The environmental pathogens are those whose primary reservoir is the environment where cows live and not the infected mammary gland (Smith and Hogan, 1993). Although new infections by environmental pathogens can occur at milking, primary exposure appears to be between milkings. Other environmental pathogens in the sub-group include *Proteus, Yeasts, Prototheca* spp and *Nocardia* spp which are of opportunistic nature (Watts, 1988). Individual cases or sporadic outbreaks of mastitis may be caused by *Pseudomonas* spp., *Arcanobacter pyogenes*, *Serratia* spp., or other unusual pathogens (Radostitis *et al.*, 2000). About 60-70% of environmental pathogen infections exist for less than 30 days. Mastitis caused by the major pathogens results in the greatest compositional changes of milk, including increases in somatic cell counts, and has the most economic impact of all causative organisms (Radostitis *et al.*, 2000).

2.2.1.2 Minor pathogens

Infections by minor pathogens cause only moderate inflammation with somatic cell counts exceeding those of uninfected glands by only two-to-threefold. Marked compositional changes in milk, or dramatic decreases in milk yield occur following udder infection by minor pathogens (Radostitis *et al.*, 2000). The agents simply colonize the teat streak canal but do not cause a clinical disease. Minor pathogens are also classified as contagious and environmental. Coagulase negative staphylococci (CNS) consisting of a variety of *Staphylococcus* spp. and *Corynebacterium bovis* are contagious pathogens (Harmon and Langlois, 1986; Radostitis *et al.*, 2000). Minor pathogens are responsible for a high somatic cell count but at the same time increasing the resistance of the udder to invasion by the major pathogens (Rainard and Poutrel, 1988; Nickerson and Boddie, 1994). This is due to elevated SCC or an anti-microbial-like substance secreted by CNS that inhibits growth of *Staphylococcus aureus*. There is also evidence of secretion of a factor that is inhibitory to growth and haemolytic patterns of *S. aureus*. However, quarters infected by CNS and *Corynebacterium bovis* are susceptible to *Streptococcus agalactiae* (Nickerson and Boddie, 1994). From this, the role of competitive inhibition in mastitis appears to be an area that needs to be explored.

2.3 Epidemiology

2.3.1 General

Mastitis is a worldwide problem and affects dairy cows and other species like lactating ewes, does, sows/gilts, wild ruminants, mares, bitches, camels, and queens. Mastitis is a multifactorial disease and results when management and environmental factors interact to increase exposure, to reduce udder resistance and to aid deposition of organisms into the teat canal (Philpot, 1984). However, many researchers on the disease complex have only restricted the disease causation to microbial infection, ignoring the other important epidemiological players like environmental and managerial factors (Radostits *et al.*, 1994). Most studies and surveys conducted the world over have concentrated on the determinants (etiology) of disease rather than the effects of diseases as determinants of production (James, 1984). For mastitis, both are important.

Mastitis is very difficult to eradicate, but its control is feasible, practical and can greatly reduce economic losses that are associated with it (Radostits and Blood, 1985). Control measures such as paying special attention to milking technique, housing, avoidance of teat injury, disinfectant teat cleaning before and after milking, and dry period treatment with antibiotics have been reported to greatly reduce the occurrence of mastitis (Radostits *et al.*, 2000). In most countries, surveys in dairy herds indicate that the prevalence of infection (mastitis) is about 50% in cows and with a quarter infection rate of about 25% (Radostits *et al.*, 2000). The average annual incidence rate of mastitis, calculated as the number of clinical quarter cases per 100 cows at risk per year including the dry period in individual herds is 10-12% in most herds but higher values, ranging from 16-65%, occur in some herds (Bartlett *et al.*, 1992). The greatest risk of acquiring clinical mastitis occurs early in lactation, usually in the first 50 days (Bartlett *et al.*, 1992). Case fatality rates vary widely depending on the identity of the causative agent for example, *Streptococcus agalactiae* mastitis is not a fatal disease, but peracute staphylococcal mastitis in a recently calved cow may be fatal (Radostits *et al.*, 2000).

2.3.2 Mastitis in Ethiopia and the region

The epidemiology of mastitis in Ethiopian dairy herds is poorly understood as very few studies have been documented addressing this aspect. The Faculty of Veterinary Medicine of Addis Ababa University has conducted a lot of undocumented work concerning the mastitis complex. A

very restricted study by Biru (1989) revealed a combined prevalence of 67.4% of both clinical and sub-clinical mastitis in a herd of 43 cows. International Livestock Center for Africa, (ILCA) conducted California Mastitis test (CMT) screening on 725 dairy herds with a total of 2735 dairy cows and established an overall prevalence of 1.2% of clinical mastitis and 37.6% of sub-clinical mastitis at cow level (ILCA, 1994). Kassa *et al.*, (1999), carried out a mastitis survey on a similar number of herds like ILCA but with 2681 dairy cows and established that prevalence of clinical mastitis, nonfunctional or blocked quarters and sub-clinical mastitis to be 1.2%, 3.8% and 38.9% respectively. In another study conducted on small holder farms in Wondogenet area of Ethiopia, 39% of the cows had mastitis with a quarter infection rate of 16% (Abdella, 1996). Hussein *et al.*, (1997), in a study conducted at the Adamitulu and Holetta livestock research centers, reported a clinical and sub-clinical mastitis prevalence of 5.3% and 19% respectively. Bishi, (1998) reported sub-clinical mastitis prevalence of 34.3% and clinical mastitis 5.5% at cow level. The prevalence was 30.6% and 27.9% for sub-clinical in large and small-scale farms and 5.7% in large and 4.4% in small-scale farms for clinical mastitis at cow level. Frese, (1999) reported an overall mastitis prevalence of 44.5% based on CMT with microbiological prevalence of 34.6% and 2.8% for sub-clinical and clinical mastitis, respectively

The neighbouring countries in the region, just like Ethiopia, have equally a poorly understood epidemiology of mastitis. Omore *et al.*, (1996), estimated the prevalence of sub-clinical mastitis to be 71% and SCC up to 1,992, 000, median being 620, 000 cells/ml of milk on small holder dairy farms in Kenya. The same study reported a clinical mastitis incidence of 13.3% per annum. Other studies (Lauerman *et al.*, 1973; Hamir *et al.*, 1978; Ngatia, (1988) estimated the prevalence of sub-clinical mastitis to be 49 %, 48% and 55% respectively. Ngatia, (1988) also studied types of production systems employed, herd composition, hygiene and mastitis control and preventive methods practiced. The annual incidence of clinical mastitis in Kenya was estimated at 2.5 – 3% (Hamir *et al.*, 1978) but could be as high as 25% in some areas (Anon, 1980). Bovine mastitis was also reported as the most frequently encountered disease in Kenyan dairy herds (Hamir *et al.*, 1978) but the types of mastitis, the severity and duration of disease and the major risk factors needed elaboration.

A study conducted on prevalence of bovine sub-clinical mastitis in Malawi established a prevalence of 63% on cow and 28% on quarter basis (Klastrup and Halliwell, 1977). Sokoine University of Agriculture in Tanzania conducted a much more recent study in the region on the

prevalence of mastitis on small holder and large-scale farms and established that 62% and 4% of the cows had sub-clinical and clinical mastitis respectively (Shem *et al.*, 2001). The same study revealed that SCC were greater than 500, 000 cells/ml of milk.

2.4 Risk factors associated with mastitis

As with most infectious diseases, the occurrence of mastitis depends on three components: exposure to microbes, cow defense mechanisms, and environmental and management factors (Suriyasathaporn *et al.*, 2000).

2.4.1 Animal (host) risk factors

2.4.1.1 Age and parity

The prevalence of infected quarters increases with age, peaking at 7 years (Schukken *et al.*, 1989). Older cows, especially after four lactations are more susceptible to mastitis (Quinn *et al.*, 1994). It is postulated that young animals have a decreased susceptibility through a more effective host defense mechanism (Dulin *et al.*, 1988).

2.4.1.2 Stage of lactation

Most new infections occur during the early part of the dry period and in the first 2 months of lactation, especially with environmental pathogens (Smith and Hogan, 1993). Schukken *et al.*, (1990) reported that the first month of lactation is the most sensitive period for mastitis risk in the cow even in the well-managed herds. This is because of increased stress as a result of depressed immunity due to metabolic changes and peak milk production during the early days following parturition. Prevalence of sub-clinical mastitis increases as stage of lactation progresses (Radostits *et al.*, 1994). Other less important risk factors include other concurrent diseases, previous mastitis history, and pre-existing intra-mammary infections.

2.4.1.3 Breed

Genetic predisposition to mastitis could be related to factors such as teat shape, sphincter tone, anatomy of the teat canal and susceptibility to weakening of the suspensory ligament (pendulous udder) (Quinn *et al.*, 1994). Teat end is important in mastitis defense for its role in preventing entry of pathogens into the mammary gland (Schukken *et al.*, 1989). Teat-end-to-floor distance is also a risk factor for clinical mastitis. Heritability estimates of teat-end-to-floor distance or udder height range from 0.2 - 0.7, which could be a consideration in the selection indices of bulls (Radostitis *et al.*, 2000). Several physical and chemical properties of the streak canal constitute the first line of defense. Once inside the teat cistern, pathogens encounter a group of non-specific bacteriostatic and bacteriocidal factors (Schukken *et al.*, 1989).

Genotypes favorable for milk production are more susceptible to mastitis than those, which are not favorable for milk production (Oltenacu and Ekesbo, 1994). Milk yield has been shown to be positively correlated with the risk of clinical mastitis (Schukken *et al.*, 1990). Pure Holsteins producing up to 20-25 kg of milk per milking and their crosses with Borana and Arsi producing 15-20 kg of milk per milking are at higher risk of contracting mastitis than the local breed cows with a low milk production potential (Girma, 2001). This assertion could as well be disputed on grounds that differences in management rather than genetic factors could lead to more susceptibility to mastitis (Radostits *et al.*, 2000). Information that is valid on comparisons between breeds in the tropical set ups is lacking and needs elaboration.

A variety of morphological, physiological, and immunological factors contribute to a cow's resistance or susceptibility to mastitis, and each of these factors is influenced to some extent by heredity (Radostits *et al.*, 2000). The immunological factors such as the level of IgA, IgG1, lactoferrin, lysozyme and phagocytes in the mammary gland are direct products of genes and have a genetic basis (Quinn *et al.*, 1994). The production of keratin in the streak canal and the physical and biochemical characteristics of keratin are important contributors to mastitis resistance (Radostits *et al.*, 2000).

2.4.1.4 Presence of lesions on the teats

Lesions when present on the teats may predispose to inadequate milking or may harbour mastitis-producing bacteria hence increasing the risk of infection (Quinn *et al.*, 1994).

2.4.1.5 Nutritional status

Nutritional programs which are associated with imbalances in anion-to cation in the dry cow diets predisposes the cows to periparturient hypocalcaemia which in turn increases the risk of intra-mammary infections, IMI (Radostits *et al.*, 1994). Vitamins A and E and selenium may be involved in the resistance to certain types of mastitis (Erskine *et al.*, 1987). Early reports found that supplementation with antioxidants such as selenium and vitamins A and E had a beneficial effect on udder health in dairy cattle by decreasing the incidence of clinical mastitis (Radostits *et al.*, 2000).

2.4.1.6 Prevalence of infection

The greater the prevalence of mastitis in the herd, the greater the new infection rate and duration of infection. This is a major feature in herds with high levels of contagious pathogens and for which no strict hygienic measures are observed as the infection spreads during milking (Calvinho *et al.*, 1998). Environmental pathogens on the other hand, may be a big problem in herds that have successfully controlled contagious pathogens if the housing conditions and associated characteristics like bedding are of low hygienic standard (Smith and Hogan, 1993).

2.4.1.7 Low somatic cell count

Since leukocytes in the udder are present to resolve the IMI, once an intramammary challenge occurs, a very low somatic cell count may predispose cows to a higher risk of clinical mastitis especially in high yielding cows (Schukken *et al.*, 1990). In an experimental study, it was shown that factors such as low peripheral leukocyte count and low SCC were associated with a more severe mastitis response (van Werven *et al.*, 1999).

2.4.1.8 Body condition score (BCS)

It was demonstrated that cows with low BCS (1 to 1.75) have higher risks of getting clinical mastitis in comparison to cows with BCS 3 to 3.75. BCS is a tool for estimating energy balance status. Cows with negative energy balance from feed restriction had a higher severity of experimental *E. coli* mastitis (Suriyasathaporn *et al.*, 2000). Cows with a low BCS are more

likely to be ketotic. In an experimental demonstration, it was shown that ketone bodies decreased the chemotactic function of the leukocytes (Suriyasathaporn *et al.*, 2000) and therefore may put a cow at risk for severe clinical mastitis.

2.4.2 Environmental and managerial risk factors

2.4.2.1 Quality and management of housing

The quality and management of housing for dairy cattle has a major influence on the types of mastitis pathogens which can infect the mammary gland and the degree of infection pressure (Radostitis *et al.*, 2000). Management and design of a housing system influence the prevalence of intra-mammary infection and the incidence of clinical mastitis. Any housing factor or management system that allows cows to become dirty or damage teats, or which causes overcrowding will result in an increase in clinical mastitis (Radostitis *et al.*, 2000). Ventilation is a critical factor to maintenance of dry conditions. Very old structures frequently have very poor ventilation, which is a major risk factor for mastitis (Smith and Hogan, 1993). A design that has free stalls built against outside walls or any solid wall should be avoided as it serves as a risk factor of mastitis (Smith and Hogan, 1993). A house which does not allow free movement of urine and other wastes causes the accumulations of urine that harbour an assortment of pathogens mostly of the environmental type which serves as a reservoir for intramammary infection. Type of bedding has a great influence on the infection rates of mastitis. Sand and other inorganic materials have low moisture content and contain few nutrients for bacteria to utilize as compared to organic materials like straw, saw dust, recycled manure and paper (Smith and Hogan, 1993). Majorities of the bacteria in the bedding are environmental bacteria, for example straw tends to have highest streptococcal counts, while sawdust and recycled manure have highest coliform counts in comparison among these bedding materials (Smith and Hogan, 1993).

2.4.2.2 Herd size

The size of the milking herd may positively be associated with an increased incidence of clinical mastitis because it is more difficult to control contagious mastitis in a herd with a greater prevalence of infection and a larger number of cow-to-cow contacts. As herd size increases, manure disposal and sanitation problems increase exposure to environmental pathogens (Bartlett *et al.*, 1992).

2.4.2.3 Milking practices

Contamination of the udder immediately before and after milking is an important risk factor for mastitis (Peeler *et al.*, 2000). Milking presents an opportunity for any pathogen present on the udder to penetrate the teat canal. Confinement in the yard after milking has been recommended because it encourages cows to remain standing while the teat ducts are patent and thus more vulnerable to penetration by mastitis pathogens present elsewhere (Blowey and Edmondson, 1995).

Udder preparation both before and after milking influences the rate of mastitis in a given herd. It has been established that farmers who use a common cloth/sponge for drying teats after cleaning the udder have a greater odds of having a high prevalence on infection than herds using individual paper towels (Dargent-Molina *et al.*, 1988). Wet teats and udders are a risk factor for increased SCC (Radostits *et al.*, 2000). Water is helpful because it is necessary for effective cleaning when the teats and udder are dirty, but at the same time it also can carry bacteria down the teat from a wet udder and thus contaminates milk during milking (Bushnell, 1984). Drying off procedure at the end of a lactation period and an active policy on drying off treatment are very important as they can reduce the level of intra-mammary infection especially with the coliforms (Thirapatsakun, 1989).

Increased number of person-hours spent milking per cow is said to be associated with a higher rate of clinical mastitis (Bartlett *et al.*, 1992). Milking machines whose teat liners are not changed after every 2500 milkings serve as a risk factor of mastitis. This act of changing liners may be cause or effect: farmers may change liners frequently because of a mastitis problem or frequent liner change may potentially damage teats (Peeler *et al.*, 2000). Further investigation is required to clarify the importance of milking machine management.

Predipping helps reduce environmental mastitis by as much as 50% in some herds, although this reduction is not observed in all herds. Failure of predipping to control environmental mastitis in all herds likely reflects the complex epidemiology of environmental pathogens (Smith and Hogan, 1993). Stripping foremilk before cluster attachment in those farms that practice machine milking and also before hand milking has been shown to be a risk factor of mastitis especially of the contagious type (*Staphylococcus aureus* mastitis). Foremilking could expose other cows at risk to mastitis pathogens in the stripped milk, in the same way as leaked milk, or through

increased contamination of the cow's teats from the dairyman's hands. The reduction in the incidence rate of clinical mastitis attainable by ceasing this practice, as indicated by the population attributable fraction (PAF), cannot be predicted until the degree to which stripping foremilk increases transmission of mastitis pathogens in the parlor, rather than increasing detection of mild cases, is known (Peeler *et al.*, 2000).

2.4.2.4 Climatic influences

The incidence of mastitis especially in the tropics is associated with the availability of rain. Girma, (2001), established that short rains coming in the months of March and April and the long rains coming from June to August positively influenced the rate of occurrence of clinical mastitis in Ethiopia. An increase in the number of hours a cow spends out in the sun protects against environmental mastitis due to a cleansing effect by the radiation of the sun and also due to a reduced exposure period of the cow to microorganisms contained on the bedding (Smith *et al.*, 1985; Schukken *et al.*, 1989).

2.4.2.5 Feeding after milking

Feeding a cow after it is milked is necessary since the cow remains standing (while feeding). Lying down immediately after milking can be a risk factor as the teats are still open. This gives the teats time to switch back to their normal anatomical shape otherwise the risk of acquiring environmental pathogens is very high (Radostitis *et al.*, 2000; Peeler *et al.*, 2000).

2.4.2.6 Exercise

Gustafson, (1993) reported that lack of exercise could affect the general health of cows. This study showed that lack of exercise in tied dairy cows resulted in increased frequency of calving related problems, mastitis and lameness compared to those cows exercising.

2.4.2.7 Traumatic influences

External trauma such as that arising from rough, tough treatment most frequently met on cows as they are driven into the milking parlour could be a risk factor to mastitis. This could be as a result

of animals suffering bruises on the teats or running through muddy and unhygienic stretches as the cows rush into the milking shed. It can predispose them to environmental pathogens (Quinn *et al.*, 1994).

2.4.3 Pathogen factors

2.4.3.1 Viability of pathogen

The ability of the pathogen to survive in the cow's immediate environment i.e. its resistance to environmental influences including cleaning and disinfection procedures, is a characteristic of each pathogen (Quinn *et al.*, 1994). Contagious mastitis pathogens are more susceptible to disinfection than environmental mastitis ones. Teat dipping, therefore checks on the contagious pathogens but has very little effect on the environmental pathogens (Fox and Gay, 1993).

2.4.3.2 Virulence factors

Since the epidermis is the first line of defense against mastitis pathogens, colonization of the skin and streak canal and the attachment to epithelial cell surfaces enable the pathogens to breach this first line of defense. The next line of intramammary defense is the neutrophil (Polymorphonuclear cells, PMN). Virulence factors that will interfere with PMN function enable a pathogen to develop acute or chronic clinical infections. *Staphylococcus aureus* possesses a variety of such factors. Protein A produced by some staphylococcus varieties binds to the fragment constant (Fc) portion of the immunoglobulin, impairing oposonization and, thereby, PMN phagocytosis (Fox and Gay, 1993). The influence of bacterial virulence factors depends, among other things, on stage of lactation and severity of infection of the intra-mammary infection and the effects elicited by the virulence factors on bovine mammary tissue (Radostitis *et al.*, 2000). The colonizing ability and potential of bacteria to adhere to mammary epithelium and initiate mastitis, is a major characteristic of many bacterial causes of mastitis e.g. *S. aureus*. The other important virulence factor is ability to elaborate toxins as seen in mastitis due to *E. coli* and *S. aureus*.

2.4.3.3 Invasive capabilities

Regarding the degree of invasiveness, for example, streptococci cause little pathological change to secretory cells within the udder but staphylococci initiate degenerative changes in the udder (Quinn *et al.*, 1994).

2.4.3.4 Resistance

Ability to resist phagoctyosis and antibacterial substances in the udder, including resistance to antibiotics will make the pathogen to be available at all times to cause infection (Quinn *et al.*, 1994).

2.5 Diagnosis of mastitis

2.5.1 Screening tests

2.5.1.1 Clinical examination and use of strip cup

Clinical examination is the most obvious and important diagnostic procedure in the detection of clinical mastitis. The clinical signs detected during clinical mastitis include swollen gland, abnormal milk (watery, with clots/flakes, blood tinged), pain on palpation and heat (Radostitis *et al.*, 2000). A strip cup on the other hand is also a very valuable tool for the detection of clinical cases. Sometime farmers with little technical knowledge about presentation of the clinical signs of mastitis and without a strip cup may strip the first few squirts of milk on the floor, clean gum boots or on any black surfaced background in order to detect any abnormality in milk, as an indicator of clinical mastitis (Quinn *et al.*, 1994).

2.5.1.2 Cell counts in milk

Direct Microscopic count (Modified Breed's smear)

This method counts leukocytes in the milk directly. Normally a volume of 0.01ml of milk is spread over a microscope slide, defatted and then stained by methylene–blue based stain. The

microscope is calibrated and then leukocytes are counted on about 50 fields to estimate the number per ml of milk. If there is a large bacterial load in the milk these may also be seen in the stained smear.

Individual indirect cell counts using CMT

This is the test of choice for field and also laboratory people and is based on the quantity of deoxyribonucleic acid (DNA) in the milk and hence the number of leukocytes and other cells present. A squirt of milk from each quarter of the udder is placed in each of the four shallow wells (cups) in the CMT paddle. An equal amount of commercial CMT reagent or 14% teepol (shell) is added to each cup. A gentle circular motion is applied to the mixture in a horizontal plane and a positive gelling reaction occurs in a few seconds with the positive samples. The consistency of the gel (i.e. the heavier the gel the higher the somatic cells in the milk and vice versa) is indicative of the leukocyte count (Schalm *et al.*, 1971; Quinn *et al.*, 1994). CMT is a very useful method though quite labor intensive and qualitative in nature (Radostits *et al.*, 1994). It can be quite rapid for the determination of mastitis status at quarter level. CMT is used as adjunct to SCC and composite culture information for the determination of quarter level status (Radostits *et al.*, 2000). Other tests dependent on the development of gels are Brabant, Winconsin and NAGase mastitis tests. The NAGase mastitis test is easily automated and is based on a cell-associated enzyme in milk, N–acetyl-D-glucosaminidase. High levels of the enzyme indicate a high cell count (Quinn *et al.*, 1994). Adenosine triphosphate (ATP) is also another screening test that can be used to detect mastitis.

Electronic somatic cell counters

Electronic somatic cell counting of suspicious milk is another screening test for the diagnosis of mastitis based on cell counts. A Coulter Milk Cell Counter does the somatic cell counts as the particles flow through an electric field.

2.5.1.3 Indirect chemical tests to detect mastitis

- Some tests are based on the increase in sodium and chloride ions in milk and consequently an increase in electrical conductivity of the milk.

• The serum albumin concentration in the milk increases if epithelial damage is present. A radial immunodiffusion test has been developed based on this fact (Quinn *et al.*, 1994).

• An anti-trypsin test measures the trypsin-inhibitor capacity of the milk. Anti-trypsin activity tends to be naturally high at the beginning of lactation due to colostrum levels, but later in a lactation the values are high only if serum anti-trypsin has leaked through damaged mammary epithelium. This method lends itself to automation.

2.5.2 Microbiological diagnosis

Microbiological diagnosis is sought in case of elevated Somatic cell counts as given by CMT/Direct or electronic somatic cell counts are done to differentiate the microorganisms involved and also to act as a confirmatory test for the CMT results. Microbiological diagnosis begins with aseptic collection of milk from quarters that are having elevated cell counts on direct cell counts and or electronic counters, CMT positive or with obvious abnormal milk secretions and can be one of the following:

2.5.2.1 Direct microscopy

The collected milk sample can be centrifuged and a stained smear made from the deposit. A Gram stain is used routinely to detect Gram-positive pathogens such as staphylococci, streptococci, Bacillus spp., and also Yeasts such as *Candida albicans* that stain deeply by crystal violet. An Modified Ziehl Neelsen-stained smear can be made if *Nocardia asteroides* is suspected and A Ziehl Neelsen- stained smear for the rare cases when bacteria such as *Mycobacterium fortuitum* or *M. bovis* are suspected (Quinn *et al.*, 1994).

2.5.2.2 Culture

The incriminated milk sample is cultured on ox or sheep blood agar, which supports growth of most mastitis pathogens. *Candida albicans* and *Aspergillus fumigatus* are able to grow on blood agar at 37 °C in 2-3 days if there is no competition from the faster-growing bacteria. A MacConkey agar plate is streaked in parallel to detect *Enterococcus faecalis* and any Gram-negative bacteria that is able to grow on the medium. If streptococci are suspected then the milk sample is plated on Edward's medium, which is selective and also acts as an indicator medium

for haemolysis and for the hydrolysis of esculin. In cases where fungal pathogens are highly suspected then it is advisable to culture the milk sample on Sabouraud dextrose agar.
Isolation is then done based on colonial morphologies and other microbiological and biochemical characteristics of the involved microorganisms (Quinn *et al.*, 1994). The anti- microbial sensitivity tests are conducted to give guidance to selection of proper drugs as this will help reduce chances of development of drug resistance (NMC, 1996).

2.5.3 pH monitoring techniques

Also in use are techniques such as pH detection that depends on the acidity/alkalinity of the milk samples. Mastitic milk and also colostrum are alkaline in nature i.e. 7.4 which is slightly above the physiological pH of 7.2, whereas normal milk's pH is approximately 6.5 - 6.7 (Radostits *et al.*, 2000). pH papers and pH meters are used to detect any changes. Acid and alcohol tests are used to check for the soundness of the milk. Any milk sample whose pH is beyond that of normal milk is considered spoilt or mastitic. Resazurin dye tests and also boiling will give an indication of the state of the milk and since mastitis has a direct effect on the shelf-life of milk, then these can be reliable tests in mastitis diagnosis.

2.5.4 Advanced mastitis diagnostic technologies

Hand instruments are now available in addition to automated electric conductivity (EC) by in-line electrodes within computerized milking parlors. The system makes it possible to monitor new infections at quarter level on a continuous milking-by-milking basis. Other recent technologies engage immunological tests for detection of antibodies to specific mastitis pathogens. Since majority of these techniques use preserved milk samples, dairy herd improvement (DHI) laboratories could offer specific pathogen screening test programs as an adjunct service (Radostits *et al.*, 2000). Other screening tests do exist and are in use in industrialised countries such as electrical conductivity, N-Acetyl-B-Glucosaminidase test (NAGase), specific antibody test, Bovine serum albumin and antitrypsin concentration assays. Screening tests do not establish the infecting agents but simply detect the inflammatory response following infection (Philpot and Nickerson, 1991). Several DHI centres have already commenced programs using a commercially available ELISA test for *S. aureus*.

2.6 Mastitis prevention and control

Mastitis like all other diseases occurs as a result of the complex interaction of the host, agent and the environment (the epidemiological triad). For any control program to be effective, it should be geared towards correcting mastitis problems associated with these three factors. Radostits *et al.*, (1994) have given the following essential components of a comprehensive udder health program:

- Employ proper milking management methods
- Proper maintenance and use of milking equipment
- Dry cow management/therapy
- Appropriate therapy during lactation
- Cull chronically infected cows
- Maintenance of a clean environment
- Good record keeping
- Monitoring udder health status
- Periodic review of the udder health program
- Setting goals for the udder health status

Philpot and Nickerson, (1991) give the following as steps to udder health management geared towards the fulfillment of epidemiological principles:

1). Elimination of existing infections

This is through employing proper milking practices/hygienic measures (such as post milking teat dipping), dry cow therapy, treatment of clinical mastitis cases and culling all chronically infected cows. These procedures help to reduce either the rate of new infection (prevention) or the duration of infection.

2). Prevention of new infections

This employs good environmental, nutritional as well as breeding management to be able to prevent new infections.

3). Monitoring udder health

This includes regular use of cow side diagnostic tests such as the strip cup test, pH papers/meters and physical examination to detect clinical mastitis, California Mastitis Test and other tests like

direct somatic cell counts, electronic cell counts to detect sub-clinical mastitis. The cow side tests are not for identifying cows for treatment but for monitoring progress and revealing deficiencies in mastitis control programs. However, any mastitis control program should have the following qualities for it to be successful:

- Must be cost effective
- Easily adaptable to the currently used dairy management systems
- Should lead to visible success by a rapid reduction in numbers of clinical cases, and a steady improvement in the parameters used for monitoring udder health status and
- Must be within the scope of the average dairy farmer's understanding (Radostits *et al.*, 1994).

2.7 Economic losses due to mastitis

The economic effects of mastitis can be divided into three categories (Houben, 1995):

- Reduced milk receipts
- Cost of treatment; and
- Premature disposal (culling).

Very limited if any set of published data is available to quantify these effects for crossbred cows under tropical conditions. Therefore, they have to be derived from various sources such as field research, secondary data from the dairy cooperatives, government veterinary services, private veterinary practices and farmers societies and associations (Dijkhuizen and Morris, 1997).

2.7.1 Decreased milk production

Significant losses in milk production from individual cows and herds have been shown to be associated with elevated somatic cell counts; higher cell counts mean greater loss. The loss of an individual cow increases from 6-30% as cell counts increase from 100,000 to 1,600,000 cells/ml while the loss of the entire herd increases from 6-29% for cell counts from 500,000 to 1,500,000 cells/ml (Philpot and Nickerson, 1991). De Graaf and Dwinger, (1996), reported crude milk production losses per cow with sub-clinical mastitis estimated at 1.56 kg per day. Milk production loss per affected quarter due to sub-clinical mastitis was estimated to be 17.6% on average (De Graaf and Dwinger, 1996). De Graves and Fetrow, (1993), give a loss in the range of up to 10-26% per affected quarters with sub-clinical mastitis. Radostits *et al.*, (2000) estimate about the same figure (10-25%) of loss in milk yield following infection by sub-clinical mastitis.

Dobbins, (1977), estimated an absolute decrease in milk production per CMT score per quarter as follows: CMT score negative as 0 kg loss, Trace as 0.27 kg loss, 1 as 0.991 kg loss, 2 as 1.76 kg loss and CMT score 3 as 2.61 kg loss in milk yield per quarter. Most estimates indicate that on average an affected quarter results in a 30% reduction in productivity and an affected cow is estimated to lose 15% of its production for the lactation following infection by sub-clinical mastitis (Radostits *et al.*, 2000). Schepers and Dijkhuizen, (1991) reported that 70% of total losses due to mastitis arise from decreased milk production. Singh and Singh, (1994) estimated that up to 68% of the total losses of mastitis in dairy cows in India are as a result of sub-clinical mastitis (reduced yield). The loss in production by an affected quarter following sub-clinical mastitis may be largely compensated by increased production in the other quarters so that the net loss to the cow may be less than expected (Radostits *et al.*, 2000). As concerns clinical mastitis, milk yield decreases substantially and losses are much larger in early than late lactation (Radostits *et al.*, 2000). Cows with multiple lactations experience greater milk production losses as compared to first lactation cows. Singh and Singh, (1994) estimate a 50% reduction in the milk yield of a cow suffering from clinical mastitis. Clinically affected quarters may not completely recover milk production in subsequent lactations but the carry-over losses are not as large as the losses from acute mastitis (Radostits *et al.*, 2000). Blowey, (1986) assumed no decrease in milk production for mild mastitis, but a 10% decrease in the remaining lactation after acute mastitis.

Somatic cell count also reflects the changes, which occur in the composition of milk. As the degree of inflammation increases, the chemical composition of milk more closely approaches that of blood because the components filter from blood circulation into the mammary gland. The yield of total solids, butter fat, solids-non-fat, casein and lactose is reduced substantially while total protein changes only slightly. Lipase, immunoglobulins, serum albumins, sodium chloride increase substantially (Philpot and Nickerson, 1991).

2.7.2 Losses due to culling

Culling due to mastitis is a result of intra-mammary infections that fail to cure when the bacteria causing the disease fail to respond to the commonly used antibiotics (resistance development). The proportion of the culls is related to the bacteria infecting the udder. For example, when *Arkanobacter pyogenes* causes mastitis, then a large number (> 80%) of affected cows are likely to be culled as compared to that by other agents. Coliform and staphylococcal mastitis substantially contribute to a relatively bigger proportion since these agents cause peracute and

gangreous form of mastitis (Dijkhuizen and Morris, 1997). Culling causes 2 kinds of losses, namely a reduced slaughter value of a cow but an increased replacement cost and lost production time following premature removal from the herd before the animal in question attains its optimal production age (Singh and Singh, 1994; Radostits *et al.*, 2000). Replacement costs following culling are estimated to contribute about 14% of the total mastitis losses (Schepers and Dijkhuizen, 1991). In most developing countries, farmers do not cull their animals suffering from mastitis because they are ignorant of the economic losses arising from the presence of this disease in their herds. This is revealed in a study to establish economic losses due to mastitis in India where there was no data available on culling and so the authors had to assume a 1% culling rate (Singh and Singh, 1994). As a result of this existing ignorance among the farmers especially in most sub-Saharan African countries on the immense economic losses due to failure to cull chronically infected cows, the information available on the losses is scanty, or at worst non-existent. In organized dairy farms, particularly as it is the case for industrialized nations, the carrier animals are culled if the mastitis problem continues or the affected quarter goes blind. Many dairy managers will cull the chronically infected cows which do not respond to therapy as the only option to clinically manage mastitis, especially control (Radostits *et al.*, 2000).

2.7.3 Treatment costs

In Sub-Sahara Africa (SSA), Asia and in the majority of Latin American countries, mastitis therapy is restricted to the clinical forms of the disease most of the time with very few elite farmers treating cows with sub-clinical mastitis. For each case of clinical mastitis in a herd population there will usually be 15 to 40 sub-clinical cases (Philpot and Nickerson, 1991). This points out how much is lost by farmers not being able to recognize and institute therapeutic measures on the sub-clinical cases. Blosser, (1979), asserts that most of the dairymen are not aware of the existence of the sub-clinical mastitis in their herds as the symptoms are not visually evident to them. Hence, much of the treatment costs incurred on many farms are due to clinical mastitis with a negligible figure attributed to sub-clinical mastitis. The failure to attend to both forms of the disease is due to lack of proper diagnostic kits, lack of skilled personnel and limited funds to purchase necessary drugs for treatment of both sub-clinical and clinical mastitis. Unlike the industrialized countries where treatment costs constitutes the cost of drugs, veterinary charges, labour costs and withdrawal of milk for at least 3 days following treatment (Dobbins, 1977), the situation in SSA is different as almost no farmer withdraws milk after treating the mastitic cows. A review of economics of mastitis by Schepers and Dijkhuizen, (1991) estimated

that 8% of total losses are due to drug costs and veterinary fees, and another 8% being due to discarded milk following treatment following a mandatory withdrawal period of 72 hours. Blowey, (1986) unlike his fellow investigators in the developed countries, asserted that instead of discarding milk following treatment for 3 days, the milk can be fed to calves and dogs and in the process avoid the loss (be given a salvage value). A part from the milk discarded following antibiotic treatment of mastitic cows, there is also unfavorable changes which occur following about of mastitis and also a decrease in the hygienic quality of the milk which necessitates such milk to be discarded for public health reasons (Thirapatsakun, 1989). Cost of drugs takes the biggest chunk of the treatment cost as there are different commercial intra-mammary preparations sold at the prevailing market prices. On average intra-mammary infusions are administered at 12/24 hourly interval for 3 consecutive days. This means that total cost of medicines equals cost of 3 treatments multiplied by the number of affected quarters/cow (Singh and Singh, 1994).

3 MATERIALS AND METHODS

3.1 Study Area

The study was conducted in the urban, peri-urban dairy farms and also in dairy farms in secondary towns production subsystems of the Addis Ababa milk shed (ILCA, 1994). This study was conducted from February to September 2001. The urban production subsystem was comprised of dairy farms located within Addis Ababa. The peri-urban production subsystem included farms in Kaliti, Sebeta and Holleta. The dairy farms in secondary towns were those located in Debre Zeit. Overall, the study area is located between 1880-2560m above sea level. It has an average annual temperature of about 21 °C, (a few other places like Debre Zeit having even extreme temperatures of about 30 °C and above in the dry season months of September to May), and an annual bimodal rainfall estimated at 1800mm. The estimated human population is 3 million (CSA, 1998).

3.2 Study population

The farms described in the production subsystems (sub-section 3.1) were included in this study. Most of the farms had predominantly crossbred cows, Friesian–local breed crosses with very high level exotic blood. Preliminary work by ILCA, (1994) estimated dairy farmers in the study area to be 10,000 and about 40,000 dairy animals. These figures were applied in this study just as rough estimates because many changes (other people joining/some living the enterprise and also dynamism in animal and dairymen) have taken place since then.

3.3 Sampling method/selection of farms/herds

A total of 51 dairy farms were selected by stratified random sampling from the study based on the sampling frame established by ILCA (ILCA, 1994). The farms selected are as shown below:
Urban dairy (UD) subsystem in the city of Addis Ababa, 9 farms,
Peri-urban diary (PD) subsystem in Sebeta, Kaliti and Holleta, 15 farms and
Dairy herds in secondary towns (DHIST), 27 farms (Table 1)

Table 1 Summary of the selected farms in the study areas

Production System	Number of Farms	Lactating cow population
DHIST (large)	5	109
DHIST (small)	22	55
Urban (large)	1	0
Urban (small)	8	20
Peri-urban (large)	7	8
Peri-urban (small)	8	171
Total	**51**	**363**

Farms of similar characteristics replaced the selected farms for one reason or another as and when necessary in a resampling procedure done during the course of the study. The main reason for replacement was refusal to cooperate in the study after the initial sampling had been done, although in some instances, the businesses had either been wound up or the animals had dried off or died. The study involved 363 lactating cows in the selected farms. This figure was determined using an assumed average mastitis prevalence of 40% across the said production subsystems as established by the previous studies conducted in the same set-up. The following formula was employed to get the target sample of milking cows above as described in Thrusfield, (1995).

$$n = \frac{1.96^2 [P_{exp} (1-P_{exp})]}{d^2}$$

In this formula, **n** is the required sample size, 1.96 is the value of Z at the 95% confidence level, P_{exp} is the expected prevalence of mastitis and d^2 the desired absolute precision level at 95% confidence level.

3.4 Study design

3.4.1 Questionnaire survey

A questionnaire was administered to all farm/herd owners or those responsible for taking care of the animals during the study period (Appendix 7.1). The management practices/risk factors/aspects considered in the questionnaire survey were grouped into four main categories:

i). Housing situation on the study farms

Information on the animal house types, floor types, roof conditions, whether or not bedding were used, space for the animals and whether or not the farms had maternity wings as separate entities.

ii). Drying off lactating cow (s)

Whether farmers practiced dry cow therapy and if so what they used, when they dried off the cows and after how long in the gestation.

iii). Milking management

Information on when the mastitic cows were milked, (whether last, first or at any given time). Whether or not the farmers did complete milking? Used teat disinfection and when this was done, i.e. whether before or after milking? What was used for disinfecting the teats? Whether udder cleaning and drying was done before/after milking or both? What was used to dry the teats, whether the use of towels was restricted to each individual cow or shared? Milkers were contracted, family members or permanently employed on the farm? Whether the milkers washed hands in between each milking.

iv). Drainage and manure handling

The data collected included the frequency of manure removal from the animal houses and where it was disposed.

Whether floors were constructed in such way so as to allow free and smooth flow of urine and other wastes from the animal houses.

3.4.2 Cross-sectional study

Prevalences of sub-clinical and clinical mastitis in the study farms were determined using the 363 cows. Clinical examination of lactating udders and California Mastitis Test were applied/used on each cow. The prevalence of clinical and sub-clinical mastitis at cow level was calculated as shown below:

$$\text{Overall prevalence of sub-clinical/clinical Mastitis} = \frac{\text{Number of cows with mastitis} \times 100\%}{\text{Total number of lactating cows examined}}$$

Quarter wise prevalence was also considered in this study as:

$$\text{Quarter wise prevalence of mastitis} = \frac{\text{Number of positive quarters} \times 100\%}{\text{Total number of quarters screened}}$$

Prevalence of blocked quarters, was expressed as a percentage of the total quarters as shown below:

$$\text{Prevalence of blind/blocked quarters} = \frac{\text{Number of quarters blind/blocked} \times 100\%}{\text{Total number of quarters in examined cows}}$$

A farm was judged small if less than 10 cows were examined, and as large when at least 10 cows were sampled (Staal and Shapiro, 1995). Presence or absence of supernumerary teats was also noted Furthermore, data on identification, parity, date of last calving (to estimate stage of lactation), leaking milk, body condition score and patency of teats were recorded.

3.4.2.1 Clinical examination/inspection of the udder

Clinical mastitis was defined at cow or quarter levels, by gross clinical signs such as udder swelling, pain on palpation, a warmer than normal udder skin (inflammatory signs) and abnormal

secretion (blood tinged milk, watery milk or milk with pus flakes and clots) from the udder or both.

The symmetry, consistency, presence/absence of supernumerary teats, presence/absence of any lesions on the teats, evidence of swelling and also congruency of the quarters were noted. Teats were also examined for their functioning status. They were categorized as either patent/functional or blocked/non-functional. Mastitis investigation was done on the functional quarters only. Blocked or non-functional quarters were simply enumerated for purposes of knowing their proportion in relation to the total quarters.

The body conditions of the study animals were scored using an ordinal scale and recorded as being of good, fair, and poor (Appendix 7.2). Clinical examination was done prior to milking to minimize exciting the cows.

3.4.2.2 California Mastitis Test

Milk samples passed as normal on clinical examination were obtained from each cow. These samples were examined using CMT. Milk which was grossly abnormal i.e. containing clots and flakes, watery or blood tinged was not screened by CMT. The screening was conducted just before milking (following clinical examination). It was made sure that the first few squirts of fore milk were discarded to avoid chances of having false positives

The CMT procedure was carried out as described in Schalm *et al.*, (1971). This test identified sub-clinical cases. Clinical cases, which were not overtly clear, were also defined by CMT. Two to three millilitres of milk from each patent quarter were taken in the corresponding cups/wells of the paddle and mixed with an equal volume of the CMT reagent. The mixture was swirled horizontally for a few seconds while observing for gel formation. The results were interpreted as described in Quinn *et al.*, (1994) and summarized in Table 2.

Table 2: Interpretation of California Mastitis Test

CMT SCORE	INTERPRETATION	VISIBLE REACTION
0	Negative	Milk fluid and normal
T	Trace	Slight precipitation
1	Weak positive	Distinct precipitation but no gel formation
2	Distinct positive	Mixture thickens with gel formation
3	Strong positive	Viscosity greatly increased. Strong gel that is cohesive with a convex surface.

3.4.3 Definition of mastitic cases and non-cases

A case definition criterion based on high cut-off points to increase the specificity and lower the sensitivity of the CMT screening and clinical examination was employed (Thrusfield, 1995). This was to avoid inclusion of many false positives since no confirmatory tests like microbial isolation and or somatic cell counts were conducted on the milk from CMT/clinical examination positive quarters. This enabled the formation of an aggregation rule to help in determination of prevalence of sub-clinical and clinical mastitis at cow and quarter levels respectively. Based on the above criterion a cow/quarter was judged as a positive case of sub-clinical mastitis if it had CMT score 1 and above in at least one quarter, otherwise negative if trace and 0 (De Graaf and Dwinger 1996). Likewise, clinical mastitis was also defined by manifestation of clinical signs, including abnormal milk, a hard or swollen udder, or both.

3.5 Economic impact of mastitis

Primary data and secondary data (published works) were both used to estimate the economic losses of mastitis. Milk production losses due to sub-clinical mastitis were determined by help of split udder results, prevalence findings and the published data while those due to clinical mastitis were estimated using secondary data and prevalence findings only. Treatment, culling, and withdrawal losses were estimated from the primary data collected by the current study.

3.5.1 Economic losses due to Mastitis

The economic losses were estimated with a financial model/function of the form:

$Y = a + b + c + d$ where Y = total mastitis losses, **a** = Reduction in milk yield, **b** = Cost of treatment, **c** = milk withdrawal losses, and **d** =losses due to culling.

Losses in milk yield and treatment costs were considered at quarter level. Loss estimation was based on some assumptions as given below:

1). A cow was assumed to have a uniform quarter production of 2.2 liters a day based on the established daily yield of 8.8 kg/day/cow with 2897 kg in a 328-day lactation period (Mekonnen *et al.*, 1985).

2). A cow suffered sub-clinical mastitis (SCM) at most for one-quarter during the lactation period. Thus the SCM lasted for 82 days out of 328 days of lactation.

3). A cow suffered clinical mastitis at most 4 times, i.e. in a total of 328 production- days the cow was at risk of contracting clinical mastitis 4 times and therefore received treatment four times.

4). Treatment was only restricted to clinical cases since majority of the farmers were not even aware of the presence of sub-clinical mastitis. Thirty percent (30%) of the clinical mastitis cows were assumed to have acute form of the disease and therefore received both intramammary infusions and parenteral treatment.

5). Following treatment, milk from treated animals was withdrawn. The situation on ground was contrary to the practice of withholding milk and any other products from antibiotic treated animals. Based on assumption 3, milk was withdrawn for a total of 12 days since the withdrawal period recommended was 3 days as recommended by most drug companies.

6). Each case of clinical mastitis lasted at least 5 days. Therefore, whatever estimates taken considered 5 days as the minimum duration.

7). Clinical cases were treated as independent and occurring on different farms. A different veterinarian attended every case of clinical mastitis that occurred.

3.5.1 1 Milk production losses

A split udder investigation was carried out on 30 crossbred dairy cows Friesian x indigenous breeds of a research herd at Holetta Agricultural Research Centre, Ethiopia. Cows were hand milked into separate buckets per quarter over a period of 8 days. All cows received the same feeding. Sub-clinical mastitis was diagnosed by California Mastitis Test. This approach was attempted to determine milk production with/without sub-clinical mastitis. Cows on the research station are milked twice, but for convenience of the investigator, late afternoon milking was used for this purpose. A cow with at least one healthy quarter (CMT score 0) was included in this trial The difference between the milk yield of 'trace', 1+, 2+, and 3+ and that of the normal quarter constituted the loss at the corresponding CMT score. The quarter milk loss was derived by multiplying the number of different CMT scores of the positive quarters by their corresponding milk production, and then averaged.

Quarter milk loss = (All quarters scored 3+ * quarter production at the same CMT) + (all quarters scored 2+ * quarter production at the same grade) + (all quarters scored 1+ * quarter production at the same) / Total number of positive quarters.

SCM losses = **a** * **b** * **c** * **d** where, **a**, was quarters affected in the positive animals, **b**, was daily milk yield per quarter, **c** the percent loss, and **d**, days in a lactation when a cow had the disease.

Clinical mastitis losses = Quarters affected x duration of a clinical case x number of times at risk x % loss per quarter. Cows with untreated clinical mastitis lose up to 50% quarter milk production (Singh and Singh, 1994). The two estimates were summed to give milk losses in Birr.

3.5.1.2 Treatment costs

Mastitis drugs and their prices were identified from veterinary drug shops and field veterinarians. No one drug was preferred to the others depending on its availability, price and also on the veterinarian's preference. The average prices of the commonly used intramammaries and parenteral antibiotics were used in the estimation. Information about veterinarians' fees through oral interview was collected from the veterinary clinics in Addis Ababa. Drug cost was summarized as cost of drugs and veterinary charges. Estimates were as follows:

Cost of intramammaries = Price per unit x treated quarters x treatment duration x number of times a cow was at risk in a lactation.

Cost of parenteral treatment = Number of cows treated x dose in cc/cow x treatment duration x number of times a cow is at risk in a lactation.

Veterinarians' fees = Number of cases x charge per case x number of times a cow is at risk in a lactaion.

Total treatment cost = cost of imtramammaries + cost of parenteral antibiotics + veterinarians' fees/case attended.

3.5.1.3 Withdrawal losses

Losses amount to cows treated x milk production per cow/day x treatment duration x milk price x risk period in a lactation.

3.5.1.4 Culling rate estimation

Culling was estimated retrospectively by considering farm dynamics in the last three years (1998-2001). Each farm owner was asked through a questionnaire the number of animals he/she (individual owners) or it had in the 3 years considered. Culling information was also retrieved from records for those farms with a record keeping system otherwise recall tendencies were relied on. Total culls irrespective of cause and also culls due to mastitis were estimated as:

$$\text{Total culling} = \frac{\text{Number culled per year due to all reasons x } 100\%}{\text{Cow population present in a year}}$$

$$\text{Mastitis culls per year} = \frac{\text{Mastitis culls per year x } 100\%}{\text{Cow population present in a year}}$$

Cow pricing was actively collected by assistance of questionnaires from the study farms and considered various aspects such as age/parity/lactation number of the cow, pregnancy status, and productivity. Since the respondents suggested a range of figures, these were only taken as rough estimates. To validate these figures, a mean survey on selected farms was remounted to verify the

earlier findings on cow prices. The prices got from the initial survey and the preceding one were averaged and used in estimating the loss incurred by culling a mastitic cow.

Culling loss = price of normal lactating cow – that of culling a cow.

3.6 Variable definition

3.6.1 Management risk factors

The risk factors considered in the model were breed, dry cow therapy, age of the cows, body condition score, stage of lactation, leaking milk, history of previous mastitis attacks, time of drying off, drying off style and teat drying. Some of these variables were binary, ordinal or categorical in nature.

3.6.2 Prevalence study

The number of cows with outcome of interest (mastitis) was noted out of the sampled population. This was a binary variable as the disease was either present or not.

3.6.3 Economics study

Data on milk reduction due sub-clinical and clinical mastitis, milk prices, cow prices, culling rate, treatment and associated withdrawal losses were considered. The variables in mastitis economics were either continuous or count in nature. These data were collected concurrently with prevalence study and questionnaire survey data except the split udder data, which was collected separately.

3.7 Data analyses

Data were stored in Ms Access databases 1997. The same software was used to form queries that could be manipulated by various statistical packages like Stata 6.0 (1984-1999), Statgraphics version plus 2.1, 1995, SPSS 10.0, SPSS Inc. (Munich) and Ms Excel 1985 - 1996.

Chi square tests were used to identify the associations between disease factors (explanatory/Predictor variables) and the disease (response/outcome variable). Only those variables with a strong association as seen from their χ^2 values at $P< 0.05$ were selected. Logistic regression was used to screen those explanatory variables with significant association with the outcome variable for their magnitude of association as seen from their odds ratios and 95% confidence intervals. The simple random-effect logistic regression model can be written as:
Logit P (Yij=1)=$\beta o + \sum \beta i$ xi + eij, where eij = R +e, R=correlation matrix, e= random error, the fixed part includes the intercept, βo and covariates xi and regressors for covariates, βi. Two univariate random-effect logistic models were fitted for each selected risk factor with sub-clinical and clinical mastitis as the outcome variables. P values less than 0.05 were considered significant. Multivariate analyses (Adjustment of odds ratios) was also done to evaluate the factors associated with sub-clinical and clinical mastitis in two other models as a final step in order to compare the odds ratios got from univariate and multivariate analysis. In a case where the difference between the two was big then there was confounding and the adjusted odds from the multivariate analysis were reported, otherwise only the multivariate analysis results were reported.

Split udder trial data was analyzed by SPSS. Unlike in the prevalence study where quarter CMT scores of 'trace' and 0 were considered negative (CMT score 0), the split udder result considered all the traces as positive and only quarter CMT score negative as 0. This approach was adopted in order to minimize chances of having imbalanced distribution of the CMT scores where a given score could have very many counts as compared to that of other scores. The relationship between CMT score and quarter milk production was analyzed using univariate analysis of variance (UNIANOVA, SPSS 10.0, SPSS Inc. Chicago). The dependent variable was the mean quarter milk production for the eight days and CMT scores (0, 1+, 2+, and 3+) were entered as independent/fixed variables.

Mastitis losses for cows with sub-clinical mastitis were determined based only on the percentage quarter loss in milk production following sub-clinical mastitis and expressed in financial terms. For clinical mastitis losses included milk loss, treatment costs, discarded milk following treatment and culling. All the losses were used in the partial budget analysis except losses due to sub-clinical mastitis. Sensitivity analysis considering three different scenarios of 100%, 80% and

60% treatment efficacy were considered to see the changes in both the net benefits and benefit cost ratios as a way of evaluating treatment as an effective way to control clinical mastitis.

4 RESULTS

4.1 Questionnaire survey

Questionnaires were administered to 51 dairy farm owners/managers. The response rate was 90 %. A few interviewees refused the use of their dairy herds for the study.

4.1.1 Farm distribution

The study was conducted on a total of 51 dairy farms (Figure 1) that were randomly selected from 3 production sub-systems namely dairy herds in secondary towns, urban and peri-urban. Overall, thirty-eight (75%) small-scale, and thirteen (25%) large-scale farms across the production sub-systems were included in the study.

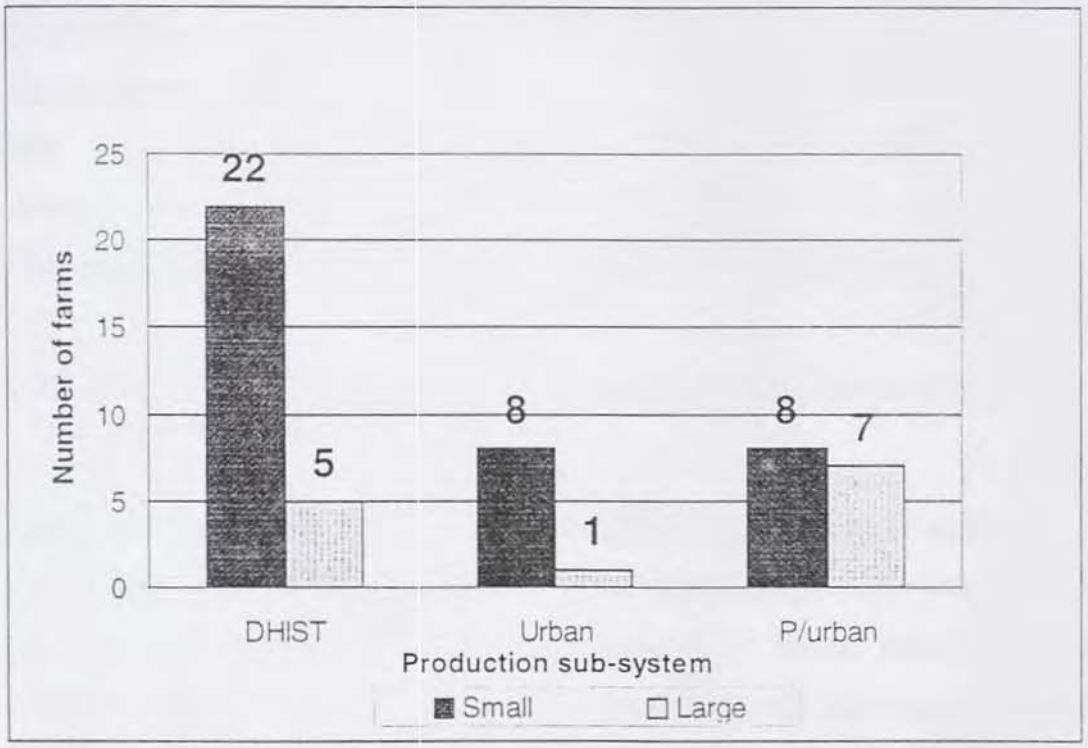

Figure 1: The distribution of farm sizes by production sub-systems in the study areas

4.1.2 Summary of descriptive statistics of continuous variables of the 51 study farms.

Mean farm size was 18.2 ha although many farms had a small acreage of 0.05 hectares (Table 3). These were small-scale farms with mostly 2 cows kept. However, a few farms especially in the peri-urban and DHIST had big land and contributed a bigger proportion of the large-scale farms.

The youngest farmer was 25 years and the oldest was 78 years. Males (71%) dominated farm ownership while females owned 27% of the farms. The remaining 2% of the study farms were either research or private company owned. The age of cows ranged from 2 to 13 years. Many of the cows were in their first parity and their ages were at least 5 years with an average cow age of 5.7 years. Parity and cow ages were only considered on 288 counts, as 75 cows had no information. As concerns months in lactation, the smallest was 1 month and the highest was 22 months.

Table 3: Summary statistics of the parameters of continuous variables in the study farms

Variable	Mean	Median	Mode	Range	SD*	SE*
Farm size (ha)	18.2	0.07	0.05	0.02-800	111.8	15.6
Farmer's age (yrs)	50.6	52	50	25-78	10.3	6
Cow numbers	16.4	3	2	1-232	2.29	1.5
Cow age (years)	5.7	5.25	5	2-13	1.65	0.13
Parity	2.7	2	1	1-9	3.59	0.1
Lactation stage	6.17	6	-***	1-22	3.59	0.2

SD* = Standard deviation, SE* = Standard error and -*** no value for mode.

4.1.3 Distribution of categorical farm-level variables among the 51 dairy farms

The proportional distribution of the categorical variables by farm sizes is shown in Table 4. The majority (98.0%) of the respondents said there were no contacts between their cows and other farmers' cows. The majority (84.3%) of the respondents started dairying through induction. Others (15.7%) acquired their knowledge through agricultural training and extension services. For ease of handling, the variables under mastitis diagnosis were merged into two levels. Nearly four fifth (78.4%) of the respondents had knowledge on mastitis and 21.6% had no knowledge. The farmers had various ways of identifying a cow with mastitis. Of those with knowledge of mastitis, 12% used diagnostic facilities such as CMT and strip cup to define a mastitic case whereas the rest depended on inflammatory signs like udder swelling and other indicators such as reduced milk yield. Temporal prediction of the frequency of occurrence of mastitis depended on the seasons of the year. For example, 10%, 19.6% and 25.5% of the respondents with knowledge of mastitis said they experienced the problem during the rainy, dry and both seasons.

respectively. Stall-feeding was the main (82.4%) grazing system practiced although a few (17.7%) farmers practiced semi-zero grazing and only one farmer indicated extensive grazing.

Table 4: The proportional distribution of the farm categorical variables by farm sizes in the study areas

Variables	Farm sizes		Combination
Contact between animals	Small=38	Large=13	All farms=51
No	37 (97.4%)	13 (100%)	50 (98.0%)
Yes	1 (2.6%)	0 (0%)	1 (2.0%)
Dairying skills			
Agriculture /extension	2 (5.3%)	6 (46.2%)	8 (15.7%)
Induction	36 (94.7%)	7 (53.9%)	43 (84.3%)
Mastitis diagnosis			
No knowledge	11 (28.9%)	0 (0 %)	11 (21.6%)
Knowledge about mastitis	27 (71.1%)	13 (100%)	40 (78.4%)
Mastitis time**			
Rainy season	4 (10.5%)	1 (7.7%)	5 (10.0%)
Dry season	4 (10.5%)	6 (46.2%)	10 (19.6%)
Both seasons	8 (21.1%)	5 (38.5%)	13 (25.5%)
	16 (42.11%)	**12 (92.3%)**	**28 (55.0%)**
Grazing system			
Zero grazing	35 (92.1%)	7 (53.9%)	42 (82.4%)
Semi-zero grazing	3 (7.9%)	6 (46.2%)	9 (17.7%)

** Variable with less than 100% column sub-totals

4.1.3.2 Dry cow practices

The dry cow practices are summarized in Table 5. Very few (13.7%) respondents did not dry off their cows. Fifty two percent (27 out of 46) of those who dried their cows did it gradually and the remaining 37.3% dried them rapidly/suddenly. Dry cow therapy was only reported in 2 (3.9%)

large farms. Incidentally, both farms were individual farms (one in DHIST and another from Kaliti in the peri-urban). The dry cow therapy used were intra-mammary tubes and lugol's iodine, which were infused in the teats by the resident veterinarians.

Table 5: Summary of dry-cow practices in the study areas

Variables	Farm sizes		Combination
	Small=38	Large=13	All farms=51
Drying time**			
1 month before calving	3 (7.9%)	0 (0%)	3 (5.9%)
2 months before calving	20 (52.6%)	13 (100%)	33 (64.7%)
Milk turns yellow	8 (21.1%)	0 (0%)	8 (15.7%)
	31 (81.6%)	**13 (100%)**	**44 (86.3%)**
Drying style**			
Gradual	15 (39.5%)	12 (92.3%)	27 (52.9%)
Abrupt/sudden	18 (47.4%)	1 (7.7%)	19 (37.3%)
	33 (86.8%)	**13 (100%)**	**46 (90.2%)**
Dry cow therapy			
No	38 (100%)	11 (84.6%)	49 (96.1%)
Intramammary &others	0 (0 %)	2 (15.4%)	2 (3.9%)

** **Variable with mixed column sub-totals (< >100%)**

4.1.3.3 Milking management/procedure

Nearly all the 51 respondents reported that they milked their animals twice a day, i.e. morning from 3.00 - 7.00 am and late afternoon from 3.00 p.m. However, few (2.0%) farmers said they milked their cows thrice, especially during early lactation for the heavy milkers. Incomplete milking was only reported in 10% of the studied farms and these were mostly small-scale as they reserved some milk for calves. Two thirds (67%) of the respondents reported that they washed hands in-between milkings. The summary of the proportional distribution of categorical variables on milking practices is shown in Table 6. Majority (82.4%) of the respondents practiced teat cleaning immediately before milking. Few (7.8%) did it both before and after milking. A small (17.7%) number of respondents said they did not clean the udder of the cow. This was commonly

practiced by those with local breeds and those who allowed calves to suckle first before milking. Most (82.4%) of those who reported cleaning the udder used water only. Only 7.8% used water with disinfectant (soap). They used tap water in a pail to wash the udder. Nearly three fifth (62.7%) of the respondents dried the udder after cleaning. Of these, 33.3% used the shared piece of cloth as towel on all milking cows in the herd while, 29.4% used a separate piece of cloth for every milking cow in the herd. In all the farms, which reported teat drying by a shared or separate towel, the practice was that the same piece of cloth used in drying both the milkers' hands and the teats.

Teat dipping was not commonly practiced on the study farms. Those farms (5 large farms) that practiced teat dipping used ammonium compounds (3.9%) and lugols iodine (5.9%). Nearly all the respondents indicated that they never practiced teat dipping post milking. Only 10.0% practiced teat dipping. Post milking teat dipping was carried by 3.9% of the respondents and 5.9% did it both before and after milking.

A mastitic cow was milked last, any time or first depending on the farmer's knowledge of mastitis. Those with better knowledge milked last (37.3%) and those with scanty information milked it either any time (52.9%) or first (2.0%). A few (7.8%) respondents refrained from milking a mastitic cow altogether.

Family members (49.0%) and employees (43.1%) or a combination (7.8%) did the milking. A very small proportion of the respondents did not wash their hands before milking. Those came from small-scale farms that kept the local breeds of cows.

Table 6: Summary of proportional distribution of milking practices in the study areas

Variables	Farm sizes		Combination
	Small=38	Large=13	All farms=51
Teat cleaning**			
Before milking	30 (79.0%)	12 (92.3%)	42 (82.4%)
Both before and after milking	4 (10.5%)	0 (0 %)	4 (7.8%)
	34 (89.5%)	**12 (92.3%)**	**46 (91.2%)**
Teat cleaning style**			
Water only	31 (81.6%)	11 (21.6 %)	42 (82.4%)
Water and disinfectant	3 (7.9%)	1 (2.0%)	4 (7.8%)
	34 (89.5%)	**12 (92.3%)**	**46 (90.2%)**
Teat drying**			
Separate towel	13 (34.2%)	2 (15.4%)	15 (29.4%)
Shared towel	13 (34.2%)	4 (30.8%)	17 (33.3%)
	26 (68.4%)	**6 (46.2%)**	**32 (62.8%)**
Teat dipping time**			
Both before and after	0 (0 %)	3 (23.1%)	3 (5.9%)
After milking	0 (0 %)	2 (15.9%)	2 (3.9%)
	0 (0%)	**5 (38.5%)**	**5 (10.0%)**
Milking mastitic cow++			
Anytime	22 (57.9%)	5 (38.5%)	27 (52.9%)
First	1 (26.3%)	0 (0)	1 (2.0%)
Last	11 (29.0%)	8 (61.5%)	19 (37.3%)
	34 (89.5%)	**13 (100%)**	**47 (92.2%)**
Milkers			
Family	24 (63.2%)	1 (7.7%)	25 (49.0%)
Employees	10 (26.3%)	12 (92.3%)	22 (43.1%)
Combination of all	**4 (10.5%)**	**13 (100%)**	**4 (7.8%)**
Hand washing			
No	4 (10.5%)	0 (0%)	4 (7.8%)
Yes	34 (89.5%)	13 (100%)	47 (92.2%)
Hand drying			
No	18 (47.4%)	7 (53.9%)	25 (49.0%)
Yes (Reusable towel)	20 (52.6%)	6 (46.2%)	26 (51.0%)

** Variable with < 100% column sub-totals and ++ Variable with mixed column sub-totals (<>100%)

4.1.3.4 Housing practices

One third (33.3%) of the houses had a design that allowed sunlight to reach the inside. The remainder (66.7%) did not but were well ventilated. Leaking roofs were observed in about 47.0% of houses. Appendix 7.1 outlines the housing practices in the study areas. The distribution of housing types and floor practices are shown in Table 7. Animal houses in the study farms were of various types. One third (33.3%) of them had wooden and or muddy walls with corrugated iron sheet roofs. Eighteen percent of the houses had stone and concrete walls. Forty nine percent of the houses had roofs but no walls or a wall on one section and the rest open. Fifty eight percent of the houses had concrete floors, while one-fifth (20.0%) had earth floors and arranged stones, respectively. Many of the houses were spacious and animals had enough space to freely turn around and rest. Maternity rooms present on twelve percent of the farms were regularly disinfected.

Table 7: Summary of housing practices distributed by farm sizes in the study areas

Variables**	Farm sizes		Combination
	Small=38	Large=13	All farms=51
Type of house			
Wooden & muddy walls + roof	16 (42.1%)	1 (7.7%)	17 (33.3%)
Concrete/stone walls + roof	4 (10.5%)	5 (38.5%)	9 (17.7%)
Other	18 (47.4%)	7 (53.9%)	25 (49.0%)
Floor type			
Concrete	19 (50.0%)	11(84.6%)	30 (58.8%)
Earth	9 (23.7%)	1 (7.7%)	10 (19.6%)
Stones	10 (26.3%)	1 (7.7 %)	11 (21.6%)
Bedding			
Nothing on the floor	34 (89.5%)	10 (19.6%)	44 (86.3%)
Bedding	4 (10.6%)	3 (5.9%)	7 (13.7%)
Maternity room disinfection			
No	37 (97.4%)	8 (61.5%)	45 (88.2%)
Yes	1 (2.6%)	5 (38.5%)	6 (11.8%)

** all variables had 100% column sub-totals

4.1.3.5 Drainage and manure handling

Appendix 7.1 summarizes the outline of drainage and manure handling practices. Ninety six percent of the respondents removed manure from animal houses daily. Two percent of the respondents reported that they removed manure from the animal houses twice a week. These houses were visibly dirty and had different species of flies around them. The rest (2%) collected manure weekly or fortnightly. Many farms had poor drainage that led to damming of slurry near or inside the animal houses. No proper disposal of collected manure was reported in 55% of the farms. Thirty one percent and 14% reported disposing the manure near animal houses and in crop farms, respectively. As for the farm hygiene/sanitation, 2%, 49% and 49% of the farms were ranked as being excellent, good and poor, respectively.

4.2 Prevalence study

A total of 363 cows were randomly sampled from the 51 study herds. Clinical examination and CMT screening were used to determine the prevalence of mastitis. A total of 1452 quarters were considered in the prevalence study but 54 (3.72%) of them were blind/non functional. The functional quarters were 1398 (96.28%). Left front and right front quarters each had 8 quarters blocked (0.55%), left rear 21 (1.45%) and right rear 17 (1.17%). Quarter CMT results indicated that 207, 111, 70, 60, and 950 quarters were CMT scores 3+, 2+, 1+, 'trace' and negative (0), respectively (Appendix 7.6).

4.2.1 Prevalence of sub-clinical mastitis

The distribution of the prevalence of sub-clinical mastitis was based on the farms, farm sizes and production sub-systems. Prevalence on production sub-system and herd size level was summarized at cow and quarter level and on farms only at cow level. Overall prevalence of sub-clinical mastitis at cow level was 46.6% (95% CI: 41.3 – 51.8%) and at quarter level 27.8% (95% CI: 25.4 – 30.2%).

4.2.1.1 Farm level sub-clinical mastitis prevalence

Appendix 7.4 summarizes the prevalence of sub-clinical mastitis by farms. Thirty-four (67%) out of fifty-one farms had at least one case of sub-clinical mastitis. Nine (24%) out of thirty-eight

small-scale farms had 100% sub-clinical mastitis. The small-scale farms with 100% prevalence of sub-clinical mastitis had either 1 or 2 cows. Distribution of small-scale farms with 100% prevalence was 4, 3 and 2 in DHIST, urban and peri-urban systems respectively. Twenty (53%) small-scale farms had prevalence of 50-100%. Seventeen (45%) small-scale farms had no sub-clinical mastitis and many of them had either one or two cows per herd.

The large-scale farms had sub-clinical mastitis and the highest prevalence in that category was 69% recorded in one farm of 44 cows situated in the peri-urban production sub-system. Five (38%) out of the thirteen large scale farms observed at least 50% prevalence while the rest had prevalences less than 50%. All large-scale farms had sub-clinical cases. The smallest prevalence recorded in this category was 11% in a farm of 18 cows situated in the peri-urban production sub-system. The urban production system had a higher prevalence of sub-clinical mastitis as compared to the other 2 production sub-systems.

4.2.1.2 Cow and quarter sub-clinical mastitis prevalence by farm sizes

Figure 2 summarizes the cow and quarter prevalences of sub-clinical mastitis at farm size level. Sub-clinical mastitis stratified by farm sizes was 48.2% (95% CI: 37.1-59.4%) and 46.1% (95% CI: 40.1-52.1%) for small scale and large-scale farms, respectively. Prevalences based on farm size were not significantly (P = 0.7336) different. At quarter level it was 28.6% (95% CI: 23.8-33.8% and 26.2%, (95%CI: 23.6-28.8%) for small and large-scale farms respectively. Quarter prevalences were not significant (P=0.403) among farm sizes.

4.2.1.3 Cow and quarter sub-clinical mastitis prevalence by production sub-systems

Figure 3 summarizes the prevalence of sub-clinical mastitis by production sub-systems. According to production sub-systems, the prevalence of sub-clinical mastitis was estimated to be 44.5% (95% CI: 36.8-52.5%), 60% (95% CI: 36.1-80.9%) and 46.9% (95% CI: 39.4 -54.5%) for DHIST, urban and Peri-urban at cow level, respectively. There was no statistical significant (P = 0.361) difference between these estimates. At quarter basis the prevalence estimates were 24.8%, (95% CI: 21.6-28.3%), 40%, (95% CI: 29.2-51.6%) and 27%, (95% CI: 23.7-28.8%) for DHIST,

urban and peri-urban sub-systems respectively. There was a significant (P= 0.006) difference between these estimates.

4.2.2 Prevalence of clinical mastitis

4.2.2.1 Farm level clinical mastitis

Appendix 7.5 summarizes clinical mastitis at farm level. Twelve (24%) out of the fifty-one farms studied registered at least a case of clinical mastitis. Large farms constituted 67% of the total number of farms, which registered at least a clinical case. However, no farm recorded above 30% clinical mastitis prevalence. The highest prevalence was 27.3% in one large farm situated in the peri-urban production sub-system. Fourteen percent was the second highest prevalence in another large farm located in the peri-urban production system. All the farms in the large farm category, with cases of clinical mastitis had a prevalence of above 5%.

Concerning the small-scale farms, the highest prevalence was 33.3% and was recorded in the urban and peri-urban production sub-systems. This was followed by 20% in two farms both of the DHIST.

4.2.2.2 Cow and quarter overall clinical mastitis prevalence

The overall prevalence of clinical mastitis was 6.6%, (95% CI: 4.3 - 9.7%) level was 2.8%, (95% CI: 2 – 3.8%) at cow and quarter levels, respectively.

4.3.2.3 Cow and quarter clinical mastitis prevalence by farm sizes

Figure 2 summarizes the prevalence of clinical mastitis at farm level. Prevalence by farm sizes, was 3.6%, (95% CI: 0.8 -10.2%) and 7.5%, (95% CI: 4.7-11.2%) for small and large scale farms respectively. There was no statistical significant (P=0.125) difference between these estimates. At the quarter level, it was 2.4%, (95% CI: 1.0-4.7%) and 2.8%, (95% CI: 1.9-3.9%). There was no significant (P=0.101) difference between these estimates.

4.2.2.4 Cow and quarter clinical mastitis prevalence by production sub-systems

Figure 3 summarizes the prevalence of clinical mastitis by production sub-systems. Stratification by production sub-system level gave clinical mastitis prevalence as 2.4%, (95% CI: 0.7-6.1%), 5%, (95% CI: 0.1-24.9%) and 10.6%, (95% CI: 6.5-16.1%) for DHIST, urban and peri-urban production sub-systems at cow level, respectively. There was statistical significant (P = 0.003) difference in the prevalence at production sub system levels. At quarter level it was 0.9%, (95% CI: 0.3-2.0%), 1.3%, (95% CI: 0.03-6.8%) and 4.5%, (95% CI: 3.1-6.3%) for DHIST, urban and peri-urban respectively. There was significant (P = 0.04) difference between these prevalence estimates.

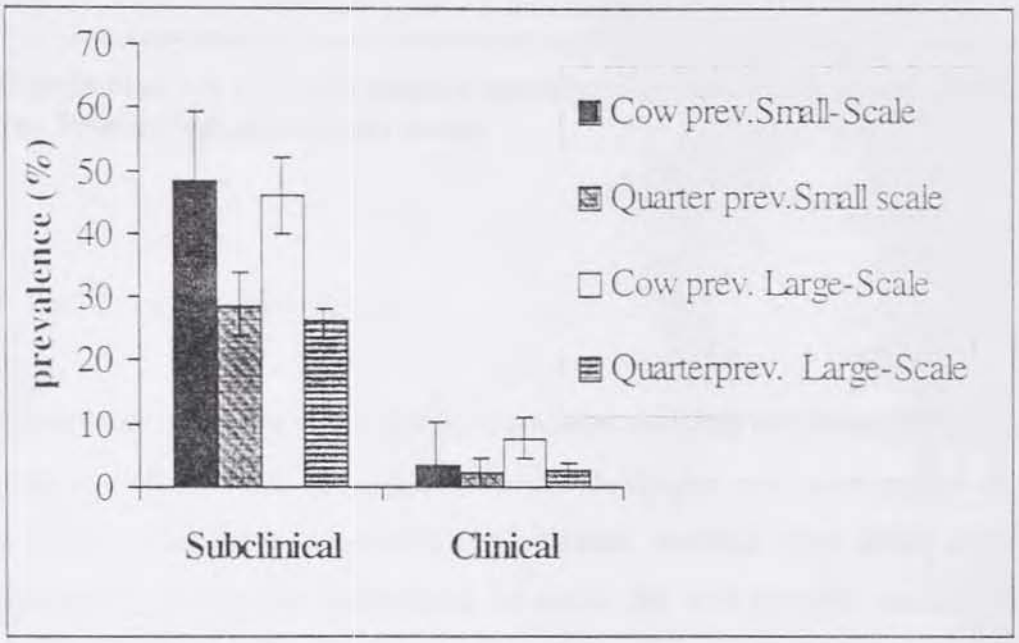

Figure 2: Distributions of cow and quarter prevalence of sub-clinical and clinical mastitis by farm sizes

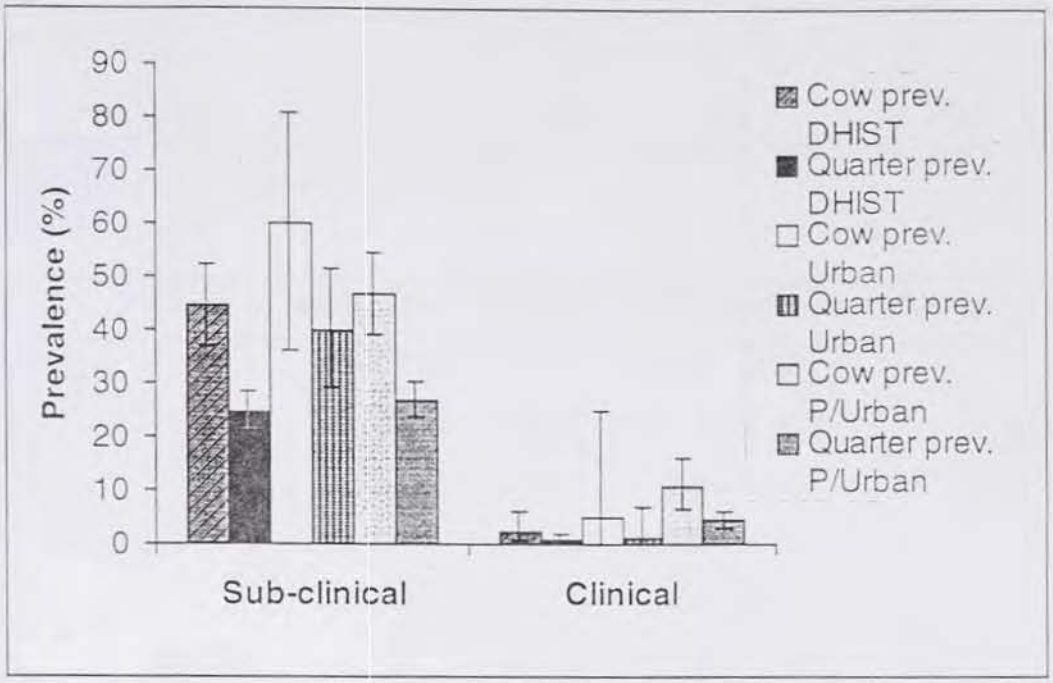

Figure 3: Distributions of cow and quarter prevalence of sub-clinical and clinical mastitis by Production sub-system levels

4.3 Risk factors of Mastitis

Explanatory/predictor variables significantly associated with the response/outcome variable were selected using chi-square into the logistic model to derive the odds ratios. Univariate and multivariate analyses for both sub-clinical and clinical mastitis were done. Only multivariate results are reported since the differences between the risk-specific coefficients were not significant.

4.3.1 Multivariate logistic regression of risk factors for sub-clinical mastitis

Results of multivariable logistic regression analysis on sub-clinical mastitis are summarized in Table 8. Age of at least 8 years, cows with at least 8 parities, cows in at least 8th month of lactation, and poor body condition were significantly ($P<0.05$) associated with sub-clinical mastitis as their odds ratios were greater than 1 and their 95% confidence intervals excluded 1. Breed, fair body conditions, leaking milk and udder problem were not significantly ($P>0.05$) associated with sub-clinical mastitis although their odds ratios were greater than 1. The rest of the

variables except cows aged 1 - 3 years and lactation stage 1-3 months, which were dropped, had odds of less than 1 and were considered protective factors.

Table 8: Summary results of multivariate analysis of associations between sub-clinical mastitis and potential risk factors

Variable	Level	odds ratio	P-value	95% confidence interval
Dry cow therapy		0.125	0.006	0.090 – 0.895
Dry off time		0.899	0.708	0.543 – 1.404
Dry off style		0.850	0.845	0.694 – 1.068
Teat drying		0.592	0.241	0.536 – 1.170
Breed		1.409	0.101	0.472 – 4.210
Age♦	Middle	1.117	0.203	0.934 – 3.656
	Old	3.009	0.000	1.151 – 12.104**
Parity	1	0.725	0.803	0.583 – 0.859
	2	0.991	0.111	0.585 – 1.336
	3	2.822	0.000	1.002 – 3.007**
Lactation	Mid	0.841	0.583	0.454 – 1.560
	Late	1.115	0.003	1.001 – 1.212**
Body score	Good	0.699	0.903	0.321 – 0.889
	Fair	1.001	0.092	0.876 – 5.339
	Poor	5.333	0.003	2.119 – 13 221**
Leaking milk		1.432	0.313	0.347 – 3.843
Udder problem		1.321	0.097	0.564 – 1.698

** Significant at 95%CI, ♦Age young 1-3 years dropped.

Age, recoded as young 1-3 years, Middle 4 - 8 years and old 8 and above years. Parity, 1= 1-3, 2 = 4-7, 3 = at least 8 and Lactation recoded as early meaning 1-3, Mid 4-7 and late 8 and above. Lactation was measured in months whereas parity by count.

4.3.4 Multivariate analysis for risk factors of clinical mastitis

Cows aged at least 4 years, cows with at least 8 parities, cows in at least 4th month of lactation, poor body conditions, problem of leaking milk, and a history of udder problems before had odds greater than 1 and their 95% confidence interval excluded 1 and were significantly ($P<0.05$) associated with clinical mastitis (Table 9). Drying off time, style of drying, teat drying and breed were protective factors with odds ratios less than 1. Good and fair body condition scores had odds ratios greater than 1 but their 95% CI included 1 hence were not significantly ($p>0.05$) associated with clinical mastitis.

Table 9: Results of multivariate analysis of association between clinical mastitis and potential risk factors

Variable	Level	odds ratio	P-values	95% confidence interval
♦				
Dry off time		0.785	0.348	0.173 – 1.400
Dry off style		0.656	0.547	0.193 – 0.968
Teat drying		0.429	0.377	0.366 – 0.857
Breed		0.909	0.335	0.679 – 1.407
Age♦	Middle	1.337	0.003	1.004 – 3.216**
	Old	2.544	0.000	1.885 – 6.314**
Parity♦	3	1.987	0.041	1.032 – 4.426**
Lactation♦	Mid	2.776	0.001	1.974 – 4.536**
	Late	4.815	0.000	3.201 – 6.992**
Body Score	Good	1.430	0.103	0.524 – 1.983
	Fair	2.247	0.07	0.875 – 4.335
	Poor	3.563	0.008	2.035 – 9.550**
Leaking		9.001	0.000	3.747 – 28.973**
Udder problem		3.625	0.000	1.779 – 9.581**

** Significant at 95% CI. ♦ Dropped variables e.g "young" age, "early" lactation, parity 1 and 2 and dry cow therapy.

Age, recoded as young 1-3 years, Middle 4-8 years and Old at least 8 years. Parity, 1= 1-3, 2 = 4-7, 3 = at least 8 and Lactation recoded as early meaning 1-3, Mid 4-7 and Late at least 8. Lactation was measured in months whereas parity by count.

4.4 Economic analysis

4.4.1 Split udder

Fifty-nine quarters had CMT score '0', and 14, 18, and 27 quarters had 1+, 2+ and 3+ respectively. CMT score 'trace' was added to CMT score 1 + for analysis. Two quarters were blind. Average milk production per quarter was 0.82 kg (95% CI: 0.42-1.22 kg) per milking. The CMT score showed significant (P=0.008) association with quarter milk production. CMT score 0, 1+, 2+ and 3+ had a mean production of 0.9188 kg (95% CI: 0.820-1.018), 0.9080 kg (95% CI: 0.662-1.154), 0.8610 kg (95% CI: 0.658-1.064) and 0.616 kg (95% CI: 0.470-0.761). The difference (loss due to SCM) in production of quarters with CMT score 1+, 2+, and 3+ compared to quarter CMT score 0 was 0.0108 kg (1.2%), 0.0578 kg (6.3%) and 0.303 kg (33%). Milk production also differed between quarters. It was higher in rear than front quarters (0.89 ± 0.06 vs. 0.71 ± 0.06 kg).

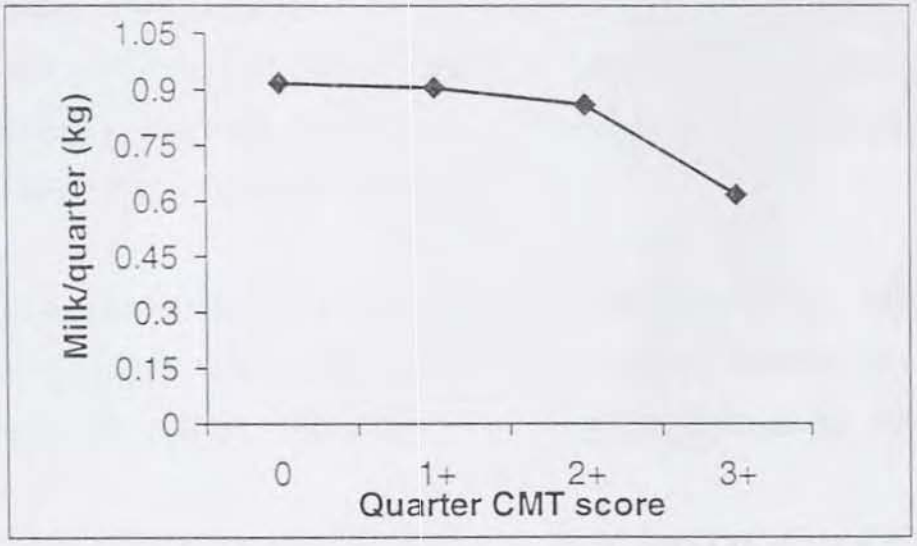

Figure 4: Quarter CMT score and its corresponding milk yield

4.4.2 Factors/determinants of mastitis losses

Mastitis losses = milk production losses + treatment costs + withdrawal losses + culling losses.

4.4.2.1 Milk production losses

Total milk losses in a lactation = Loss due to sub-clinical mastitis + loss due to clinical mastitis.

Sub-clinical mastitis (SCM) losses

Findings of the split udder experiment and the prevalence results estimated milk loss per quarter due to sub-clinical mastitis. Four hundred forty eight (448) quarters were positive on prevalence study, out of which 207, 111 and 130 (70 with 1+ and 60 with trace) were CMT scores 3+, 2+ and 1+, respectively (Appendix 7.6). Quarters with CMT scores 3+, 2+, and 1+ produced 0.616 kg, 0.861 kg and 0.908 kg respectively.

$$\text{Mean quarter production with SCM} = \frac{(207 \times 0.616) + (111 \times 0.861) + (130 \times 0.908)}{448} = 0.761 \text{ kg}$$

Loss per quarter due to SCM was 0.919 – 0.761 = 0.157 kg (17.1%).

SCM losses = 448 x 8.8/4 x 328/4 x 0.171 = 13820.0832 kg per lactation. The loss was 13820.0832 ÷ (82 x 448) = 0.3762 kg per quarter or 1.505 (0.3762 x 4) kg per cow per milking. Loss per cow per day was estimated at 3.01 Birr/day. In 82 days the cow was affected she loses 246.79 Birr. Milk was priced 2 Birr per kilogram.

Clinical mastitis (CM) losses = 39 x 5 x 4 x 8.8/4 x 0.5 = 858 kg. Where 39 are the number of quarters, 4- was the risk period in a lactation, each with a duration of 5 days. Loss per cow per day due to CM = 858kg ÷ (39 x 20) x 4 = 4.4 kg (8.8 Birr) per day and 176 Birr in 20 days she suffers.

Table 10: Summary of milk production losses in a lactation

Number of quarters (a)	% loss in ¼ production (b)	¼ production without mastitis/lactation (c)	Loss made (d)=(c) *(b)
SCM (169 cows) 388	17.1	69995.2	13820.0832
CM (24 cows) 39	50	1716	858
		71711.2 kg	**14678.0832 kg**

Total milk production losses amounted 14678.0832 x 2 =29356.1664 Birr per lactation.

The loss per quarter per day due to mastitis was 14678.0832 ÷71711.2 x 100 = 20.5% (Table 10).

The loss made = 0.205 x 2.2 x 2 = 0.902 Birr per quarter per day where 2 was milk price/kg.

4.4.2.2 Treatment cost

Veterinary charges and cost of medicines comprised this. The commonly used mastitis drugs were Multiject, Mastitis-2, Gentamast and Alfasan priced

6.20, 6.25, 14.75 and 6.60 respectively.

The average price =

$$\frac{6.20 + 6.25 + 14.75 + 6.60}{4}$$

= 8.45 Birr per one intra-mammary.

A cc of parenteral antibiotics costed 0.80 Birr. A cow weighed about 250-300 kg, hence a minimum of 25 cc for 3 days at a dose rate of 1mg/kg body weight.

8.45 x 39 x 3 x 4 = 3954.6 Birr (39 treated quarters, 3-treatment duration and 4 number of times a cow was at risk).

Parenteral treatment cost 30/100 x 24 x 25 x 0.8 x 4 x 3 = 1728 Birr (30/100 proportion of cows with acute mastitis, 24 cows treated, 25-dose rate and 0.8 charge/dose 3 duration of treatment).

Total drug cost = 3954.6 + 1728 = 5682.6 Birr.

Veterinarians' charges = 24 x 15 x 4 = 1440 Birr (24 cows treated, 15 veterinarians' charge per case and 4, the number of times a cow at risk).

Total treatment cost = 1440 + 5682.6 = 7122.6 Birr and 74.19 Birr per cow one time treatment.

4.4.2.3 Withdrawal losses

Milk from 24 cows with clinical mastitis was discarded following treatment.

Withdrawal loss = 24 x 8.8 x 0.5 x 2 x 3 x 4 = 2534.4 Birr (24 treated cows, 8.8 average daily yield per cow, 0.5 lose due to CMT, 3 withdrawal period, days and 4 risk period).

4.4.2.4 Culling/cow pricing

Culling information was retrieved from retrospective data, which considered the past three years. The results are summarized in Table 11.

Table 11: Summary of cow population, total culls and mastitis culls in relation to production sub-system in the last 3 years

Production sub-system	Cows at risk for culling 3 years ago	Total culls last (3 years)	Mastitis culls in last 3 years	Mastitis culls (%)
DHIST	258	57	11	4.26
Urban	36	10	0	0
P/urban	935	298	79	8.45
Total	**1229**	**365**	**90**	**7.32**

Annual mastitis culling rate was 7.23%. Annual total culling rate was and 29.7%. A normal lactating cow was valued 1500-4500 Birr. The average 3000 Birr was used. Culls were valued at 700-2300 Birr. The average (700 + 2300)/2 =1500 Birr was used. Expected number of mastitis culls from 363 cows at 7.32% culling rate was 25

Amount received from culls was 25 x 1500 =37500 Birr and for 25 normal cows was 75000 Birr.

Mastitis culling loss = 75000-37500=37500 Birr.

Total losses of mastitis = 29356.1664 + 7122.6 + 2534.4 + 37500 = 76513.1664 Birr. The loss per cow per lactation is 210.8 Birr.

Milk loss, culling, treatment and withdrawal contributed 38.4%, 49%, 9.3% and 3.3% to the total mastitis losses, respectively (Figure 5).

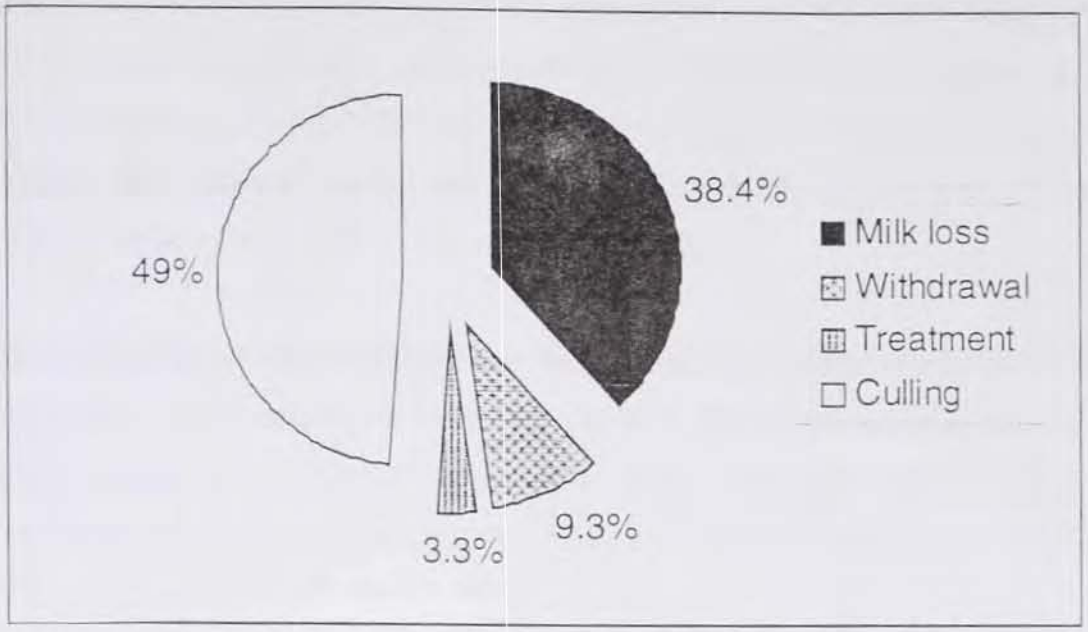

Figure 5: The contribution of each component to total mastitis losses

4.4.3 Partial budget analysis of the farms in the study

The partial budget analysis with treatment of clinical mastitis cases only was performed. All estimated losses due to mastitis were therefore included in the partial budget analysis either on the costs or benefits side with exception of sub-clinical mastitis (Table 12).

Table 12: Summary of costs and benefits due to the farms in the study areas

Costs	Amount (Birr)	Benefits	Amount (Birr)
Extra costs		**Extra Benefits**	
Drug costs	5682.6	Increased milk sales (Cases treated)	1716
Veterinary fees	1440.0		
Revenue forgone		**Costs saved**	
Withdrawal losses	2534.40	Culling	37500
Total costs (TC)	**9657.00**	**Total benefits (TB)**	**39216**

Net benefits were estimated as TB-TC, and was 29559, and the Benefit cost ratio (BCR), TB/TC was 4.06. A sensitivity (break-even) analysis to determine the effect of treatment on net benefits and BCR at 100%, 80% and 60% efficacious treatment was carried out. If 100% treatment efficacy was assured, this would reduce treated clinical cases to zero and therefore net benefits

and BCR remain as in Table 12. Treatment efficacy of 80% only reduces clinical mastitis by 80%. Twenty- percent cases are not cured and result in a proportionate loss in milk yield of 343.2 Birr and culling loss of 7500 Birr. At 80% cure rate, total benefits are lowered from 39216 to 31372.8 Birr. The net benefits and BCR at 80% cure rate are therefore 21715.8 (31372.8-9657) Birr and BCR of 3.25, respectively.

When treatment is only 60% effective, this reduces clinical mastitis by 60%. There is a 40% loss from both milk production and culling. At 60% effective treatment, culling benefits and milk sales reduced to 22500 and 1029.6 Birr, respectively. The total benefits are 23529.6 Birr. Therefore at that level of success, the net benefits are 13872.6 Birr (23529.6-9657) and BCR is 2.44, respectively.

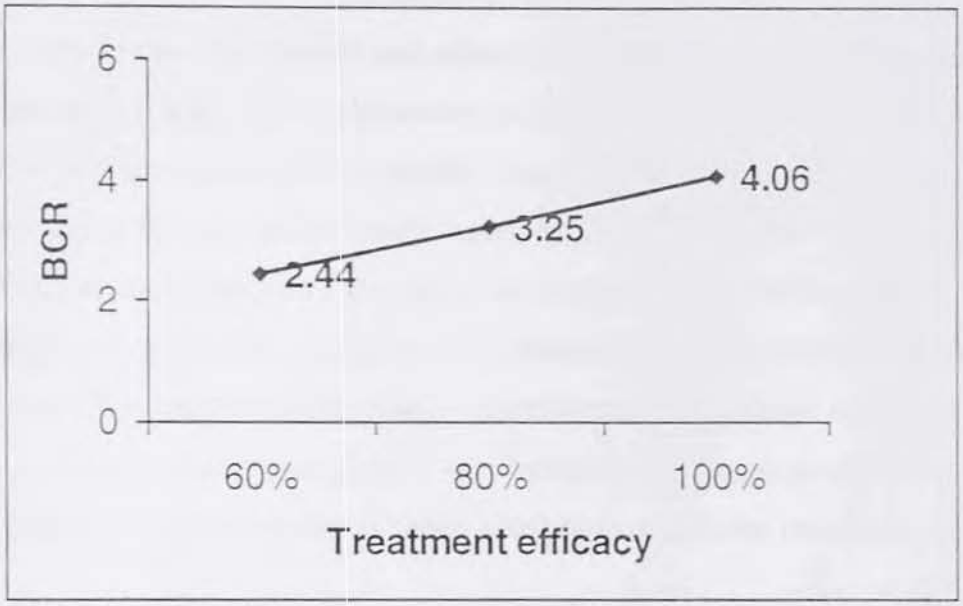

Figure 6: The treatment efficacy and associated changes in BCR

It is clear from this figure that benefit cost ratio and by extension net benefits increase with increase in treatment efficacy and vice versa.

5 DISCUSSION AND CONCLUSIONS

5.1 Discussion

Cow ages were from 2-13 years. These cows had a mean parity of about 3 although many of them were of single parity and some old ones with up to 9 parities. There was no correlation between parity and cow age and it was not uncommon to encounter several cases of older cows with a single parity. This meant that either these cows got their first calf late in life or their inter-calving interval was longer than the expected 12 months. Similar results have been reported by Omore *et al.*, (1996) working on small holder dairies in Kenya and Hussein *et al.*, (1999) who investigated mastitis in the same set-up as the present study.

There were cows in their first month and others twenty-second month of lactation. The lactation length is supposed to be 328 days (Mekonnen *et al.*, 1985). Cows that were milked more than 328 days had gone beyond this established length. Omore *et al.*, (1996) also reported that many of the small holder dairy farmers milked their cows beyond 20 months. Mekonnen *et al.*, (1985) established that in most state run dairy farms in Ethiopia, inter-calving periods were as long as 421 – 497 days with a possible consequence of shortened productive herd life and a lowered life time production. The most probable reasons could be that the farmers are in dire need of income and the only available source was their cows. Furthermore, due to poor nutrition, many of these cows had probably developed anestrus hence silent heat, which the farmers could not detect.

Questionnaire survey addressed a number of aspects including general aspects, dry cow, milking management, housing and drainage and manure handling. All respondents, except one reported no contacts between cows from different herds. This enabled farm owners to control the spread of mastitis and other diseases between herds. When mastitic cows mix with susceptible ones it is possible for the disease to spread through flies (Fox and Gay, 1993). Many such results have been reported in the central highlands of Ethiopia especially by the many unpublished theses conducted by the Faculty of Veterinary Medicine of Addis Ababa University, Bishi, (1998), Frese, (1999) and Hussein, (1999).

Many respondents started dairying by induction possibly by seeing their neighbours with a dairy cow and because many of the males who dominated the industry had been working together as military officers and had time to exchange ideas about dairying. A few other farmers, however having been linked to agricultural research stations started by learning some techniques in those institutions or through the advice of extension agents.

Many respondents could detect mastitis. However, they could only detect the clinical form of the disease. This meant that all sub-clinical mastitis cases went undetected due to lack or no knowledge of necessary diagnostic facilities required like CMT plates and reagent. Only two research farms and one private enterprise farm had the CMT screening facilities. However, screening for sub-clinical mastitis in the three farms was done only when the reagent was available or if milk was rejected on failing the alcohol test before processors took it. Sub-clinical mastitis was a major problem, but incidentally it seemed to go unnoticed despite the fact that it robbed these farmers quite a big deal of money. Mekonnen *et al.*, (1985), Hussein *et al.*, (1997), Bishi (1998), Hussein, (1999), Frese, (1999) and Kassa *et al.*, (1999) in their investigations also reported the problem of sub-clinical mastitis and how difficult it is, to diagnose this form of mastitis.

Time when mastitis occurred varied from respondent to respondent. It could not be quite clearly concluded that season had an influence on mastitis as many of the respondents did not keep records when mastitis occurred. This was contrary to local reports that the rainy season increased incidence of mastitis (Girma, 2001). Therefore, the findings on the temporal prediction of mastitis as given by season need prospective studies to ascertain whether clinical cases increased or decreased with the seasons of the year.

Many farmers practiced zero grazing and hence cows were in total confinement. Large-scale farms with big tracts of land practiced semi-zero grazing. Confined cows do not exercise and are prone to calving-related problems, lameness and periparturient diseases which serve as risk factors to mastitis (Gustafson, 1993). The stall fed animals mostly received dry hay and hence were deprived of vitamin A as hay has very little of β-carotene a precursor of vitamin A (Erskine *et al.*, 1987). Vitamin E, selenium and phosphorus are also abundant in green fodder. Vitamin A aids in the regeneration of epithelial surface and inadequate supply leads to poor and delicate barrier in the teat streak canal, which can easily be penetrated by the bacterial pathogens.

Experimental studies have demonstrated that supplementation with vitamins A, E and selenium reduced the risk of acquiring mastitis (Erskine *et al.*, 1987). A part from the exercise and nutritional benefits, allowing animals to graze during the day allows time for effective cleaning of the barn and time for the animal house to dry before the animals come back in the late afternoon and this reduces the moisture in animal houses and the risk of contracting infections of the environmental origin (Smith and Hogan, 1993).

Many farmers dried off their cows exactly two months before parturition. Cows were dried off gradually or abruptly. Sudden/abrupt drying causes more infections than the intermittent drying (Philpot and Nickerson, 1991). Omore *et al.*, (1996) also reported instances of some people continuing to milk their cows until a few weeks before calving and stopped suddenly when milk changed to colostrum. This was due to lack of sound knowledge on drying cows. Thus, they continued receiving a small income through milk sales. Dry cow therapy was practiced in only 2 farms. Selective or no dry cow therapy increases the risk of acquiring mastitis in the subsequent lactation especially with *Streptococcus agalactiae* (Dargent-Molina *et al.*, 1988).

Milking was done twice on the study farms, except where there were heavy milkers in the early stages of lactation. Before the animals were milked, the udder was washed with cold water without soap or disinfectant on most of the farms. Udder cleaning without antiseptics does not free the teats off microorganisms (Peeler *et al.*, 2000). This served as a risk factor of mastitis as water with microorganisms drips into the teat cup (Schukken *et al.*, 1990). Teats were dried with a shared towel (33.3%) and with separate towel (29.4%). Drying with a shared towel has been demonstrated as a means of spread of mastitis especially of the contagious type (Fox and Gay, 1993). The decision to use a shared towel by some farmers was due to ignorance of the possible outcome and had financial implications also.

Post milking teat dipping was largely not reported on the study farms. Only about 10% practiced it. Other works elsewhere in the region also reported no teat dipping schedules (Omore *et al.*, 1996; Shem *et al*, 2001). A mastitic cow was milked according to how the farmer was informed of mastitis. Those with better understanding of mastitis milked mastitic cows last whereas the majority milked them any time or first. Milking mastitic cows first or any time enables mastitic pathogens to spread through the milkers hands or formites like milking buckets that are not properly cleaned, dirt and contaminated water (Fox and Gay, 1993). Sub-clinical mastitis cases

were milked irrespective of the infectious status because of lack of detection. Hand milking was predominantly done and family members, employees and or a combination did this. Family members dominated the milking on small-scale farms whereas employees were concentrated on the large-scale farms. The employees especially the contract milkers could easily aid the spread of mastitic pathogens from one herd to the other. Others have reported this mode of spread of mastitis in Ethiopia (Bishi, 1998; Hussein, 1999; Mathias, 1999). The milkers washed their hands at the time of udder cleaning and dried it with the same piece of cloth that dried the udder.

Various house types were in use. Majorities were wooden and or muddy walled houses and roofed type but no walls. Very few animal houses had concrete or stone walls. The housing system in use was a reflection of the financial standing of the farmer. The concrete walls symbolized a good financial base and vice versa. These houses had concrete floors, earth floor or arranged stones. Concrete floors are better as they can easily be cleaned and allow management of urine and faeces than the arranged stones or earth floors. Many floors with arranged stones were found in urban production sub-system and were visually unhygienic with swarms of flies of various species. Such farms had poor manure handling. The ammonium compounds from urine predispose to respiratory diseases, which if not checked can spread rapidly through the town population. Many floors had no bedding material. A few had hay and grass. The bedding were mixed with urine and faeces and were potential risk factors for environmental mastitis. Reports have indicated the risk of using straw and other plant material where hygiene of animals is limited as they accumulate wastes from animals and hence encourage growth and proliferation of environmental micro-organisms (Barkema *et al.*, 1998).

The prevalence study reported a proportion of blind quarters of 3.72% which agrees well with 3.8% reported by Kassa *et al.*, (1999). Earlier reports in Ethiopia showed that 10% (i.e. 2.5% quarters) of cows in the large-scale farms especially the State Dairy Farms have at least one blind quarter (Mekonnen *et al.*, 1985). The high proportion of blind quarters could be attributed to improper milking practices and inadequate veterinary attention leading to wrong or no treatment, which in most cases failed to cure clinical mastitis. It would be appropriate to mention that the best way out of the problem would be through realistic and efficient management coupled with proper facilities but not by simple haphazard therapeutical procedures, which are not properly instituted. The prevalence of sub-clinical and clinical mastitis results reported by this study was not in agreement with earlier prevalence estimates in Ethiopia. The estimates were either above

or below those by other investigators. Biru, (1989) reported a combined mastitis prevalence of 67.4% at cow level, which was higher than the present findings of 46.6% and 6.6% for cow sub-clinical and clinical mastitis, respectively. Lower prevalence estimates than the current estimates have been reported in the same production sub-systems (ILCA, 1994; Abdella, 1996; Hussein *et al* 1997; Bishi, 1998; Hussein, 1999 and Kassa *et al.*, 1999). Various findings have been based on stratification of farms as small or large-scale and production sub-systems. Bishi, (1998) reported cow sub-clinical mastitis prevalence of 35.3% and 34.3% for small and large-scale farms, respectively. As for clinical mastitis prevalence, it was 4.4 and 5.7% at cow level for small and large-scale farms, respectively. Kassa *et al.*, (1999) using the same methodology as that of the present study, reported a quarter prevalence of 40% in urban and peri-urban systems and 25.1% in DHIST. The present study reported quarter sub-clinical prevalence of 24.8%, 40% and 26.1% for the DHIST, urban and peri-urban respectively. The findings of this study show that small-scale farms were affected more by sub-clinical mastitis as compared to large-scale farms. The most probable reason could be that large-scale farms had many employees who were able to observe the necessary hygiene measures required than the small-scale farms, which relied on family labour. Clinical mastitis was the opposite of this as many cases occurred on large than on small-scale farms. At the production sub-system level, prevalence of sub-clinical mastitis was higher in the urban production sub-system than in the other two. Urban production sub-system had a problem of manure handling due to lack of adequate land space for better disposal. Clinical mastitis was more prevalent in the peri-urban than in DHIST and urban production systems.

A recent study in Tanzania reported cow sub-clinical mastitis prevalence of 62% and clinical mastitis of 4% (Shem *et al.*, 2001). The clinical mastitis estimate was however, lower than what the current study established. Cow sub-clinical prevalence on the small holder dairy farms in Kiambu District of Kenya was estimated at 71% (Omore *et al.*, 1996). Other studies in Kenya (Lauerman *et al.*, 1973; Hamir *et al.*, 1978; Ngatia, 1988) have reported cow sub-clinical mastitis prevalence which was above what the current study established. Another study in Malawi reported 63% and 28% of cow and quarter sub-clinical mastitis respectively (Klastrup and Halliwell, 1977).

The current study had short falls since prevalence estimates were determined by screening tests only. Clinical measurement on a continuous scale (indirect somatic cell counts through CMT), is a problem as a result of overlap between healthy and diseased individuals based on the cut off

points. There was a likelihood of over and or under estimation than would be if microbial isolation was used as a gold standard. However, caution in the definition of a sub-clinical case was taken by raising the cut-off points for the CMT scores where a positive case was defined by CMT score 1 and above otherwise negative (De Graaf and Dwinger, 1996). The study set a high cut off-point criterion to identify cases of sub-clinical and clinical mastitis to avoid inclusion of many false positive cows. However, with this criterion, false negatives could not be avoided. Some of the "Trace" scores, which were considered negative, would have yielded a minor/major pathogen isolates on culturing. Hence, these were probably cases that were missed out (false negatives). The strength of the decision to avoid this was based on the fact that local findings indicated that CMT score of 1 and above gave a very high probability of isolating a microorganism (Hussein *et al.*, 1997).

The results indicated that based on quarter level, the frequency of occurrence of sub-clinical (388 quarters) mastitis was almost 10 times that of clinical mastitis (39 quarters). This is a lower estimate as compared to 25% quarter and 50% cow infection rates in many mastitis surveys world wide (Radostitis *et al.*, 2000). Considering the number of cows involved in both sub-clinical and clinical mastitis, the quarter infection rate was 2.29 and 1.62 quarters per cow for sub-clinical and clinical mastitis respectively.

Cows aged at least 8 years, with at least 8 parities, at least 8th month of lactation and poor body condition scores had a positive statistical association with sub-clinical mastitis. Cows aged at least 4 years, with at least 8 parities, cows in at least 4th month of lactation, with poor body condition scores, with a problem of leaking milk and a history of udder problem had a higher risk of contracting clinical mastitis. Protective factors for both sub-clinical and clinical mastitis were breed, good and fair body conditions, teat drying, dry cow therapy, time for drying off and dry off style. Dry cow therapy was however dropped in the final model for clinical mastitis. However, it was not possible to tell whether for example it was drying off cows at exactly 2 months before calving or later that offered protection. The same was for drying off style (gradual versus sudden), teat drying (shared versus separate towel) and breed (local verses cross). Other workers have reported mixed findings on mastitis risk factors although many of them considered this in the context of clinical mastitis and did bacterial isolation. Peeler *et al.*, (2000) reported an increase in clinical mastitis incidence in herds that had at least a cow with leaking milk inside or outside the milking parlour. Cows in their early and mid lactation and thin cows had increased

risk of clinical mastitis (Suriyasathaporn *et al.*, 2000). Schukken *et al.*, (1989) demonstrated that older cows with at least 4 parities were prone to mastitis than young cows. The same result was reported by Dulin *et al.*, (1988). The findings of this study on parity, leaking milk and body condition scores were in agreement with the findings reported elsewhere (Peeler *et al.*, 2000; Suriyasathaporn *et al.*, 2000). Leaking milk on entering the parlor and leaking milk at other times are important risk factors of mastitis because this could increase the exposure for other cows in the parlor to mastitis pathogens, as bacteria from infected quarters may be present in the milk (Peeler *et al.*, 2000). Similarly, leaking milk outside the parlor may result in increased levels of mastitis pathogens in the environment. Alternatively, cows that leak milk outside the parlor may themselves be at greater risk of mastitis, because they may have wider teat canals, which is associated with an increased risk of penetration by mastitis pathogens (Lacy-Hubert and Hillerton, 1995). It is an established fact that breed, dry cow therapy, and use of separate towels is a protective factor of mastitis. These findings were confirmed by the current study. Dargent-Molina *et al.*, (1988) also reported that use of dry cow therapy and shared towel protected cows against mastitis. Mastitis research in Ethiopia revealed that parity above 3, cows in at least 4 months of lactation and older cows have identified as risk factors of mastitis (Hussein *et al.*, 1999). Otherwise, the subject of mastitis risk factors still remains barren and therefore calls for more prospective studies under the local conditions to be able to come up with much more information before any effective preventive or control strategy can be adopted.

A split udder investigation to determine losses due to sub-clinical mastitis revealed that on average, a quarter produces 0.82 kg (95% 0.42-1.22 kg) per milking hence a total of 6.56 kg per cow per day. This was lower compared to the established 8.8 kg per cow per day, an average of 2.2 kg per quarter (Mekonnen *et al.*, 1985). The lower yield in those experimental animals was not surprising, as the animals selected had sub-clinical mastitis and were not for production purposes but for research. The selection criterion did not consider the stage of lactation and parity of these animals but the infection status irrespective of the 2 factors, an issue that would help explain the low productivity. Quarters with CMT scores 0, 1+, 2+ and 3+ yielded 0.9188 kg, 0.9080 kg, 0.8610 kg and 0.616 kg per milking, respectively. Loss per quarter was subsequently 0 kg, 0.0108 kg, (1.2%) 0.0578 kg (6.3%) and 0.303 kg (33%) for CMT score 0, 1+, 2+ and 3+, respectively. The loss due to CMT scores 1+, and 2+ are less than those of CMT score 3+. This is because CMT 1+ and 2+ are less established compared to those of CMT score 3+ which are long standing and well established with extensive destruction of the udder tissue hence the big losses.

Similar work reported that the production loss was 0 kg, 0.27 kg, 0.991 kg, 1.76 kg and 2.61 kg for CMT scores 0, 'trace', 1+, 2+ and 3+ respectively (Dobbins, 1977). Other possible reasons for the lower estimates could be poor management, inferior genetics coupled with nutritional inadequacies leading to lower productivity. The tropical heat stress was another factor, which could explain the lower figures. The average quarter milk loss due to sub-clinical mastitis for the crossbred cows in Ethiopia was estimated to be 17.1%, which was comparable to 17.6% reported in Costa Rica (De Graaf and Dwinger, 1996). Otherwise, no local research reports on quarter milk production losses due to sub-clinical mastitis are available for comparison purposes.

Mastitis losses were estimated to be 76513.1664 Birr i.e. 210.8 Birr per cow per lactation. This loss could easily be reduced or totally avoided with proper mastitis control measures in place. Milk production losses contributed 38.4% of the total losses. Sub-clinical mastitis contributed 94% and clinical mastitis 6% of the milk losses. Sub-clinical mastitis losses contribute 36.1% of the total losses which, are primarily due to reduced milk production. Primary findings and reviews have reported higher percentages than this. Scheppers and Dijkhuizen, (1991) in a review of mastitis losses since 1970 reported that 70% of mastitis losses are due to reduced milk yield. Singh and Singh (1994) reported that reduced milk yield contributed 68% of the total mastitis losses. Singh and Singh, (1994) overestimated the milk losses as they assumed that a cow remained infected throughout an entire lactation phase an assumption that was not wholly in this present work.

Mastitis annual culling rate was estimated at 7.32%. This was lower compared to other local reports. Mekonnen *et al.*, (1985) and Workineh, (1998) who reported mastitis culling rates of 10 and 8.9%, respectively. Mastitis culling in relation to total culling of dairy cows was 24.7% as compared to 22.5% (Workineh, 1998). Culling contributed 49% of the losses. Other works on mastitis losses assumed a culling rate of 1% and this resulted in lower rates than that of this study (Singh and singh, 1994). Culling contributed immensely to mastitis losses due to poor cure rates following treatment. The reason for poor cure rates could have been as a result of incomplete treatment regimens and probably due to incurable mastitis types like *Arkanobacter pyogenes* (Radostits et al., 2000). Culling a cow due to mastitis causes triple loss to the farmers because of lost production time due to early culling, genetic resource loss for the higher yielders and the associated replacement costs. However, the cost of reduced time in production and genetic resource loss following culling could not be easily quantified although they are of importance in

such work. Treatment cost accounted for 9.3% of the total mastitis losses. Scheppers and Dijkhuizen, (1991) reported a treatment cost inclusive drugs and veterinary charges of 8%. This was almost agreeing with the current estimate. The high percentage was expected since mastitis control strategies in the Sub-Saharan Africa are non-existent or where practiced they are poorly instituted hence of little consequence. This in return increases the budgets for treatment. Withdrawal losses were 3.3%. Scheppers and Dijkhuizen, (1991) reported 8%, which is a little, bit higher than what the present study reported. Nobody however, discarded all the milk from treated cows on the study farms. It was therefore, evident that consumption of antibiotic laden milk by people was rampant on the study farms.

The partial budget analysis outcome indicated that farmers are making some profit from their cows despite clinical mastitis as the net benefits were greater than 1 and the Benefit cost ratio (BCR) also greater than 1. The net benefits of 29559 implied that farmers could make that difference by effectively controlling clinical mastitis through treatment. On the other hand, benefit cost ratio of 4.06 meant that incurring a treatment cost of 1 Birr due to a case of clinical mastitis resulted in a return of 4.06 Birr. However, this was an ideal situation where treatment was assumed to be 100% effective. Otherwise, there is always uncertainty about treatment efficacy, which is unavoidable. A treatment efficacy of 80% for example, reduces net benefits and BCR by 20%. The net benefits are 21715.8 Birr and the BCR is 3.25 from 29559 and 4.06, respectively when cure rates are 100%. This decrease is accounted for by the cases not cured and culling benefits that reduced to only 80%. At 60% efficacious treatment, reduced clinical mastitis by 60%. Forty percent of the cases are not cured and hence a drop in milk yield of 40%. Culling rate of 40% would still be done. Generally, treatment that is less than 100% effective lowers the returns to farmers since BCR decreases with decrease in treatment efficacy. It is therefore evident that treatment alone can not effectively control clinical mastitis and hence the need to supplement this with other control strategies.

5.2 Conclusions and recommendations

Mastitis and especially sub-clinical mastitis is a problem, which escapes the notice of farmers. Diagnosis of sub-clinical mastitis is almost non-existent due to lack of the necessary kits (California Mastitis Test reagent mostly). No control strategies of mastitis are in use currently.

These therefore, require clear-cut preventive and or control measures to be instituted in order to reduce the high mastitis prevalence and minimize the associated economic losses. In view of this, the following recommendations are suggested:

1). Milking and general hygiene practices

- Farmer education (awareness campaigns) on the control of spread of mastitis e.g. of use of separate towels on each milking cow instead of using shared towels and dry cow therapy adoption could be of use.
- Milking of mastitic cows last to avoid spread of mastitis through contaminated milkers' hands.
- Farmers should attempt to improve hygienic standards like use of post milking teat dipping using iodine solutions and also use of detergents like soap, which can cheaply be acquired. Farmers could also use milking salves to apply to teats before and after milking to reduce teat abrasions/lacerations (teat lesions).
- Encourage regular screening of cows for sub-mastitis in order to improve surveillance activities for any control strategy to be adopted. This is an area that calls for an integrated approach by all stakeholders like the dairy farmers, the Government through the relevant Ministries, Researchers and Field Veterinarians. The lack of the required diagnostic kits (California Mastitis Test reagent and other facility-aided diagnosis) should be addressed through favourable institutional policies. Otherwise, the continued scarcity of this will continue delaying mastitis surveillance and control.

2). Improve the management level to alleviate the identified risk factors

- Culling of cows with problem of leaking milk and those with chronic mastitis, which does not respond to therapy. Older cows to be culled also to as they stand a higher risk of contracting clinical mastitis.
- Improved feeding to help offset the negative energy imbalances, which predispose to poor body condition scores. The feeding should also be encouraged after milking, as many of the cows tend to lie down immediately after being milked.
- Lactation to be strictly 328 days or shorter in order to adhere to good drying practices instead of sudden drying which increases the risk of occurrence of mastitis.

- Housing practices, manure handling and water and sewerage management to be improved.

3). Therapeutic interventions

This should come as the very last option as it is sought to help control those cases that occurred despite the preventive and other control strategies listed above. The strategies include:

- Encouraging regular antimicrobial sensitivity testing to select effective antibiotics and also help reduce the problem of resistance development towards commonly used antibiotics. This is a policy issue that the Addis Ababa Dairy Producers Association in collaboration with relevant Ministry can help enforce. Research results/recommendations have to be implemented, and where they are deficient new prospective studies conducted on this subject.
- Treatments given to be timely and complete as many of the clinical cases encountered were chronic. Farmers to seek the advice and help of professionals on all matters to do with treatments to mastitic cows.

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7 APPENDICES

Appendix 7.1: Management factor's questionnaire

a). Herd description and farm structure

Farm owner---------- Sex-------- Age------ years

Farm physical address---------- tel. (if any)

Location: a) urban----------

b) Peri-urban----------

c) Dairy herds in secondary towns----------

Farm size---------- Ha.

Herd structure a) Cattle---------, b) Goats, --------c) Sheep------d) other-----

Breeds of cattle owned---------, ----------, ----------, ---------- and---------

Number of each (cattle) breed----------, ---------, --------, -------, and -------

Cows-------, Heifers --------, Bulls --------, Oxen ------and calves

Farm fenced? Yes----- No------

Is there frequent contact between your animals and other herds? Yes----- No -----

b). General aspects

How did you acquire skills to raise dairy cattle? a) Through agricultural training --------

b). Through extension agents --------

c). Through induction -------

How do you know that a cow has mastitis?

a). Swollen udder, painful on touch and abnormal secretions -------

b). Reduced milk yield -------

c). Use of Strip cup--------

d). CMT screening --------

e). Combination of all the above--------

f). Can not tell-------

What grazing type do you practice on your farm? a). Stall (zero grazing)------ b). Semi-zero grazing------ c). Extensive system------

When do you experience many episodes of mastitis? a). During the rain season---- b). In the dry season------ c). Both seasons-------d). Never experienced---------

c). Dry cow practices

Do you practice dry cow therapy? Yes ------ No -------

When do you dry off your cows? a). 2 months before calving -------

b). 1 month before calving ---------

c). When milk turns yellow ---------

d). Not at all --------

How do you dry cows? a). Gradually/intermittently --------

b). Abruptly/suddenly---------

What do you use for drying off cows? a). Nothing --------

b). Intra-mammaries -------

c). Lugols iodine -------

d). Both chemicals above --------

d). Milking procedure/management practices

How many times do you milk your cows in a day? a). Once --------

b). Twice -------

c). Thrice/more -------

What time do you clean teats? a). Before milking -------

b). After milking --------

c). Both 'a' and 'b' above-------

d). No udder cleaning -------

What do you clean teats with? a). Water alone--------

b). Water + Disinfectant/Antiseptic-------

c). Dry towel---------

What do you use for drying teats? a). Separate towel/cow-------

b). Shared towel --------

c). Dried by milkers' hands------

d). Left to air dry-------

When do you dip teats? a). Before milking-------

b). After milking---------

c). Both 'a' and 'b'-------

d). No dipping--------

What do you use for teat dipping? a). Ammonium compounds--------

b). Lugols iodine/tincture of iodine--------

c). Iodophors----------

d). Other (specify)-------

When do you milk cows with mastitis? a). First------

b). Last------

c). Any time-------

Are cows milked completely? Yes------ No------

Who milks cows? a). Family members only------

b). Contract milkers-------

c). Employees of the farm------

d). Both 'b' and 'c'-------

e). A combination of all above------

Do they wash between milking every cow? Yes----- No-----

What is used for hand drying? a). Reusable hand towel-------

b). Disposable towel-------

c). Hands air dried-------

e). Milk when hands still wet-------

e). Housing practices

What type of house is in use? a). Closed (roof + wooden walls)---------

b). Closed (roof + concrete walls)---------

c). Open, covered (roof, but no walls)-------

d). Other (specify) -------

What is the floor type? a). Concrete------

b). Earth-----

c). Arranged stones------

Is the roof rainproof? Yes----- No------

Does sunshine reach inside animal houses? Yes----- No------

Does the construction of houses allow for sufficient ventilation? Yes ---- No -----

Are cubicles spacious enough (220 * 110cm)? Yes-----No-------

What is the bedding type in animal houses? a). Grass--------

b). Wood shavings-------

c). Straw/maize stover---------

d). Nothing on the floor---------

Are there dry cow/maternity facilities on the farm? Yes------ No----

Are the dry cow/maternity facilities regularly cleaned and disinfected? Yes----- No -----

f). Drainage and manure handling/general farm hygiene practices

Does the house allow free and unhindered movement of slurry (animal waste)? Yes----- No ------

How frequent is manure collected from animal houses? a). Daily----- b). Twice a week----- c). Weekly------ d). Fortnightly------e). Other (specify)-------

Where is the collected manure disposed off? a). Near animal houses----- b). in the crop farms near by----- c). Littered all over the farm----- d). No proper disposal system-----

Do you have a problem of flies next to the animals' house/on animals? Yes----- No------

What is the general sanitary situation on the farm? a). Excellent------ b). Good------ c). Satisfactory----- d). Poor-------

Appendix 7.2: Clinical examination/inspection form

Date of examination ---------- Area/Animal serial No.----------

Owner's name---------- Address----------

Animal identification: Breed----------Age---------- Calving date---------- Parity----------

- Any previous history of udder problem/infection? Yes-------- No-------

- **Physical Examination**

i). General examination: a). Demeanour: Discharges from udder/no discharges

Leaking milk/no leaking milk

b). Body condition: i). Good----, ii). Fair---- and iii). Poor----

ii). Palpation of teat canal, teat cistern, wall of teat, udder skin and glandular tissue of each quarter for;

a) udder skin temperature: warm/cold

b) swelling: swollen/not swollen

c) patency of teats: patent/blocked

d) Gross milk quality: i). Watery-------- ii). Blood tinged------- iii). Clots/flakes in milk------ and iv). Normal milk---------

e)

- **Form of clinical mastitis**

  i). Acute-------, ii). Sub-acute---------- and iii). Chronic--------

- **Remarks/comments**----------

Appendix 7.3: Economic data questionnaire sheet

Farm owner---------- Sex------- Age------ Years

Occupation---------- Farm physical address--------- Tel (if any)---------

Location of the farm:

a). Urban------

b). Peri-urban------

c). Dairy herds in secondary towns

I). Mastitis Treatment data

Is mastitis a problem on this farm? Yes----- No------

What is done to a mastitic cow when identified?

a). Nothing-----

b). Treated------

c). Not treated but culled------

d). Continued to be milked-----

II). Producer Prices

How much on average do you sell a liter of milk? Please mention the figure------ Birr.

On average how much is a normal lactating dairy cow? Please mention the figure----- Birr.

How many animals have you culled on this farm in the last i). 1 year------? ii). 2 years-------? iii). 3 – 5 years--------? Please mention the number. (to be checked for those with records).

Out of the said number of culls, how many cows were culled due to mastitis? Please mention number------

How much are you paid for a culled cow? Please mention the figure----- Birr.

iii). Veterinarians/Drug shops

How much do you charge per treatment when you go out? Please mention figure-------

How many times do you treat a case of mastitis?

a). Once-----

b). Twice-----

c). Thrice-----

d). Five/more times------

What is used for treating mastitis on this farm?

a). Nothing-------

b). Intramammary tubes-----

c). Parenteral antibiotics + corticosteroids------

d). Lugols iodine------

e). Other (specify)------

In case you have to use intra mammary tubes how many do you use on an affected quarter? Please mention number-------

If you supplement intramammaries with parenteral antibiotics, how much do you charge per cow? Please mention figure for Oxytetracycline---- Pen and strep--- Amoxiccillin---- other-----

Appendix 7.4: Farm sub-clinical mastitis prevalence

Farm	Prod	n	Positive	Negative	Missing	SCM Prev.
1	DZ	6	3	3	0	50
2	DZ	1	0	1	0	0
3	DZ	2	1	1	0	50
4	P	44	29	13	2	69.04
5	P	2	0	2	0	0
6	U	2	1	1	0	50
7	P	2	2	0	0	100
8	P	18	2	16	0	11.11
9	DZ	1	1	0	0	100
10	DZ	1	1	0	0	100
11	DZ	57	21	36	0	36.84
12	DZ	1	0	1	0	0
13	P	28	16	12	0	57.14
14	U	7	5	2	0	71.43
15	DZ	22	7	15	0	31.82
16	DZ	5	3	2	0	60
17	U	2	2	0	0	100
18	U	2	1	1	0	50
19	U	1	1	0	0	100
20	DZ	2	2	0	0	100
21	U	3	1	2	0	33.33
22	DZ	3	1	2	0	33.33
23	DZ	2	0	2	0	0
24	P	28	9	19	0	32.14
25	P	3	1	2	0	33.33
26	P	1	0	1	0	0
27	DZ	2	2	0	0	100
28	DZ	2	0	2	0	0
29	DZ	1	0	1	0	0
30	DZ	1	0	1	0	0
31	U	1	0	1	0	0
32	P	1	0	1	0	0
33	DZ	2	0	2	0	0
34	P	8	5	3	0	62.5
35	DZ	4	3	1	0	75
36	U	2	2	0	0	100
37	DZ	2	0	2	0	0
38	U	1	0	1	0	0
39	P	1	0	1	0	0
40	DZ	5	4	1	0	80
41	DZ	1	0	1	0	0
42	DZ	7	5	2	0	71.43
43	DZ	1	0	1	0	0
44	DZ	13	8	5	0	61.54
45	P	17	9	8	0	52.94
46	DZ	2	1	1	0	50
47	DZ	1	0	1	0	0
48	P	1	1	0	0	100

49	P	13	6	7	0	46.15
50	DZ	17	10	7	0	58.82
51	P	11	3	6	2	33.33

Appendix 7.5: Farm clinical mastitis prevalence.

Farm No.	Prodn.sys.	Sample size	Positive	Clin. prev
1	DZ	6	0	0
2	DZ	1	0	0
3	DZ	2	0	0
4	P	44	5	0.113
5	P	2	0	0
6	U	2	0	0
7	P	2	0	0
8	P	18	0	0
9	DZ	1	0	0
10	DZ	1	0	0
11	DZ	57	0	0
12	DZ	1	0	0
13	P	28	4	0.143
14	U	7	0	0
15	DZ	22	0	0
16	DZ	5	1	0.2
17	U	2	0	0
18	U	2	0	0
19	U	1	0	0
20	DZ	2	0	0
21	U	3	1	0.333
22	DZ	3	0	0
23	DZ	2	0	0
24	P	28	3	0.107
25	P	3	1	0.3333
26	P	1	0	0
27	DZ	2	0	0
28	DZ	2	0	0
29	DZ	1	0	0
30	DZ	1	0	0
31	U	1	0	0
32	P	1	0	0
33	DZ	2	0	0
34	P	8	0	0
35	DZ	4	0	0
36	U	2	0	0
37	DZ	2	0	0
38	U	1	0	0
39	P	1	0	0
40	DZ	5	1	0.2

41	DZ	1	0	0
42	DZ	7	0	0
43	DZ	1	0	0
44	DZ	13	1	7.69
45	P	17	2	0.118
46	DZ	2	0	0
47	DZ	1	0	0
48	P	1	0	0
49	P	13	1	7.69
50	DZ	17	1	5.88
51	P	11	3	0.273

Appendix 7.6: Summary quarters CMT scores

LF CMT scores	RF CMT scores
0 = 230	0 = 227
1 = 17	1 = 12
2 = 23	2 = 19
3 = 24	3 = 32
4 = 51	4 = 55
9 = 18	9 = 18
Total = 363	Total =363
LR CMT scores	**RR CMT scores**
0 = 227	0 = 233
1 = 17	1 = 14
2 = 17	2 = 11
3 = 27	3 = 28
4 = 49	4 = 52
9 = 26	9 = 25
Total = 363	Total = 363

* CMT scores 0, 1, 2, 3, 4, and 9 represent negative, trace, 1+, 2+, 3+ and blind/clinical mastitic quarter.

CMT score 3+ = 207 quarters, CMT score 2+ = 111 quarters CMT score, 1+ = 70 quarters and CMT score trace = 60 quarters.

Appendix 7.7: Curriculum vitae

BIODATA

Name:	Erick Ouma Mungube
Date of birth:	25th May 1970
Place of birth:	Busia, Kenya
Nationality:	Kenyan
Marital status:	Single

Language (Spoken/written): English, Kiswahili and Luhya

Permanent Address: P.O Box 8403 Ronald Ngala Post Office, Nairobi.

EDUCATIONAL BACKGROUND

1978 – 1985:	Primary education Kenya ceritificate of education (KCPE)
1986 – 1989:	Secondary Education Kenya certificate secondary education (KCSE)
1990 – 1997:	University of Nairobi Bachelor of Veterinary Medicine (BVM)
2000 – 2001:	Free University of Berlin/Addis Ababa University, Joint MSC Program in Tropical Veterinary Epidemiology & Preventive Medicine. Courses covered-Veterinary Epidemiology (Qualitative & quantitative epidemiology),

Bisotatics, Veterinary public Health, Livestock Economics Animal health and Preventive medicine, GIS, Livestock Production systems, and Tropical animal diseases.

Special skills: Computer literate. Good working knowledge with MS Word, MS Access, MS Excel, Statgraphics and Stata. Some knowledge in GIS

Research interests: Bacterial especially zoonotic and haemoparasitic diseases

Publications

1). Mungube, E.O., Tenhagen, B.A., Kassa,T., Regassa, F., Kyule, M.N., Shiferaw, Y (2002): Relationship between CMT scores and quarter milk production in Ethiopian Crossbred dairy cattle. A paper to be presented at the XXII World Buiatrics Congress. Hannover, Germany, 18-23 August, 2002.

Work experience

1997 – 1999: Private Veterinarian based in Busia and Nairobi.

Membership: Registered Veterinarian by the Kenya Veterinary Board and Kenya Veterinary Association.

Referees

1). Dr Baumann Maximillan, P. O
Course Co-ordinator, postgraduate studies
in International Animal Health
Luisenstrasse 56 D-10117, Berlin
Tel. + 49-30-20936125
Fax + 49-30-20936349
Official e-mail: Tropvet@city.vetmed.fu-berlin.de
Personal e-mail mbaumann@city.vetmed.fu-berlin.de

2). Dr Joseph, K. Litamoi
c/o FAO, OAU/IBAR Project

Vaccine Specialist, PANVAC

P.O Box 5536 Addis Ababa, Ethiopia/or Box 501 Kitale,

Kenya/or Box 53260 Nairobi.

Tel. 251-1-338001

Fax 251-1-338844

Official e-mail: panvac@telecom.net.et

Personal e-mail: litamoi@africaoline.co.ke

3). Dr Moses, N. Kyule

International Animal Health

Luisenstrasse 56 D-10117, Berlin

Tel. + 49-30-20936125
Fax + 49-30-20936349

Official e-mail - kyule@city.vetmed.fu-berlin.de

Appendix 7.8: Declaration sheet

I, the undersigned, declare that the thesis is my original work and has not been presented for degree in any University.

Name: Erick Ouma Mungube

Signature.....

Date of submission: 23 November 2001

This thesis has been submitted for examination with our approval as University advisors

1). Dr Bernd-Alois Tenhagen Freie Universität Berlin

2). Dr Tesfu Kassa Institute of Pathobiology, Addis Ababa University

3). Dr Fekadu Regassa Addis Ababa University

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Erick Ouma

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