

Institute of Animal Breeding and Animal Husbandry with Veterinary Clinic

Faculty of Agriculture

(Dean: Prof. Dr. agr. habil. W. Merbach)

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Investigations of factors affecting the udder health status of dairy cows in Thuringia

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Submitted for the degree of Doctor of Agricultural Sciences

by

Abdel-Aziz Ahmed Fadel-El-Moula

(B.V.Sc.; M.Sc.)

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A dissertation

For the award of the degree
Doctor agriculturarum (Dr. agr.)

By
Abdel-Aziz Ahmed Fadel-El-Moula
(B.V.Sc.; M.Sc.)

Born in 01.09.1963
Barakat, Sudan

Reviewers: Prof. Dr. habil. H. Swalve
Prof. Dr. E. von Borell
Dr. habil. G. Anacker

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Halle/Saale 2002

Dedication

To

My parents, brothers and sisters

My sweetie children, Rayan, Omer and Ayat

With love

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ABBREVIATIONS

a.p.	ante partum
ADR	Arbeitsgemeinschaft Deutscher Rinderzüchter
ATP	Adenosintriphosphate
<i>C. bovis</i>	<i>Corynebacterium bovis</i>
C.V.	Coefficient of Variation
CMT	California Mastitis Test
CNS	Coagulase Negative Staphylococci
DMRT	Duncan's Multiple Range Test
DVG	Deutsche Veterinärmedizinische Gesellschaft
<i>E. coli</i>	<i>Escherichia coli</i>
EPS	Esculin Positive Streptococci
FMI	Finish Milk Inspection
FQL	Fore Quarter Left
FQR	Fore Quarter Right
GLM	General Linear Model
HQL	Hind Quarter Left
HQR	Hind Quarter Right
IDF	International Dairy Federation
IMI	Intra-Mammary Infection
LKV	Landeskontrollverband
LS	Least Square
MLP	Milchleistungsprüfung
n.s.	not significant
OR	Odds Ratios
p.p.	post partum

REML	Restricted Maximum Likelihood
<i>S. aureus</i>	<i>Staphylococcus aureus</i>
S.E.	Standard error
SAS	Statistical Analysis System
SCC	Somatic Cell Counts
SCS	Somatic Cell Score
<i>St. agalactiae</i>	<i>Streptococcus agalactiae</i>
<i>St. dysgalactiae.</i>	<i>Streptococcus dysgalactiae</i>
Std.	Standard Deviation
TLL	Thüringer Landesanstalt für Landwirtschaft
TMR	Total Mixed Ration
TVL	Thüringer Verband für Leistungs- und Qualitätsprüfung in der Tierzucht e.V.
VIT	Vereinigtes Informationssystem Tierhaltung

1 INTRODUCTION

In recent years the demand for liquid milk increased tremendously worldwide, due to increased population growth. The consumers in the industrialized countries demanding food not only to be economical, but also safe and sound in respect to animal welfare and the environment. Klaas, (2000) conducted a field study in Germany and found that the number of dairy farms decreased at the time that herd size grow-up. The average number of the dairy herds in the season 2000/2001 in Thuringia (TVL-annual report, 2000) which enrolled in the MLP-organization was 135.0, which comprises 132946 lactating cows producing an average 7198 kg milk, 307 kg fat (4.26%) and 247 kg protein (3.44%) (ADR, 2001). The average somatic cells count was 205×10^3 cells/ml in 572 dairy farms.

Mastitis is the costliest disease of dairy industry today with an annual losses in USA estimated to be 200 \$ per cow (David and Shearer, 1996). Whereas in Germany mastitis losses/cow/year estimated to be 285 \$ broken down as 165 \$ representing a 10% loss of the mean yield, 20\$ treatment costs and 100 \$ reduced useful life (Hamann, 2001).

In this study, 48 dairy farms were included as a part of an udder improvement project in collaboration with Thuringia center of agriculture (TLL), with the following themes:

1. Surveying the spectrum of mastitis causing pathogens in a cow level and udder quarter and the frequency of the pathogens with respect to herd size, number and stage of lactation, year-season and farm management and hygienic factors.
2. Studying the effects of herd size, lactation number, stage of lactation, year-season and farm management and hygienic factors with respect to contagious and environmental bacteria isolated on infection rate and lactation SCC.
3. Investigating the effects of herd size, lactation number, stage of lactation, year-season and farm management and hygienic factors depending on the class of lactation SCC on test-day milk yield.
4. Over-viewing IMI in heifers, investigating the effect of positive findings and time of sampling before and after calving and the effects on future milk yield and SCC.
5. Identifying, assessing and quantifying the risk factors associated with IMI and high SCC.

2 LITERATURE REVIEW

2.1 Intra-mammary infection (IMI)

In most countries, dairy cattle breeding programs are directed toward milk production traits. Although these traits are of primary economic importance, functional traits such as longevity fertility and udder health are of increased interest to producers to improve herd profitability.

Mastitis is defined as an infection of the udder, caused by bacteria entering the quarter through the teat end (Rodenburg, 1990), and according to the US national mastitis council's current concepts of bovine mastitis (1996): mastitis is an inflammation of the mammary gland in response to injury for the purpose of destroying and neutralizing the infectious agents and to prepare the way for healing and return to normal function. Inflammation can be caused by many types of injury including infectious agents and their toxins, physical trauma or chemical irritants (Jones and Bailey, 1998). Mastitis is one of the most common dairy diseases (Rajala-Schultz et al. 1999) because of its high incidence (Seegers et al. 1997a and Seegers et al. 1997b). The economic consequences of mastitis either clinical or sub-clinical include loss of milk production, loss of milk sales, increased culling rates, and cost for veterinary treatments, in addition to that high SCC in milk affect the price of milk in many payment systems that are based on milk quality (Schukken et al. 1997). Milk cell count has been used extensively as an indicator of the infection status of the mammary gland (Hillerton, 1999). The German Veterinary Medicine Association (DVG, 1994) categorized the udder health status as shown in table 1.

Table 1: Categorization of udder health status (DVG, 1994)

Cell count per ml milk	Pathogenic organisms	
	Negative	Positive
$< 100 \times 10^3$	Normal secretion	latent infection
$> 100 \times 10^3$	Non-specific mastitis	mastitis

The legal maximum bulk tank SCC is lower in other dairy exporting countries than USA (Smith and Hogan, 1998). Canada has a limit of 500×10^3 cells/ml, in the European community, Norway, Switzerland, Australia and New Zealand the maximum bulk tank SCC is 400×10^3 cells/ml. In those countries, SCC is calculated as a geometric mean of

successive milk shipments over several weeks, therefore, it is expected to be lower than arithmetic mean (Shook and Ruegg, 1999).

2.2 Classes of mastitis

2.2.1 Clinical mastitis

Clinical mastitis is defined as an infection of the udder that results in visible changes in the udder quarter and milk (Rodenburg, 1990), may it be acute, sub acute or chronic. The development of clinical mastitis in dairy cows can be detected with high sensitivity and specificity in advance of visible changes in foremilk or udder tissue by determining the electrical conductivity of the foremilk (Milner et al. 1997). Weller et al. (1992) and Pösö and Mantysaari (1996) stated that the genetic correlations between clinical mastitis and SCS among different lactations were positive and moderate to high (varied from 0.37 for the first lactation to 0.68 for the third lactation). Whereas Mrode and Swanson (1996) estimated a genetic correlation between SCC and incidence of mastitis of 0.7. Peeler et al. (2000) in a study to assess the level of clinical mastitis and to quantify risk factors associated with the incidence rate of clinical mastitis in U.K, found a mean incidence rate of clinical mastitis of 22.8 cases per 100 cows/year. They also reported that the incidence rate of clinical mastitis increased when farmers reported that they had straw yard housing for milking cows (compared with cubicle housing), mucked out the calving area less frequently than once per month, when they had greater than 50% replacement rate and when always practiced post-milking teat disinfection. Barkema et al. (1999) attributed the increase in the incidence rate of clinical mastitis in herds practicing post-milking teat disinfection to *E. coli* infections. While Wilson and Kingwill (1975) and Wilesmith et al. (1986) claimed that the incidence rate of clinical mastitis in Great Britain has declined from an estimated 120 cases per 100 cows/year in 1960 to approximately 40 cases per 100 cows/year in 1986 due to a reduction in mastitis caused by contagious pathogens particularly *S. aureus*, *St. agalactia* and *St. dysgalactia* through the introduction of improved control measures. Booth (1988) reported that the reduction in the prevalence of contagious pathogens resulted in a decrease of the average bulk milk SCC from 573×10^3 cells/ml to 352×10^3 cells/ml. But Barkema et al. (1998) showed in a recent study that there was no association between bulk milk SCC and incidence rate of clinical mastitis. Aarestrup and Jensen (1997) found that the presence of bacteria in a quarter before parturition increased the risk of IMI for the lactating cow. And the variability in the prevalence and the duration of intra mammary infection according to the bacterial species

occurred around the first parturition. Lescourret and Coulon (1994) and Schukken et al. (1997) reported that mastitis has many economic consequences among which are loss of milk production, loss of milk sales, increased culling rates and cost for veterinary treatments, in addition to that high SCC in milk affects the price of milk. Rajala-Schultz et al. (1999) studied the effect of clinical mastitis on milk yield in dairy cows, they found that the daily loss during the first 2 weeks after the occurrence of mastitis varied from 1.0 kg to 2.5 kg and the total loss over the entire lactation varied from 110 kg to 352 kg; cows with mastitis did not reach their pre mastitis milk yields during the remainder of the lactation after onset of the disease. Rupp and Boichard (1999) indicated that SCC is a more accurate measure of udder health than records of clinical mastitis. Because SCC are generally routinely recorded in most milk recording systems, in the time that clinical mastitis events are not routinely recorded in most countries except in Scandinavian countries and the field data may not be accurate, complete or standard. In addition to that the heritability of SCC is much greater (0.15) than that of clinical mastitis (0.02-0.03) and SCC also reflects incidence of sub-clinical infections. Trinidad et al. (1990) studied the prevalence of IMI in unbred and primigravid dairy heifers, they found that 97% had IMI and 29% showed clinical symptoms, 75% of the quarters were infected. Presence of mammary inflammation in young dairy animals could be deleterious to the future milk production as the mammary tissue development occurs to the large extent during the first gestation (Anderson, 1985 and Tucker, 1987). Etherington et al. (1996) reported that 6.8% of the culling rate of cows in Ontario-Canada was due to mastitis. Mastitis also found to reduce both milk production (Fetrow et al. 1991) and reproductive performance in a lactating cow (Cullor, 1990; Moore et al. 1991; Moore and O'Connor, 1993). Barker et al. (1998) demonstrated that cows with clinical mastitis during early lactation exhibited a prolonged interval until first service (94 days) compared with animals with no clinical mastitis (71 days). Additionally, cows with clinical mastitis between the first service and the establishment of pregnancy had increased number of days open and a two fold increase in services/conception. Rupp and Boichard (2000) stated that without clinical signs of mastitis during the first month of lactation and with a first test day a SCC lower than 400×10^3 cells/ml. they also claimed that the risk of first clinical mastitis was highest around the second calving in lactation starting in summer and for high-yielding cows. The probability of clinical mastitis occurring increased continuously as initial SCC increased. they also concluded that cows with the lowest initial SCC had the lowest risk for clinical mastitis without any intermediate optimum.

A group of researchers (Emanuelson et al. 1998; Weller et al. 1992; Lund et al. 1994 and Pösö and Mäntysaari, 1996) reported that direct selection against clinical mastitis is difficult because in most countries other than the Nordic ones clinical mastitis event is not widely recorded. And because the corresponding heritability of the trait is very low close to 0.02, while Heringstad et al. (1999) estimated heritability of clinical mastitis in Norwegian cattle to be 0.035.

2.2.2 Subclinical mastitis

Rodenburg (1990) showed that 97% of all cases of mastitis are sub-clinical which do not involve visible changes to the quarter or the milk it produces. While Reneau and Packard (1991) reported that approximately 70 to 80% of the mastitis cases are sub-clinical.

Sub-clinical mastitis is found to be associated with decreased milk yield, also a positive relationship clinical mastitis with milk yield has been found (Dohoo and Martin, 1984; Fetrow et al. 1991). Laevens et al. (1997) indicated that the measurement of SCC from dairy herd improvement programs is used worldwide as an indicators of sub-clinical mastitis. Ruffo et al. (1978) and Harmon and Reneau (1993) reported in different studies that IMI have been recognized as major factors that influence SCC. Milk from healthy udder quarters was found to have an average value of SCC between 23×10^3 - 50×10^3 cells/ml depending on the breed and the physiological status of the animal (Klaas, 2000). The milk yield starts to drop with an increase in SCC over 100×10^3 cells/ml (Korhonen and Kaartinen, 1995). They also showed that the increase in SCC to a level more than 100×10^3 cells/ml resulted in 18% reduction in milk yield. De Graaf and Dwinger (1996) estimated the crude milk production losses per cow with sub-clinical mastitis as 1.56 kg/day for daily milk yield, and the milk production loss per affected quarter due to sub-clinical mastitis was estimated to be 17.6% on average. They concluded that the decrease in milk production in heifers with sub-clinical mastitis did not differ significantly from the decrease in production in older cows. Sub-clinical mastitis is also known to affect the reproductive performance of the animals. Schrick et al. (2001) found that cows with sub-clinical mastitis before the first service had an increase of days to first service (74.8 ± 2.7 d), days open (107.7 ± 6.9 d) and services per conception (2.1 ± 0.2) compared with the control (67.8 ± 2.2 d, 85.4 ± 5.8 d and 1.6 ± 0.2 ; $p < 0.05$).

2.3 Etiology and Epidemiology

Mastitis is known to be established as a result of the reaction of three bio-systems namely the causative agent, the animal and the environment in which the animal lives. Sandholm

and Korhonen (1995) reported that the primary and secondary body defense mechanisms prevent the pathogenic microbes from entering the mammary gland through the teat canal orifice. They also indicated that the concentrations of the antibacterial factors in the udder secretion are under genetic control and depend on the lactation stage and udder health. The environmental factors such as management, feeding, hygienic status, bedding, milking and the virulence of the organism contribute to the disease. Lesile (1996) reported that stress factors such as isolation of an individual and mixing groups of cows have been shown to increase somatic cells count in the absence of mastitis, moreover it has been reported that there was no increase in SCC.

2.4 Causative agents

2.4.1 Classes of mastitis pathogens

Several researchers (Bramley, 1985; Wendt et al. 1994; Smith and Hogan 1995) concluded that mastitis causing organisms can be classified into two main groups: Contagious pathogens which spread by means of hands, milking units and include *S. aureus*, *St. agalactiae*, and *Mycoplasma*. Environmental organisms which live in the cow's environment and are always present, they include *E. coli*, *St. dysg.*, *St. ubris*. Buzalski and Pyörälä (1995) stated that contagious mastitis is mainly caused by Staphylococci and shows high cell count in bulk milk whereas environmental mastitis results in a high number of clinical cases, but the cell count in the bulk milk is usually not high. Another group of mastitis causing organisms called minor pathogens (Keown, 1997) and include *C. bovis* and CNS. Buzalski and Seuna, (1995) reviewed the results of the microbiological examinations of milk samples that were done in Finish milk inspection laboratories in 1991 and reported the frequency of mastitis causing organisms as given in table 2.

Table 2: Frequency of mastitis causing organisms (FMI, 1991)

Bacterial species	No. of samples	%
<i>St. agalactia</i>	1389	0.63
<i>St. dysg.</i>	9397	4.29
<i>St. ubris</i>	10767	4.91
<i>β-haemolytic streptococci</i>	1553	0.71
<i>S. aureus</i>	42546	19.42
CNS	30417	13.88
<i>E. coli</i>	3178	1.42
<i>Klebsiella</i>	722	0.33
<i>Pseudomonas aeruginosa</i>	144	0.07
<i>Actinomyces pyogenes</i>	1272	0.58
Yeast, moulds and fungi	1224	0.56
Other	10615	4.84
Total	113224	51.67
No growth	105892	48.33
All samples	219116	100.00

2.4.2 Mode of transmission

Several research studies concluded that the contagious organisms spread during the milking process (Bramley, 1985; Smith and Hogan, 1995; Bray and Shearer, 1996) causing an infection of the udder as a result of entering the teat canal (Rodenburg, 1990). The former authors also showed that scar or connective tissue replacing the destructed milk secreting tissues and result in a permanent loss of the productive ability. Sandholm and Korhonen (1995) reported that the udder becomes infected through the teat canal which represents a physical barrier to the penetration of bacteria. They also added that when the udder is dilated the risk of infection is high. An infected mammary gland can act as a reservoir for mastitis microbes (Davidson, 1961; Barnes et al. 1987). Pre-partum heifer infections have been attributed to the feeding of mastitic milk to heifer calves and allowing heifers to suckle each other (Mc Donald, 1982), however, in another studies it was found that feeding contaminated milk did not increase the prevalence of IMI at parturition over control heifers fed milk free from contagious organisms (Barto et al. 1982; Bushnell,

1989). Kirk (1996) presented that the high risk of contagious organisms can be from the movement of animals onto the dairy herd as they may carry in a pathogen which did not exist or they may themselves not have immunity to pathogens already exist. Chrystal et al. (1999) stated that nearly all IMI occur as a result of micro organisms passing through the teat canal, and that wider teat diameters were associated with higher SCS. On the other hand, David and Shearer (1986) reported that the environmental organisms mainly live in the animal's environment like rumen and udder. The organism can also be found in feces, polluted water and bedding material. The inflammation results from the cow's reaction to the bacterial irritation and the progress of the infection depends on the ability of bacteria to adapt to milk environment and on various virulence factors (Ali-Vehmas and Sandholm, 1995).

2.5 Contagious pathogens

2.5.1 *S. aureus*

Bray and Shearer (1986) reported that *S. aureus* Lives in the udder and on the skin surfaces of an infected cow. Ali-Vehmas and Sandholm (1995) showed that the organism can produce capsular material, hemolysin and β -lactamase when incubated in mastitic milk and are transmitted from infected quarters to uninfected quarters during the milking process (Risco et al.1999). Bray and Shearer (1986) found that *S. aureus* is one of the organisms responsible for about 95% of IMI. Bramley and Dodd (1984) found that *S. aureus* is the most prevalent and costly of the major mastitis pathogens and can result in both clinical and sub-clinical mastitis. Roberson et al. (1994) found that the mean prevalence of *S. aureus* IMI in high prevalence herds (>10%) to be 30% where as the mean prevalence of *S. aureus* IMI in a low prevalence (<5%) herds was 2%. Trinidad et al. (1990) isolated *S. aureus* from 37% of all cases and 14.9% of the quarters. White and Mc Donald (1961); Oliver and Mitchell (1983) and Pankey et al. (1991) reported that the prevalence of *S. aureus* IMI in primiparous cows at parturition to range from 2-50%. The prevalence of *S. aureus* IMI in pre-partum heifers varied considerably among different regions and herds, Daniel et al. (1986) and Pankey et al. (1991) found a very low prevalence of *S. aureus* IMI. While Aarestrup and Jensen (1991) found no evidence of *S. aureus* infection at all. Other researchers (Trinidad et al. 1990 and Nickerson et al. 1995) reported a relatively high prevalence. Waage et al.(1999) in a study of dairy heifers found that *S. aureus* was most frequently isolated organism from quarters (44.3%). Trinidad et al.(1990) reported 20% of all infected quarters was *S. aureus* .In Latvia a study was conducted by Jemeljanovs et

al.(1999) showed that 55.17% of all cases of udder inflammation of 439 cows udder secretion were caused by *S. aureus* . Gentilini et al. (1994) discovered that *S. aureus* is considered one of the most etiologic agents in Argentina. Jones and Ward (1989) found that of 20% Staphylococci isolated, 14 were *S. aureus*, and that cows immunization by *S. aureus* experimental vaccine increased their resistance and decreased SCC in comparison with the control groups (Jemeljanovs and Bluzmanis, 2000). (Lucey and Rowlands, 1984; Erb, 1985 and Firat, 1993) reported that *S. aureus* IMI reduced milk yield 230 Kg, while the somatic cells count found to be $900 \times 10^3/\text{ml}$ compared to $200 \times 10^3/\text{ml}$ of non- *S. aureus* infection (Buelow, unpublished thesis, 1993 cited by Zepeda et al. 2000). Barkema et al. (1999) presented that the incidence rate of mastitis caused by *S. aureus* was mostly related to factors associated with bulk milk SCC.

2.5.2 *St. agalactiae*

St. agalactiae belongs to the group of pyogenic hemolytic streptococci and serologically to Lancefield's group B (Buzalski and Seuna, 1995). *St. agalactiae* is an obligatory organism of the cow's udder, mastitis caused by it spreads particularly during the milking through the equipment, and is highly contagious, either chronic or recurrent, often the cell count of the milk remains quite low (Pyörälä, 1995). Morin and Hurley (1999) stated that *St. agalactiae* inhibits ducts and cisterns of the mammary gland. It causes an inflammation which blocks the ducts, leading to decreased milk production and increased SCC. Barkema et al.(1999) reported a 0.004 incidence rate of mastitis of *St. agalactiae* and as was associated with management practices. The US national Mastitis Council (1996) published that *St. agalactiae* as a contagious bacteria is transmitted from infected quarters to uninfected quarters during the milking process. Jemeljanovs and Bluzmanis (2000) showed that 14.85% of the mastitis cases in Latvia was *St. agalactiae*. The organism was reported to have the highest interclass correlation within a cow for natural logarithm SCC (Barkema et al.1997). In the forties of the last century it was reported that feeding milk containing *St. agalactiae* to heifers calves and subsequent suckling among heifers would result in IMI by this major contagious pathogen at first parturition (Roberson et al. 1994). Ma et al. (2000) found that in milk collected from Holstein cows after IMI with *St. agalactiae*, post infection milk had significantly higher somatic cells count ($849 \times 10^3/\text{ml}$) than pre-infection milk ($45 \times 10^3/\text{ml}$). In a study for mastitis control Bray and Shearer (1986) found that *St. agalactiae* lives in the udder and can not exit outside the gland for a long period, it is

susceptible to penicillin and once eliminated usually does not return to the herd unless infected cows are purchased.

2.6 Environmental pathogens

2.6.1 *St. dysg.*

St. dysg. is one of the major pathogens belongs to the Lancefield's group C, *St. dysg.* is no longer included in the Streptococci group, but retained the name in the mastitis field (Buzalski and Seuna, 1995). The organism lives almost anywhere: in the udder, rumen and feces and in the barn, its spread can be stopped by dipping the whole teat to the base of the udder (Bray and Shearer, 1986). The pathogen is most prevalent in the examined quarter milk samples from 1500 heifers with clinical mastitis before or within 14d after parturition (Jonsson et al. 1991). Pyörälä (1995) stated that the identification of the organism is based primarily on a biochemical reaction and can be isolated from summer mastitis. Sansdholm and Payörälä (1995) found that the incidence of *St. dysg.* increases in herds where teat dipping and dry cow therapy are applied. Whereas, Payörälä and Myllys (1995) reported that the organism is highly susceptible to Penicillin and its derivatives. On the other hand Buzalski and Payörälä (1995) showed that herds infected with *St. dysg.* appears as high cell counts in the bulk milk. Payörälä and Buzlski (1995) found that the organism is found to be associated with teat lesions. In the study conducted by Barkema et al. (1997) it was shown that a lower intra-class correlation within herd (0.03) was detected between the frequency of the organism and SCC (log). Waage et al. (1999) found that the frequency of *St. dysg.* was 18.2% of 1040 heifer's quarters samples affected with clinical mastitis and that was collected prior or within 14 d after parturition. Aarestrup and Jensen (1997) discovered a strong association between IMI with *St. dysg.* before parturition and IMI with *St. dysg.* after parturition. Whereas Barkema et al.(1999) found a strong positive correlation between the incidence rate of clinical mastitis caused by *St. dysg.* and that caused by *S. aureus*. They also added that the incidence rate of mastitis caused by *St. dysg.* was related to nutrition, milking technique and machine milking. Østerås et al. (1999) stated that a cow had an infection or identification of a major pathogen 45 ± 32 days prior to drying off and a series of composite milk $SCC > 100 \times 10^3$ /ml before sampling.

2.6.2 *E. coli*

E. coli is an environmental polluted organism. It lives in feces, polluted water and bedding materials, it is not susceptible to antibiotics (Bray and Shearer, 1986). The organism belongs to the family Enterobacteriaceae. The injury of the teat canal often leads to acute mastitis caused by *E. coli* (Buzalski and Pyörälä, 1995), and hence it is considered to be an environmental pathogen (Radostits et al. 1994). Hogan and Smith (1987) found that the microorganisms may be eliminated before or shortly after onset of clinical symptoms, therefore the host defense system appears to eliminate *E. coli* efficiently (Hill et al. 1978), especially when IMI occurs late in lactation (Hill and Shears, 1979). Recurrent clinical episodes were found in 9.1% of quarters with mastitis caused by *E. coli* (Lam et al. 1996; Lipman et al. 1994), whereas Waage et al. (1999) found the frequency of *E. coli* to be 6.4% from infected quarters. *E. coli* was one of the most prevalent pathogens in the study of Jonsson et al. (1991). Döpfer et al. (1999) discovered that in 4.77% of all episodes of clinical mastitis caused by *E. coli*, persistent IMI caused by the same *E. coli* strain. Jones and Ward (1989) reported that *E. coli* was the predominant cause of mastitis in early and late lactation. Barkema et al. (1999) stated that the incidence rate of clinical mastitis caused by *E. coli* was mostly related to housing, hygienic measures and machine milking.

2.7 Minor pathogens

2.7.1 CNS

CNS were previously called micrococci, species most often isolated from CNS mastitis are *S. hyicus*, *S. simulans*, *S. epidermidis*, *S. warners*, *S. xylophilus*, *S. hominis*, *S. haemolyticus* and *S. chromogenes* (Buzalski and Seuna, 1995). Mastitis caused by them occurs at all stages of lactation but is most common during drying-off and soon after calving and considered milder than *S. aureus* mastitis because they possess less virulence factors than *S. aureus* (Bramley, 1991). CNS bacteria can often cause teat infection which cause only a slight increase in milk cells count, mastitis occurs particularly in heifers. Jones and Ward (1989) found that of 20 Staphylococci isolated four were CNS, which were seen in cows soon after parturition and caused 14% cases of mastitis. A similar finding was reported by Pankey et al. (1996), they stated that CNS were isolated from 21.8% of the heifers in Waikato. Studies in USA have reported that up to 90% of heifers quarters are infected before parturition and 70% were infected with CNS (Trinidad et al. 1990). Aarestrup and

Jensen (1997) found that *S. chromogenes* was the bacterial species isolated most often before parturition (15% of quarters). Whereas Waage et al. (1999) found that of the most prevalent isolates of the CNS were *S. simulans* (53.7%), *S. hyicus* (14.8%) and *S. chromogenes* (14.8%). They also concluded that CNS were the main cause of sub-clinical IMI. Laevens et al. (1997) concluded in a study that a single isolation of CNS was resulted in statistically increase in SCC with least square mean SCC (\log_e -transformed) as 3.97.

2.7.2 *C. bovis*

C. bovis is a relatively common causal agent of a mild mastitis, it requires oleic acid present in milk to grow (Buzalski and Seuna ,1995). This organism is considered to be a typical contaminant of milk flowing from the udder (Mantere-Alhonen, 1995). Classified as environmental pathogen that usually causes considerably less somatic cells count elevation (Keown, 1997). Laevens et al.(1997) indicated that a single isolation of *C. bovis* was associated with a numerical increase in somatic cells count. However, Sheldrake et al. (1983) and Rainard et al.(1990) in different studies concluded that a single isolation of *C. bovis* considered to be a false-positive result. Barkema et al.(1997) found in a study that *C. bovis* had the highest intra-class correlation within herd (0.11) with the natural logarithm of SCC.

2.8 Factors influencing determinants of IMI

2.8.1 Factors influencing frequency of pathogens and infection rate

Infectious mastitis is present when the pathogen and the inflammatory changes were detected in the secretion, whereas non specific mastitis is present when there were inflammatory changes but no pathogen in the secretion and a latent infection is present when the secretion contained pathogens but had normal cell count (IDF, 1987). Waage et al. (2000) analyzing data of 1122 infected quarters that were clinically affected found that after treatment the reexamination results showed 22% non functional quarters, 14% still affected by clinical mastitis and 12% affected by sub-clinical mastitis. Hogan and Smith (1987) stated that the percentage of quarters infected with environmental streptococci is low and seldom exceeds 10% of quarters. A group of researchers (Linde et al.1980; Brooks and Barnum, 1984; Pankey et al.1985; Watts, 1988; Woodward et al.1988) concluded that in herds in which post-milking teat antisepsis is not practiced, it is not unusual for *C. bovis* to be isolated from more than 60% of quarter milk samples and the new infection rate of such organism was nearly 30 times higher than that of *St. agalactiae* which is attributed to

teat colonization and subsequent contamination of milk samples. Kingwill et al. (1970) (cited after Peeler et al. 2000) stated that the reduction in the incidence rate of mastitis in Great Britain is attributed to the reduction in mastitis caused by contagious pathogens through the introduction of improved control measures. Shoshani and Berman (1998) assessed sub-clinical mastitis by deviation in milk yield and suggested that there are episodic aggravations in mammary health that do not evolve into mastitis but may induce significant losses in milk yield and quality.

2.8.1.1 Herd size

It was earlier suggested that there was a relation between the farm performance and the farm structure (van Asseldonk et al. 1998). Herd size was observed as a risk factor for mastitis with a significant influence (Waage et al. 1998). Although herd size was found to have no significant effect on the occurrence of mastitis in the study of Costa et al. (1998), but Smith et al. (2000) stated that small herds reported more cows leaving for mastitis than high medium and low medium herd size. Wilesmith et al. (1986) claimed that the incidence of mastitis declined with increasing herd size.

2.8.1.2 Year-Season

Waage et al. (1999) in their study of the bacteria associated with mastitis in dairy heifers found that the proportion of *S. aureus* and *Actinomyces pyogenes* were highest and the proportion of CNS were lowest in late autumn and early winter. The proportion of *E. coli* was highest in summer, they concluded that the relative percentage were significantly affected by season. Jonsson et al. (1991) who examined quarter milk samples of 1500 heifers with mastitis before or 14d after parturition, stated that the relative percentages of some organisms were significantly affected by season. Jones and Ward (1989) in their study of the cause of mastitis in dairy cows in Wisconsin, detected mastitis with approximately equal frequency throughout the year. Hogan et al. (1989) in their field survey of clinical mastitis in low SCC herds showed that the rate of infection was different among seasons of the year. Shpigel et al. (1998) reported that the incidence of mastitis in Israeli dairy herds was lower in summer months.

2.8.1.3 Lactation number

The US national mastitis council (1997a) showed that the rate of streptococcal infection increases progressively as the lactation number increases. Schaeffer and Solbu (1987) who investigated the Norwegian red cattle, reported that a first lactation cows had a 10%

probability of having mastitis, which was roughly the same for second, third and fourth lactation, provided that they did not have mastitis in the previous lactations. While cows that had mastitis in the immediately previous lactation, had double this probability of having mastitis again. A fourth lactation cow that had mastitis in the three previous lactations had a 62% probability of having mastitis in the fourth lactation. They also concluded that there does not seem to be an age effect on the probability of mastitis occurrence and any cow that has not had mastitis previously has a 10-11% chance of having mastitis in the current lactation regardless of parity number. Analogous findings were reported by Firat (1993) who analyzed data dealing with susceptibility of clinical mastitis in successive lactations and indicated that cows with mastitis in the preceding lactation were almost twice susceptible to mastitis in the current lactation than those without mastitis in the preceding lactation with probabilities of 0.46 and 0.29, respectively. Fetrow et al. (1991) reported that the carry-over effect of mastitis from one lactation to the next found to be statistically significant but small. Nickerson et al. (1995) found in a Louisiana study of 116 pregnant and unbred Jersey heifers with collected samples from four herds that the bacterial infection were present in 97% of heifers and 75% of quarters, and there were 2.8 infected quarters per animal. Shpigel et al. (1998) observed an increase in the incidence of mastitis as the lactation number increases till the fifth lactation then start to decrease. Hogan et al. (1989) stated that the incidence of mastitis caused by environmental bacteria in the first and second lactation is greater than in older cows. Different from the result that obtained by Zadoks et al. (2001) who found that the rate of infection with *St. uberis* was lower in first and second parity cows than in older cows and was depending on the stage of lactation in one herd. Fleischer et al. (2001) found a significant relationship between the previous 305 days milk yield and the incidence of mastitis.

2.8.1.4 Stage of lactation

It is known that the risk of environmental mastitis infection is highest during early lactation and decreases as the lactation advances. The US national mastitis council (1997b) stated that the rate of IMI is higher during the dry period than during lactation, and during the first 75 days postpartum the rate of infection is higher than it is during the remainder of lactation. The percentage of infected quarters with environmental streptococci at any one point is generally low and seldom exceeds 10% of quarters. In an early study, Munch-Petersen (1970) stated that 22% of all quarters in heifers were already infected by the first

day of lactation, and by the end of the first week of the lactation the infection decreased to 9.4%. Trinidad et al. (1990), reporting a US study, found that up to 90% of heifers had quarters infected before parturition, while other researchers in the USA and Europe (Munch-Petersen, 1970; Meaney, 1981; Oliver and Mitchell, 1983; Pankey et al. 1991 and Matthews et al. 1992) claimed that the IMI rate in heifers was moderate (13 to 39%). Jones et al. (1998) stated that the last 7-10 days before calving or early lactation is the time of greatest susceptibility to new environmental streptococci infections.

2.8.1.5 Farm management factors

The US national mastitis council's fact sheet (1997b) states that housed cows are at greater risk for environmental mastitis compared to cows on pasture. And that post milking teat barrier dips reduce new coliform IMI but their efficacy against the environmental streptococci and contagious pathogens appears to be lower than that of germicidal preparations. They showed also that backflushing of the milking unit does not control environmental mastitis. Additionally, malfunctioning milking machines which result in frequent liner slips and teat impacts can increase cases of environmental mastitis. Washburn et al. (2002) compared seasonally calved Holstein and Jersey cows in confinement or pasture systems and found that cows in confinement had 1.8 times more cases of clinical mastitis and 8 times the culling rate for mastitis than did cows on pasture. Jones and Bailey (1998) reported that purchased heifers from another source could harbor mastitis pathogens and should be sampled for bacteriological culture after calving and should be isolated from the other milking animals until tested negative. In the past decade, hygiene and management practices have been provided as standard program to control IMI (Neave et al. 1969). Radostits et al. (1994) summarized the control measures of mastitis among which pre-milking udder hygiene, post-milking teat dipping and environmental control during the dry and calving periods are to be mentioned. Each of these control measures is aimed at the management of specific pathogen types. Natzke (1981); Pankey (1989); Boddie et al. (1993) and Malinowski (2000) concluded that pre-milking udder hygiene and teat dipping are aimed at reducing infections mainly caused by contagious pathogens and preventing new infections and to a lesser extent at preventing infections that might be caused by environmental pathogens. While Smith et al. (1985) and Todhunter et al. (1995) showed that the environmental management during the transition and calving periods is targeted primarily at preventing new infection with environmental streptococcal species and Coliform bacteria e.g. *E. coli*, *Klebsiella* spp. Over half of the environmental

pathogens acquired during the dry period persist to lactation. Sargeant et al. (2001) claimed that producing high quality milk will require effective udder health programs at the herd level. Management practices at the time of dry-off and during the dry period are essential in this respect.. Peeler et al. (2000) in their study of risk factors associated with clinical mastitis in low SCC British dairy herds found that the incidence of mastitis increases when milking cows were housed in straw yard, cows were standing in the yard after milking, which always practiced post-milking teat disinfection and had greater than 50% replacement rate. They discovered also that the incidence of mastitis was lower when the gathering yard used before milking was scraped at least twice a day. Oliver et al. (2001) demonstrated that pre-and post-milking teat disinfections with phenolic combination were significantly more effective in preventing new IMI than was post-milking teat disinfections only. They also added that pre-milking teat disinfections with phenolic combination in association with good udder preparation and post-milking teat disinfections can further reduce the occurrence of new IMI by numerous mastitis pathogens during lactation. A similar conclusion was reported by Saloniemi and Kulkas (2001) who described the mastitis control in Finland. They recommended post-milking teat dipping as control tool in herds with contagious udder pathogen problem. Hogan and Smith (1987) in their practical look at environmental mastitis concluded that no single uniform management procedure effectively prevents environmental mastitis under controlled conditions. Rodenburg (1990) claimed that high energy or high protein diets do not increase or decrease the number of new mastitis infections, however, feeding high producing cows for maximum production does increase stress on the udder and may cause infected cows to flare-up. Rodenburg also showed that too small stalls subjected animals to teat injury. In free-stall barns cows are less likely to lie in the dirt and the lying area is always of adequate size.

2.8.2 Factors influencing levels of SCC

The measurement of SCC from dairy improvement programs is used worldwide as an indicator of sub-clinical mastitis (Ostensson, 1993) because of its relatively high genetic correlation with mastitis which was estimated to be ~ 0.7 (Mrode and Swanson, 1996) and an important criterion of quality payment systems. As an indicator for the hygienic quality of milk and for the mastitis status in a given herd (DVG, 1989), cow SCC is used to trace sub-clinically infected cows (Laevens et al. 1997), is relatively easy to record and has a higher heritability ($h^2=0.11$) than mastitis incidence ($h^2\sim 0.04$) (Mrode and Swanson, 1996). Philipsson et al. (1995) concluded that it is possible to improve resistance to mastitis by

selecting for a low SCC, due to the higher heritability of the SCC. Philipsson added that selection based on the heritability of the SCC was more efficient than selection directly on mastitis. Results of several studies indicated that SCC is a more accurate measure of the udder health, as it is routinely recorded in most milk recording systems (Rupp and Boichard, 1999). Ma et al. (2000) stated that post-infection milk had a significantly higher SCC (849×10^3 cells/ml) than pre-infection milk (45×10^3 cells/ml) in experimentally intramammary infected Holstein cows. A high SCC was found to decrease the value of milk intended for manufacturing, has adverse effects in cheese making, reduces curd firmness and decreases cheese yield, and increases fat and casein loss in whey (Politis and Ng-Kwai-Hang, 1988a; Politis and Ng-Kwai-Hang, 1988b; Barbano et al. 1991; Klei et al. 1998).

2.8.2.1 Herd size

Herd size and SCC were declared to be negatively related, and larger herds had lower SCC than smaller herds (Norman et al. 2000; Oleggini et al. 2001; Van Schaik et al. 2002). Lafi et al (1994) found that the mean value of SCC was negatively associated with herd size. Norman et al. (2000) added that herd size and SCC were negatively related and large herds had a lower SCC. Peeler et al. (2000) stated that herds with greater than 50% replacement rate indicate that herd size was increasing culling for some reasons including high individual cow SCC.

2.8.2.2 Year-season

Season of calving is reported to have a significant effect on milk SCC and SCS (Corbett, 1998; Rodriguez et al. 2000). However, Liebe et al. (1996) reported no influence of season on SCC of German brown cows. Leslie (1996) found that SCC were lowest during winter and highest during the summer months of July and August, he attributed the seasonal variations to the effect of housing and temperature changes on infection status. Kelly et al. (2000) found a significant seasonal influence on milk SCC, with cows calving in spring having a $SCC > 160 \times 10^3$ cells/ml with higher proportions of polymorphonuclear leukocytes in the total milk SCC than milk from autumn calving cows. Norman et al. (2000) estimated the mean herd SCC to be lower during October through January (280×10^3 to 300×10^3 cells/ml) than during July and August (340×10^3 cells/ml). Rupp et al. (2000) illustrated that regardless of the lactation stage, SCC were higher in summer and lower in autumn of the milk SCC in French dairy breeds. Whereas Allore et al. (1997) found that SCC were significantly higher in spring than in fall. However, Jemeljanovs and Bluzmanis (2000)

determined a seasonal effect on SCC. They claimed that SCC/ml milk was less in summer, a little more in autumn and more high in spring and most SCC encountered in winter. Season was suggested to have no significant influence on SCC in healthy mammary glands (Malinowski, 2001).

2.8.2.3 Lactation number

Several studies revealed a significant effect of the cow age and the lactation number on the level of milk SCC (Corbett, 1998; Kelly et al. 2000; Seker et al. 2000; Haile-Mariam et al. 2001). Kiiman and Saveli (2000) studied the factors affecting milk SCC and reported that milk SCC increased with increasing lactation number, in the first lactation SCC was 285×10^3 whereas in the second, third and fourth lactations were 321×10^3 , 461×10^3 and 477×10^3 , respectively. Godollo and Tanszek (2000) reviewed 98 scientific publications related to physiological and environmental factors influencing SCC. They reported that the number of lactation significantly affect the SCC in milk. A similar conclusion was realized by Labohm et al. (1998) who found that lactation number influence the SCC in a statistically reliable extent. But attributed the rise in SCC above 100×10^3 to infected quarter. Leslie (1996) reported that higher SCC have been found in the milk of older cows. Hortet and Seegers (1998) investigated the relationship between SCC and variation in milk production at the cow level, they indicated that at the test-day level an average loss of 0.4 kg milk in primiparous cows and 0.6 kg in multiparous by each 2-fold increase of SCC above 50×10^3 cells/ml. At the lactation level, the average trend was a loss of 80 kg of milk in primiparous and 120 kg in multiparous by each 2-fold increase of the geometric mean of SCC above 50×10^3 cells/ml. Similar results were published by Hortet et al. (1999) who found that the reduction in milk yield in kg increased with parity and with days in milk to the extent that the reduction in milk yield was 0.32 kg per 100×10^3 cells/ml increase in SCC, 0.63 kg per 200×10^3 cells/ml SCC and 1.13 kg decrease in milk per 600×10^3 cells/ml increase in SCC. This result is in joint agreement to that of Jemeljanovs and Bluzmanis (2000) in their study of somatic cell and micro-organisms contents in milk. They revealed that SCC in milk increased in clinically healthy cows with the increase in the age. The further interpretation of these findings is that: if 90% of the 2nd lactation cows had up to 200×10^3 cells/ml, then only 63.4% of the older than the 4th lactation cows had such level of SCC and 18.1% had more than 500×10^3 cells/ml SCC. These findings supported the results published earlier by Tyler et al. (1989) who stated that primiparous and multiparous cows were similarly showed production losses due to the increase in SCC. In primiparous cattle

with SCC range 403×10^3 - 665×10^3 had 5.22 kg decrease in test-day milk yield whereas multiparous cows with the same range had 3.01 kg reduction in milk yield. Koldeweij et al. (1999) found a geometric mean for SCC of 63.1 in the first lactation and 107.2 in the later lactations. They also found an individual milk yield loss of 1.29 kg/day for each unit increase in $\log_{10}(\text{SCC})$ for cows in the first lactation and 2.04 kg/day milk yield decrease per unit $\log_{10}(\text{SCC})$ for cows in the later lactations. Kiiman and Saveli (2000) found a significant ($p < 0.001$) effect of lactation number on milk SCC, they found that in the first lactation the milk SCC was $285 \times 10^3/\text{ml}$, in the second and third lactation $321 \times 10^3/\text{ml}$ and $461 \times 10^3/\text{ml}$ respectively. Laevens et al. (1997) stated no significant effect of lactation number on SCC when cows were bacteriologically negative and the least square mean of SCC for first, second and third lactations were 3.80, 3.93 and 3.97, respectively. Schepers et al. (1997) estimated the variance components for SCC, they illustrated the shape of the SCC curve which was flat for the first lactation cows compared with the shape of the SCC curve for cows in the subsequent lactations.

2.8.2.4 Stage of Lactation

A group of researchers reported that SCC and milk yield traits vary the stage of lactation (Vech et al. 1989; Corbett, 1998; Labohm et al. 1998; Kelly et al. 2000; Rupp et al. 2000) and with test-day (Haile-Mariam et al. 2001). Schepers et al. (1997) showed that stage of lactation affected the SCC, since the logarithm SCC was high at the beginning of the lactation, dropped to a minimum between 40 and 80 days postpartum and then steadily increased until the end of lactation. Carnier et al. (1997) stated that from a genetic view point, SCS in early lactation behaves differently from those in later stages of lactation. Williams et al. (1991) claimed that stage of lactation had a pronounced effect on milk SCC, with the level being high in early lactation, low in mid-lactation and high again in late lactation. However, Rodriguez et al. (2000) stated that milk SCS typically reaches a minimum early in lactation and then rises, but lactations starting between October and December had the highest fall of SCS at the beginning of lactation, and smallest increase thereafter. Early results were obtained by Emanuelson et al. (1988) who found a significant effect of the stage of lactation on SCC of morning milk samples from cows over 18 months and concluded that stage of lactation must be taken into account when establishing normal values for ATP as an indicator of mastitis. Seker et al. (2000) found that a positive CMT score increased in Brown-Swiss cows with higher yield and at the 4th and 6th month of lactation. Kirk et al. (1996) indicated that sub-clinical infection with minor pathogens

(primarily CNS.) had no significant effect on average SCC during early and mid lactation. Laevens et al. (1997) obtained least squares mean SCC for first, second and third parity bacteriological negative cows as 3.80, 3.93 and 3.97 respectively, with no significant effect of parity, stage of lactation and parity, stage of lactation interaction, however the effect was significant when including the data of both infected and bacterial free cows.

2.8.2.5 Farm management factors

In the past decade, the standard mastitis control program has provided hygienic and management practices to control IMI (Neave et al. 1969), a decrease in bulk milk SCC is an indicator of the success of the control program (Suriyasathaporn et al. 2000). Yalcin et al. (1999) studied the impact of mastitis control procedures in Scottish dairy herds, and concluded that udder preparation involving washing was associated with higher SCC and had detrimental effects on the efficacy of post-milking teat disinfections. Smith and Ely (1997) reported that free-stall bedding did not significantly affect milk quality, with no difference in linear SCS among the herds studied. They also showed that herds fed inside the free-stall barn or under covered roof had higher milk production and lower SCS than those fed outside. However, Bewley et al. (2001) stated in a comparison of free-stall barns used by modernized Wisconsin dairies that herds with four-row free-stall barns had higher production than herds with six-row barns and that the average linear was SCS significantly ($p < 0.05$) lower in new four-row barns than six-row barns (2.71 vs. 2.95). Omore et al. (1999) assessed the impact of a clinical trial of three mastitis control strategies among which improved udder hygiene in smallholder dairy farms in Kenya, they concluded that the trial had some impact in lowering the prevalence of contagious pathogens by 18%, but found no significant increase in milk yield or lowered SCC. Barkema et al. (1998) reported about post-milking teat disinfections and good milking management as important factors for the prevention of a high bulk milk SCC. Godollo and Tanszek (2000) indicated that technological environment, feeding and milking are known to interfere with changes in SCC. Mazzucchelli et al. (2000) gave an account of the changes in the management of a Spanish herd of cows affected by mastitis by making a dietary adjustment, an improvement of the housing management and improving the design of milking parlors and management of milking. These changes resulted in a reduction of the milk SCC from 380×10^3 cells/ml to 200×10^3 cells/ml. Kiiman (2001) indicated that the adequate pre-milking cow preparation was essential to milk SCC as well as over-milking ($p < 0.001$). He

also stated that the effect of milking equipment was not statistically significant for milk SCC.

2.8.3 High milk yield

Gröhn (2000) studied the relationship between disease and milk production, he found that high milk yield predisposed a cow to certain diseases particularly mastitis. Whitaker et al. (2000) found that there was a positive association between bulk milk SCC and mastitis rate. Haile-Mariam et al. (2001) estimated the correlation between test-day yield and SCC, they stated that genetic correlations between yield and log SCC were positive at the beginning and negative at the end of the first lactation, in the second and third lactations genetic correlations were nearly zero at the beginning of the lactation but negative at the end, however, environmental correlations were always negative. The authors attributed the positive correlations to the fact that high producers are more susceptible to mastitis than cows with average or low production whereas the negative correlations in the second half of the first parity and later parities due to the mastitis cause high SCC and udder damage resulting in reduced milk yield. These findings support results presented by Gröhn et al. (1995) who claimed that cows with mastitis are often higher yielding cows, which produce more milk even having contracted the disease, compared to their healthy and generally lower yielding herd-mates.

3 MATERIALS AND METHODS

3.1 Construction of the project

As a part of the FST- project of udder health improvement, 48 dairy farms were included in a side co-operative research project with the institute of animal breeding and husbandry with veterinary clinic, Martin-Luther University, Halle-Wittenberg and the Thuringia center of agriculture (TLL) to investigate the effects of contagious and environmental bacteria and management procedures on milk performance traits and udder health. The farms included in the project are among 572 dairy farms in the state which are members of the-MLP-organization. Milk samples are regularly tested by the LKV. Herd size, herd management, housing, feeding regime and husbandry system data were collected by a questionnaire. Milk performance data was supplied by the national data center (VIT) at Verden. Regularly collected milk samples were subjected to bacteriological investigations at the Animal Health Service-Mastitis Laboratory at Bad Langensalza (Thuringia). Determination of the somatic cells were done at the milk laboratory of the TVL-Thuringia by means of Fossomatic device (Fossomatic-5000[®], Fa. Foss Electric, Denmark).

3.2 Farms description and Management

3.2.1 Herd size

During the year 98-99 the herd size of Thuringia dairy farms was between 72-1074 lactating cows of part A, i.e. those that completed their lactation period, and 21-278 lactating cows of part B, i.e. those that are at part of their lactation.

3.2.2 Housing system

Among the 48 farms studied, 49% use loose housing with plan floor, 27% use loose housing stall barns with slatted floor and 24% were found to use other types of housing systems, e.g. tie-stall barns. Bedding was differed between straw, rubber and deep straw which was used in loose barns of some farms.

3.2.3 Feeding regime

In the farms surveyed, it was found that 66.67% of the farms use mobile method of feeding. Whereas 20.51% of the farms use stationary feeding method and 12.82% of the farms use both mobile and stationary methods. It was also found that 60.97% of the farms use TMR to feed their animals compared to 34.15% using a base ration to be fed with mobile or stationary feeding method and concentrate to be fed with an automatic system.

And 4.88% of the farms use TMR, base ration fed with mobile or stationary feeding method and concentrate to be fed with an automatic system. However, 67.57% of the farms investigated found to use silage the whole year, 21.62% use green fodder in summer and 10.81% use both silage and green fodder. Field accessibility was allowed for dry cows in 36.96% of the farms, 19.56% of the farms allow grazing for both dry and lactating cows. Whereas 43.48% of the farms surveyed found to manage their animals at zero grazing.

3.2.4 Milking system

Of the 48 farms surveyed 64% use Side by Side and herring bone milking units, 10% had carousel, 11% of the farms used pipes systems and 4% of the farms had combined types of milking systems.

3.2.5 Hygienic measures

Before and after milking, milking units are subjected to sanitization using backflushing, air wash, and bath or spray techniques. The udder of a lactating cow is subjected to thorough cleaning before milking using single service paper towels or dried cloth. Teats are disinfected using teat dipping. Drying cows off at the end of the lactation period is performed either by means of antibiotics as found in 89.80% of the farms or without as in 10.20% of the farms studied. In the former class, 65.91% of the farms practiced drying off for all animals and 34.09% used antibiotics only for bacteriologically positive animals. Mastitis test performed regularly by means of rapid mastitis test (CMT).

3.3 Review of the data

3.3.1 Performance data

Test-day data that were recorded in the period 1998-2000 were included in the study, supplied by the central data bank (VIT) through the FST-Institution of agriculture. Data on the following traits were included:

1. Milk yield/lactation.
2. Fat yield /lactation.
3. Protein yield/lactation.
4. Test-day SCC.
5. Test-day milk yield.
6. Fat percentage.
7. Protein percentage.

8. Lactose percentage.

In addition to the farm number, animal herd-book number, sire herd-book number, dam herd-book number, date of calving, lactation number and reason of culling were available.

3.3.2 Questionnaire data

The questionnaire intended for the collection of management data was prepared in the institute of animal breeding and husbandry with veterinary clinic, Martin Luther University Halle-Wittenberg and the information requested was supplied by the farm managers, which include the following data:

1. Origin of the cow.
2. Housing system.
3. Milking system.
4. Feeding method.
5. Type of udder cleaning before milking.
6. Inter-milking sanitization method of the milking units.
7. Teat disinfection.

3.3.3 Bacteriology data

Milk samples, which were collected before and after calving, were subjected to bacteriological investigations and the pathogens isolated were assorted to eight species and bacterial groups namely:

1. *S. aureus*
2. *St. agalactiae*
3. *St. dysg.*
4. *E. coli*
5. *C. bovis*
6. CNS
7. EPS
8. Others (*Pseudomonas aeruginosa* *Actinomyces pogenes*, yeast, spore forming bacteria ...etc)

The bacteriological data included the animal and herd number which later was used to merge the data with the data from the central data base. Date of sampling, site of sampling i.e. udder quarter or udder and the result of the investigation of the sample (positive or

negative) also were included. The types of bacteria later were classified into two groups, contagious and environmental.

3.4 Methods of the analysis

3.4.1 Sample collection, bacterial isolation and identification and SCC

Milk samples intended for bacteriological investigations are collected and cultured according to the regulations released by the German veterinary association (DVG, 1980). After the primary cleaning of the udder, the tip of the teat was sterilized with 70% ethanol alcohol and dried up with clean and disposable swabs. The first strips of fore milk were discarded, then 30 ml milk aseptically taken in sterile tube which was held in slant position during stripping. Milk samples were contain 0.18 gm boric acid in dough form as conservative material; samples were investigated within four hours after collection or cooled in 4⁰C to be investigated in the second day. After thorough mixing of the sample, inoculums of 0.01 ml placed in Columbia agar plate supplemented with 5% sheep, and with Edward's culturing loop streaks are made. Aerobes grow within 24 hours of incubation at 37⁰C, the media is incubated again for 24 hours, if the growth of the colony is not clear. Identification of mastitis bacteria merely depends on the growth behavior of the colony morphology, formation of color pigments, esculin splitting and hemolysin formation, gram stain reaction and CAMP-Test.

For determination of somatic cells 0.2 ml from the sample after being stained with Ethidium bromide was taken and transferred to a glass container on a rotary table where it was mixed with a preheated buffer and dye and stirred well. Part of the mixture was transferred to the periphery of a rotating disc which serve as an object plane for the microscope. The film was illuminated by a Xenon arc lamp, the light passed through lenses and a blue filter. Red light emitted from the cells was led through a different filter to a slit and photo-multiplier. Each cell produces an electrical pulse which is fed to an amplifier. The printout of the count was multiplied by 1000 to give cells/ml.

3.4.2 Data processing

The study included data on 10742 dairy cows which were obtained from three sources, namely test-day data including SCC and milk yield, bacterial investigation results included the identified types of bacteria from the milk samples and management data collected by the questionnaire (data on the origin of the cow, housing systems, feeding methods, milking systems, between milking sanitization methods of milking units, types of udder

cleaning before milking, teat disinfections). The three sources of the data were merged into one data set by means of a statistical program using the SAS package (SAS-Institute, Inc.1996) and developed in the institute of Biometrics. The number of animals and their records were distributed into the following classes according to the factors shown in table 3.

Table 3: Factors distribution of the data

Factor	Class	Definition
Herd size	5	Small (< 200 cows), medium small (200-400 cows), medium (401-600 cows), medium large (601-800 cows) large (> 800 cows)
Year-season	8	Summer 98, autumn 98, winter 98/99, spring 99, summer 99, autumn 99, winter 99/2000 and spring 2000
Lactation number	4	Lac.1, Lac.2 Lac.3 and >Lac. 3
Stage of lactation	3	Early (1-100 days), middle (101-200 days) and late (>200 days)
Origin of the cow	2	Farm bred and purchased
Milking equipment	3	Pipe system, carousel and milking parlor
Feeding method	3	Mobile, stationary and both methods
Type of udder cleaning	2	Moist, dry
Housing system	4	Loose housing stalls with slat floor, loose housing stalls with plan floor and others
Inter-milking sanitization of the milking units	6	Backflushing, air wash, spraying, bath, combinations or not used
Teat disinfection	2	Teat dipping or not used

In addition to that the data on the logarithmic SCC were averaged and classified into four classes within the entire lactation (<3.22, 3.22-4.47, 4.48-5.73.and >5.73). And from the bacterial investigation result the infection rate was calculated as the number of pathogens discovered in the udder quarter and five groups were obtained as follows:

1. Infection rate=1 when all quarters are free from bacteria.
2. Infection rate=2 when bacteria was discovered in all four quarters.
3. Infection rate=1.75 when bacteria was discovered in three quarters.

4. Infection rate=1.50 when bacteria was discovered in two quarters.
5. Infection rate=1.25 when bacteria was discovered in one quarter.

3.4.3 Data analysis

For all analyses, the SAS package (SAS institute Inc. 1996) was used. Means, standard deviations, and C.V. of the variables, namely, performance data, somatic cell count, infection rate and test-day milk yield were computed using the procedure MEANS.

3.4.3.1 Distribution of contagious and environmental groups of bacteria

The frequencies of the contagious and the environmental groups of bacteria were computed using the procedure FREQ incorporated in SAS. A χ^2 -test was performed to test the significance of bacteria encountered in the udder quarters and the udder in accordance of the factors studied.

3.4.3.2 Models describing determinants of IMI

To achieve the normal distribution of the SCC, SCC were transformed into the logarithmic form. Investigation of the factors affecting logarithmic somatic cell counts, test-day milk yield and infection rate were performed using the procedure MIXED of SAS on the basis of restricted maximum likelihood method (REML). This procedure incorporated both fixed and random effects of the studied factors in linear models. Means were tested for significances with the aid of F-test.

3.4.3.2.1 Model describing infection rate and logarithmic SCC

$$Y_{ijkmnop} = \mu + \text{Lacn}_i + \text{Lacs}_j + \text{Yeas}_k + \text{Far}_m + \text{Bactr}_n + \text{Lacn}_i * \text{Bactr}_n + \text{Lacs}_j + \text{Bactr}_n \\ \text{Yeas}_k * \text{Bactr}_n + \text{Far}_m * \text{Bactr}_n + \text{Anim}_o + e_{ijkmnop}$$

Where,

$Y_{ijkmnop}$	=mean infection rate or logarithmic SCC of oth animal (o=1,2,3,.....),
μ	=over all mean,
Lacn_i	=fixed effect of the ith lactation number (i=1,2,3, >3),
Lacs_j	=fixed effect of the jth stage of lactation (j=1,2,3),
Yeas_k	=fixed effect of the kth year-season (k=1-8),
Far_m	=fixed effect of the mth herd size (m=1-5),
Bactr_n	=fixed effect of the nth bacterial group (n=1-3),
$\text{Lacn}_i * \text{Bactr}_n$	=fixed effect of the ith lactation number*nth bacterial group (i=1-4*n=1-3),

$Lacs_j * Bactr_n$	=fixed effect of the jth stage of lactation* nth Bacterial group ($j=1-3 * n=1-3$),
$Yeas_k * Bactr_n$	=fixed effect of the kth year-season* nth Bacterial group ($k=1-8 * n=1-3$),
$Far_m * Bactr_n$	=fixed effect of the mth herd size* nth Bacterial group ($m=1-5 * n=1-3$),
$Anim_o$	=random effect of oth animal ($o=1,2,3, \dots$),
$e_{ijkmnop}$	=residual effect,

the other management and hygienic factors were replacing the herd size factor in the model.

3.4.3.2.2 Model describing test-day milk yield

$$Y_{ijkmnop} = \mu + Lacn_i + Lacs_j + Yeas_k + Far_m + log_n + Lacn_i * Log_n + Lacs_j * Log_n + Yeas_k * Log_n + Far_m * Log_n + Anim_o + e_{ijkmnop}$$

Description of the factors in the model

$Y_{ijkmnop}$	=mean test-day milk yield of oth animal ($o=1,2,3, \dots$),
μ	=over all mean,
$Lacn_i$	=fixed effect of the ith lactation number ($i=1,2,3, >3$),
$Lacs_j$	=fixed effect of the jth stage of lactation ($j=1,2,3$),
$Yeas_k$	=fixed effect of the kth year-season ($k=1-8$),
Far_m	=fixed effect of the mth herd size ($m=1-5$),
Log_n	=fixed effect of the nth class of logarithmic SCC ($n=1-4$),
$Lacn_i * Log_n$	=fixed effect of the ith lactation number* class of logarithmic SCC ($i=1-4 * n=1-4$),
$Lacs_j * Log_n$	=fixed effect of the jth stage of lactation* nth class of logarithmic SCC ($j=1-3 * n=1-4$),
$Yeas_k * Log_n$	=fixed effect of the kth year-season* nth class of logarithmic SCC ($k=1-8 * n=1-4$),
$Far_m * Log_n$	=fixed effect of the mth herd size* nth class of logarithmic SCC ($m=1-5 * n=1-4$),
$Anim_o$	=random effect of oth animal ($o=1,2,3, \dots$),
$e_{ijkmnop}$	=residual effect,

the other management and hygienic factors were replacing the herd size factor in the model.

3.4.3.3 Factors affecting heifers IMI

The effect of the positive bacterial findings and time of sampling on SCC (log) and daily milk yield of the first lactating cows were analyzed using the procedure GLM of the computer software SAS (SAS institute Inc.1996) in accordance with the following statistical model:

$$Y_{kmnop} = \mu + Bd_k + Bu_m + Gt_n + Anim_o + e_{kmnop}$$

Where,

Y_{kmnop}	=mean first stage of lactation SCC or milk yield of the oth animal (o=1,2,3.....),
μ	=over all mean SCC or milk yield,
Bd_k	=fixed effect of the kth time of sampling (k=-10,-20,-30,-40,+10,+20,+30 and +40 days from the calving date),
Bu_m	=fixed effect of the mth result of investigation (m=positive (1), negative (2)),
Gt_n	=fixed effect of the nth bacterial group (n=contagious (1), environmental (2) or bacteria free sample(3)),
$Anim_o$	=random effect of oth animal (o=1,2,3,.....),
$e_{abijkmnop}$	=residual effect,

DMRT was used with factors that had significant effects on the mean of the traits studied.

3.4.3.4 Risk factors associated with IMI and high SCC

For the investigation of the relationship between the probabilities of the occurrence of IMI and elevated SCC in response to some environmental and management factors, logistic regression analysis was performed in which logistic regression (events/trials) model was used. In the model each dependent variable can accept any value, whereas, the independent variable (explanatory) values ranged between 0 and 1. The unknown parameter β was estimated by the method of maximum likelihood, the procedure logistic of the computer packet SAS (SAS institute Inc.1996) was employed to estimate the unknown parameters, a χ^2 -test was used to examine the statistical significance. And after many trials the following models were chosen:

Model 1: Entrance of IMI

$$\text{Logit}(\rho_{ijklmnopqrz}) = b_o + pa_i + ln_j + cs_k + ml_l + sc_m + hs_n + mt_o + uc_p + di_q + zt_r + log_z$$

Where,

$\rho_{ijklmnopqrz}$	=probability of occurrence of IMI,
b_o	=constant,
pa_i	=fixed effect of the ith herd size (I=1-2),
ln_j	=fixed effect of the jth season of calving (j=1-2),
cs_k	=fixed effect of the kth stage of lactation (k=1-2),
ml_l	=fixed effect of the lth pathogen group (l=1-2),
sc_m	=fixed effect of the mth source of the herd (m=1-2),
hs_n	=fixed effect of the nth housing system (n=1-2),
mt_o	=fixed effect of the oth system of milking (o=1-2),
uc_p	=fixed effect pth method of udder cleaning (p=1-2),
di_q	=fixed effect of qth inter-milking sanitization methods (q=1-2),
zt_r	=fixed effect of rth teat disinfection (r=1-2),
log_z	=fixed effect of zth class of SCC (log) (z=1-2),

Model 2: High SCC

$$\text{Logit}(\rho_{ijklmnopqrz}) = b_o + pa_i + ln_j + cs_k + ml_l + sc_m + hs_n + mt_o + uc_p + di_q + zt_r + log_z$$

Where,

$\rho_{ijklmnopqrz}$	=probability of high threshold of SCC,
b_o	=constant,
pa_i	=fixed effect of the ith herd size (I=1-2),
ln_j	=fixed effect of the jth lactation number (j=1-2),
cs_k	=fixed effect of the kth season of calving (k=1-2),
ml_l	=fixed effect of the lth stage of lactation (l=1-2),
sc_m	=fixed effect of the mth pathogen group (m=1-2),
hs_n	=fixed effect of the nth source of the herd (n=1-2),
mt_o	=fixed effect of the oth housing system (o=1-2),
uc_p	=fixed effect pth method of udder cleaning (p=1-2),
di_q	=fixed effect of qth inter-milking sanitization method (q=1-2),
zt_r	=fixed effect of rth teat disinfections (r=1-2),

4 RESULTS

4.1 General survey of the farms

The study was conducted to investigate the factors that could have an influence on udder health status and consequently the individual production. The study covered the period from June 1998 to April 2000. Milk samples from cow in 48 dairy farms in the FST that were enrolled in the state udder health improvement program were randomly collected and subjected to bacteriological investigation. The result of the bacteriological investigation together with the relevant test-day milk yield and SCC were statistically analyzed.

4.1.1 Herd size

The farms studied had 25710 lactating cows registered in the MLP-organization. The number of the lactating cows contributed to the study was accounted to be 10741 i.e. 41.78% of the total animals in the 48 dairy farms. Table 4 shows the class distribution of the cows in the farms. It could be clearly indicated that most of the farms are medium to large having between 15.46 to 37.30% of the animals. Whereas 18.75% of the farms having 37.30% of the animals.

Table 4: Class distribution of the lactating cows

Herd size	Range of cows	No. of animals	%	No. of farms	%
Small	< 200	696	02.71	05	10.42
Medium small	200-400	4343	16.89	16	33.33
Medium	401-600	3976	15.46	08	16.67
Medium large	601-800	7105	27.64	10	20.83
Large	> 800	9590	37.30	09	18.75
Total		25710	100	48	100

4.1.2 The over-all means of the traits

The arithmetic means and standard deviation of studied traits were shown in table 5.

Table 5: Arithmetic means, Std. and C.V. of the performance traits and infection rate

Trait	n	Mean	Std.	C.V.%
Lactation length (days)	18579	297.51	15.61	05.25
Milk (kg)/lactation	18117	6855.07	1605.40	29.94
Fat (kg)/lactation	18117	291.94	67.08	29.10
Protein (kg)/lactation	18117	233.66	52.40	29.08
Milk yield/test day	9496	23.82	7.16	34.26
Fat %	9496	4.19	0.50	11.32
Protein %	9496	3.45	0.30	04.02
Lactose %	9480	4.79	0.21	03.98
SCC (*10 ³)	7829	317	231	73.03
SCC (log)	7829	5.39	0.30	11.97
Infection rate	21331	1.16	0.28	25.29

4.1.3 Frequency of contagious and environmental pathogens

Figure 1 displays the frequency of the individual bacterial types that have been discovered in the udder quarter's and udder's samples. The total positive findings were estimated to be 15701 and 3765, respectively which represented 27.57 and 49.66% of the total samples collected from each site. *S. aureus* and CNS were the most frequently isolated pathogens from the udder quarter's and udder's samples (35.50/28.70% and 32.70/26.60%, respectively). However, *St. dys.* and EPS scored a higher frequency in the udder's samples than in the udder quarter's samples (13.90/12.90 vs. 10.60/9.0%, respectively). Meanwhile the frequency of mastitis pathogens that have been discovered in the foremilk samples were distributed in two main groups; contagious and environmental groups. Figure 2 shows that

the contagious pathogens discovered in the udder quarters as well as in the udder's samples scored a high frequency compared to the environmental pathogens. The difference was significant ($\alpha < 0.05$).

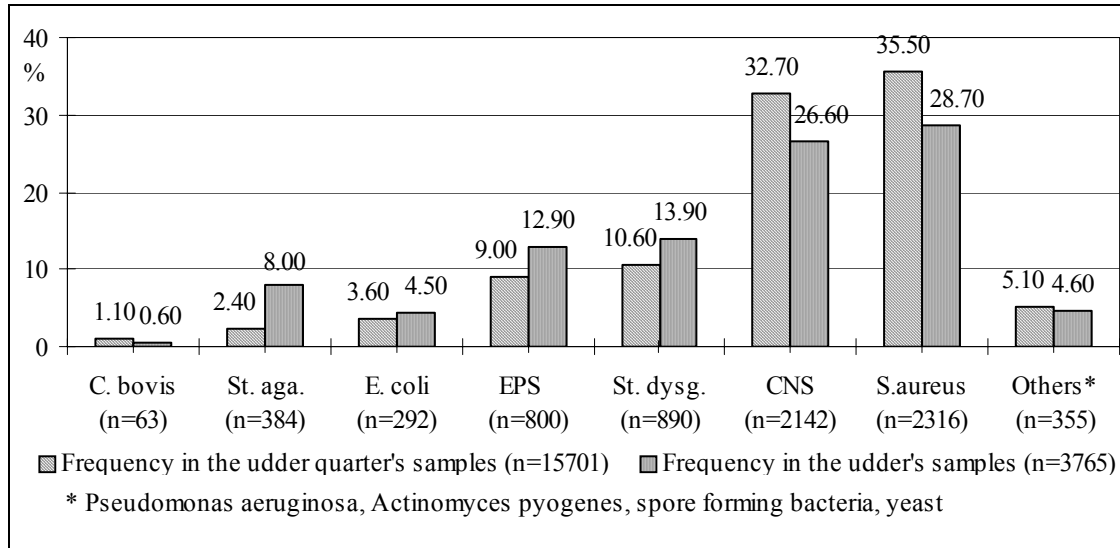


Figure 1: Frequency distribution of the bacterial species in udder quarter's and udder's samples ($p=0.001$)

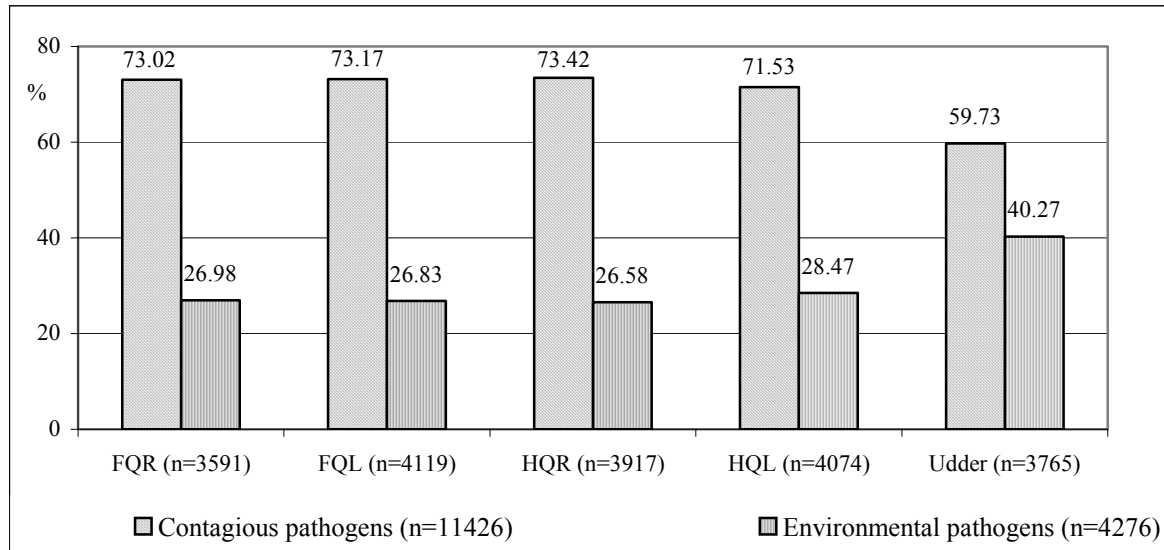


Figure 2: Frequency distribution of contagious and environmental pathogens in udder quarter's and udder's samples ($p=0.001$)

4.2 Factors affecting determinants of IMI

Herd size, year-season, lactation number and stage of lactation in addition to the farm management and hygienic measures were the factors studied. These factors were handled to test their effect on the frequency of the contagious and the environmental pathogens,

infection rate SCC (log) as well as the milk yield as an important factors determining the udder health status of a lactating cow.

4.2.1 Herd size

4.2.1.1 Frequency of contagious and environmental pathogens

The result revealed that the positive findings were higher in the small and large herd size (33.70 and 32.20%, respectively), lower in the medium small herd size (23.91%). However, the frequency in the medium and medium large classes was not greatly differing (28.79 and 30.24%, respectively). On respect of the pathogens groups, table 6 shows that the frequency of the contagious pathogens was higher compared to the environmental pathogens. Based on the relative values, the frequency was extremely higher in the small, medium small and large herd size (75.12, 74.12 and 72.47%, respectively). And based on the absolute value, the frequency was higher in the small herd size (25.32%). The frequency was lower in the medium and medium large herd size (67.64 and 65.19%, respectively). Converse to that were the frequencies of the environmental pathogens. A χ^2 -test revealed a significant ($p=0.001$) variation between the groups.

Table 6: Frequency distribution of contagious and environmental pathogens according to the herd size

Herd size	Number of the positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Small (n=5069)	1708 (33.70%)	25.32	75.12	08.38	24.88
Medium small (n=7934)	1897 (23.91%)	17.76	74.64	06.06	25.36
Medium (n=13459)	3875 (28.79%)	19.47	67.64	09.32	32.36
Medium large (n=13338)	4033 (30.34%)	19.78	65.19	10.56	34.81
Large (n=24384)	7851 (32.20%)	23.36	72.54	08.86	27.52
χ^2 -test ($\alpha=0.05$)	0.001				

4.2.1.2 Infection rate

The study indicated that infection rate and independent on the bacterial status was highly significantly ($p=0.0001$) influenced by the herd size (Table 7). Large herd size had a significantly low infection rate (1.24) compared to small herd size (1.31). It was also found that infection rate decreased steadily as the number of animals in the herd increased. Dependent on the bacterial status figure 3 shows that small herd size had a higher incidence rate with respect to the contagious pathogens than large herd size. The difference of the

infection rate between the two extremes classes was 8%. It could also be observed that infection rate with either contagious or environmental pathogens went down as the class of herd size was higher.

Table 7: LS-means and S.E. of infection rate according to the herd size and irrespective of the bacterial status ($p=0.0001$)

Herd size	Infection rate
Small	1.31 \pm 0.01
Medium small	1.28 \pm 0.01
Medium	1.27 \pm 0.01
Medium large	1.25 \pm 0.01
Large	1.24 \pm 0.01

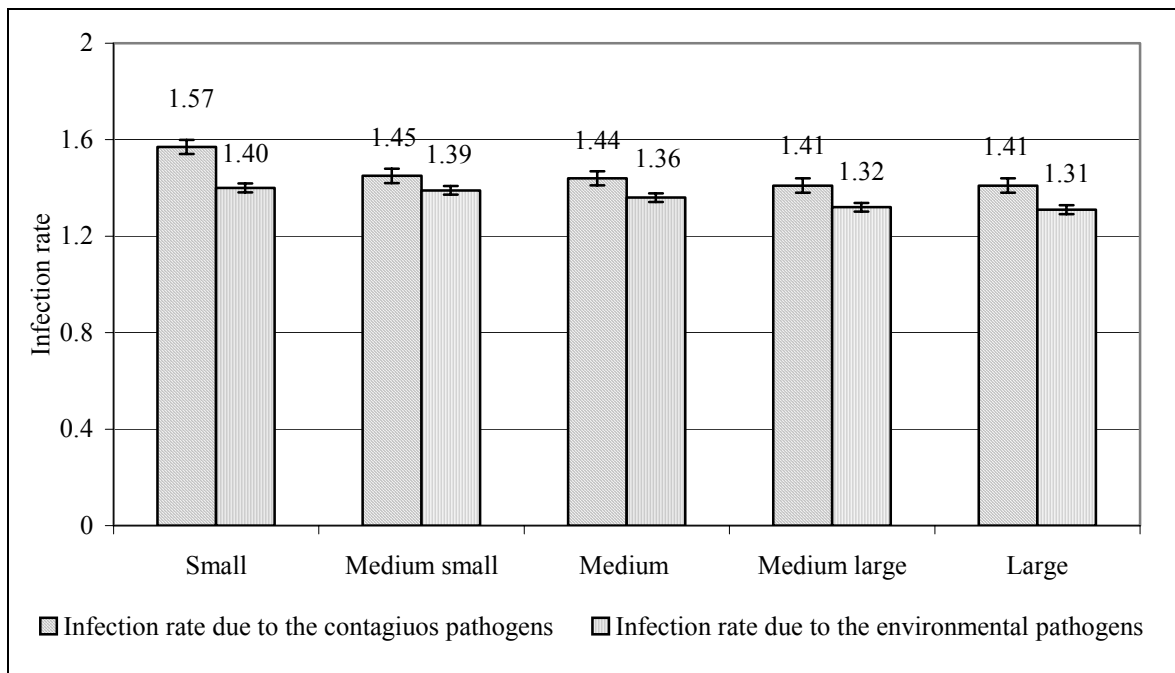


Figure 3: LS-means and S.E. of infection rate dependent on herd size and bacterial status ($p=0.0001$)

4.2.1.3 Logarithmic SCC

Table 8 shows that LS-means SCC and independent on the bacterial status was highly significantly ($p=0.0001$) varied. SCC showed a decreasing rate as the herd size increased from small to large herd size. The difference in the SCC was significant between small and large herd size. Meanwhile, there was no statistical difference in the LS-mean SCC of the medium small and the medium classes of the herd size. Bacterial types, herd size exerted a

highly significant ($p=0.0001$) effect on the logarithmic SCC. Contagious bacteria elevated the level of SCC in all classes of the herd size except in the medium large where the level due to the effect of the environmental pathogens (4.86) was higher than that due to the effect of the contagious pathogen. Small herd size had the over-all higher SCC due to the effect of both contagious and environmental bacteria (5.27 and 4.99, respectively). Whereas the large herd size scored the lower level of SCC with respect to the same groups of bacterial (4.60 and 4.59, respectively). Samples without specific findings had the lower level of SCC except in the medium large herd size, where the level of SCC was higher than the level due to the effects of the bacterial groups (Figure 4).

Table 8: LS-means and S.E. of SCC (log) with respect to the herd size and independent on the bacterial status ($p=0.0001$)

Herd size	SCC (log)
Small	5.06±0.06
Medium small	4.91±0.03
Medium	4.92±0.03
Medium large	4.73±0.05
Large	4.58±0.03

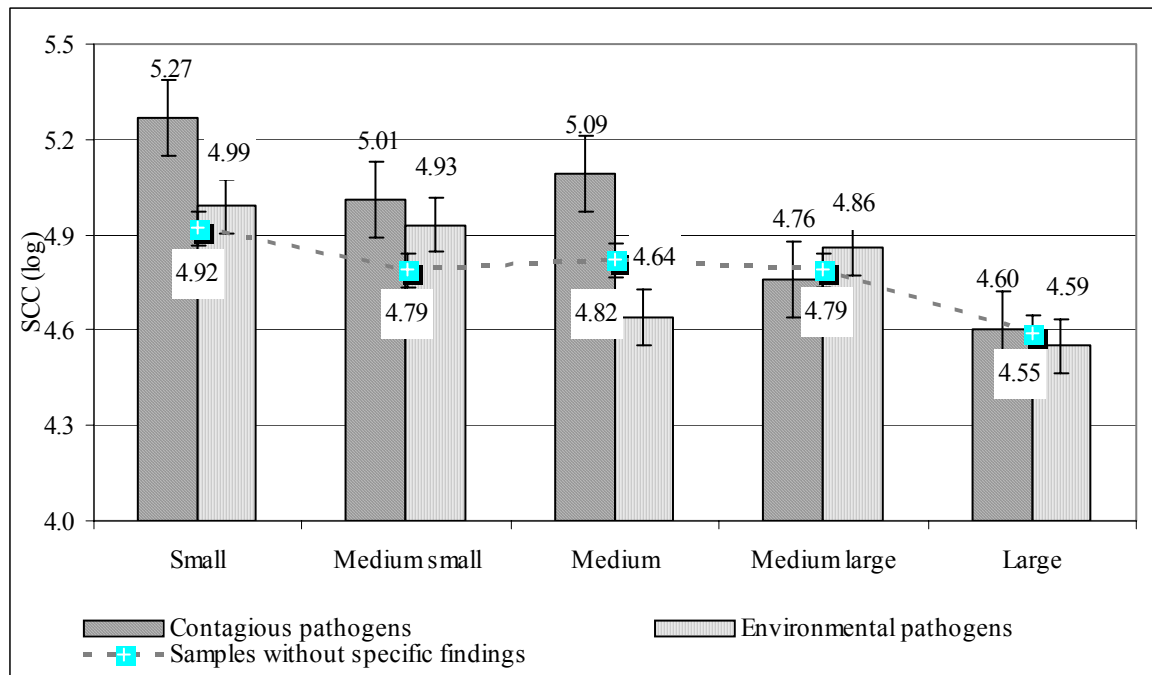


Figure 4: LS-means and S.E. of SCC (log) according to herd size and bacterial status ($p=0.0001$)

4.2.1.4 Test-day milk yield

Table 9 reveals that test-day milk yield showed a high significant ($p=0.0001$) variation in the different herd size classes. Milk yield was higher in the medium, medium large and large classes' herd size. That was significantly higher than in the small and medium small herd sizes. With the increase of the herd size, milk yield increases in a rate of 1.60 kg. Large class herd size scored the most higher daily milk yield (24.94 kg) that was nearly the same as in the medium and medium large classes herd size. Whereas the medium small class herd size had the lower milk yield (23.30 kg). Which was statistically the same as that of the small class herd size (23.35 kg). Class of the logarithmic SCC, herd size interaction significantly influenced the test-day milk yield. Milk yield decreases as the level of SCC increases. Medium large-class herd size obtained the over-all higher daily milk yield (28.28 kg) with low class of SCC than the other classes herd sizes. However, with high class of SCC (>5.73), the large class herd size was superior (22.22 kg) to the other classes' herd size (Figure 5). With the middle classes of SCC, milk yield was fairly the average of the extremes classes of SCC. That means with 2 folds increase of the SCC, milk yield decrease in a daily rate of 0.69 kg.

Table 9: LS-means and S.E. of test-day milk yield (kg) according to the herd size and irrespective of SCC (log) ($p=0.0001$)

Herd size	Test-day milk yield (kg)
Small	23.35 \pm 0.30
Medium small	23.30 \pm 0.22
Medium	24.31 \pm 0.16
Medium large	24.27 \pm 0.19
Large	24.94 \pm 0.18

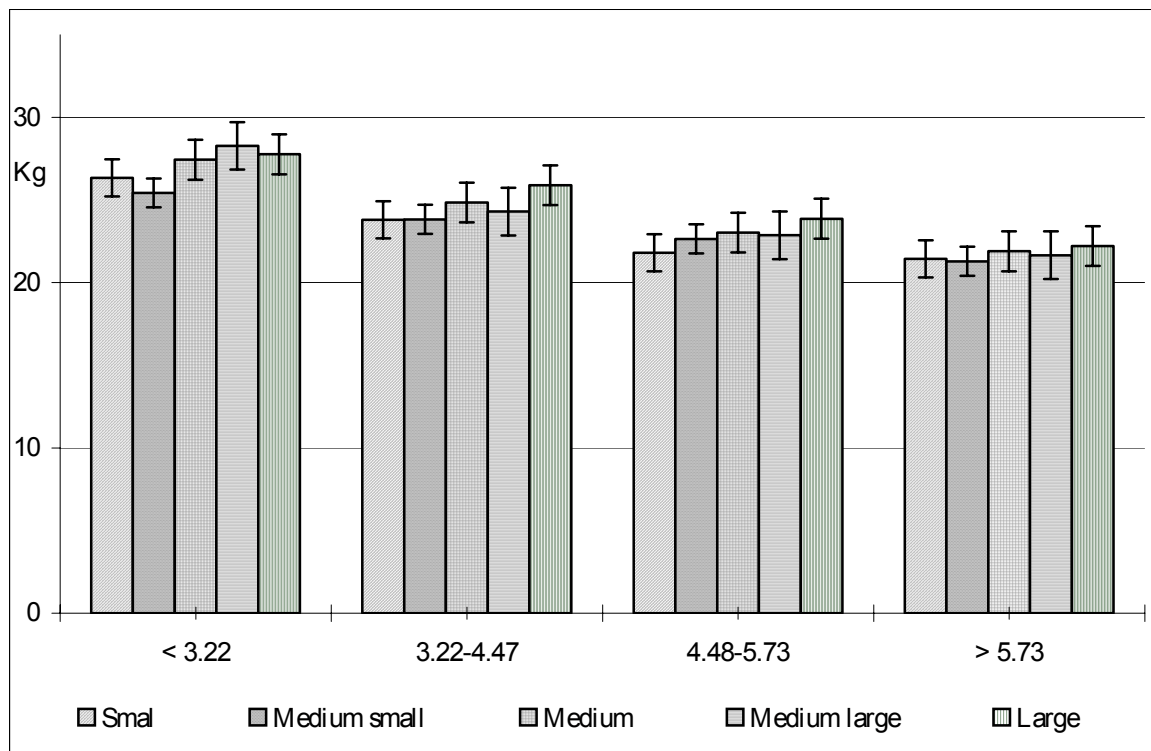


Figure 5: LS-means and S.E. of test-day milk yield (kg) with respect to herd size and SCC (log) ($p=0.0153$)

4.2.2 Year-season

4.2.2.1 Frequency of contagious and environmental pathogens

Figure 6 presents the number of positive findings as well as the relative distribution of the contagious and the environmental pathogens. The results found that the most frequent isolates were in winter 98/99, summer 99 and spring 2000 (32.51, 32.92 and 32.45%, respectively). Which were not statistically different from the frequency in autumn 99 and winter 99/2000 (31.90 and 30.79%, respectively). However, the lower finding was obtained in summer 98 (23.98%). The frequency of the contagious pathogens were higher than the frequency of the environmental pathogens. Higher frequency of the contagious pathogens was discovered in autumn 99 (75.48%) and the lower frequency was in winter 98/99 (65.20%). On the other hand, higher frequency of the environmental pathogens was indicated in winter 98/99 (34.80%) and the lower frequency was in autumn 99 (24.52%). In the other seasons the frequencies of the pathogens groups followed the same trend i.e. higher frequency of the contagious pathogens than the environmental pathogens and were nearly not different.

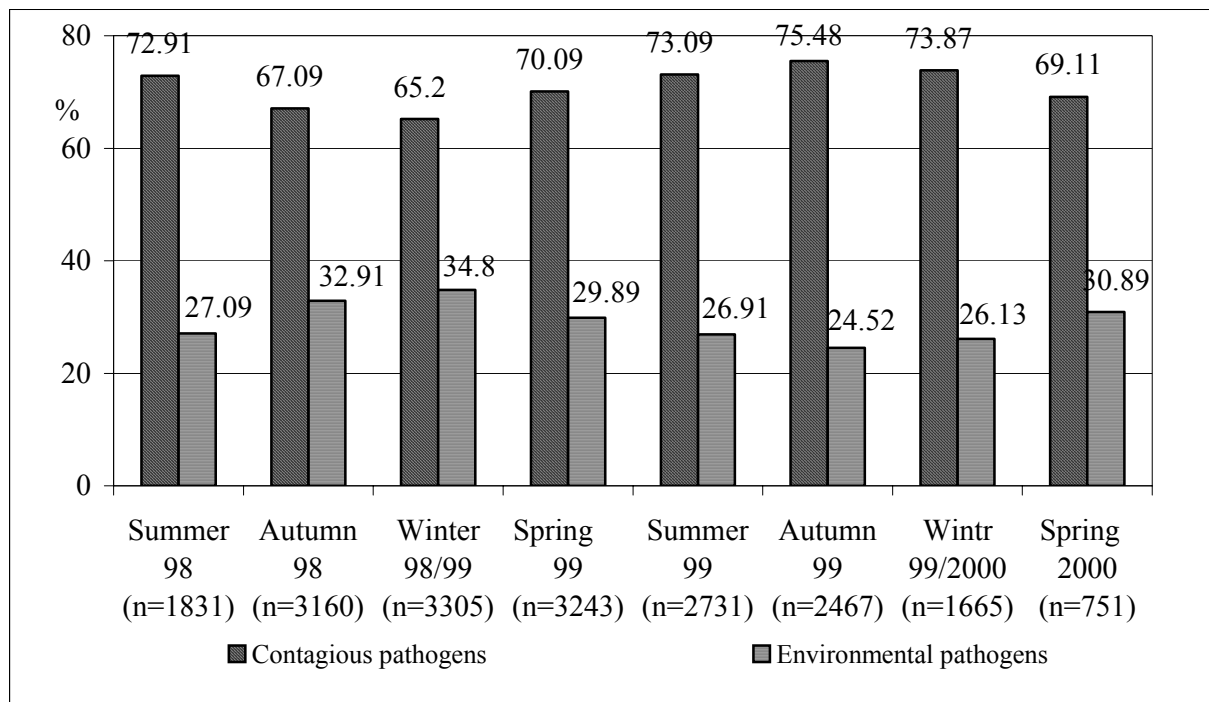


Figure 6: Frequency distribution of contagious and environmental pathogens among year-season ($p=0.001$)

4.2.2.2 Infection rate

Independent on the bacterial status, infection rate was highly significantly ($p=0.0001$) affected by the year-season. The LS-mean infection rate was the over-all higher during autumn 99 and lower during spring 2000 (Figure 7). It could be observed that the LS-means infection rate increased from summer 98 to autumn 98 (1.23 and 1.27, respectively). And a slight increase from autumn 98 to winter 98/99 (1.27 and 1.28, respectively) which was not maintained during spring 99 (1.27). Meanwhile the infection rate revealed a high significant ($p=0.0001$) variation due to the effect of the year-season bacterial status interaction (Figure 8). The contagious pathogens produced a higher infection in all year-season classes. The over-all higher infection rate due to the contagious pathogens was in summer 99 (1.51) and the lower infection rate due to the same bacterial group was in spring 2000 (1.27). The tendency of a higher infection rate due to the environmental pathogens was in autumn 99 (1.40) and spring 2000 for the lower value (1.17).

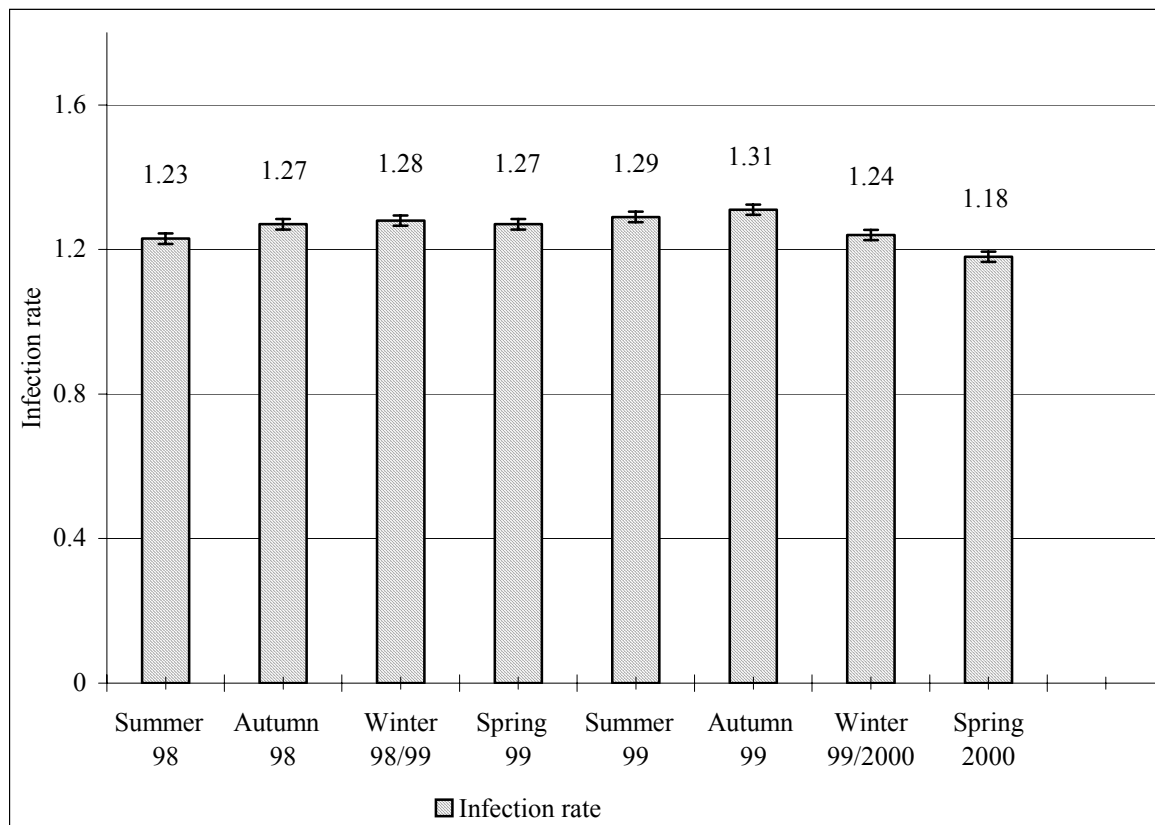


Figure 7: LS-means and S.E. of infection rate according to year-season and independent on bacterial status ($p=0.0001$)

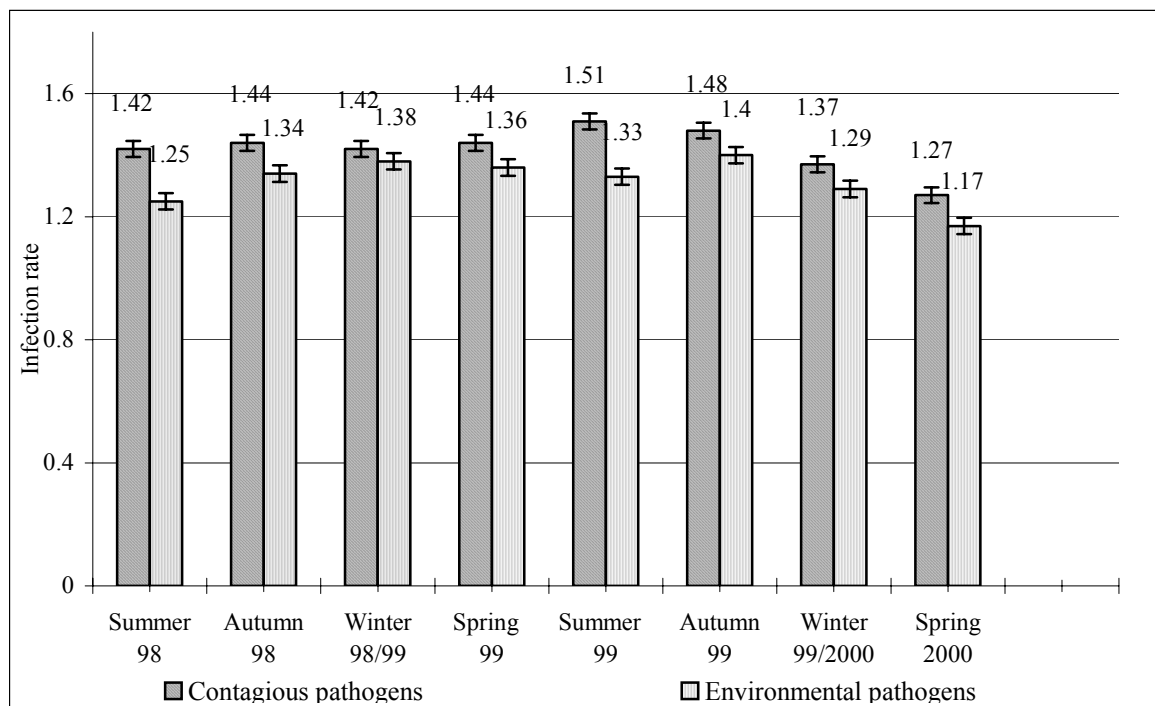


Figure 8: LS-means and S.E. of infection rate dependent on year-season and bacterial status ($p=0.0001$)

4.2.2.3 Logarithmic SCC

A highly significant ($p=0.0001$) variation of the SCC (log) was noticed due to the effect of the year-season (Figure 9). The result revealed that LS-mean SCC showed an increasing trend from summer 98 to autumn 98 (4.52 to 4.61). And a fair increase from autumn 98 to winter 98/99 (4.61 to 4.66). A decreasing pattern was noticed from winter 98/99 to spring 99 (4.66 to 4.63) which increased again during summer 99 (4.63) to reach the maximum value during spring 2000 (4.78). That was the same LS-mean in summer 99. However, the lower LS-mean SCC was found in autumn (4.59). The effect of the year-season dependent on the bacterial status was significant ($p=0.001$). The contagious pathogens initiated a significantly over-all higher SCC than that due to the effect of the environmental pathogens. High LS-mean SCC due to the effect of the contagious pathogens was experienced in winter 99/2000 and spring 2000 (4.78). But a lower LS-mean SCC was obtained in summer 98 that was insignificantly different from autumn 98 (4.67 and 4.68, respectively). On the other hand, a higher LS-means SCC due to the effect of the environmental pathogens were indicated in winter 98/99, autumn 99, summer 99 and spring 2000 (4.67, 4.65, 4.63 and 4.63, respectively). And a lower value was revealed in summer 98 (4.50) which was significantly lower than that in autumn 98 (4.54). However, SCC due to the effect of the non-specific findings exhibited a zigzag pattern during the season-year with means lie between 4.39-4.62. And the SCC curve was consistently under the SCC curves due to the effect of the contagious and the environmental bacteria except in autumn 98 when the level was higher than that due to the effect of the environmental pathogens (Figure 10).

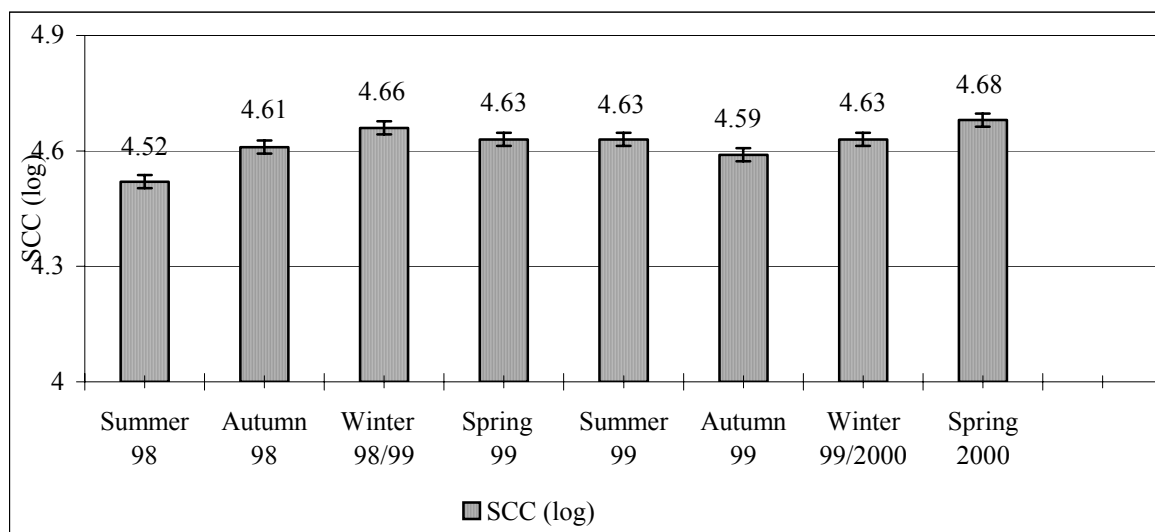


Figure 9: LS-means and S.E. of SCC (log) with respect to year-season ($p=0.0001$)

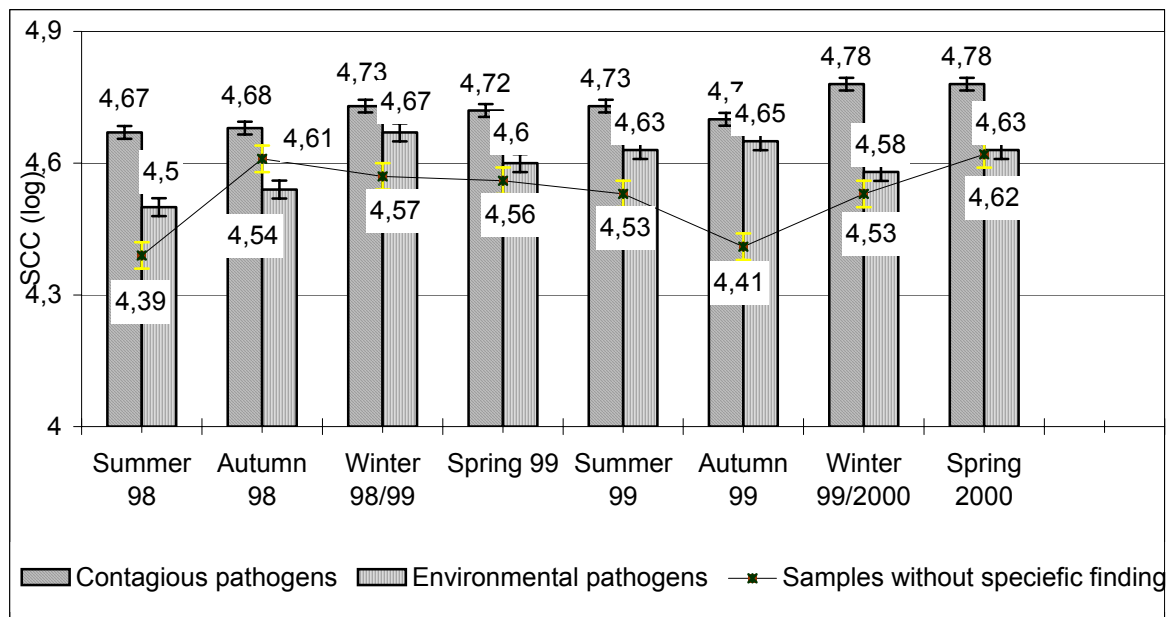


Figure 10: LS-means and S.E. of SCC (log) according to year-season and bacterial status ($p=0.001$)

4.2.2.4 Test-day milk yield

The year-season and independent on the level of SCC was found to affect the test-day milk yield ($p=0.0001$). Daily milk yield was found to increase in an increasing rate from summer 98 through the season in the year 99 and 2000 to spring 2000 (Figure 11). It could be observed that from summer 98 to autumn 98 there was 0.27 kg increase in the daily milk yield. And 0.17 kg decrease in the daily milk yield from autumn 98 to winter 98/99. Then the yield increased again in spring 99 and thereafter. With respect to the year-season class of SCC (log) interaction the test-day milk yield was highly significantly affected ($p=0.0001$). Daily milk yield practiced a decreasing trend with the increase in the level of the SCC (Figure 12). The over-all higher yield was in spring 02 (30.00 kg) and the lower yield was in summer 98 (25.94 kg). However, with the higher class of SCC (>5.73) milk yield was higher during autumn 2000 (23.11 kg) and lower in winter 98/99 (19.19 kg).

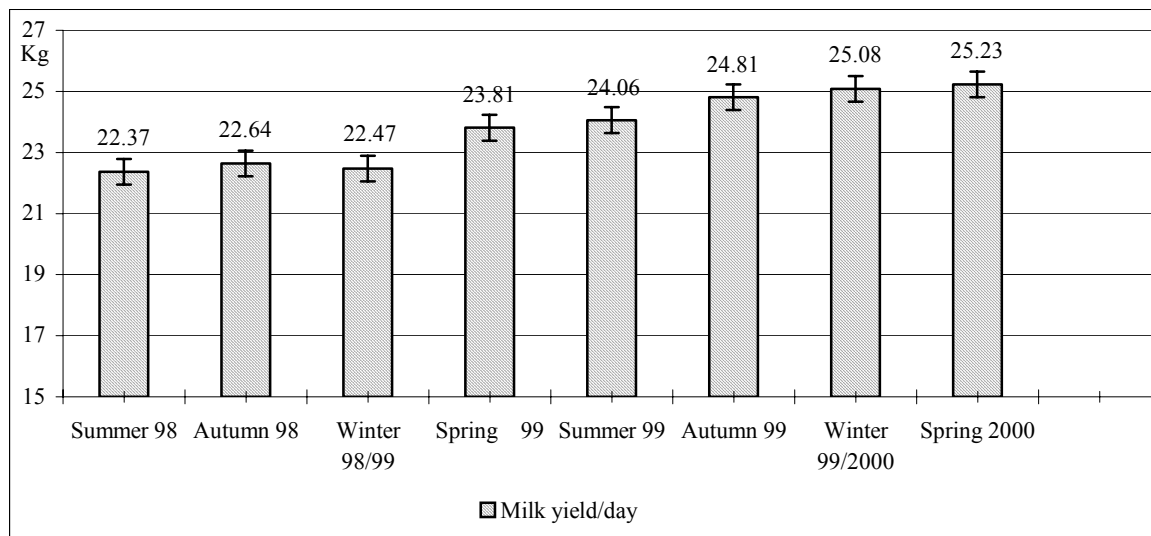


Figure 11: LS-means and S.E. of test-day milk yield (kg) dependent on year-season and irrespective of SCC (log) (p=0.0001)

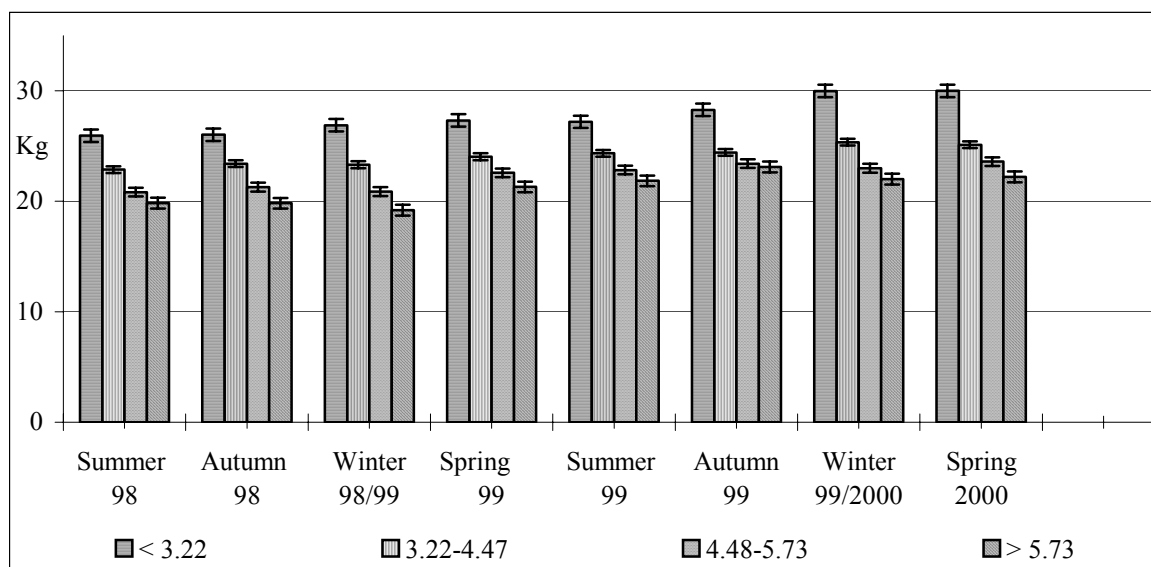


Figure 12: LS-means and S.E. of test-day milk yield (kg) with respect to year-season and SCC (log) (p=0.0001)

4.2.3 Lactation number

4.2.3.1 Frequency of contagious and environmental pathogens

Table 10 shows the distribution of the bacterial findings among the lactations. The positive findings steadily decreased from the first to the second lactation (32.06 to 27.05%, respectively). Then gradually increased in the third lactation to 27.98% and decreased again in the lactations after the third one (27.93%). Contagious pathogens were frequently isolated in the first lactation (72.93%) and found to be of a lower frequency in the

lactations after the third (64.28%). On the other hand, environmental pathogens were found to be higher in the lactations after the third (35.72%) and lower in the first lactation (27.07%). However, in the second and third lactations the frequencies of the environmental bacterial were not greatly differing (31.71 and 32.22%, respectively).

Table 10: Frequency distribution of contagious and environmental pathogens according to lactation number

Lactation number	Number of positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Lac.1 (n=36621)	11742 (32.06%)	23.38	72.93	08.79	27.07
Lac. 2 (n=11448)	3097 (27.05%)	18.47	68.29	08.58	31.71
Lac. 3 (n=8252)	2309 (27.98%)	18.96	67.78	09.02	32.22
>Lac. 3 (n=6505)	1817 (27.93%)	17.95	64.28	09.98	35.72
χ^2 -test ($\alpha=0.05$)	0.001				

4.2.3.2 Infection rate

The infection rate was shown to decrease from the first to the third lactation and thereafter as was shown in (Table 11). The difference in the mean infection rate among the lactations was highly significant ($p=0.0001$). Figure 13 reveals the effect of the lactation number on the infection rate dependent on the bacterial status. The effect was found to be highly significant ($p=0.0001$). In the figure, it could be clearly observed that the infection rate decreased as the animal goes older when it was infected with the environmental bacteria. From the second to the third lactation the mean infection rate due to the contagious pathogens was not changed (1.46). Which was lower than that in the first lactation (1.53) and the lactations after the third one (1.40).

Table 11: LS-means and S.E. of infection rate according to lactation number and irrespective of bacterial status ($p=0.0001$)

Lactation number	Infection rate
Lac.1	1.32±0.01
Lac.2	1.27±0.01
Lac.3	1.26±0.01
>Lac.3	1.24±0.01

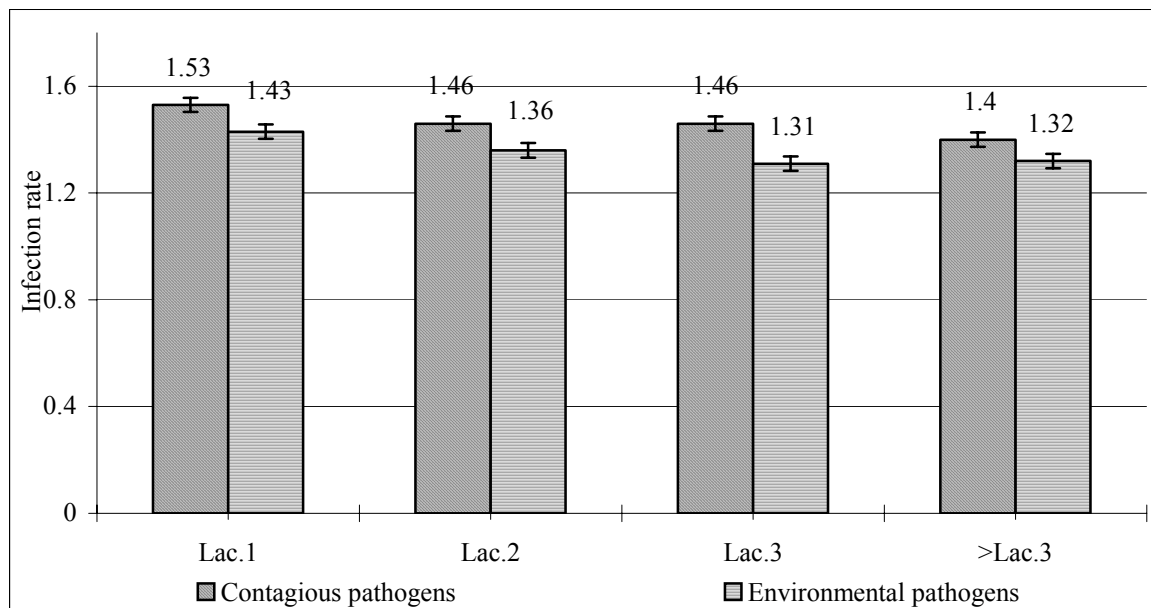


Figure 13: LS-means and S.E. of infection rate dependent on lactation number and bacterial status (p=0.0001)

4.2.3.3 Logarithmic SCC

The lactation SCC was significantly (p=0.0001) increased with the increase in the number of lactations (Table 12). Mean SCC was indicated higher (5.27) in the older cows (>Lac.3) and lower (4.48) in heifers (Lac.1). With respect to the lactation number, bacterial status interactions (Table13) the mean SCC was found to increase in a step wise manner as the lactation number increased after the first lactation in concern of both the contagious and the environmental pathogens. With the difference being significant (p=0.0002).

Table 12: LS-means and S.E. of SCC (log) with respect to lactation number and independent on bacterial status(p=0.0001)

Lactation number	SCC (log)
Lac. 1	4.48±0.02
Lac. 2	4.79±0.03
Lac. 3	5.04±0.03
> Lac. 3	5.27±0.03

Table 13: LS-means and S.E. of SCC (log) with respect to lactation number and bacterial status (p=0.0002)

Bacterial status	Lactation number			
	Lac. 1	Lac. 2	Lac. 3	>Lac. 3
Contagious bacteria	4.57±0.03	4.81±0.04	5.26±0.04	5.51±0.05
Environmental bacteria	4.42±0.04	4.81±0.05	4.97±0.06	5.10±0.07
Samples without specific findings	4.37±0.02	4.56±0.02	4.97±0.03	5.17±0.03

Contagious bacterial was found to elevate the level of SCC above the level that was reached due to the effect of the environmental bacteria. The level of the SCC in one way or another depends on the presence of a pathogen, this was observed in the cases where no specific mastitis causing bacteria were found which had the lower SCC.

4.2.3.4 Test-day milk yield

The lactation number revealed a significant (p=0.0001) effect on the milk yield/day (Table 14). The milk yield was increased from the first lactation onwards irrespective of the level of the SCC. The test-day milk yield was found to increase in an increasing rate as the lactation number increased till the third lactation where it reached the peak (31.12). Then started to decrease in a decreasing rate with the lower class of SCC (<3.22). As was revealed in table 15 the daily milk yield was highly significantly (p=0.0001) affected by the lactation number dependent on the level of SCC. In the entire lactation, milk yield decreased in an increasing rate as the level of SCC increased. At the higher class of SCC (>5.73) the yield was increased in a decreasing rate as lactation number increased.

Table 14: LS-means and S.E. of test-day milk yield (kg) according to lactation number and irrespective of SCC (log) (p=0.0001)

Lactation number	Test-day milk yield (kg)
Lac. 1	21.12±0.14
Lac. 2	24.33±0.16
Lac. 3	25.70±0.17
>Lac. 3	25.80±0.20

Table 15: LS-means and S.E. of milk test-day milk yield (kg) according to lactation number and SCC (log) (p=0.0001)

Class of SCC	Lactation number			
	Lac. 1	Lac. 2	Lac. 3	>Lac. 3
<3.22	23.33±0.26	28.57±0.34	31.12±0.43	30.20±0.54
3.22-4.47	21.15±0.14	25.42±0.17	26.39±0.20	26.91±0.23
4.48-5.73	20.37±0.14	22.51±0.17	23.61±0.19	23.81±0.21
>5.73	19.65±1.00	20.83±0.23	21.68±0.24	22.28±0.26

4.2.4 Stage of lactation

Stage of lactation is defined as the intervals within the lactation. In the present study the lactation is classified into three stages each of 100 days. And the study considered also the period before calving for the estimation of the bacterial frequency.

4.2.4.1 Frequency of contagious and environmental pathogens

The percentages of the positive bacterial findings with respect to the stage of lactation were significantly (p=0.001) different. The frequency of the contagious pathogens was found to be the highest in the samples collected from heifers' a.p (74.04%). But in the early stage of lactation the frequency of the contagious pathogens was 70.45% and 57.00% in the middle stage of lactation. However, the frequency increased to 69.74% in the late stage of lactation. On the other hand, the reverse condition was showed by the environmental pathogens. They increased with the decreased frequency of the contagious pathogen before calving, early and middle stages of lactation (21.47, 29.55 and 43.00%, respectively). Whereas the frequency of the environmental pathogens decreased to 30.26% in the late stage of lactation. A χ^2 -test between the contagious and the environmental pathogens were significant ($\alpha<0.05$) as was shown in table 16.

Table 16: Frequency distribution of contagious and environmental pathogens according to time of sampling

Time of sampling	Number of positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Before calving (n=11864)	3447 (29.05%)	21.51	74.04	06.24	21.47
Early stage of lactation (n=44199)	13657 (30.90%)	21.77	70.45	09.13	29.55
Middle stage of lactation (n=2082)	486 (23.34%)	13.30	57.00	10.04	43.00
Late stage of lactation (n=4195)	1246 (29.70%)	20.71	69.74	08.99	30.26
χ^2 -test ($\alpha=0.05$)	0.001				

4.2.4.2 Infection rate

Table 17 reveals that although the mean infection rate irrespective of the bacterial groups was slightly higher in the early stage of lactation (1.28). But the difference from the middle and late stages of lactation (1.27) was statistically not significant ($p>0.05$). The same applies to the effect of the stage of lactation with respect to the contagious and the environmental pathogens. No statistical variations were encountered ($p>0.05$), even though the contagious pathogens were found to cause a higher infection rate in the early stage of lactation (1.47) that was higher than the infection rate caused by the environmental pathogens (1.37). With the progress from the middle to the late stage of lactation the mean infection rate due to the effect of either the contagious or the environmental pathogens was not changed (Figure 14).

Table 17: LS-means and S.E. of infection rate according to stage of lactation and irrespective of bacterial status ($p>0.05$)

Stage of lactation	Infection rate
Early	1.28±0.01
Middle	1.27±0.01
Late	1.27±0.01

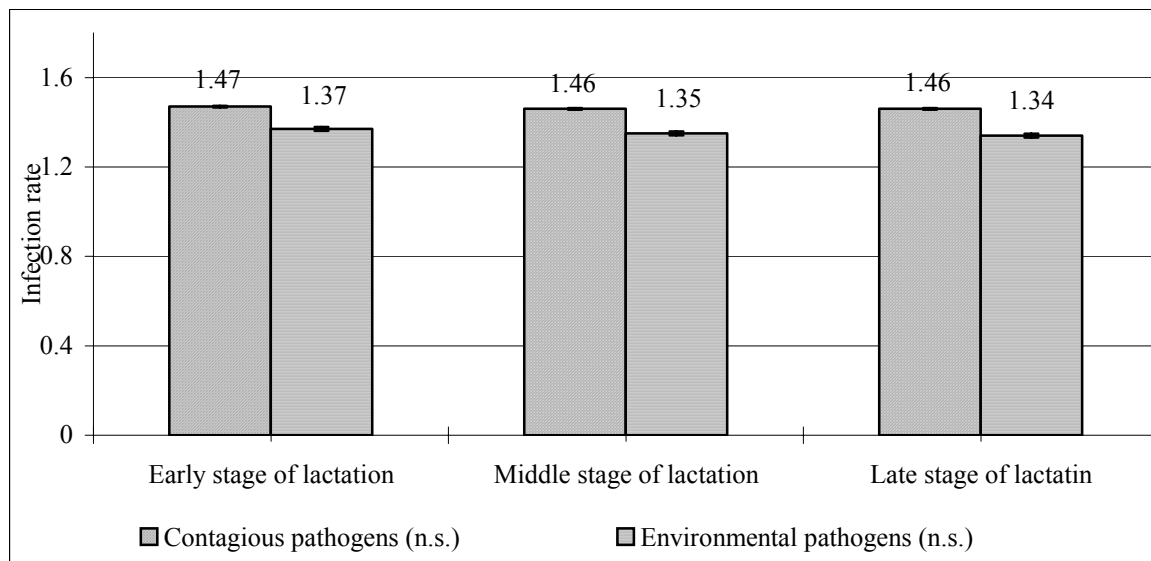


Figure 14: LS-means and S.E. of infection rate dependent on stage of lactation and bacterial status ($p=0.4273$)

4.2.4.3 Logarithmic SCC

The stage of lactation and irrespective of the bacterial status was found to have a highly significant ($p=0.0001$) effect on the SCC. And as appears in table 18 the LS-mean SCC increased gradually from the early stage of lactation (4.85) to the middle stage of lactation (4.87). And it reached the higher level in the late stage of lactation (4.96). Although the stage of lactation*bacterial status interaction was not significantly affecting the SCC (Table 19). However, the LS-mean SCC due to effect of the contagious as well as the environmental bacteria increased as the lactation advanced. It was also observed that the increase in the LS-mean SCC due to the effect of the environmental bacteria was not very different from the normal physiological increase in the SCC.

Table 18: LS-means and S.E. of SCC (log) with respect to stage of lactation and independent on bacterial status ($p=0.0001$)

Stage of lactation	SCC (log)
Early	4.85±0.02
Middle	4.87±0.03
Late	4.96±0.03

Table 19: LS-means and S.E. of SCC (log) with respect to stage of lactation and bacterial status and ($p>0.05$)

Bacterial status	Stage of lactation		
	Early	Middle	Late
Contagious bacteria	5.00±0.03	5.01±0.03	5.11±0.04
Environmental bacteria	4.79±0.04	4.79±0.04	4.90±0.05
Samples without specific findings	4.71±0.02	4.75±0.02	4.84±0.02

4.2.4.4 Test-day milk yield

The test-day milk yield in this study was tested separately: independent and dependent on the level of SCC. Independent on the class of the SCC the LS-mean milk yield/day was varied significantly (0.0001) with the stage of lactation. In the early stage of lactation the LS-mean milk yield/day was significantly higher (28.41 kg). This quantity was decreased to 24.79 kg/day in the middle stage of lactation. On the other hand, only 19.52 kg was the LS-mean milk yield/day produced in the late stage of lactation (Table 20). Figure 15 presents the effect of the stage of lactation on the daily milk yield tested with different classes of SCC. The effect was found to be highly significant ($p=0.0001$). The result obtained showed that within the same stage of lactation the LS-mean daily milk yield revealed a decreasing pattern as the level of the SCC increased. In the early term of the lactation the LS-means milk yield/day was fairly higher than that scored in the late term of the lactation at a lower class of SCC (31.42 vs. 24.85 kg). The LS-means milk yield/day in the middle stage of lactation were relatively the mean of the early and late stages of the lactation. Towards the end of lactation when the somatic cell was at the higher level the LS-mean daily milk yield was very low (15.16 kg).

Table 20: LS-means and S.E. of test-day milk yield (kg) according to stage of lactation and irrespective of SCC (log) ($p=0.0001$)

Stage of lactation	Test-day milk yield (kg)
Early	28.41±0.14
Middle	24.79±0.15
Late	19.52±0.18

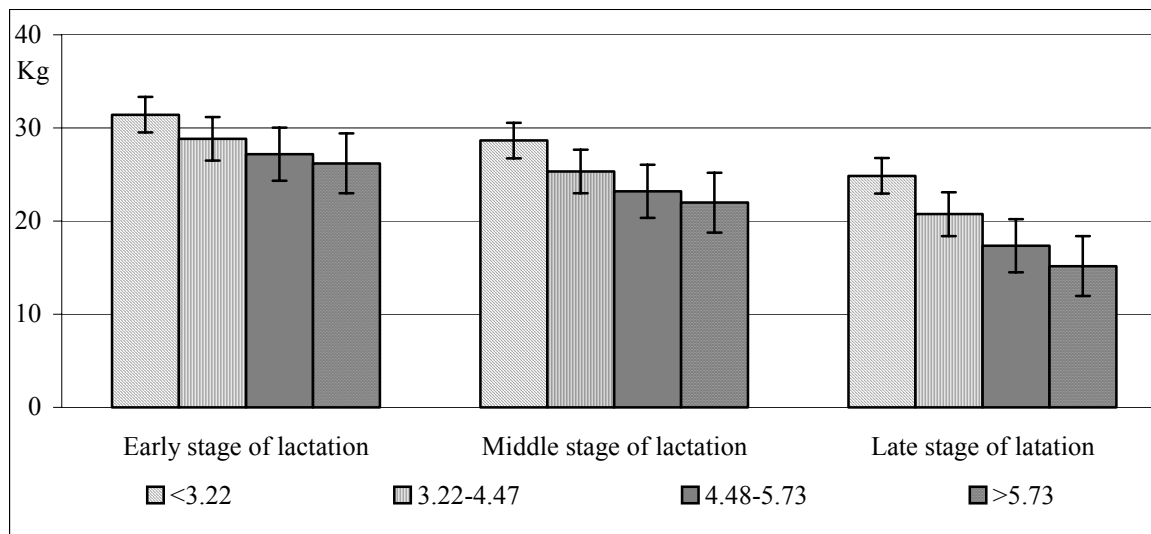


Figure 15: LS-means and S.E. of test-day milk yield (kg) with respect to stage of lactation and SCC (log) (p=0.0001)

4.2.5 Origin of the cow

4.2.5.1 Frequency of contagious and environmental pathogens

The performed χ^2 -test revealed a significant difference of the frequency of the positive samples obtained from the farm bred and the purchased animals (p=0.001). The percentage frequency of the positive samples detected in the purchased animals was higher (37.53%) than that of the farm-bred animals (29.87%) as was shown in table 21. In concern of the bacterial groups, contagious pathogens were of higher frequency in the farm bred animals (72.28%) compared to 64.97% in the purchased animals. However, the frequency of the environmental pathogens was merely the reciprocal of the frequency of the contagious pathogens. It revealed a higher frequency in farm-bred animals (35.03%) than that in the purchased animals (27.72%).

Table 21: Frequency distribution of contagious and the environmental pathogens according to origin of the cow

Origin of the cow	Number of positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Farm bred (n=50106)	14966 (29.87%)	21.59	72.28	08.28	27.72
Purchased (n=5720)	2147 (37.53%)	24.38	64.97	13.15	35.03
χ^2 -test ($\alpha=0.05$)	0.001				

4.2.5.2 Infection rate

Infection rate was obtained as a type of a bacterium presents in one or more of the udder quarters. Table 22 shows that purchased cows had a significantly (0.0001) higher LS-mean infection rate (1.33) than the farm bred cows (1.29). Whereas table 23 illustrates that the LS-mean infection rate due to the environmental pathogens was higher in the purchased cows (1.57) at the time that the effect of the interacted factors was highly significant ($p=0.0001$) on the infection rate. It was also observed that the LS-mean infection rate due to the contagious pathogens did not differ between the two groups of cows. Infection rate due to the environmental pathogens on the farm-bred cows was lower among all groups (1.40).

Table 22: LS-means and S.E. of infection rate according to origin of the cow and irrespective of bacterial status ($p=0.0001$)

Origin of the cow	Infection rate
Farm bred	1.29±0.00
Purchased	1.33±0.01

Table 23: LS-means and S.E. of infection rate according to origin of the cow and bacterial status and ($p=0.0001$)

Bacterial status	Origin of the cow	
	Farm bred	Purchased
Contagious bacteria	1.52±0.00	1.52±0.01
Environmental bacteria	1.40±0.01	1.57±0.02

4.2.5.3 Logarithmic SCC

Table 24 demonstrates that farm-bred cows had a significantly (0.0001) higher LS-mean SCC (4.95) than the purchased cows (4.83). Though, Figure 16 exhibits that the effect of the origin of the cow interacted with the bacterial status on the SCC was found to be highly significant ($p=0.0001$). Within each group of the cows the LS-means SCC were significantly different. SCC was higher in the two groups of cows infected with contagious pathogens. Samples in which no specific bacteria were discovered had a lower SCC between the groups. Farm bred cows that were infected with environmental bacteria showed a slightly higher level of SCC (4.91) than the level of the purchased cows (4.87).

Table 24: LS-means and S.E. of SCC (log) with respect to origin of the cow and independent on the bacterial status (p=0.0001)

Origin of the cow	SCC (log)
Farm-bred	4.95±0.02
Purchased	4.83±0.02

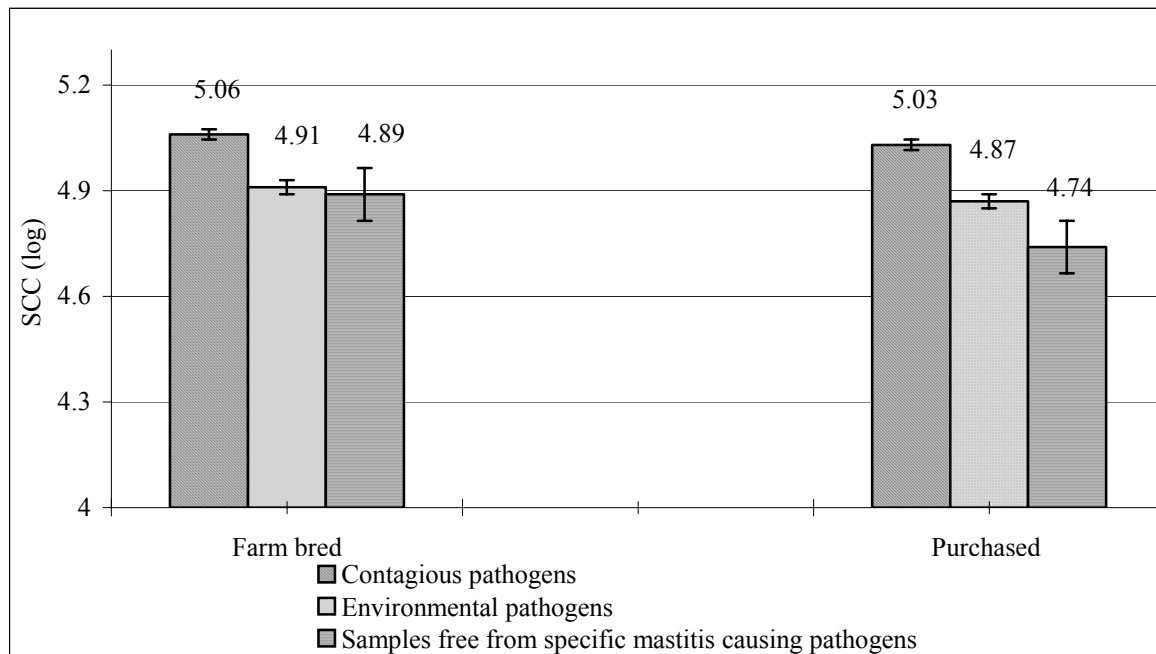


Figure 16: LS-means and S.E. of SCC (log) according to origin of the cow and bacterial status (p=0.0001)

4.2.5.4 Test-day milk yield

The study found that the daily milk yield was highly significantly (0.0001) higher (24.78 kg) in the farm-bred cows compared to the daily production of the purchased cows (22.39 kg) as was indicated in table 25. When origin of the cow was incorporated in the statistical model as a factor in an interaction with the class of the logarithmic SCC the effect was found to be highly significant (p=0.0001) as was presented in table 26. Within each group of cows daily milk yield shows a decreasing trend as the level of SCC increases. Farm bred cows were superior in the daily milk production with respect to all classes of SCC. Farms whose heifers were purchased scored a daily milk yield that could be ordered in the second place with respect to all classes of SCC. And when the level of SCC was mostly higher (>5.73) the LS-mean daily milk yield dropped to 16.26 kg.

Table 25: LS-means and S.E. of test-day milk yield (kg) according to origin of the cow and irrespective of SCC (log) (p=0.0001)

Origin of the cow	Test-day milk yield
Farm bred	24.78±0.09
Purchased	22.39±0.12

Table 26: LS-means and S.E. of test-day milk yield (kg) according to origin of the cow and SCC (log) (p=0.0001)

SCC (log)	Origin of the cow	
	Farm-bred	Purchased
<3.22	28.74±0.31	23.90±1.20
3.22-4.47	25.48±0.11	22.61±0.39
4.48-5.73	23.25±0.10	19.13±0.37
>5.73	21.51±0.16	16.26±0.57

4.2.6 Housing system

Animals participated in this investigation were housed in one of three housing systems. Namely slat floor loose housing system with straw or rubber bedding. Plan loose housing system that was also bedded with straw or rubber. And the third system includes tie-stall barn and/or combination of one or more of the former types.

4.2.6.1 Frequency of contagious and environmental pathogens

Frequencies of the mastitis causing bacterial that have been isolated from the udder quarters and the udder's samples were found to be significantly (p=0.001) different between the houses kept cows. The pattern of the distribution was that contagious pathogens had a higher percentage of the positive samples in both sites of isolation. Cows housed in a loose housing with plan floor harbors more contagious pathogens (68.42%). Compared to 66.68% of the contagious pathogens that were discovered in the sampled cows kept in the loose stall with slat floor (Table 27). Cows housed in the other housing system encountered an extremely higher percentage of pathogens in their samples (73.93%). The reverse to this distribution was that of the environmental pathogens.

Table 27: Frequency distribution of contagious and environmental pathogens according to housing system

Housing system	Number of positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Loose housing with slat floor (n=20597)	6138 (29.80%)	19.87	66.68	09.93	33.32
Loose housing with plan floor (n=20524)	5589 (27.23%)	18.63	68.42	08.60	31.58
Other (n=5022)	1542 (30.70%)	22.70	73.93	08.00	26.07
χ^2 -test ($\alpha=0.05$)	0.001				

4.2.6.2 Infection rate

LS-mean infection rate was found to be significantly ($p=0.0001$) varied between the stalls kept cows (Table 28). Cows housed in either slat or plan floor loose housing barns had a higher infection rate (1.26 and 1.27, respectively). These results were significantly higher than those of the cows kept in barns other than loose housing (1.22). On the other hand Figure 17 shows that with respect to the bacterial status, housing system was significantly ($p=0.0001$) affected the LS-means infection rate. Cows kept in a plan floor loose housing were subjected to a higher infection with contagious pathogens (1.45) than those managed in other housing systems (1.43). The latter group of cows were found to be less threatened with the environmental pathogens (1.22), compared to those live in a slat or a plan floor loose housing (1.36 and 1.38, respectively).

Table 28: LS-means and S.E. of infection rate according to housing system and irrespective of the bacterial status ($p=0.0001$)

Housing system	Infection rate
Loose housing with slat floor	1.26±0.01
Loose housing with plan floor	1.27±0.01
Other	1.22±0.01

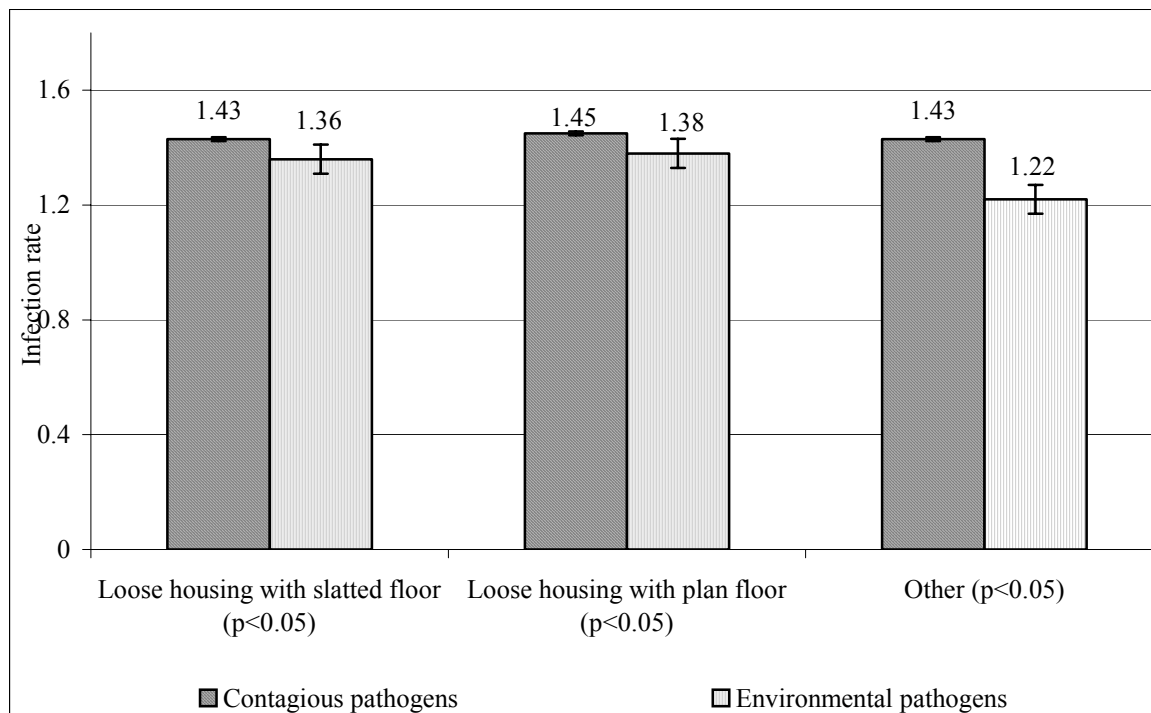


Figure17: LS-means and S.E. of infection rate dependent on housing system and bacterial status (p=0.0001)

4.2.6.3 Logarithmic SCC

It can be observed in table 29 that the SCC showed a high significant ($p=0.0001$) variation in the cows housed in different barns. On ignoring the bacterial effect, loose housing with slat floor reduced the LS-mean SCC to a lower level (4.77). Whereas, barns other than the loose housing elevated the LS-mean SCC to a higher level (5.05). But not very high than the level obtained in the loose housing with plan floor (4.95). Likewise the effect of the bacteria and the housing system interaction on SCC was similarly highly significant ($p=0.0001$) (Table 30). However, the result reveals that the LS-mean SCC was higher in cows kept in houses other than the loose barn and inflicted with contagious pathogens (5.29) than those housed in slat and plan floors loose housing (5.02 and 5.13, respectively). The LS-mean SCC due to a non-specific infection was significantly higher in loose housing kept cows (4.86 and 4.80) than those kept in other housing (4.41).

Table 29: LS-means and S.E. of SCC (log) with respect to housing system and independent on the bacterial status ($p=0.0001$)

Housing system	SCC (log)
Loose housing with slat floor	4.77±0.02
Loose housing with plan floor	4.95±0.02
Other	5.05±0.04

Table 30: LS-means and S.E. of SCC (log) with respect to housing system and bacterial status (p=0.0001)

Bacterial status	Housing system		
	Loose housing with slat floor	Loose housing with plan floor	Other
Contagious pathogens	5.02±0.03	5.13±0.02	5.29±0.13
Environmental pathogens	4.89±0.04	4.83±0.04	4.70±0.08
Samples without specific findings	4.86±0.02	4.80±0.01	4.41±0.24

4.2.6.4 Test-day milk yield

Although, the daily milk yield independent on the SCC level was highly significantly (0.0001) different among the housing systems (Table 31). But the result indicated a non-significant effect of the housing systems dependent on the SCC level on test-day milk yield (p>0.05). Cows housed in barns other than loose housing produced a relatively low milk yield/day (21.76 kg) than their counterparts housed in loose housing (23.16 and 23.21 kg). The test-day milk yield was statistically the same with respect to housing system logarithmic cell count interaction (Figure 18). However, with a low level of SCC the milk yield was higher and vise versa.

Table 31: LS-means and S.E. of test-day milk yield (kg) according to housing system and irrespective of SCC (log) (p=0.0001)

Housing system	Test-day milk yield (kg)
Loose housing with slat floor	23.16±0.08
Loose housing with plan floor	23.21±0.10
Other	21.76±0.23

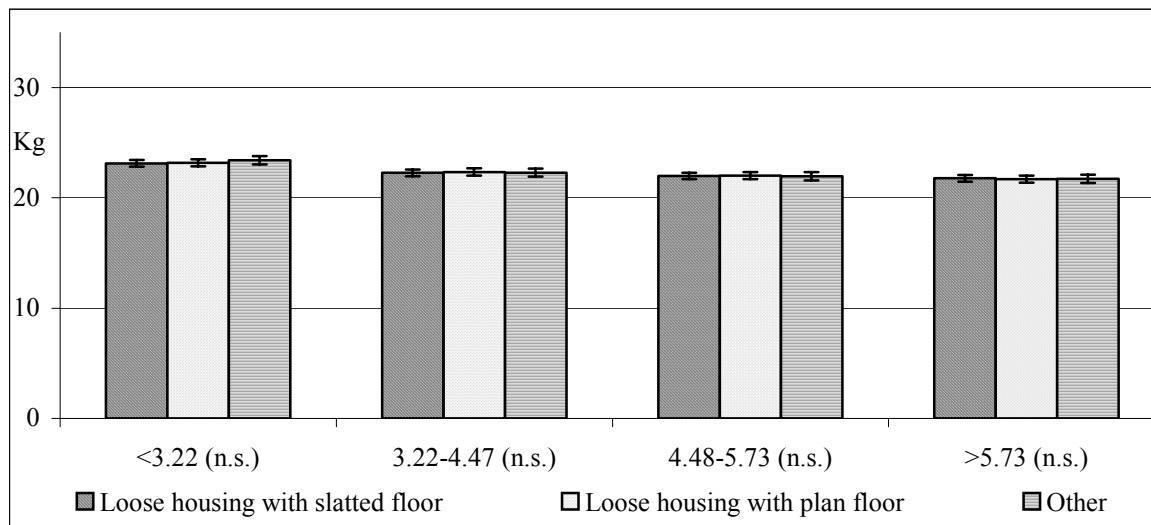


Figure 18: LS-means and S.E. of test-day milk yield (kg) with respect to housing system and SCC(log) ($p>0.05$)

4.2.7 Milking system

Farms included in the study had three types of milking systems, either pipe system, carrousel or milking parlors. Most of the farms investigated (64 %) found to use the later milking unit.

4.2.7.1 Frequency of contagious and environmental pathogens

Table 32 shows a significant difference among the three milking systems in sense of the bacteria isolated from the udder quarter's and the udder's samples. The result discovered that the contagious pathogens irrespective of the farm milking system scored a higher frequency on absolute and relative bases. The frequency of the findings discovered in the animals milked with the pipe system was higher (35.45%). While the lower frequency was found in animals milked using milking parlor (29.33%). The study also found that the frequency of the contagious pathogens was higher with the use of the carrousel system (75.10%) than with the use of the other milking systems. Whereas the frequency of the environmental pathogens was higher with the use of the pipe system (31.56%).

Table 32: Frequency distribution of contagious and environmental pathogens according to milking system

Milking system	Number of positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Pipe system (n=959)	339 (35.45%)	24.26	68.44	11.88	31.56
Carrousel (n=22687)	7336 (32.34%)	24.29	75.10	08.05	24.90
Milking parlor (n=32180)	9438 (29.33%)	20.13	68.63	09.20	31.37
χ^2 -test ($\alpha=0.05$)	0.001				

4.2.7.2 Infection rate

Irrespective of the bacterial findings a significant ($p=0.003$) variation in the LS-mean infection rate was encountered due to the effect of the Milking systems (Table 33). The LS-mean infection rate was significantly higher (1.34) in animals milked using the pipe system than those milked in either the carrousel or the milking parlor units (1.30 and 1.29, respectively). Similarly, milking system was positively interacting with the bacterial status to influence the infection rate. This was found to be highly significant ($p=0.0001$). Animals their milk flow through the pipe system were found to have a high infection rate with contagious pathogens as well as with environmental pathogens (Table 34). Infection rate with contagious bacteria in the animals milked using pipe system was higher (1.63) than those milked using carrousel (1.57) and milking parlor (1.48). Environmental pathogens were found to cause a high infection rate in the animals used pipe system to pass their milk into the collection tanks (1.49). Whereas milking parlor and carrousel units were found to have a lower LS-mean infection rate due to the environmental pathogens effect (1.44 and 1.36, respectively) compared to the former unit.

Table 33: LS-means and S.E. of infection rate according to milking system irrespective of bacterial status ($p=0.003$)

Milking system	Infection rate
Pipe system	1.34±0.01
Carrousel	1.30±0.01
Milking parlor	1.29±0.01

Table 34: LS-means and S.E. of infection rate according milking system and bacterial status (p=0.0001)

Bacterial status	Milking system		
	Pipe system	Carrousel	Milking parlor
Contagious bacteria	1.63±0.03	1.57±0.01	1.48±0.01
Environmental bacteria	1.49±0.05	1.36±0.01	1.44±0.01

4.2.7.3 Logarithmic SCC

Table 35 demonstrates that the SCC was significantly ($p<0.05$) varied among the milking systems. The LS-mean SCC was significantly higher in animals milked in the milking parlor (4.93) than those milked in the carrousel units (4.89) and the pipe system (4.84). The study also indicated that the SCC showed a high significant ($p=0.0001$) variability as a result of the effect of the milking system bacterial status interaction (Table 36). Animals whose milking unit was the pipe system, and were found to be infected with contagious bacteria scored the higher LS-mean SCC (5.34). Milking parlor and carrousel follow this finding (5.11 and 5.05, respectively). However, milking parlor-milked animals that were infected with environmental bacteria elevated the logarithmic SCC to 4.89. This was not greatly differing than that recorded in carrousel and pipe system milked animals (4.88 and 4.76, respectively).

Table 35: LS-means and S.E. of SCC (log) with respect to milking system and independent on bacterial status (p=0.0182)

Milking system	SCC (log)
Pipe system	4.84±0.08
Carrousel	4.89±0.02
Milking parlor	4.93±0.01

Table 36: LS-means and S.E. of SCC (log) with respect to milking system and bacterial status (p=0.0001)

Bacterial status	Milking system		
	Pipe system	Carrousel	Milking parlor
Contagious bacteria	5.34±0.09	5.05±0.03	5.11±0.02
Environmental bacteria	4.76±0.05	4.88±0.02	4.89±0.03
Samples without specific findings	4.42±0.20	4.74±0.05	4.80±0.01

4.2.7.4 Test-day milk yield

With the use of the carousel unit and independent on the SCC milk yield/day was highly significantly ($p=0.0001$) higher (24.49 kg) than the daily milk yielded with the use of the milking parlor (22.98 kg) and pipe system (19.84 kg) (Table 37). Likewise, the two factors in interaction significantly ($p=0.0001$) affecting the LS-mean test-day milk yield. The trait was found to decrease as the SCC increased. Cows milked in a carousel unit were practically produced a high daily milk yield (28.58 kg) when the SCC level was low. But with the high level of SCC (>5.73) animals milked in the carousel and the milking parlor performed fairly the same quantity (21.33 and 21.39 kg, respectively). When the SCC is of the middle classes animals that were milked in the carousel units produced a high daily milk than those milked using pipe system unit and milking parlor (Figure 19).

Table 37: LS-means and S.E. of test-day milk yield (kg) according to milking system and irrespective of SCC (log) ($p=0.0001$)

Milking system	Test-day milk yield (kg)
Pipe system	19.84 \pm 0.46
Carrousel	24.49 \pm 0.10
Milking parlor	22.98 \pm 0.07

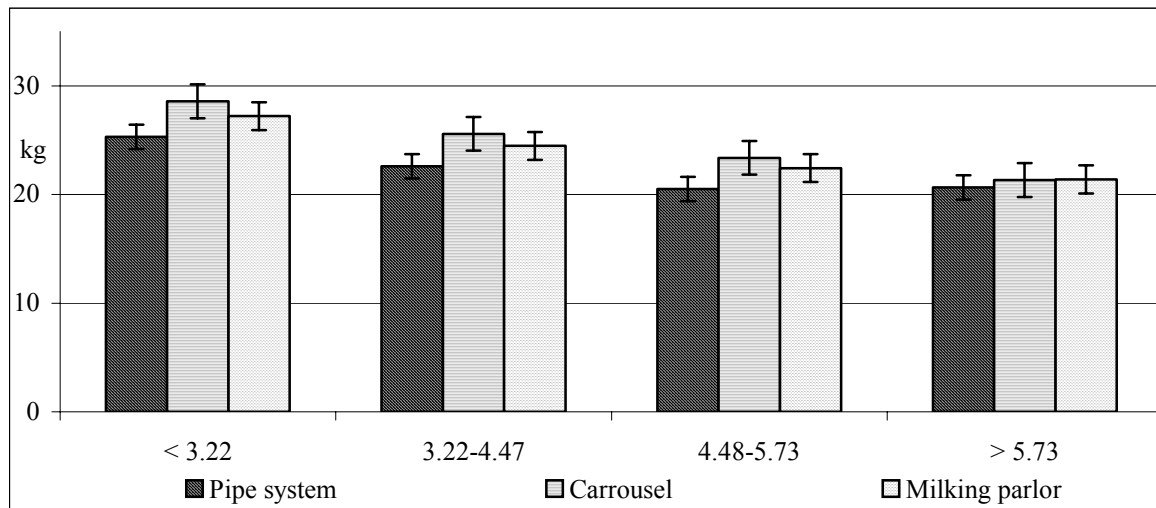


Figure 19: LS-means and S.E. of test-day milk yield (kg) with respect to milking system and SCC (log) ($p=0.0001$)

4.2.8 Feeding method

Dairy farms participated in the study were reported to use three methods to feed their animals: mobile, stationary or in some farms both methods. These methods were run in the analysis of variance models to test their effects on the determinants of the IMI.

4.2.8.1 Frequency of contagious and environmental pathogens

Table 38 presents the distribution of the two groups of mastitis causing pathogens in accordance with the feeding methods. The statistical difference among the methods were found to be insignificant ($p>0.05$). The result also showed that sampled animals that were fed with both feeding methods showed a higher frequency of the positive samples (32.43%). Whereas in the mobile and stationary methods the frequency was statistically the same. Contagious pathogens were frequent in the samples of the animals in the farms practice both methods of feeding (71.57%). However, the frequency of the environmental pathogens based on the relative values was higher in the farms intended to use a stationary feeding method (29.41%). And based on the absolute values the frequency was higher in animals fed with both methods (09.22%).

Table 38: Frequency distribution of contagious and environmental pathogens according to feeding method

Feeding method	Number of positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Mobile (n=35799)	10802 (30.17%)	21.18	70.21	08.99	29.79
Stationary (n=18422)	5572 (30.25%)	21.35	70.59	08.90	29.41
Both (n=3666)	1189 (32.43%)	23.21	71.57	09.22	28.43
χ^2 -test ($\alpha=0.05$)	0.126				

4.2.8.2 Infection rate

Analysis of variance revealed a significant ($p=0.0001$) statistical effect of the feeding methods on the infection rate irrespective of the bacterial type (Table 39). The LS-mean infection rate was higher in the farms use both methods of feeding (1.30). That was followed by the farms use mobile method of feeding (1.29). However, the farms use stationary method of feeding was found to have a lower LS-mean infection rate (1.27). And as previously stated with the other management factors the LS-means infection rate due to the effect of the contagious microorganisms was higher. It was declared that mobility of the

feed and the contagious bacterial infection was resulted in a higher LS-mean infection rate (1.51). But not very high than that of the animals fed with both methods (1.50). The LS-mean infection rate due to the effect of the environmental pathogens was not different in the farms use mobile and stationary feeding method. And higher (1.43) in farms use both methods as was indicated in table 40. And the differences among the factors were being significant ($p=0.0007$).

Table 39: LS-means and S.E. of infection rate according to feeding method and irrespective of bacterial status ($p=0.0001$)

Feeding method	Infection rate
Mobile	1.29±0.01
Stationary	1.27±0.01
Both	1.30±0.01

Table 40: LS-means and S.E. of infection rate according to feeding method and bacterial status ($p=0.0007$)

Bacterial status	Feeding methods		
	Mobile	Stationary	Both
Contagious pathogens	1.51±0.01	1.49±0.01	1.50±0.01
Environmental pathogens	1.39±0.01	1.39±0.01	1.43±0.02

4.2.8.3 Logarithmic SCC

As was demonstrated in table 41 it could be noticed that independent on the bacterial status the methods of feeding was highly significantly ($p=0.0001$) influenced the SCC. The LS-mean SCC was significantly higher (5.98) when the methods of feeding were both mobile and stationary. However, with the use of the mobile method the value of the LS-mean SCC was lower than in the former method (4.83). But higher than that scored with the use of stationary method (4.57). The result also revealed that the methods of feeding used in the farms was poor in reducing the effect of the bacteria. As the LS-mean SCC was found to be higher in animals infected with either contagious or environmental bacteria and fed with a mobile, stationary or both methods. The difference was tested to be not significant ($p>0.05$). The use of both feeding methods was claimed to be extra-ordinary in elevating the level of SCC irrespective of the pathogens (Figure 20).

Table 41: LS-means and S.E. of SCC (log) with respect to feeding method and independent on bacterial status ($p=0.0001$)

Feeding method	SCC (log)
Mobile	4.83±0.02
Stationary	4.57±0.02
Both method	5.98±0.03

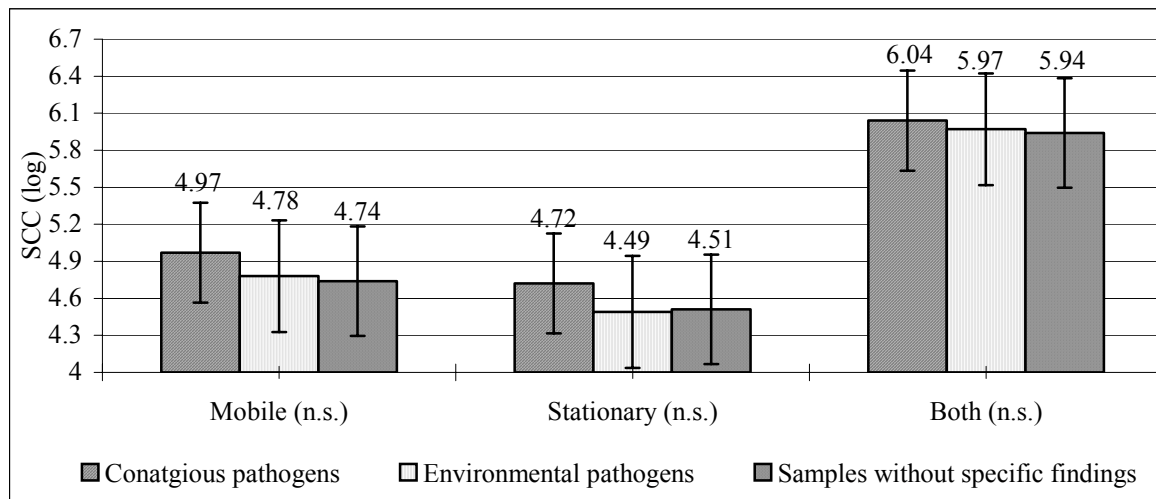


Figure 20: LS-means and S.E. of SCC(log) according to method of feeding and bacterial status ($p>0.05$)

4.2.8.4 Test-day milk yield

Feeding methods were significantly ($p=0.0001$) influenced the daily milk yield (Table 42). The LS-mean milk yield/day was 25.94 kg in farms intended to use stationary method. Farms use both method had 1.43 kg less milk yield/day than the former ones. While farms use mobile method scored the lower LS-mean milk yield/day (21.31 kg). The effect of the feeding methods and the class of the logarithmic SCC was found to be significant ($p<0.05$). The trend was not differing from the other factors as the milk yield decreases with the increase of the SCC. With the middle level of SCC the LS-mean milk yield/day was higher (25.23 kg) in the farms use both methods of feeding than those that use mobile or stationary method (24.90 and 24.19 kg, respectively). However, at a high level of SCC the LS-mean milk yield/day was statistically not differing (Table 43).

Table 42: LS-means and S.E. of test-day milk yield (kg) according to feeding method and irrespective of SCC (log) (p=0.0001)

Feeding method	Test-day milk yield (kg)
Mobile	21.31±0.10
Stationary	25.94±0.09
Both	24.51±0.20

Table 43: LS-means and S.E. of test-day milk yield (kg) according to feeding method and SCC (log) (p=0.0065)

Class of logarithmic SCC	Feeding method		
	Mobile	Stationary	Both
<3.22	28.12±0.38	28.38±0.31	-
3.22-4.47	24.90±0.13	25.19±0.15	24.23±0.43
4.48-5.73	23.08±0.16	23.38±0.21	23.00±0.27

4.2.9 Type of udder cleaning before milking

Before milking the udder of a lactating cow was subjected to a thorough cleaning using towels, disposable papers or tissue. These types of cleaners could be dry or moist by means of water or diluted disinfections. The study classified the means of the udder cleaning according to the state of their use into: moist udder cleaning and dry udder cleaning.

4.2.9.1 Frequency of contagious and environmental pathogens

Table 44 shows the frequency of the contagious and the environmental pathogens discovered in the udder quarter's and udder's samples. It can be clearly viewed that the frequency of the contagious bacteria was higher in the samples where the moist udder cleaning were used (73.57%). Compared to those which used the dry udder cleaning (69.42%). And the opposite is correct for the environmental pathogens. Still the frequency of the positive samples was higher with the use of the moist udder cleaning (32.05%) than with the use of the dry udder cleaning (28.46%). The Chi-square test revealed a significant difference (p=0.001) between the two types.

Table 44: Frequency distribution of contagious and environmental pathogens according to type of udder cleaning

Type of udder cleaning	Number of positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Moist (n=28614)	9170 (32.05%)	23.58	73.57	08.47	26.43
Dry (n=26873)	7648 (28.46%)	19.76	69.42	08.70	30.58
χ^2 -test ($\alpha=0.05$)	0.001				

4.2.9.2 Infection rate

The study was attaining a high significant ($p=0.0001$) effect of the udder cleaning types on the infection rate (Table 45). Farms, which were intending to use moist udder cleaning had a higher LS-mean infection rate (1.30) than those that were practicing dry udder cleaning (1.29). Meanwhile the type of the udder cleaning before milking and the bacterial status interaction exerted a high significant effect ($p=0.0001$) on the infection rate (Table 46). The infection rate was higher (1.54) due to the effect of the contagious pathogens in case of using moist cleaning. While the dry cleaning reduced the infection rate with the contagious bacteria to 1.49. Nevertheless, the case was different with the environmental bacteria, since the infection rate was higher where the dry cleaning was used (1.44) than where the moist cleaning was used (1.40).

Table 45: LS-means and S.E. of infection rate according to type of udder cleaning and irrespective of bacterial status ($p=0.0001$)

Type of udder	Infection rate
Moist	1.30 \pm 0.01
Dry	1.29 \pm 0.01

Table 46: LS-means and S.E. of infection rate according to type of udder cleaning and bacterial status ($p=0.0001$)

Bacterial status	Type of udder cleaning	
	Moist	Dry
Contagious bacteria	1.54 \pm 0.01	1.49 \pm 0.01
Environmental bacteria	1.40 \pm 0.01	1.44 \pm 0.01

4.2.9.3 Logarithmic SCC

A high SCC was found in farms whose means of udder cleaning was moist (5.10) which was significantly ($p=0.0001$). Different from the level of the SCC in farms use dry means of udder cleaning (4.80) (Table 47). And dependent on the bacterial status the type of the udder cleaning was found to affect the SCC significantly ($p=0.0001$). A high SCC was revealed due to the infection with contagious as well as environmental pathogens in animals their udders were cleaned by moist means of udder cleaning. Dry udder cleaning let to a high SCC in case of contagious pathogens infection (5.01). That was higher than the infection with the environmental pathogens (4.72). SCC was found to be higher in animals in which no specific pathogen was isolated but their udders were cleaned by means of moist udder cleaning (5.01) compared to 4.66 LS-mean SCC in farms where dry udder cleaning were used (Figure 21).

Table 47: LS-means and S.E. of SCC (log) with respect to type of udder cleaning and independent on bacterial status ($p=0.0001$)

Type of udder cleaning	SCC (log)
Moist	5.10 \pm 0.02
Dry	4.80 \pm 0.02

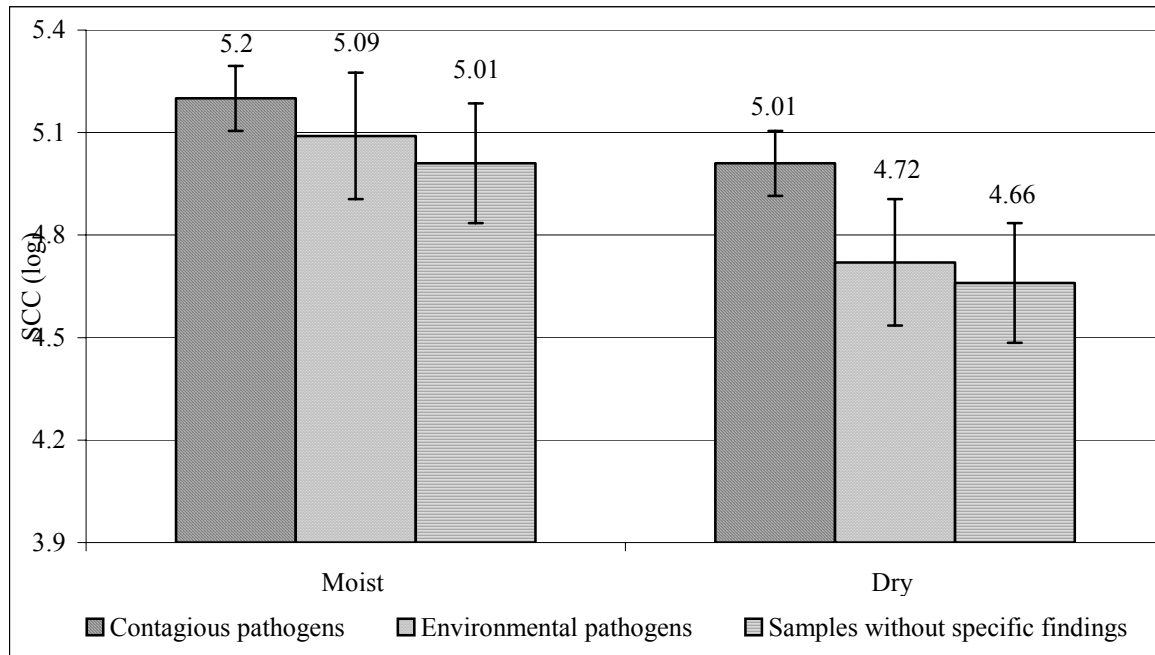


Figure 21: LS-means and S.E. of SCC (log) according to type of udder cleaning and bacterial status ($p=0.0001$)

4.2.9.4 Test-day milk yield

In table 48 it can be seen that the mean milk yield/day was significantly ($p=0.0001$) higher in the farms which use moist means of udders cleaning (24.87). Than those use a dry mean of udder cleaning (22.67kg). Likewise the types of the udder cleaning and the class of the SCC interaction were highly significantly ($p=0.0001$) affecting the daily milk yield (Table 49). When the SCC level was low then the animals whose udders were cleaned by means of moist cleaners produced a pronounce high milk/day (30.34 kg). Than their counterparts which were cleaned by means of dry udder cleaning (26.86 kg). In the case of the udder cleaning types the general trend was not violated because the daily milk yield decreases when the level of the SCC increases. And in all classes of SCC the performance of the animals that their udders were cleaned by means of moist cleaning were best than those their udders were cleaned by means of dry cleaning.

Table 48: LS-means and S.E. of test-day milk yield (kg) according to type of udder cleaning and irrespective of SCC (log) ($p=0.0001$)

Type of udder cleaning	Test-day milk yield (kg)
Moist	24.87±0.09
Dry	22.67±0.07

Table 49: LS-means and S.E. of test-day milk yield (kg) according to type of udder cleaning and SCC (log) ($p=0.0001$)

Class of logarithmic SCC	Type of udder cleaning	
	Moist	Dry
<3.22	30.34±0.36	26.86±0.30
3.22-4.47	26.52±0.12	23.91±0.11
4.48-5.73	23.74±0.11	21.97±0.11
>5.73	21.61±0.17	21.30±0.18

4.2.10 Inter-milking sanitization method of milking units

The study investigated the effects of the sanitization methods between milking on the mammary gland infection. The methods were classified into six types namely backflushing, air wash, bath (Tub), spraying and when two or more of these methods combined together

the type was designated as other. Whereas the 6th class was the non-use of the inter-milking sanitization.

4.2.10.1 Frequency of contagious and environmental pathogens

Table 50 shows the distribution of the positive samples and consequently the bacterial groups among the farms which, use different methods of inter-milking sanitization. The difference was found to be significant ($p=0.001$). A higher percentage of the positive samples was found in animals whose milking units were sanitized by the use of air wash (43.30%). Followed by backflushing (33.83%), bath (30.73%) and the other methods (28.43 %). However, a lower findings were discovered when spraying was used (21.81%). Whereas when no sanitizer was used the percentage of the positive findings was the highest (49.09%). The general pattern of the frequencies of the pathogens groups were that the contagious pathogens consistently higher than the environmental pathogens (Table 50). The contagious pathogens were pronounced when backflushing was used (80.28%) whereas the use of combining sanitizers reduced the contagious to 76.93%. On the other hand air wash was effective in reducing the effect of the contagious pathogens (62.14%). However, the frequency of the environmental pathogens was lower where the backflushing was used (19.72%) followed by the use of combination of sanitizer (23.07%).

Table 50: Frequency distribution of contagious and environmental pathogens according to inter-milking sanitization method

Sanitization method	Number of positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Backflusing (n=11584)	3919 (33.83%)	27.16	80.28	06.67	19.72
Air wash (n=3044)	1318 (43.30%)	26.91	62.14	16.39	37.86
Bath (n=11660)	3583 (30.73%)	20.68	67.29	10.11	32.91
Spraying (n=19004)	5059 (26.62%)	17.81	66.91	08.81	33.09
Other (n=9377)	2666 (28.43%)	21.87	76.93	06.56	23.07
Not used (n=1157)	568 (49.09%)	34.57	70.42	14.52	29.58
χ^2 -test ($\alpha=0.05$)	0.001				

4.2.10.2 Infection rate

Table 51 displays the effect of the inter-milking sanitization methods on the infection rate. The LS-mean infection rate was higher in farms ignore to use the inter-milking sanitization

of the milking unit (1.34). Similar frequency was encountered in farms that use backflushing sanitizer (1.33). The use of combining methods reduced the infection rate to 1.26 which was significantly different from the former means of sanitization. The use of air wash and spraying were resulted in an infection rate that was not differing (1.28). Whereas bathing the milking unit was lead to a middle LS-mean infection rate (1.27). Table 52 presents that an infection with contagious and environmental pathogens was encountered in animals that their milking units were sanitized. And was found to exert a high significant effect ($p=0.0001$). The contagious pathogens induced a high infection rate in animals kept in farms that use backflushing, air wash and bath sanitizers (1.58, 1.56 and 1.53, respectively). However the infection rate was lower with the use of spraying and other combinations (1.47 and 1.44 respectively). Generally the infection rate was relatively higher when the inter-milking sanitization was not used (1.61). On comparing with the environmental pathogens infections it was clearly indicated that the inter-milking sanitization affect the infection rate. The extent was that the plateau was lower than the infection with the contagious bacteria (table 52). A high LS-mean infection rate was exerted with the environmental bacteria found in the milking unit sanitized by spraying (1.44).

Table 51: LS-means and S.E. of infection rate according to inter-milking sanitization method and irrespective of bacterial status ($p=0.0001$)

Inter-milking sanitization method	Infection rate
Backflushing	1.33±0.01
Air wash	1.28±0.01
Bath (Tub)	1.27±0.01
Spraying	1.28±0.01
Other	1.26±0.01
Not used	1.34±0.01

Table 52: LS-means and S.E. of infection rate according to inter-milking sanitization method and bacterial status (p=0.0001)

Inter-milking sanitization	Contagious pathogens	Environmental pathogens
Backflushing	1.58±0.01	1.41±0.01
Air wash	1.56±0.05	-
Bath (Tub)	1.53±0.01	1.35±0.01
Spraying	1.47±0.01	1.44±0.01
Other	1.44±0.01	1.40±0.02
Not used	1.61±0.01	1.41±0.02

4.2.10.3 Logarithmic SCC

Sanitization of the milking units was found to affect the SCC (p=0.0001). The LS-mean SCC was the over-all higher (5.11) in farms which ignore the use of the inter-milking sanitization of the milking units. This was significantly different than that in farms used spraying (4.21). The use of the other methods of sanitizations were ended-up with different LS-means SCC that were ranged between 4.77-4.98 (Table 53). Consistently the inter-milking sanitization methods bacterial status interaction were significantly influenced the SCC (p=0.0001). Farms ignored the use of the inter-milking sanitizer their animals had a high level of SCC. The development of SCC with the other methods is that the LS-mean SCC was higher when any of the sanitization methods was used. The farms were found to be threatened by the contagious bacteria compared to the environmental bacteria (Table 54). Samples that were confirmed to be without specific findings scored different levels of the SCC. It was a little bit higher in farms practiced no inter-milking sanitization (5.04) than in farms which use backflushing (4.91) followed by those that use spraying (4.87).

Table 53: LS-means and S.E. of SCC (log) with respect to inter-milking sanitization method and independent on bacterial status (p=0.0001)

Inter-milking sanitization method	SCC (log)
Backflushing	4.98±0.02
Air wash	4.83±0.02
Bath (Tub)	4.77±0.02
Spraying	4.21±0.12
Other	4.96±0.02
Not used	5.11±0.02

Table 54: LS-means and S.E. of SCC (log) with respect to inter-milking sanitization methods and bacterial status (p=0.0001)

Inter-milking sanitization	Contagious pathogens	Environmental pathogens	Samples without specific findings
Backflushing	5.04±0.05	4.92±0.06	4.91±0.03
Air wash	4.35±0.13	4.27±0.12	4.01±0.32
Bath (Tub)	4.88±0.03	4.79±0.05	4.65±0.02
Spraying	5.08±0.03	4.99±0.04	4.87±0.02
Other	5.14±0.03	4.68±0.05	4.66±0.02
Not used	5.22±0.03	5.07±0.02	5.04±0.06

4.2.10.4 Test-day milk yield

High significant variations (0.0001) in the daily milk yield were obtained due to the effect of inter-milking sanitization (Table 55). The LS-mean milk yield/day was higher in farms use backflushing (25.55 kg) versus those use the other methods (22.50 kg). The difference was significant. In the other farms the mean daily milk yield was statistically the same. Figure 22 reveals the effect of the sanitization methods of the milking units on the daily milk yield in different classes of SCC. It was found to be highly significant (p=0.0001). In this aspect the result was concluded that irrespective of the sanitization methods the daily milk yield decreased as the level of SCC increased. However with the use of air wash the mean daily milk yield was higher (31.15 kg) when the level of the logarithmic SCC was lower (<3.22). Among the other sanitization methods Backflushing seem to have no clear effect on the factors that having constrained the milk yield. As the yield with the middle classes of SCC was not greatly differing. Spraying showed a clear and distinct effect on milk yield independent on the level of SCC. Bathing of the milking units and the combination of more sanitization methods were resulted in improved mean daily yield depending on the class of the SCC. Because the yield was relatively high with the low and the middle classes of SCC.

Table 55: LS-means and S.E. of test-day milk yield (kg) according to inter-milking sanitization method and irrespective of SCC (log) (p=0.0001)

Inter-milking sanitization method	Test-day milk yield (kg)
Backflushing	25.55±0.14
Air wash	23.97±0.77
Bath (Tub)	23.09±0.13
Spraying	23.41±0.11
Other	22.52±0.11
Not used	23.06±0.21

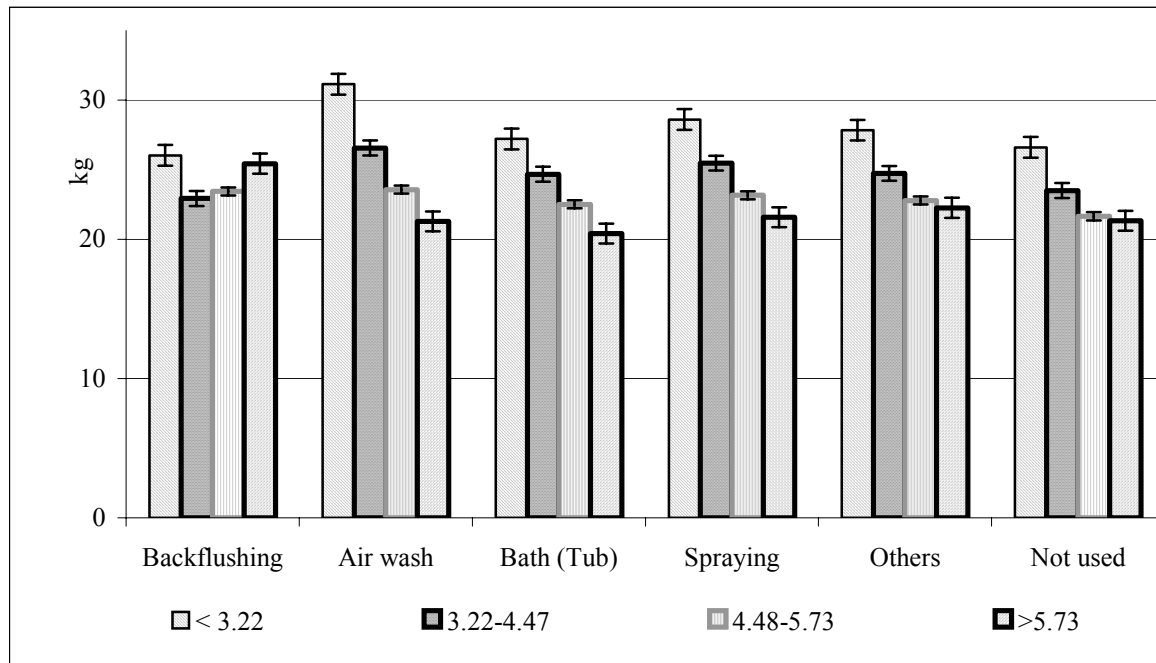


Figure 22: LS-means and S.E. of test-day milk yield (kg) with respect to inter-milking sanitization method and SCC (log) (p=0.0001)

4.2.11 Teat disinfection

Teat disinfection as a method to reduce the effect of the mastitis causing pathogens are normally practiced in many dairy farms. In this study teat dipping was tested against no teat dipping. And various results of the factors determining the extent of IMI were obtained.

4.2.11.1 Frequency of contagious and environmental pathogens

The frequency of the positive samples discovered in animals whose teats were not dipped was higher (35.98%). From which the frequency of the contagious bacteria was 80.08%

and that of the environmental bacteria was 19.92%. Whereas the samples taken from the teat-dipped quarters scored 29.38% as positive samples distributed as 68.80% contagious pathogens and 31.20% environmental pathogens (Table 56).

Table 56: Frequency distribution of contagious and environmental pathogens according to type of teat disinfection

Teat disinfection	Number of positive samples	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Teat dipping (n=45038)	13232 (29.38%)	20.21	68.80	09.17	31.20
Not used(n=10788)	3881 (35.98%)	28.81	80.08	07.17	19.92
χ^2 -test ($\alpha=0.05$)	0.001				

4.2.11.2 Infection rate

Teat disinfection was indicated to influence the state of the infection ($p=0.0001$). Teat dipping reduced the LS-mean infection rate to 1.29 vs. 1.32 when no teat dipping was employed (Table 57). Depending on the bacterial status (Table 58) the infection rate was significantly affected ($p=0.0001$) to the point that the contagious pathogens had a wide spread infection in animals whose teats were not dipped (1.60). Whereas the teat dipping was resulted in a lower infection rate (1.49). The LS-mean infection rate due to the environmental bacteria was proved higher in animals whose teats were dipped (1.42) than was in the other group (1.37).

Table 57: LS-means and S.E. of infection rate according to type of teat disinfection and irrespective of bacterial status ($p=0.0001$)

Teat disinfections	Infection rate
Teat dipping	1.29±0.01
Not used	1.32±0.01

Table 58: LS-means and S.E. of infection rate according to type of teat disinfection and bacterial status and ($p=0.0001$)

Bacterial status	Teat disinfection	
	Teat dipping	Not used
Contagious bacteria	1.49±0.01	1.60±0.01
Environmental bacteria	1.42±0.01	1.37±0.01

4.2.11.3 Logarithmic SCC

Table 59 exhibits that the LS-mean SCC was significantly ($p=0.0001$) higher (5.08) in farms which did not practice teat dipping. Compared to those that use teat dipping (4.88). The result also indicated that teat disinfection depending on the bacterial status was significantly affected the SCC (Table 60). The LS-mean SCC was tended to increase when no teat dipping was used and it was pronounced when infection with contagious bacteria was encountered (5.19). However, the SCC went downwards where teat dipping was used (5.06). In addition, in the existence of the environmental pathogens effect, the teat dipping lowered the SCC (4.83) compared to the non-use of the teat dipping (5.12). The difference between the two groups was found to be highly significant ($p=0.0001$).

Table 59: LS-means and S.E. of SCC (log) with respect to type of teat disinfection and independent on bacterial status ($p=0.0001$)

Teat disinfection	SCC (log)
Teat dipping	4.88±0.01
Not used	5.08±0.02

Table 60: LS-means and S.E. of SCC (log) with respect to and type of teat disinfection and bacterial status ($p=0.0001$)

Bacterial status	Teat disinfection	
	Teat dipping	Not used
Contagious bacteria	5.06±0.02	5.19±0.03
Environmental bacteria	4.83±0.03	5.12±0.02
Samples without specific findings	4.75±0.01	5.00±0.07

4.2.11.4 Test-day milk yield

Table 61 reveals that farms practiced teat dipping had a significantly ($p=0.0001$) higher mean milk yield/day (25.04 kg) than for farms adopt no teat dipping (23.18 kg). Table 62 shows the effect of teat disinfection depending on the class of SCC on the mean daily milk yield. Which was being highly significant ($p=0.0001$). With all classes of SCC the mean daily milk yield was found to be higher where teat dipping was employed than where no teat dipping was used. In the lower level of SCC (<3.22) and with the use of the teat dipping the mean daily milk yield was higher (27.23 kg). The higher mean milk yield (24.42 kg) which was produced when no teat dipping was practiced and the level of SCC was lower was not

differing from that produced with middle class of SCC and practicing teat dipping (24.51 kg). The most lower mean daily milk yield (19.50 kg) was noticed when no teat dipping was used and SCC (log) was higher (>5.73).

Table 61: LS-means and S.E. of test-day milk yield (kg) according to type of teat disinfection and irrespective of SCC (log) ($p=0.0001$)

Type of teat disinfection	Test-day milk yield
Teat dipping	25.04 \pm 0.14
Not used	23.18 \pm 0.06

Table 62: LS-means and S.E. of test-day milk yield (kg) according to type of teat disinfection and SCC (log) ($p=0.0001$)

Class of logarithmic SCC	Teat disinfections	
	Teat dipping	Not used
<3.22	27.23 \pm 0.30	24.42 \pm 0.93
3.22-4.47	24.51 \pm 0.11	22.76 \pm 0.30
4.48-5.73	22.56 \pm 0.10	19.50 \pm 0.32
>5.73	21.56 \pm 0.16	17.34 \pm 0.49

4.3 Factors affecting heifers IMI

Heifers are the initiative of the future-breeding herd. And to reach the maximum profitable production, heifer should be well bred and managed. The study investigated the effect of the time of sampling and positive findings a.p. and p.p. on the first lactation SCC and test-day milk yield.

4.3.1 Effects of positive findings, time of sampling and bacterial groups on first stage of lactation SCC

Table 63 reveals that bacterial findings significantly ($p<0.05$) affect the SCC. Positive findings raised the mean SCC compared to the sample free from specific findings. It was also observed that the mean SCC was significantly ($p<0.05$) differing with the time of sampling (i.e. 10-40 days a.p. and p.p.). Table 64 shows that the mean SCC was higher for heifers sampled 20 days a.p. (4.91) than those that were sampled 20 days p.p. (4.47). Whereas the mean SCC was consistently higher in heifers that were sampled 40 days p.p. (4.93) compared to those that were sampled 40 days a.p. (4.40). On the other hand, SCC increased gradually as the sampling time increased from the point of the delivery (Table

64). The effect of the bacterial groups on the SCC was insignificant ($p>0.05$). Although the mean SCC due to effect of the bacterial group a.p. was higher than that detected p.p. (Table 65). However samples without specific findings were significantly lower than the infected ones and it was higher p.p. (4.35) than was a.p. (4.30).

Table 63: Effect of bacterial findings (a.p. and p.p.) on mean SCC (log)*

Time of sampling	Positive findings	Negative findings
40d before calving	4.53 ^a	4.08 ^b
40d after calving	4.46 ^a	4.25 ^b

* Means with different superscript letters are significantly different ($p<0.05$).

Table 64: Effect of time of sampling on mean SCC (log)* and test-day milk yield (kg)*

Time of sampling	SCC (log)	Test-day milk yield (kg)
10 d a.p.	4.59 ^{ab}	24.12 ^b
20 d a.p.	4.91 ^a	24.40 ^b
30 d a.p.	4.58 ^{ab}	24.81 ^{ab}
40 d a.p.	4.40 ^b	25.85 ^a
10 d p.p.	4.46 ^b	24.47 ^a
20 d p.p.	4.47 ^b	24.13 ^a
30 d p.p.	4.78 ^{ab}	23.94 ^a
40 d p.p.	4.93 ^a	23.84 ^a

* Means with different superscript letters are significantly different ($p<0.05$).

Table 65: Effect of bacterial groups on SCC (log)*

Bacterial group	Mean SCC (log)	
	a.p.	p.p.
Contagious pathogens	4.61 ^a	4.48 ^a
Environmental pathogens	4.58 ^a	4.48 ^a
Samples without specific findings	4.30 ^b	4.35 ^b

* Means with the same superscript letters are not significantly different ($p>0.05$).

4.3.2 Effect of time of sampling on first stage of lactation test-day milk yield

As was shown in table 64 the time of sampling a.p. significantly ($p < 0.5$) affect the daily milk yield and consequently the lactation milk yield. The result reveals that as the infection was occurred in a later time a.p. the daily milk yield would not be affected. However, infection that was occurred near to the point of delivery affected the daily milk yield and a loss of 1.73 kg was observed. Infection which was occurred p.p. had no significant effect on the test-day milk yield although a loss of milk yield of 0.63 kg between the yield correspond to the sampling 10 days p.p. and those that were collected 40 days p.p.

4.4 Risk factors associated with IMI

Table 66 shows that of the risk factors investigated only the stage of lactation and the housing system had no significant effect on the of entrance of IMI. The results revealed that herd size with less than 200 cows had 1.41 times more risk of encountering IMI than those with more than 200 cows. And the summer-calving cows were 1.01 times more subjected to the infection than the winter-calving cows. However, there was no significant difference in the risk of entrance of IMI between the early and the late stages of lactation. The study also showed that the contagious pathogens were highly significantly different ($p = 0.0001$) in the chance of causing IMI than the environmental pathogens ($OR = 1.61$). And the purchased heifers were highly different ($p = 0.0001$) in subjection to IMI than the farm bred heifers ($OR = 1.47$). On investigating the effect of the housing system on the IMI it was found that tie-barns had insignificantly 1.07 more chance of predisposition to IMI than loose housing systems. In the time that pipe-system was 1.06 more risky in predisposing animals to IMI than carrousel and milking parlors, the difference was significant ($p = 0.02$). Moist cleaning of the udder before milking had fairly significantly 1.6 times the chance of encouraging IMI than the dry udder cleaning. The non-use of inter-milking sanitization of the milking units was three times more risky in aggravating the udder health conditions than its use. And the disregarded usage of teat dipping was found to raise the risk of IMI to 2.08 times compared to the routine usage of post-milking teat dipping the difference was highly significant ($p = 0.0001$). The chance of elevation of SCC (log) above 5.73 in response to IMI was 1.5 times compared to the level below 5.73.

Table 66: Factors associated with IMI, degree of freedom (D.F.), regression coefficients (b), S.E., χ^2 -test, Odds ratios (P) and 95% confidence limits (Ψ)

Factor	D.F.	b	S.E.	χ^2 -test ($\alpha < 0.05$)	P	Ψ
Herd size > 200 cows	-					
Herd size < 200 cows	1	0.101	0.010	0.0001	1.405	0.886-1.923
Winter	-					
Summer	1	0.039	0.011	0.0002	1.013	0.943-1.082
Late stage of lactation	-					
Early stage of lactation	1	0.014	0.013	0.2882	1.014	0.988-1.041
Environmental pathogens	-					
Contagious pathogens	1	0.476	0.010	0.0001	1.610	1.578-1.642
Farm bred heifers	-					
Purchased heifers	1	0.387	0.032	0.0001	1.472	1.383-1.567
Loose housing system	-					
Tie-system	1	0.028	0.024	0.2282	1.073	0.928-1.218
Carrousel and milking parlor	-					
Pipe system	1	0.061	0.025	0.0164	1.063	1.011-1.117
Dry udder cleaning	-					
Moist udder cleaning	1	0.469	0.024	0.0001	1.598	1.525-1.676
Inter-milking sanitization (used)	-					
Inter-milking sanitization (not used)	1	1.095	0.056	0.0001	2.988	2.678-3.334
Teat dipping (used)	-					
Teat dipping (not used)	1	0.732	0.057	0.0001	2.079	1.861-2.323
SCC (log) < 5.73	-					
SCC (log) > 5.73	1	0.403	0.044	0.0001	1.495	1.371-1.632

4.5 Risk factors associated with high SCC

Table 67 shows that herd size, lactation number, pathogens group, udder cleaning methods before milking, inter-milking sanitization method and teat disinfection were the factors that showed a significant effect on the risk of levels of SCC. A herd size with less than 200

cows raised the risk of a high level of SCC with 1.001 times than a herd size with more than 200 cows. In the time that cows in their fourth lactation or more had 2.32 times risk of having a high threshold of SCC compared to cows in their third lactation or less. However, summer-calving cows revealed 1.01 more risk of having a higher SCC than winter-calving cows and animals in the early stages of lactation had 1.03 risk of having high SCC when compared to animals in the late stages of lactation. However, the difference was not significant. The contagious pathogens revealed 1.15 times risk of elevating the level of SCC than environmental pathogens. Whereas the purchased heifers were significantly raised the risk of elevating the SCC level compared to the farm bred heifers (OR=1.06, $p>0.05$). Animals housed in tie-barns showed to be at 1.02 times risk of getting a high level of SCC than animals housed in loose-system. Also cows their udders cleaned with a moist means of cleaning were in a high risk of having a high level of SCC (OR=1.23, $P=0.05$) than cows their udders were cleaned with dry means of udder cleaning. Ignored inter-milking sanitization of the milking units raised the chance of elevated the threshold of SCC with 1.29 times ($p=0.003$) this risk could be decreased if inter-milking sanitization of the milking units should have been used. And the disuse of post-teat dipping could have 2.35 times risk of predisposing to a high level of SCC compared to the trial of using teat dipping, the difference was highly significant.

Table 67: Factors affecting SCC (log), degree of freedom (D.F.), regression coefficients (b), S.E., χ^2 -test, Odds ratios (P) and 95% confidence limits (Ψ).

Factor	D.F.	b	S.E.	χ^2 -test ($\alpha < 0.05$)	P	Ψ
Herd size > 200 cows	-					
Herd size < 200 cows	1	-0.060	0.020	0.0032	1.001	0.904-1.098
Lac. ≤ 3	-					
Lac. ≥ 4	1	0.842	0.054	0.0001	2.322	2.089-2.581
Winter	-					
Summer	1	0.009	0.020	0.6507	1.009	0.970-1.050
Late stage of lactation	-					
Early stage of lactation	1	0.033	0.026	0.2099	1.033	0.982-1.088
Environmental pathogens	-					
Contagious pathogens	1	0.139	0.022	0.0001	1.149	1.101-1.198
Farm bred heifers	-					
Purchased heifers	1	0.060	0.066	0.3656	1.062	0.933-1.209
Loose housing system	-					
Tie-system	1	0.021	0.050	0.6711	1.021	0.927-1.125
Dry udder cleaning	-					
Moist udder cleaning	1	0.205	0.047	0.0001	1.227	1.118-1.346
Inter-milking sanitization (used)	-					
Inter-milking sanitization (not used)	1	0.256	0.087	0.0031	1.292	1.090-1.531
Teat dipping (used)	-					
Teat dipping (not used)	1	0.855	0.113	0.0001	2.352	1.883-2.937

5 DISCUSSION

In dairy production system, efforts should be directed towards the factors that mostly affect the mammary health status and milk quantity and quality of a producing cow. Mammary health status and milk production efficiency are influenced by several factors that determine whether the individual should remain within the producing herd and subjected to intensive treatment or leave it.

Factors influencing determinants of IMI

Several factors were found to be affecting the IMI, the frequency of the causing pathogen and the infection rate, which would be reflected in an increase SCC whereas high milk yield is claimed to be a predisposing factor to IMI. These factors on the other hand, lie under the influence of intrinsic factors within the animal and extrinsic factors as a result of exposure to the environmental factors in which the animal lives. The present study investigated the effects of the herd size, year-season, lactation number, stage of lactation, farm managements and hygienic factors.

Frequency of pathogens and infection rate

The present study was based on 64542 randomly collected fore milk samples. 56960 were udder quarter's samples, among which 22.87% were positive samples in FQR, 26.23% in FQL, 24.95% in HQR and 25.95% in HQL. while the frequency of positive samples at the cow level were 49.66% of 7582 of the total samples collected from the whole udder as mixed strips from each quarter. The result indicated that 77.22% of the animals were infected, contrary to the result of Trinidad et al. (1990) who found 95% of the animals studied had IMI. The most frequently isolated pathogens from both udder quarter's samples and udder's samples were *S. aureus* and CNS. This result is in line with Trinidad et al. (1990), Nickerson et al. (1995) and Waage et al. (1999). The frequencies of *E. coli* in the udder quarter's samples and udder's samples were not greatly differing (3.6 and 4.5%, respectively) which was nearly the same frequency as obtained by Waage et al. (1999) and lower than the results which were published by Lam et al. (1996) and Lipman et al. (1994). The frequency of *C. bovis* isolates was lowest in the udder quarter's samples and udder's samples (1.1 and 0.6%, respectively). It was found that 72.79% of the pathogens in the udder quarters were contagious and 27.21% were environmental pathogens. Which is consistent with the conclusion of Chrystal et al. (1999) who stated that nearly all cases of IMI occur as a result of the contagious microorganisms passing through the teat canal. And

as the environmental pathogens are those present in the environment of the animal and could easily be controlled by improving the management practices, the management of the studied farms will be following the management procedure hypothesized that less problem is expected.

Herd size

Farms studied are fairly of large herd size and to test their effect they were classified into five classes according to the number of the lactating cows they owned. The study revealed that the small and large herds scored a higher frequency of the IMI causing pathogens compared to the medium and medium large herds. Whereas the medium small herd size had a lower frequency of the pathogens. However, contagious pathogens decreased gradually from the small to the medium large herd and then increased slightly in the large herd size. This may indicate that medium and medium large size farms were subjected to some modernization in husbandry systems and milking systems and changes of management practices resulted in reducing the frequency of pathogens, specifically the contagious pathogens. The opposite was the trend of the environmental pathogens. Although the frequency of the positive findings was higher in large herds, but still the mean infection rate was lower (Table 68).

Table 68: LS-means and S.E. of infection rate with respect to the herd size

Herd size	Infection rate
Small (< 200)	1.31±0.01
Medium small (200-400)	1.28±0.01
Medium (401-600)	1.27±0.01
Medium large (601-800)	1.25±0.01
Large (> 800)	1.24±0.01

In the small farms, infection rate was highest, and decreased gradually to reach the lowest value in the large farms. The results are in agreement with Wilesmith et al. (1986), they stated that the incidence of mastitis declined with increasing herd size.

Year-season

In the present study summer 99, winter 98/99 and spring 2000 were the seasons of most frequent IMI causing pathogens (32.92, 32.51 and 32.45%, respectively) whereas summer

1998 had lowest isolates (23.98%) and no significant difference was detected between autumn 1999 and winter 99/2000 (31.90 and 30.79%, respectively) (Table 69).

Table 69: Absolute and relative percentages of IMI causing pathogens among year-season.

Year-season	Positive samples (%)	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Summer 98	23.98	17.48	72.91	06.50	27.09
Autumn 98	27.50	18.77	67.09	09.05	32.91
Winter 98/99	32.51	21.20	65.20	11.31	34.80
Spring 99	29.74	20.84	70.09	08.90	29.91
Summer 99	32.92	24.06	73.09	08.86	26.91
Autumn 99	31.90	24.08	75.48	07.82	24.52
Winter 99/2000	30.79	22.74	73.87	08.05	26.13
Spring 2000	32.45	22.43	69.11	09.78	30.13

Likewise, similar frequencies were obtained in autumn 98 and spring 99 (27.50 and 29.74%, respectively). The absolute frequencies resulted were: lower frequency of contagious pathogens in summer 98 which slightly increased in autumn 98 to reach a higher level in winter 98/99, which was not maintained in spring 99. Summer and autumn of the year 99 were the seasons of the most and frequent contagious pathogens, which was consistently decreased in winter 99/2000 and spring 2000. Meanwhile, environmental pathogens showed different trends, with a higher frequency in winter 98/99 and a lower frequency in summer 98. However, the frequencies remained unchanged in spring 99 and summer 99 and were not statistically different to autumn 99, winter 99/2000 and spring 2000. The estimated pathogen frequencies caused significantly different infection rates in year-season. A higher mean infection rate was discovered in autumn 99 (1.31) than in autumn 98 (1.27). However, the lower infection rate was in spring 2000 (1.18) which was lower than in spring 99 (1.27). The study also found that contagious pathogens caused a significantly pronounced infection in summer 1999 (1.51) as compared to that occurred due to the same pathogens in the other year-seasons. However, in summer 98, autumn 98, autumn 99 and winter 98/99 the infection rates with contagious pathogens were relatively higher. Which was not different from infection rate caused by the environmental pathogens in autumn 1999. The infection rate due to the contagious pathogens in the study could be

attributed to the presence of a higher frequency of *S. aureus* and *St. agalactia*. This finding is in accordance with Waage et al. (1999) who found that the proportion of *S. aureus* and *Actinomyces pyogenes* were higher and the proportion of CNS was lower in late autumn and early winter and Wilesmith et al. (1986) who stated that the incidence rate of mastitis in Great Britain has declined from 120 cases/100 cows to 40 cases /100 cows due to reduction of mastitis caused by contagious pathogens particularly *S. aureus* and *St. agalactia*. Likewise, it conforms to Bray and Shearer's (1986) who asserted that *S. aureus* is one of the organisms responsible for about 95% of IMI. Bramley and Dodd (1984) reached another similar conclusion in their description of *S. aureus* as being the most prevalent pathogen. This is in contrast to the findings of Buzalski and Pyörälä (1990) who concluded that contagious mastitis mainly caused by Staphylococci show high cell count in bulk milk, whereas environmental mastitis results in a high number of clinical cases, but the cell count in the bulk milk is usually not high.

Lactation number

With regard to the lactation number; primiparous cows were at high risk of being attacked with mastitis pathogen than multiparous cows (OR 1.30, $p=0.0001$) and the frequency of the pathogens was higher in the first lactation (32.06%) and decreased in the second lactation (27.05%). This could be revealing that some cows had left the herd and/or the entrance of pathogens free animals into the herd. In addition to the treatment of the infected cases. The frequency showed slight increase in the third lactation (27.98%), which was not different from the frequency in the lactations after the third (27.93%). But the frequency of the contagious pathogens compared to the environmental pathogens was higher. A higher threshold of contagious pathogens was showed in the first lactation, indicating that heifers are highly subjected to infection compared to older cows. And the first pregnancy might be a predisposing factor to infection as some of the contagious pathogens are opportunistic and flare-up when the defense mechanism is distressed. Some research findings were different, for Shpigel et al. (1998) observed an increase in the incidence of mastitis, as the lactation number increases till the fifth lactation then starts to decrease. Facts supporting the above observation were published by Nickerson et al. (1995) who found in Louisiana that bacterial infection were present in 97% of the heifers and 75% of the quarters, and Zadoks et al. (2001) who found that the rate of infection with *S. uberis* was lower in first and second parity cows than in older cows. Similar trends were also observed with regard to the infection rate, which was found to be higher (1.53) due to

the effect of the contagious pathogens. This may explain the previous result of the pathogen frequency, in that there was no difference in the infection rate with contagious pathogens in the second and third lactations, but after the third lactation infection rate with contagious pathogens was the lowest (1.40). On the other hand infection rate with environmental pathogens decreased as the cow gets older, following the same order of pathogens frequencies. Supporting results for that were presented by Hogan et al. (1989) who stated that the incidence of mastitis caused by environmental bacteria in the first and second lactation is greater than in that occurred in the older cows.

Stage of lactation

It is not uncustomary to investigate the udder status of the heifer before parturition and cows in their dry period. In this study the frequency of the IMI causing pathogens before calving were estimated (table 70).

Table 70: Absolute and relative percentages of IMI causing pathogens among stage of lactation

Time of sampling	Positive samples (%)	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Before calving	29.05	21.51	74.04	06.24	21.47
First stage of lactation (1-100 days)	30.90	21.77	70.45	09.13	29.55
Second stage of lactation (101-200 days)	23.34	13.30	57.00	10.04	43.00
Third stage of lactation (> 200 days)	29.70	20.71	69.74	08.99	30.26

The frequency of the contagious pathogens before calving was found to be 74.04% of the positive cases, whereas 21.47% was the frequency of the environmental pathogens. In the first stage of lactation the frequency decreased to 70.45% for contagious pathogens and increased to 29.55% for environmental pathogens. More decreasing was observed in the second stage of lactation for the contagious pathogens that was compensated by environmental pathogens. Towards the end of the lactation the contagious pathogens increased to 69.74% and the environmental pathogens decreased to 30.26%. That could be due to the exhaustion of the intrinsic immune system and/or to the fact that large number of the pathogens continuously washed by the act of milking. And that might be lowering

towards the end of lactation. The difference of the frequencies during the successive stages of lactation was statistically significant ($\alpha < 0.05$). The categorization to contagious and environmental pathogens displays different trends in that in the period a.p. and the first stage of lactation the frequency of the contagious and the environmental pathogens was higher than in the second terms of lactation. This could be ascribed to the fact that heifers are more prone to infection than younger lactating cows and the subjection of the pathogen to be washed-up during milking in the second stage, which is the period of peak production. Infection rates with either contagious or environmental pathogens were found to be statistically not differing, but generally higher in the first stage of lactation (1.47 and 1.37 for contagious and environmental pathogens, respectively). In the second and last stages of lactation infection rates remained the same. Several research findings also confirm this result; the US-National mastitis council (1997) affirmed that during the first 75 days postpartum the rate of infection is higher than it is during the remainder of lactation. Jones et al. (1998) stated that the last 7-10 days before calving or early lactation is the time of greater susceptibility to new environmental streptococci infections, and Trinidad et al. (1990) reported in a study in USA that up to 90% of heifer quarters were infected before parturition.

Farm management and hygienic factors

These are considered to be among the main risk factors, as they predispose the animal to IMI. Our study investigated the influences of the origin of the cow, housing systems, milking techniques, feeding methods, udder cleaning methods, inter-milking sanitization methods as well as post-milking teat disinfection on IMI.

On investigating the effect of the origin of the cow on the frequency of the pathogens, the statistical test revealed a significant difference. Since the frequency was higher in the purchased herds than in the farm-bred herds (37.53% vs. 29.87%, respectively). However, the frequency of the contagious pathogens was extremely higher in the purchased animals than in the farm-bred animals (24.38 vs. 21.59%, respectively). The trend was also for the environmental pathogens (13.14 vs. 08.28%, respectively). Infection rate with contagious pathogens was not different in both groups of animals (table 71).

Table 71: LS-means and S.E. of infection rate with respect to the origin of the cow and the bacterial status

Bacterial status	Farm bred	Purchased
Contagious bacteria	1.52±0.00	1.52±0.01
Environmental bacteria	1.40±0.01	1.57±0.02

Which indicates that the contagious pathogen could maintain its virulence irrespective of the breed or management procedures. However foreign animals were more prone to infection with environmental pathogens than farm bred animals, which may signify the fact that new comers could harbor and bring the pathogens. This fact was also stated by Jones and Bailey (1998) who reported that purchased heifers from another source could harbor mastitis pathogens. In addition to the general husbandry practices towards the young herd are among the factors that could contribute to the recorded variation.

Housing system, on the other hand plays a role in influencing the determinants of IMI. Generally housed animals are exposed to environmental pathogens, and in such a case the type of the bedding is the determinant factor. In the present study, higher percentage of positive samples was discovered in animals housed in loose housing with slat floor. In all types of housing systems, the contagious pathogens were fairly higher than the environmental pathogens. Nevertheless, environmental pathogens were higher in slat floor's loose housing kept animals than those kept in the plan floor's loose stalls. This could be due to high prevalence of claws disease causing pathogens and/or space dependent fact. As when the available space in the stall is not fairly enough to allow the animal to move or to stand freely, the animal will ultimately lie down with the risk of contacting with the environmental pathogens found in stalls bedding. Mean infection rate with contagious pathogens was significantly higher (1.45) in the plan floor's loose stall housed animals, followed by animals kept in slat floor loose housing (1.43) which was the same infection rate as that in the animals kept in other stall types. Infection rate with environmental pathogens was also higher in the former type of housing. But with regard to the later type of housing, it was found higher in plan floor loose housing stall's kept animals (1.38) than slat floor's loose stall's animals (1.36). Though with insignificant difference. Among the other housing types is the tie-stall barn in which animals are always under threatening of both contagious and environmental pathogens, as the stanchion limited the movement of the animals and subject the teat to injury. And in the loose barns,

again exit the problem of lying on the rubbery floor or straw bedding. Well maintained and bedded loose-stalls and well drained dry lots minimize possible contamination of the teat ends from IMI causing pathogens, if compared with animals managed in pasture. This justification is supported by Peeler et al. (2000) who found that the incidence of mastitis increase in milking cows housed in straw yard, as well as those standing in a yard after milking. And Rodenburg (1990) who showed that too small stalls subjected animals to teat injury, in free-stall barns cows are less likely to lie in dirty and it is always of adequate size.

Milking techniques were also considered as one of the factors that can affect the udder status of the cow. Pipeline milked animals had significantly ($p=0.001$) higher frequency of pathogens (35.45%) and consequently higher mean infection rate (1.34). If pipelines are not correctly and regularly cleaned and rinsed with a plenty of water this will lead to the bacterial lodgment and raise the problem of inter-pipe pathogen transmission. This could be warranted as managerial problem. Animals milked in carrousel units had higher mean infection rate with contagious pathogens (1.57) than those milked in milking parlor (1.48). Whereas mean infection rate with environmental pathogens was higher in milk parlor (1.44) than carrousel (1.36). As the better cleaning and disinfection of the later milking unit always leads to reduce the effect of the environmental pathogens. This difference could be assumed managerial in nature as the superior management of the milking units is assumed to improve the udder health status of the milking herds. On the other hand, faulty management will devastate the condition, besides sampling error that should always be taken into consideration.

Feeding methods were found to be of insignificant effect on the frequency of IMI pathogens ($p> 0.05$). Although farms using both mobile and stationary methods of feeding were found to have a higher frequency of contagious pathogens than those using only a stationary method or only a mobile method of feeding. The resulting mean infection rate with contagious pathogens in animals fed with these systems was not higher than that estimated for animals fed with mobile only. However, infection rate with environmental pathogens was higher in animals fed with both mobile and stationary feeding system. When either of these systems is considered the result was equal mean infection rate. These consequences are believed to be slightly dependant on the kind of feeding system, but to a great extend on the nature of the feeding and feeding equipments. This is in addition to how far these equipments are well cleaned after feeding in order to prevent the carry-over

of contaminants whether contagious that can be transmitted through hands and equipments or environmental which live in a suitable environment created by such faulty management processes.

Table 72: Absolute and relative percentages of IMI causing pathogens between the methods of udder cleaning

Method of udder cleaning	Positive samples (%)	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Moist	32.05	23.58	73.57	08.47	26.43
Dry	28.46	19.76	69.42	08.70	30.58

To achieve an optimum and a quality production, it is of paramount importance to clean the udder of the cow before commencing the milking process. In this study two types of udder cleaning were routinely performed, moist and dry. The use of the moist cleaning the frequency of the positive findings was found to be 32.05% vs. 28.46% for the dry cleaning (table 72). In both cases the contagious pathogens scored high frequency compared to the environmental pathogens. But it was absolutely higher with the use of moist than with dry cleaning methods. Infection rate follow a similar pattern as the mean infection rate in animals whose udders were cleaned by moist means had a significantly higher ($p=0.0001$) infection rate with contagious pathogens (1.54) than those whose udders were cleaned by dry cleaning (1.49). Mean infection rate with environmental pathogens was higher in dry udder's cleaned animals (1.49) than moist udder's cleaned animals (1.44). This variation could be attributed to the fact that moist cleaning can predispose the animal to IMI. And as the IMI causing pathogens enter the udder through the teat opening, milking wet teat increase considerably the chance of forcing bacteria into the quarter. Also when disposable towel used to dry the teat of more than a single cow, this will overwhelm the condition and allow bacteria to be transmitted between the cows.

Table 73: LS-means and S.E. of infection rate with respect to inter-milking sanitization method and bacteria status

Bacterial status	Backflushing	Air wash	Bath	Spraying	Other	Not used
Contagious	1.58±0.01	1.56±0.05	1.53±0.01	1.47±0.01	1.44±0.01	1.61±0.01
Environmental	1.41±0.01	-	1.35±0.01	1.44±0.01	1.40±0.02	1.41±0.02

The study was also dealt with the inter-milking sanitization methods of the milking units which, were used as means of reducing the causal of IMI. It was found that 49.09% of the

collected samples were positive in the farms ignore to the use the inter-milking sanitization compared to 31.30% in farms practicing inter-milking sanitization. These frequencies resulted in a higher mean infection rates of the animals milked in milking units that were not subjected to sanitization. Compared to those practicing inter-milking sanitization. These findings can disclose the importance of the sanitization as a routine management practice in order to control or to reduce IMI. On revising the investigated sanitization methods, it could be concluded that combination of one or more of these methods were seem to be effective in reducing the mean infection rate (table 73) if not then spraying was the most effective method in reducing contagious pathogens. And Bath was effective in reducing the environmental pathogens. Backflushing was bring into being to be ineffective in reducing the infection rate with either contagious or environmental pathogens, this judgment was reported by the US-National mastitis council's fact sheet (1997) as backflushing of the milking unit does not control environmental mastitis.

Table 74: Absolute and relative percentages of IMI causing pathogens between types of teat disinfection

Teat disinfection	Positive samples (%)	Contagious (%)		Environmental (%)	
		Absolute	Relative	Absolute	Relative
Teat dipping	29.38	20.21	68.80	09.21	31.20
Not used	35.98	28.81	80.08	07.17	19.92

Of the other hygienic measures that have been adopted in the inspected dairy farms are the post-milking teat disinfection. Two groups of farms were surveyed; the first group that routinely uses teat dipping and the other pay no attention to the teat dipping. It was obviously realized that teat dipping reduced the frequency of both contagious and environmental pathogens (table 74). This was end-up with a lower mean infection rate (1.49) compared to 1.60 mean infection rate due to the contagious pathogens in animals which, their managers ignore using teat dipping. However teat dipping was found to be slightly effective against environmental pathogens, this could show that teat dipping is not equally effective against all types of IMI causing pathogens. This result presentation could be compared Radostits et al. (1994) who summarized the control measures of mastitis and concluded that each of these control measures is aimed at the management of specific pathogen type. Natzke, (1981); Pankey, (1989); Boddie et al. (1993); and Malinowski, (2000) concluded that teat dipping is aimed at reducing infections mainly caused by contagious pathogens and preventing new infections and to a less extent preventing

infections might be caused by environmental pathogens. Oliver et al. (2001) demonstrated that pre-and post-milking teat disinfection with phenolic combination was significantly more effective in preventing new IMI than was post-milking teat disinfections only.

SCC

To approximate a normal distribution SCC which are an indicator of the udder health status was transformed into a logarithmic form.

Table 75: LS-means and S.E. of SCC (log) according to bacterial status

Bacterial status	SCC (log)
Contagious pathogens	4.97±0.03 ^a
Environmental pathogens	4.86±0.03 ^b
Samples without specific findings	4.76±0.02 ^c

The study found that the level of SCC was significantly ($p=0.0001$) higher due to the effect of contagious pathogens than that due to the effect of environmental pathogens (table 75). This result is in joint agreement with the result of Buzalski and Pyörälä, (1990) who stated that contagious mastitis shows a high cell count in bulk milk, whereas environmental mastitis results in a high number of clinical cases, but the cell count in the bulk milk is usually not high. Meanwhile, samples without specific findings showed the lowest mean SCC compared to those with contagious or environmental bacteria. This could demonstrate the role-played by the pathogens in elevating the level of SCC. This finding was not different from the result of Hogan et al. (1987) who found that the mean logarithmic somatic cell count was consistently lower for bacteriological negative udder quarter.

Herd size

Table 76: LS-means and S.E. of SCC (log) according to herd size

Herd size	SCC (log)
Small (< 200)	5.06±0.06
Medium small (200-400)	4.91±0.03
Medium (401-600)	4.92±0.03
Medium large (601-800)	4.73±0.05
Large (> 800)	4.58±0.03

The result showed that the herd size and the herd size, bacterial status interaction were significantly influenced the level of SCC. It was also found that the level of the SCC and independent on the bacterial status was pronounced in small herds size compared to the other herd sizes (table 76). This indicates that medium and large farm sizes were receiving more attention and changes of management procedures including housing systems, milking techniques and mastitis control programs that were resulted in reducing the effect of the pathogens and consequently lowering the level of the SCC. This finding is in accordance with Lafi et al (1994) who found that the mean value of SCC was negatively associated with herd size and CNS, *S. aureus* and *C. bovis* were the most prevalent pathogens. Norman et al. (2000) stated that herd size and SCC were negatively related; large herds had a lower SCC.

Year-season

Table 77: LS-means and S.E. of SCC (log) according to year-season

Year-season	SCC (log)
Summer 98	4.52±0.02
Autumn 98	4.61±0.02
Winter 98/99	4.66±0.02
Spring 99	4.63±0.02
Summer 99	4.63±0.02
Autumn 99	4.59±0.02
Winter 99/2000	4.63±0.02
Spring 2000	4.68±0.04

The mean SCC (log) during the study period found to be significantly varied between year-season and independent of the bacterial groups. Similar results were published by Corbett (1998) and Rodriguez et al. (2000), but disagreed with Liebe et al. (1996). Higher LS-mean logarithmic SCC was achieved in spring 2000, which was fairly higher than the mean in the same season of the year 99. Nevertheless, in summer the mean SCC was higher in the year 99 than that in the year 98. And in autumn, the year 98 SCC scored the higher mean than that in the year 99. On the other hand, relatively higher mean SCC was discovered in winter 98/99, which was slightly higher than the mean SCC of the year 99/2000, and the lowest mean was in autumn99 (table 77). Dependent on the bacterial groups, mean SCC due to the effect of contagious pathogen infection was higher in winter 99/2000 and spring

2000 (4.78). However, in winter 98/99 and summer 99, the mean SCC was not differing (4.73). The lowest mean SCC due to the effect of contagious bacteria was indicated in summer 98 (4.67), which was slightly lower than the mean in autumn 99 (4.70) and similar to the mean SCC due to the effect of the environmental pathogens in winter 98/99, which is the highest mean in this group. In summer 98 environmental pathogens caused the lowest mean SCC, however, a higher mean was discovered in spring 2000 (4.63). Samples without specific findings had the lowest SCC in all seasons except in autumn 98 (4.61), which was higher than the effect of the environmental pathogens in the same season. This seem to be reasonably consistent with the frequency pathogens discovered and infection rate with contagious and environmental pathogens. This is together with the seasonal variations due to the effect of the housing, bedding and temperature changes on the infection status. And the high level of CNS in the year 1999 and the year 2000 compared to the year 1998. While the frequency of the contagious pathogens e.g. *S. aureus* was higher during the year 1998. CNS are claimed to cause a tremendous increase in SCC as stated in the study of Laevens et al. (1997) who concluded in a study that a single isolation of CNS resulted in a statistically significant increase in SCC with a least square SCC of 3.97. But disobeys with the findings of Booth (1988) who showed that the reduction in the prevalence of the contagious pathogens resulted in a fall in the average bulk milk SCC from 573×10^3 cells/ml to 352×10^3 cells/ml. However a third researcher found no association between level of SCC and incidence rate of mastitis (Barkema et al. 1988).

Lactation number

Table 78: LS-means and S.E. of SCC (log) according to lactations number

Bacterial status	Lac. 1	Lac. 2	Lac. 3	> Lac. 3
Contagious bacteria	4.57±0.03	4.81±0.04	5.26±0.04	5.51±0.05
Environmental bacteria	4.42±0.04	4.81±0.05	4.97±0.06	5.10±0.07
Samples without specific findings	4.37±0.02	4.56±0.02	4.97±0.03	5.17±0.03

A distinctive significant effect was observed of the lactation number on SCC independent and with respect to the contagious and the environmental bacteria. The level of SCC increased with the increase in lactation number (table 78), this revealing that with the increase of the lactation number increased the number of multiparous cows, consequently increased the susceptibility to the infection, which might elevate the value of SCC. This finding is in close agreement with the findings of Kiiman and Saveli (2000) who studied

the factors affecting milk SCC, reported that milk SCC increased with increase in lactation number and that of Labohm et al. (1998) who found that number of the lactation influence the SCC in a statistically reliable extent, but attributed the rise in SCC above 100×10^3 to the infected quarter. Koldeweij et al. (1999) found a geometric mean of SCC was 63.1 in the first lactation and 107.2 in the later lactations. The plain rise in the SCC in the samples without specific findings could be due to the effect of the non specific bacteria and/or due to the physiological effect as it was found that SCC increases as the cow get older. This was pointed out by Leslie (1996) who reported that higher SCC have been found in the milk of older cows. And Jemeljanovs and Bluzmanis (2000) who revealed that SCC in the milk increased to clinically health cows during age increasing to the extent that if 90% of second lactation cows had up to 200×10^3 cells/ml, then only 63.4% of older than fourth lactation cows had such level of somatic cells count and 18.1% of these cows had more than 500×10^3 cells/ml SCC.

Stage of lactation

Table 79: LS-means and S.E. of SCC (log) according to stage of lactation

Stage of lactation	SCC (log)
Early (1-100 days)	4.85±0.02
Middle (101-200 days)	4.87±0.03
Late (> 200 days)	4.96±0.03

The study revealed a significant effect of the stage of lactation on SCC independent of bacterial status. It was found that SCC increased as the lactation advanced (table 79). This result is in agreement with Schepers et al. (1997); Carnier et al. (1997) and Rodriguez et al. (2000) who achieved that SCC increased with the advance in lactation. But different result was published by Williams et al. (1991). However, with respect to the bacterial status, the effect was insignificant. Nonetheless, the SCC showed slight increase as the lactation advanced and the rise in the SCC due to the effect of the contagious bacteria was obviously higher than that due to the effect of the environmental and non-specific bacteria. This result is comparable with Kirk et al. (1996) who indicated that sub-clinical infection with minor pathogens (primarily CNS) had no significant effect on the average SCC during early and mid lactation. And that of Rodriguez et al. (2000) who stated that milk SCS typically reaches a minimum early in lactation and then rises. Converse result is that of Schepers et al. (1997) who showed that the stage of lactation affected the SCC, since the

logarithm of SCC was high at the beginning of the lactation, dropped to a minimum between 40 and 80 days post partum and then steadily increased until the end of lactation. And that of Williams et al. (1991) who claimed that the stage of lactation had a pronounced effects on milk SCC, with level being high in early lactation, low in mid-lactation and high again in late lactation. The relatively higher SCC in the first stages of lactation could be due to the higher infection rate. Whereas, in the last term of lactation could be due to the reduction of the milk yield towards the end of lactation and consequently the rise of the SCC.

Farm management and hygienic factors

It is declared that a farm with a low bulk milk SCC is a title of a successful management. Among the management factors exhibited a significant effect on mean log SCC are origin of the cow, housing system and milking techniques. Whereas the type of udder cleaning, inter-milking sanitization methods and post-milking teat disinfection are the hygienic measures that showed a significant effect on the mean log SCC with regard to the contagious and environmental bacteria.

Although the infection rate was higher in the animals that were purchased, but the mean log SCC was not higher as that in the farms use the farm bred replacer. And the SCC level due to the effect of the pathogens was statistically different between and within groups. This could be a reflection of the management adopted for the animals in the farm and IMI control programs. In addition to the types of pathogens discovered and/or the age of the animals investigated.

The use of the loose-barns other than the slat floor or the plan floor was found to worse the condition of SCC. However, the condition is retained in the view that contagious bacteria were considered. Animals housed in loose-barns with slat floor had high LS-mean log SCC due to the effect of environmental bacteria, which was slightly higher than that in the animals kept in loose stalls with plan floor. In this sense the question of bedding types and conditions should be raised. But different implication was presented by Smith and Ely (1997) who reported that free-stall bedding did not significantly affect milk quality, with no difference in linear SCS among the herds studied.

The milking techniques that were implemented in farms investigated exerted a significant variation on the mean logarithmic SCC. Independent of the bacterial types, high LS-mean SCC (log) was obtained for milking parlor than for carrousel and pipe system. Whereas,

the use of the carousel unit was found to be effective in reducing the mean log SCC due to the infection with contagious bacteria compared to the other milking units. And that was due to the methods used for cleaning and disinfections of this unit. And in the case of the infection with environmental and non-specific bacteria, pipe system control great number of the pathogens and resulted in a low mean log SCC than in other systems. This variation could be managerial in nature, as a recent study specified that milking equipment was not statistically significant to the milk SCC, (Kiiman, 2001). Mazzucchelli et al. (2000) accounted that the changes in the management of a Spanish herd of cows affected by mastitis by improving the design of milking parlors and management of milking resulted in a reduction of the milk SCC from 380×10^3 cells/ml to 200×10^3 cells/ml.

Feeding methods with respect to the pathogens were created no clear effect on the mean log SCC. Although farms that used both methods of feeding methods experienced a higher level of mean log SCC. Sampling error should not be excluded, as the farms use combination of feeding methods are relatively fewer in number. Stationary method was the best in reducing the level of SCC. If the contagious pathogens were under strict management control, then the rise in the SCC level could be due to the effect of the environmental pathogens that could contaminate the feeding equipment. Best example is *St.dysg*. Which was found to be closely associated with the nutrition and feeding equipments in the study of Barkema et al. (1999).

Table 80: LS-means and S.E. of SCC (log) according to type of udder cleaning and bacterial status

Bacteria status	Moist	Dry
Contagious bacteria	5.20±0.03	5.01±0.02
Environmental bacteria	5.09±0.04	4.72±0.04
Samples without specific findings	5.01±0.01	4.66±0.01

Moist udder cleaning was found to aggravate the condition of the udder health and might be threatened by contagious or environmental pathogen's infections, which, were resulted in a higher level of mean log SCC (table 80). This outcome is supported by Yalcin et al. (1999), he concluded that udder preparation involving washing was associated with higher SCC.

Of the other hygienic measures studied is the inter-milking sanitization methods. Which explored a highly significant ($p=0.0001$) variations in mean log SCC. LS-mean SCC was

pronounced in farms practicing no inter-milking sanitization (5.11) compared to those using inter-milking sanitization (4.75). This could be a reflection of the reduction of the pathogens by the act of the sanitizer preparations. Nonetheless, and due to the effect of IMI causing pathogens and nonspecific pathogens, LS-mean logarithmic was significantly varied. Again the effects of pathogens in the farm that pays no attention to the usefulness of the effect of inter-milking sanitization was deleterious. Since the mean log SCC was consistently higher due to the bacterial effect. Air wash was the most effective method in reducing the effect of both contagious and environmental pathogens and end-up with a low level of log SCC compared to the other methods. Backflushing and spraying were weak in controlling the effect of the mastitis causers and to lower the level of log SCC. However, the relatively higher mean log SCC corresponded to the contagious bacteria could be indicative of the narrow spectrum effect of the sanitizer preparations used. This could be compared with the report of Radostits et al. (1994) who summarized the control measures of mastitis; he added that each of these control measures is aimed at the management of specific pathogen type.

Teat disinfection was routinely practiced in most of the farms surveyed and was found to have a significant effect on log SCC dependent as well as independent on the bacterial status. As the mean log SCC was higher in farms ignoring teat dipping and when their herds got infected with IMI causing bacteria. Whereas, the use of teat dipping reduce the effect of contagious as well as environmental bacterial and consequently resulted in a lower level of SCC. This emphasizes the importance of the use of teat dipping as one of the hygienic measure in controlling the infectious pathogens and lowering the milk SCC. Different studies handled this task of which Barkema et al. (1998) who reported about post-milking teat disinfections as an important factors for the prevention of a high bulk milk SCC. Boddie et al. (1993); Natzke, (1981); Pankey, (1989) and Malinowski (2000) who concluded that teat dipping is aimed at reducing infections mainly caused by contagious pathogens and preventing new infections and to a less extent preventing infections might be caused by environmental pathogens.

Test-day milk yield

Daily milk yield is assumed to be the indicator of the lactation production of the cow. The study revealed that high milk yield could be a predisposing factor to the infection. Test-day milk yield was found to be significantly ($p=0.0001$) decreasing as the level of SCC increase; this could explain a negative relation between milk yield and SCC. This finding

is consistent with the result of Geishauser et al. (1999). The study considered the effect of the factors affecting daily milk yield with relevance to the different classes of log SCC. It was reached that the daily milk decreased with the increase in the SCC.

Table 81: LS-means and S.E. of test-day milk yield among year-season

Year-season	Test-day milk yield (kg)
Summer 98	22.37±0.17
Autumn 98	22.64±0.14
Winter 98/99	22.47±0.12
Spring 99	23.81±0.13
Summer 99	24.06±0.13
Autumn 99	24.81±0.14
Winter 99/2000	25.08±0.16
Spring 2000	25.23±0.27

Independent on the level of SCC year-season exerted a significant ($p=0.0001$) effect on the mean daily milk yield (table 81). The over-all higher daily milk yield was obtained in winter 99/2000 and spring 2000, followed by summer 99 and autumn 99. Whereas, in summer 98, autumn 98 and winter 98/99 LS-mean test-day milk yield was statistically not differing. However, the lowest yield was recorded in summer 98. On the other hand, test-day milk yield showed a significant variation with respect to the level of SCC. It decreased in increasing rate with the increase in the SCC level. The difference was found to be greater with one fold increase in SCC in spring 2000 (5.73kg) than that in spring 99 (3.28kg). And in summer 98 (3.02kg) than in summer 99 (2.85). This variation could be due to the variation in the management measures between the years and/ or to the level of the infection rate and the type of the infecting organisms. Meanwhile, infection rate between seasons could be due to the effect of the temperature changes and housing. This finding was not different from that by Corbett, (1998); Rupp et al. (2000); Rodriguez et al. (2000) and Kelly et al. (2000).

Milk yield was found to increase in a decreasing rate irrespective of the SCC level from the first lactation to the third lactation, then the average milk yield started to decrease again. Dissimilar from the result obtained by Jones et al. (1984). However, the yield decreased in a decreasing rate as the SCC increased within lactation. This result agreed the result of Hortet and Seegers (1998) and that of Hortet et al. (1999). The variations detected are

perhaps due to the effect of the infection between the lactations that leads to the elevated the SCC.

Within the lactation, test-day milk yield was found to decrease as the level of SCC increase; the difference was significant ($p=0.0001$). With one fold increase in SCC, there was a decrease in milk yield of 0.19 kg. The difference could be attributed to the effect of the infection rate and consequently the SCC. A group of researchers attained comparable results (Vecht et al. 1989; Corbett, 1998; Labohm et al. 1998; Kelly et al. 2000; Rupp et al. 2000 and Haile-Mariam et al. 2001).

Farm managements and hygienic practices studied were found to have a significant effect on the test-day milk yield with respect to the level of SCC. As with the environmental factors, milk yield decrease as the level of SCC increased. It was agreed that the implementation of mastitis control programs were the most suitable means of lowering the SCC level and resulted in an optimum performance. Of the management factor that was positively affecting the milk yield is the origin of the cow. It was found that farm bred cows produced 0.24kg more daily milk than foreign cows with the same level of SCC. This is signifying the superiority of the farm management of heifers and the preparation for the future milk production than purchased heifers which might import the mastitis pathogens with. This could be comparable to the result of Jones and Bailey (1998).

According to the housing systems, Animals housed in loose stalls produced 1.43kg more daily milk than those housed in the other types of stalls. However, with respect to the level of SCC, although the yield decreased as the level of SCC increased, but no statistical variation was detected within the types of housing. This could be due the relation between infection rate, SCC and milk yield. Grohn (2000) discovered this positive relationship. Among the milking units studied, animals milked in carrousel unit yielded higher daily milk than those milked with pipe system and milking parlor. Sanitization of the milking units was explained to reduce the effect of the mastitis causing pathogens and consequently improving the yield performance.

Feeding methods were tested as an affecting factor on the daily milk yield. The effect was significant; stationary fed animals produced significantly higher milk than those fed with mobile method, dependent and independent of the SCC level. Feeding equipments contamination should be relayed on when looking for the reason of this difference.

Udder cleaning before milking found to have a significant effect on SCC and milk yield. The result indicated that the use of dry means of udder cleaning was approved better than moist cleaning methods, as the reduction of milk yield with the increase of SCC was lower with the former mean than with the later mean of the udder cleaning. The result was not different from the results of Radostits et al. (1994); Boddie et al. (1993); Natzke, (1981); Pankey, (1989) and Malinowski (2000). Independent of the SCC; milk yield/day was significantly higher when backflushing inter-milking sanitization is considered, conversely, lower than that and statistically equal yield was obtained with respect to the other inter-milking sanitization methods. Whereas, with respect to the level of SCC, air wash and spraying scored the higher milk yield.

The result also, explored the importance of using post-milking teat disinfection. Milk yield followed the general trend of reduction with the increase of SCC. However, teat dipping reduced the effect of SCC on milk yield, compared to the ignoring the use of post-milking teat disinfection. With the use of teat dipping and with high level of SCC, milk yield lowered with a rate of 1kg/day, whereas, with no use of teat dipping milk yield lowered with a rate of 2.36 kg/day with the same level of SCC. This could give hints to the role played by disinfectants and sanitizers in reducing the effect of the mastitis pathogens. Several publications supported this finding Radostits et al. (1994); Barkema et al. (1998); Oliver et al. (2001); and Saloniemi and Kulkas, (2001).

The relatively high daily milk yield that have been produced with the factors which were actually intended to reduce the mastitis pathogens effect but showed higher infection rate and SCC. Could be due to the farmers interference and/or intensive antibiotic treatment and the mean daily milk yield was found to be higher than when these factors experienced low infection rate and SCC. This indicating the successful handling of the cases. This point is in close agreement with Dohoo and Martin, (1984). They found a positive relationship between milk yield and clinical mastitis, which was attributed to the positive effect of the mastitis interference on milk production.

Heifer IMI

The study investigated the effect of the bacterial findings, time of sampling and bacterial pathogen groups on the first stage of lactation SCC of the heifer's a.p. and p.p.

Mean SCC was found to be significantly higher a.p. and p.p. for positive samples than negatives ones. And for the contagious than the environmental infection. However there was a higher rise in SCC when the animal infected a.p. than p.p. Mean test-day milk yield

was observed to decrease as the examination carried around the time of the parturition. This result indicated the risk of IMI for the lactating cow, as stated by Aarestrup and Jensen (1997). The finding proved that infection early in the heifer life which was indicated with the rise in SCC and decreased milk yield might result in damage to the developing mammary tissue and consequently reduced milk production, and/or cause the heifer to fail in reaching her maximum milk production potential. The result also indicated that the treatment of infected young herds before calving can reduce the probability of new IMI and will be usefulness for the future herds production. Owens et al. (2001) indicated that treatment of heifers in the trimester will reduce the chances of new intramammary infections occurring after treatment and persisting to calving. Nickerson et al. (1995) found that the mean SCC was 50% lower at calving for treated heifers, and milk yield over the first 2 months of lactation was 10% greater than that of untreated controls.

Risk factors associated with IMI and high SCC

The present study identified several risk factors for IMI and consequently a high level of SCC. Small herds (<200 cows) had 1.405 times risk of IMI and 1.001 times risk of a high threshold of SCC than large herds (>200 cows), the difference was highly significant ($p=0.0001$ and 0.0032 , respectively). This result indicating that small herds are at a relatively high risk of contracting IMI than large herds, comparable findings are those of Wilesmith et al. (1986); Lafi et al (1994) and Norman et al. (2000). It was also found that summer calving cows had a significantly ($p=0.0002$) high chance of being infected with mastitis and one time risk chance of having a high level of SCC than their winter herd-mates. This result is in joint agreement with Schultze, (1985) and Waage et al. (1998), they stated that calving in summer was associated with greater risk for mastitis than was calving at other times of the year. And also adding that the incidence of IMI before parturition has been considerably higher during warm than during cool weather. But a contrary result was that of Solbu, (1983), he observed a lowest rate of IMI in cows calving during summer. Cows at the early and late stage of lactation were at equal chance of IMI ($OR=1.014$, $p=0.2882$), indicating that during the entire lactation the risk of IMI was not varied. The study proved that contagious pathogens were significantly a greater risk factor for IMI as well as a high threshold of SCC ($OR=1.61$ and 1.15 $p=0.0001$, respectively) than environmental pathogens. This finding is closely related to the result of Peeler et al. (2000). Of the management factors that have associated with IMI is the origin of the cow, purchased heifers are at 1.47 times and 1.06 times chances of being attacked with IMI and

high level of SCC compared to farm-bred heifers. The difference was highly significant ($p=0.0001$) for IMI and insignificant for the threshold of SCC ($p=0.3656$). This could demonstrate the association between the mastitis in the purchased heifers and the incidence of mastitis in the herds from which they originated. Similar emphasis was that Waage et al. (1998). Tie-up system of housing was found to encourage the IMI than loose housing system (OR=1.07, $p=0.2282$) and 1.02 times chance of a high threshold level of SCC ($p=0.6711$). It could be clearly observed that there was no statistical difference. Cows milked in pipe system was at 1.03 times risk for IMI than cows milked in carrousel units and milking parlor, the difference was fairly significant ($p=0.0164$). Barkema et al. (1999) reported that milking units are among the risk factors for IMI. Of the hygienic factors investigated was the udder cleaning, moist udder cleaning compared to dry udder cleaning was at 1.6 times as risk factor for IMI and 1.23 times chance of encouraging a high threshold of SCC. The difference was highly significant ($p=0.0001$). The risk could be associated with the contagious infection. When inter-milking sanitization of the milking units was not used the risk for IMI was nearly three times than when the sanitization was used ($p=0.0001$) and the chance for a high threshold of SCC was 1.29 ($p=0.0031$). Indicating that inter-milking sanitization of the milking units prevent the IMI between milking and the other. Post-milking teat-dipping was illustrated as an important factor in controlling the IMI, the risk of non use of teat-dipping was 2.08 times for IMI and 2.35 times for a high threshold level of SCC, in both cases the difference was highly significant ($p=0.0001$). Teat disinfection was a risk factor in the study of Neave et al. (1969); Peeler et al. (2000).

Conclusion

1. Herds monitored in this study had a controlled environmental mastitis but still at high risk of IMI due to the effect of the contagious pathogens.
2. The most frequently isolated pathogens were those of the contagious group e.g. *S. aureus* and CNS, which let to significantly higher infection rates as well as higher level of SCC in the farm studied. At the time that the hygienic methods are directed towards one type of bacterium multi-isolates were discovered, i.e. more than species in one sample, this indicated that the use of multifunctional hygienic practices could assist in relieving the conditions.
3. Herd size was proving to affect the degree of infection to the extent that small herd size was more threatened with udder infection than large herd size, with consequent increased SCC level and decreased daily milk yield.

4. Primiparous cows were more affected than multiparous with the udder infection, this resulted in daily yield difference of 4.68 kg between the first parity cows and cows that had more than three parities, however, when the difference compared with high class of SCC it was dropped to 2.63 kg.
5. Within lactation, infection rate significantly different, it was higher in the first stage then decreased thereafter. This means that attention should be directed towards improvement of the udder health before or shortly after commencing the lactation, specifically for the contagious causers of udder infection. However, mean SCC and milk yield were affected with the type of pathogens and level of SCC in some instances, otherwise the normal physiological effects were the dominating effects.
6. Origin of the herd significantly influenced IMI determinants factors, farm bred cows performed better than foreign ones as the latter group scored significantly higher mean infection rate and lower mean daily milk yield.
7. Although the infection rate was higher in animals kept in loose housing with slat floor, but the mean SCC was not the highest, and plan floor's loose housing was showed to be more convenient.
8. As the farms investigated considered to be of special structure in aspect of herd size, carrousel and milking parlor units were evidencing the goal, infection rates were relatively low compared with pipe system, which showed higher probability of predisposing the animals to IMI, SCC was moderate and milk yield was considerably high.
9. The use of hygienic procedures are of utmost importance in reducing the effect of udder's causing pathogens and consequently reducing the mean infection rate, moist udder cleaning overwhelmed the condition and two times risky in predisposing to IMI, meanwhile dry udder cleaning gave best results, low mean infection rate and lower mean SCC.
10. Different methods of inter-milking sanitizations of the milking units were practiced and compared to the non-use of inter-milking sanitizations, the concluded result was: every method used resulted in fair reduction of the mean infection rate and mean SCC, within the methods: combinations of two or more methods were better than using solely one methods, if not available then bathing of the milking units, spraying and air wash should be given the priority.
11. Of the other hygienic methods tested was the post teat disinfections, teat dipping brought the expected results compared with non-teat dipping, significant difference

in mean infection rate, SCC and 1.86kg difference in daily milk yield. Contagious bacteria played the great role in elevating the rate of infection as well as that of SCC compared to environmental bacteria and that was resulted in lowering the mean daily milk yield. This concluded that in the future efforts should be directed towards hindering or minimizing the effect of such group of bacteria.

12. The study concluded also that and according to the result that the situations in the large class farm size is better than that in small class farm size in sense of infection rate and mean SCC. Nevertheless, the daily milk yield was only of 1.59 kg different between the two extremes classes.
13. Although the udder health status showed a remarkable improvement through the applications of some hygienic and management methods on the milking units and the stalls, but these should not be relied on as sole influencing factors, other environmental factors revealed negative effect on the udder health status like stage and number of lactation and season of the year. So the plans of the handling and treatments as well as the mastitis control programs should be oriented and implemented in accordance with such factors.
14. Results of investigation of the heifers mastitis explored that they were highly sensitive to infection than older cows, and their infection whether a.p. or p.p. reflected obvious effect on the milk yield, SCC was also varied according to the type of the infecting pathogen as well as the time of infection.
15. The unique fact of the current study was that measurements were taken in 48 commercial dairy farms with large herd size and management practices were carefully monitored.
16. As it was consummated from the results that the study could conclude that udder inflammation or mastitis in any degree of its occurrence has a negative effect not only on the performance of a cow but also on the milk it produced
17. Environmental and management factors are risk factors for IMI and a high threshold level for SCC and should be handled with a great care when attempting to control IMI.
18. The study suggests further investigation of the genetic correlation of the traits investigated and mastitis.

6 SUMMARY

The objective of the present field study was to investigate the factors that influence the udder health status in Thuringia. 48 dairy farms were randomly selected for the study. In the period from June 98 to April 2000 64542 milk samples from 10741 dairy cows were randomly collected and subjected to a bacteriological investigation. The relevant recorded performance data were obtained from the national data center (VIT) at Verden. Suitable statistical analysis models were selected to test the effect of the management and the hygienic factors on infection rate, SCC and daily milk yield.

The prevalence of the infection was 27.57% of the quarters and depending on the number of the affected quarters infection was detected in 77.21% of the animals. It was also found that 49.66% of the samples from the whole udder were positive. *S. aureus* and CNS were the most frequently isolated contagious pathogens with an udder and quarter prevalence of 28.70/35.50% and 26.60/32.70%, respectively. Followed by *St. dys.* and EPS (environmental pathogens) with an udder and quarter prevalence's of 12.90/13.90% vs. 9.0/10.60%, respectively.

Throughout the lactations infection rate was higher in primiparous cows and lower in multiparous cows (1.32 and 1.24, respectively). However, SCC was higher in multiparous cows (5.27) and lower in primiparous cows (4.48). The daily milk yield followed the same trends as the SCC.

Within lactation infection rate was higher in early stage of lactation (1.28) and lower thereafter (1.27). SCC was lower early in the lactation (4.85) and increased thereafter to reach 4.96 in the late stage of lactation. Daily milk yield reached the peak in the early stage of lactation (28.41 kg) and was lower in the late stage of lactation (19.52 kg).

Concerning the management factors, farm bred cows had a significantly lower mean infection rate than purchased cows (1.29 and 1.33, respectively). The mean SCC was higher in farm bred cows (4.95) and lower in the purchased cows (4.83). However, daily milk yield was higher in farm bred cows than purchased cows (24.78 and 22.39 kg, respectively).

IMI due to the effect of the herd size was significantly varied. Infection rate was 1.31 in the small herds and 1.24 in the large herds. The mean SCC (log) was 5.06 in the small herds and 4.58 in the large herds. Whereas the daily milk yield ranged between 23.30-24.94 kg.

Autumn 99 was the season of a higher infection rate (1.31) and spring 2000 was the season of a lower infection rate (1.18). SCC was significantly varied according to the year-season. The means SCC (log) were 4.68 and 4.66 during spring 2000 and winter 98/99, respectively and 4.52 was a lower value of SCC during summer 98. High daily milk yield was 25.23 kg in spring 2000 and a lower yield was 22.37kg in summer 98.

Housing system significantly influenced the mean infection rate, it was higher (1.27) in the animals housed in a loose stall with plan floor than those their stall with a slat floor (1.26). Whereas infection rate was lower in animals housed in stall other than loose stall (1.22). The mean SCC was significantly lower in the loose housing (4.77 and 4.95) and higher in the stall other than loose housing (5.05). Whereas the daily milk yield was higher in the animals managed in loose stall (23.21 and 23.16 kg) and lower in animals housed in the other stall (21.76 kg).

The milking systems were found to be affecting the infection rate. Infection rate was higher with the use of pipe system (1.34) less than that with the use of carrousel unit (1.30) and lower with the use of milking parlor (1.29). The opposite trends were the behavior of the SCC which was higher in case of milking parlor (4.93), lower when pipe system was used (4.84), the difference was significant. However, the daily milk yield was extremely higher with use of carrousel unit (24.49 kg) and lower with the use of pipe system (19.84 kg).

A significant difference was detected in the mean infection rate, SCC and daily milk yield due to the effect of the feeding methods. Infection rate was higher in the farms use both mobile and stationary methods of feeding (1.30) and lower in the farms use either mobile or stationary methods (1.29 and 1.27, respectively). SCC was 4.83 in farms use mobile methods, 4.57 in those use stationary methods and 5.98 in the farms use both methods. The daily milk yield was 21.31 kg in the animals fed with mobile methods, 25.94 kg in the animals fed with stationary method and 24.51 kg in those fed with both methods.

The difference in the mean infection rate of animals their udders cleaned with moist and dry methods was 0.01 which were 1.30 and 1.29 respectively. The mean SCC were 5.10 and 4.80 for the animals cleaned with moist and dry cleaning methods respectively. The mean daily milk yield was 24.87 and 22.67 kg in the two methods in the order.

On comparing the inter-milking sanitization of milking units, with the non-use of inter-milking sanitization it was found that the mean infection rate was higher when the method was not used (1.34) lower when practiced (1.26-1.33). The same trend was detected in the

case of the SCC, it was higher (5.11) in the farms ignoring inter-milking sanitization and lower in farms employing sanitization of milking units (4.21-4.98). The daily milk yield was lower in the animals their milking units were not sanitized (23.06 kg) and higher when milking units were sanitized (23.09-25.55 kg).

Application of teat dipping resulted in a reduced infection rate (1.29). Whereas the non-use of the teat dipping resulted in an infection rate of 1.32 with a significant difference. The SCC was 5.08 in the animals their teats were not dipped and 4.88 in those their teats were dipped. The daily milk yield was 25.04 kg when the animal's teats were dipped and 23.18 kg when the teat dipping was ignored.

Heifers subjected to udder infection ante partum were prone to develop a high SCC later in the lactation. And the mean daily milk yield was lower than when the infection was occurred post partum.

The risk for developing IMI and consequently a high threshold level of SCC was increased when the attacking pathogen was a contagious than when it was an environmental, in small herds than in large herds, in alter cows than younger ones and in the early stage of lactation. Summer calving cows were at higher risk of IMI than winter calving cows also the purchased heifers compared to the farm bred counterparts. The probability for encountering IMI was higher, in a tie-stall housed cows and with the use of pipe milking unit. Meanwhile it was found that the moist mean of udder cleaning, the non use of inter-milking sanitization preparation and a level of SCC (log) level higher than 5.73 were associated with increased risk for IMI.

7 Zusammenfassung

Die vorliegende Feldstudie hat zum Ziel Faktoren zu ermitteln, die auf den Eutergesundheitszustand von Kühen Einfluss nehmen. 48 Milcherzeugungsbetriebe aus dem Freistaat Thüringen wurden zufallsgemäß für die Studie ausgewählt. Im Zeitraum von Juni 1998 bis April 2000 wurde 64542 Milchproben von 10741 Milchkühen erfasst und einer bakteriologischen Untersuchung unterzogen. Weitere Leistungs- und Gruppierungsmerkmale stammen aus dem zentralen Datenspeicher für Deutsche Holstein des VIT-Verden bzw. einem Fragebogen, der selbst entwickelt, den Betrieben zur Beantwortung zugeleitet wurde. Mit Hilfe statistische Modelle wurden die Effekte von Managements- und hygienischer Faktoren auf die Infektionsrate (IMI), den Gehalt an somatischen Zellen und die tägliche Milchleistung analysiert. Als Prozeduren wurden MIXED, GLM und Logistik (SAS Institute Inc. 1996) verwendet, die eingegebenen Mittelwerte sind least Square.

Bei 27,57% der Viertel und abhängig von den Anzahlbetroffenen Vierteln konnten 77,21% der Tiere Infektionen nachgewiesen werden. Bei Untersuchung von Milchproben aus dem Gesamtgemelk wurde mit einer Häufigkeit von 49.60% positiver Befund erhoben. *S. aureus* und CNS waren die am häufigsten isolierten Krankheitserreger mit einer Häufigkeit von 35,50 (Viertelproben)/28,70% (Gesamtsagemelkproben) bzw. von 32,70/26,60%, gefolgt von den *Str. dysgalactiae* und von EPS (Umwelterreger) mit entsprechenden Häufigkeiten von 13,90/12,90% bzw. 10,60/9,0%.

Die Infektionsrate war bei den erstmals kalbenden Kühen höher und bei den multiparen Kühen niedriger (1,32 bzw. 1,24). Demgegenüber waren jedoch die Zellzahlen bei den multiparen Kühen höher (5,27) und bei den erstmals gebärenden Kühen niedriger (4,48). Die tägliche Milchleistung folgte den gleichen Tendenzen wie die Zellzahlen.

Innerhalb der Laktation war wie die Infektionsrate im frühen Stadium der Laktation höher (1,28) und danach geringfügig niedriger (1,27). Die Mittelwerte der Zellzahlen erreichten im frühen Stadium der Laktation niedrige Werte (4,85), um danach anzusteigen und im späten Stadium einen Wert von 4,96 zu erreichen. Die tägliche Milchleistung erreichte ihre Spitze im frühen Stadium der Laktation (28,41 kg) und war im späten Stadium der Laktation niedriger (19,52 kg).

Hinsichtlich der Managementfaktoren hatten Kühe aus Betrieben mit eigener Jungrinderaufzucht eine signifikant niedrigere mittlere Infektionsrate als solche aus Betrieben mit Färsenzukauf (1,29 und 1,33). Die Zellzahlmittelwerte waren in den Betrieben mit eigener Aufzucht höher (4,95) gegenüber solchen mit zugekauften Tieren (4,83). Jedoch war die

mittlere tägliche Milchleistung in Betriebe mit eigener Reproduktion höher als bei Zukauf von Färsen (24,78 und 22,39 kg).

Die Herdengröße nahm erheblichen Einfluss auf die IMI. In den kleinen Herden betrug die Infektionsrate 1,31 und in den großen Herden 1,24. Die Mittelwerte der logarithmierten Zellzahl erreichten in den kleinen Herden 5,06, in den großen Herden 4,58. Die mittlere tägliche Milchleistung variierte zwischen 23,30-24,94 kg/Kuh.

Bezüglich des Jahres-Saison-Effektes konnte für Herbst 1999 eine hohe Infektionsrate (1,31) und für Frühling 2000 eine niedrige Infektionsrate (1,18) festgestellt werden. Die somatische Zellzahl wurde erheblich zwischen den Jahres-Saisonklassen verändert. Hohe Mittelwerte von 4,68, 4,66 wurden für Frühling 2000 und Winter 98/99 registriert, während nur 4,52 als Mittelwert für den Sommer 98 registriert wurde. Einer hohen täglichen Milchleistung von 25,23 kg im Frühjahr 2000 stehen 22,37 kg im Sommer 98 gegenüber.

Das Haltungssystem beeinflusste die mittlere Infektionsrate signifikant bei geringen Differenzen. Sie war höher (1,27) bei Tieren, die in einem Laufstall mit planbefestigten Boden gehalten wurden als bei solchen aus Ställen mit einem Spaltenboden (1,26). Die niedrigste wurde bei Tieren festgestellt, die nicht in Laufställen (1,22) gehalten wurden. Die Zellzahlmittelwerte waren signifikant niedriger bei den Gruppen mit Laufstallhaltung (4,77(planbefestigten)-4,95(Spaltenboden)) gegenüber andere Haltungsformen (5,05). Bei Laufstallhaltungsformen war die tägliche Milchleistung der Kühe höher (23,21 und 23,16 kg) gegenüber Tieren aus anderen Haltungsformen (21,76 kg).

Die Art des Melksystems beeinflusst die Infektionsrate. Sie war bei Nutzung von Rohrmelkanlagen am höchsten (1,34), geringer unter den Bedingungen des Melkkarussells (1,30) und am niedrigsten bei den verschiedenen Formen von Melkständen (1,29). Gegensätzliche Tendenzen wurden beim Verhalten der Zellzahlen ermittelt, bei denen für Melkständen ein signifikant höheres Niveau (4,93) gegenüber Rohrmelkanlagen ermittelt wurde (4,84). Jedoch war die tägliche Milchleistung bei Nutzung von Melkkarussells (24,49 kg) höher als bei Rohrmelkanlagen (19,84 kg). Signifikante Unterschiede bezüglich des Effektes der Fütterungsmethoden wurden bei der Infektionsrate, den Zellzahlen und der täglichen Milchleistung ermittelt.

Betriebe, die im Fragebogen ausweisen sowohl mobile als auch stationären Fütterungssysteme anzuwenden, hatten die höchste mittlere Infektionsrate (1,30), gefolgt von denen mit mobiler (1,29) und stationärer Fütterung (1,27). Die mittleren Zellzahlen betrugen 4,83 bei den Betrieben mit mobilen Methoden, 4,57 bei denen mit stationären und 5,98 bei solchen, die beide Methoden verwendeten. Die tägliche Milchleistung betrug 21,31 kg bei den Tieren, die

mobil, 25,94 kg bei den Tieren, die stationär und 24,51 kg bei denen, die unter Nutzung beider Systeme gefüttert wurden.

Die Differenz bezüglich der Infektionsrate erreichte zwischen Tieren, deren Euter feucht bzw. trockenen gereinigt wurden lediglich 0,01, bei Mittelwerten von 1,30 bzw. 1,29. Als Zellzahlmittelwerte konnte für beide Reinigungsmethoden 5,10 (feucht) bzw. 4,80 (trocken) ermittelt werden. Die tägliche Milchleistung betrug 24,87 bzw. 22,67 kg bei beiden Methoden.

Die Zwischendesinfektion des Melkzeugs hatte einen senkenden Effekt auf die mittlere Infektionsrate der Kühe (1,26-1,33) gegenüber solchen Betrieben, bei denen dieses Verfahren nicht angewendet wurde (1,34). Die gleiche Tendenz wurde für den Gehalt an somatischen Zellen ermittelt. Die Zellzahl war in den Betrieben (5,11) höher, die keine Zwischendesinfektion betrieben und in den Betrieben wesentlich niedriger, die dieses Verfahren einsetzten (4,21-4,98).

Die Anwendung der Zitzendesinfektion nach dem Melken verringerte die Infektionsrate (1,29), während bei Entfallen dieser Maßnahme eine signifikant höhere Infektionsrate von 1,32 auftrat. Die Zellzahlen waren mit 5,08 bei den Tieren, deren Zitzen nicht gedippt wurden, signifikant höher gegenüber solchen deren Zitzen desinfiziert wurden. Die mittleren täglichen Milchleistungen betrugen 25,04 kg bei Zitzendesinfektion und 23,18 kg im Falle des Weglassens dieser Maßnahme.

Färsen, bei denen ante partum eine Euterinfektion nachgewiesen werden konnte, zeigten in der darauf folgenden Laktation erhöhte Zellzahlen, verbunden mit niedrigeren täglichen Milchleistungen.

Unter Nutzung von der Prozedur Logistik konnte bei Nachweis von kontagiösen Erregern eine höhere Wahrscheinlichkeit für das Auftreten von IMI, verbunden mit einem höheren Schwellenniveau für die Zellzahl, gegenüber Umweltkeimen ermittelt werden. Weiterhin konnten höhere Wahrscheinlichkeiten für Nachweis IMI, die mit höheren Zellzahlen korrespondierten, bei den Faktoren Herdengröße für die Faktorstufe kleine Herden, Abkalbsaison für Sommerkalbung, Laktationsnummer für höhere Laktationen, Haltungssystem für Anbindstall, Euterreinigungsverfahren für die Anwendung feuchter Tücher und Zwischendesinfektion des Melkzeugs wenn dieses Verfahren nicht angewendet wird, festgestellt werden.

REFERENCES

- Aarestrup, F.M. and Jensen, N.E. (1997): prevalence and duration of intra mammary infection in Danish heifers during the pre partum period. *J. dairy sci.* 80:307-312.
- Ali-Vehmas, T. and Sandholm, M. (1995): The bovine udder and mastitis: Balance between bacteria and host-The bacteria's point of view. Univ. of Helsinki, Faculty of vet. Med. ISBN: 951-834-047-1 pp 49-74.
- Allore, H.G.; Oltenacu, P.A. and Erb, H.N. (1997): Effects of season, herd size and geographic region on the composition and quality of milk in the northeast. *J. dairy sci.* 80(11):3040-3049.
- Anacker, G. and Jänicke, Aj.U. (1999): Färsenmastitis. Abschlussbericht. Thüringer Landesanstalt für Landwirtschaft. Themen-Nr. :21-07-510/99.
- Anderson, R.R. (1985): Mammary gland. Lactation (pp 3). B.L. Larson, ed. Iowa State Univ. Press, Ames. (Cited by Trinidad, P.; Nickerson, S.C. and Alley, T.K. (1990): Prevalence of intra-mammary infection and teat canal colonization in unbred and primigravid dairy heifers. *J. Dairy sci.* 73:107-114.).
- Arbeitsgemeinschaft Deutscher Rinderzüchter, ADR (2001): Rinderproduktion in der Bundesrepublik Deutschland 2000, Bonn.
- Barbano, D.M.; Rasmussen, R.R. and Lynch, J.M.(1991): Influence of milk somatic cell count and milk age on cheese yield. *J. Dairy sci.* 74:369-388.
- Barnes-Pallesen, F.D.; Blachmer, P.; Britten, A.; Bushnell, R.B.; Van Damme, D.M. and Welcome, F. (1987): Laboratory and field handbook on bovine mastitis. Natl. Mastitis Counc., Arlington, VA.
- Barkema, H.W.; Schukken, Y.H.; Lam, T.J.G.M.; Beiboer, M.L.; Benedictus, G. and Brand, A. (1998): Management practices associated with low, mid and high bulk milk somatic cell count. *J. dairy sci.* 81:1917-1927.
- Barkema, H.W.; Schukken, Y.H.; Lam, T.J.G.M.; Beiboer, M.L.; Benedictus, G. and Brand, A. (1999): Management practices associated with the incidence rate of clinical mastitis. *J. dairy sci.* 82:1643-1654.
- Barkema, H.W.; Schukken, Y.H.; Lam, T.J.G.M.; Beiboer, M.L.; Wilmink, H.; Benedictus, G. and Brand, A. (1998): Incidence of clinical mastitis in dairy herds grouped in three categories by bulk milk somatic cell counts. *J. dairy sci.* 81(2):411-419.
- Barkema, H.W.; Schukken, Y.H.; Lam, T.J.G.M.; Galligan, D.T.; Beibore, M.L. and Brand, A. (1997): Estimation of interdependence among quarters of the bovine udder with sub-clinical mastitis and implication for analysis. *J. dairy sci.* 80:1592-1599.
- Barker, A.R.; Schrick, F.N.; Lewis, M.J.; Dowlen, H.H. and Oliver, S.P. (1998): Influence of clinical mastitis during early lactation on reproductive performance of Jersey cows. *J. dairy sci.* 81:1285-1290.
- Barto, P.B.; Bush, L.J. and Adams, G.D. (1982): Feeding milk containing *S. aureus* to calves. *J. dairy sci.* 65:271.

- Bewley, J.; Palmer, R.W. and Jackson-Smith, D.B. (2001): A comparison of free-stall barns used by modernized Wisconsin dairies. *J. dairy sci.* 84:528-541.
- Boddie, R.L.; Nickerson, S.C. and Adkinson, R.W. (1993): Evaluation of teat germicides of low iodine concentration for prevention of bovine mastitis by *S. aureus* and *St. agalactiae*. *Prev. Med.* 16:111-117.
- Booth, J.M. (1988): Progress in controlling mastitis in England and Wales. *Vet. Res.* 122:299-302.
- Bramley, A.J. (1985): The sources of mastitis pathogens for a dairy herd and their control. *Kieler Milchwirtschaftliche Forschungsberichte* 37:375-385.
- Bramley, A.J. (1991): Mastitis physiology or pathology? *Flem. Vet. J.* 62(1):3-11.
- Bramley, A.J. and Dodd, F.H. (1984): Reviews of the progress of dairy science: mastitis control-progress and prospects. *J. dairy res.* 51:481-
- Bray, D.R. and Shearer, J.K. (1996): Mastitis control. Uni. Florida, dept. dairy and poultry, institute of food and agric. Sci.
- Brooks, B.W. and Barnum, D.A. (1984): The susceptibility of bovine udder quarters colonized with *C. bovis* to experimental infection with *S. aureus* or *St. agalactiae*. *Cand. J. Comp. Med.* 48:146-150.
- Bushnell, R.B. (1989): Pasteurization of milk and colostrum fed to dairy calves. *Proc. Eastern States Vet. Conf., Gainesville, FL. Eastern States Vet. Assoc., Gainesville, FL.* 112-
- Buzalski, T.H. and Pyörälä, S. (1995): The bovine udder and mastitis: Monitoring and management of udder health at the farm. Univ. of Helsinki, Faculty of vet. Med. ISBN: 951-834-047-1: 252-260.
- Buzalski, T.H. and Seuna, E. (1995): The bovine udder and mastitis: Isolation and identification of pathogens from milk. Univ. of Helsinki, Faculty of vet. Med. ISBN: 951-834-047-1. 121-141.
- Carnier, P.; Bettela, R.; Cassandro, M.; Gallo, L.; Mantovani, R. and Bittante, G. (1997): Genetic parameters for test day somatic cell count in Italian Holstein Friesian cows. 48th Annu. Meet. Europ. Assoc. Anima. Prod. Vienna-Austria-August 25-28, 1997 Session IV: Mastitis control programmes. 1-5.
- Chrystal, M.A.; Seykora, A.J. and Hansen, L.B. (1999): Heritabilities of teat-end shape and teat diameter and their relationships with somatic cell score. *J. dairy sci.* 82:2017-2022.
- Corbett, R.B. (1998) : The use of somatic cell counts in mastitis management. *Proc. 37th Annu. Mtg., natl. Mastitis council, National mastitis council, Inc., Madison, WI.* 51-55.
- Costa, E.O.; Riberio, A.R.; Watanabe, E.T. and Melville, P.A. (1998): Infectious bovine mastitis caused by environmental organisms. *Zentralblatt für Veterinärmedizin.* 45(2):65-71.

- Cullor, J.S. (1990): Mastitis and its influence upon reproductive performance in dairy cattle. Proc. Int. Symp. Bovine Mastitis, Indianapolis. IN. Natl. Mastitis coun., Inc. and Am. Assoc. Bovine Pract., Arlington, VA. 176-180.
- Daniel, R.C.W.; Barnum, D.A. and Leslie, K.E. (1986): Observation on intra-mammary infections in first calf heifers in early lactation. Can. Vet. J. 27:112-115.
- David, R.B and Shearer, J.K. (1996): Mastitis control. Co-operative extension service, institute of food and agricultural sciences, university of Florida. DS-7.
- Davidson, I. (1961): Observation of the pathogenic staphylococci in a dairy herd during a period of six years. Res. Vet. Sci. 2:22-40.
- De Graaf, T. and Dwinger, R.H. (1996): Estimation of milk production losses due to sub-clinical mastitis in dairy cattle in Costa Rica. Prev. Vet. Med. 26(3-4):215-222.
- Deutsche Veterinärmedizinische Gesellschaft (DVG, 1980): die subklinische Mastitis des Rindes und Vorschläge für bundeseinheitliche Richtlinien zu ihrer Bekämpfung. DVG, Fachgruppe Milchhygiene des Arbeitsgebietes Lebensmittelhygiene, Kiel.
- Deutsche Veterinärmedizinische Gesellschaft (DVG, 1989) :Leitlinien zur Bekämpfung der Mastitis des Rindes als Bestandsproblem. 2.Aufl. Kiel.
- Deutsche Veterinärmedizinische Gesellschaft (DVG, 1994) :Leitlinien zur Bekämpfung der Mastitis des Rindes als Bestandsproblem. Sachverständigenausschuss (Subklinische Mastitis) des Arbeitskreises Eutergesundheit der Fachgruppe Milchhygiene des Arbeitsgebietes Lebensmittelhygiene, 3.Aufl. Kiel.
- Dohoo, I.R. and Martin, S.W. (1984): Disease, production and culling in Holstein-Friesian cows. IV- Effect of disease on production. Prev. Vet. Med. 2:755-770.
- Döpfer, D.; Barkema, H.W.; Lam, T.J.G.M.; Schukken, Y.H. and Gaastra, W. (1999): Recurrent clinical mastitis caused by *Escherichia coli* in dairy cows. J. dairy sci. 82:80-85.
- Emanuelson, U. and Oltenacu, P.A. (1998): Incidence and effects of diseases on the performance of Swedish dairy herds stratified by production. J. dairy sci. 81(9): 2376-2382.
- Emanuelson, Ulf; Thomas, Olsson; Tiina, Mattila; Goran, Astrom; and Olof, Holmberg (1988): Effects of parity and stage of lactation on adenosine triphosphate, somatic cell count and antitrypsin content in cows' milk. J. dairy res. 55(1):49-55.
- Erb, H.N. (1985): Path model of reproductive disorders and performance, milk fever, mastitis, milk yield and culling in Holstein cows. J. dairy sci. 68:3337-3349.
- Etherington, W.G.; Kinsel, M.L. and Marsh, W.E. (1996): Relationship of production to reproductive performance in Ontario dairy cows: Herd level and individual animal descriptive statistics. Theriogenology 46:935-959.
- Fetrow, J.; Mann, D.; Butsche, K. and McDaniel, B. (1991): Production losses from mastitis: carry over from the previous lactation. J. dairy sc. 74:833-839.
- Firat, M.Z. (1993): An investigation into the effects of clinical mastitis on milk yield in dairy cows. Livest. Prod, Sci. 36:311-321.

- Firat, M.Z. (1993): Susceptibility of clinical mastitis in successive lactations. *Livestock prod. sci.* 34(1-2):175-180.
- Fleischer, P., Metzner, M., Beyerbach, M., Hoedemaker, M. and Klee, W. (2001) : The relationship between milk yield and the incidence of some diseases in dairy cows. *J. dairy sci.* 84,2025-2035.
- Geishauser, T.; Querengasser, K.; Nitschke, M. and Sorbiraj, A. (1999): Milk yield, somatic cell counts, and risk of removal from the herd for dairy cows after covered teat canal injury. *J. dairy sci.* 82(7):1482-1488.
- Gentilini, E.; Denamiel, G.; Pérez Monti, H.; Marco, C. and López Amoedo, M. (1994): Mastitis Bivina: Perfiles de sensibilidad de cepas de *Staphylococcus* spp. Por el método de antibiograma semicuantitativo en agar frente a 10 antimicrobianos. *Vet. Arg.* Vol.10:314-321.
- Godollo, S.I.E. and Tanszek, S.J. (2000) : Factors influencing somatic cell count in milk. 2-physiological and environmental factors. *Tejgazdasag.* 60:16-25.
- Gröhn, Y.T. (2000): Milk yield and disease: towards optimizing dairy health and management decisions *Bovine-Practitioner.*34(1):32-40.
- Gröhn, Y.T.; Ducrocq, V. and Hertl, J.A. (1997): Modeling the effect of a disease on culling: an illustration of the use of time-dependent covariate for survival analysis. *J. dairy sc.* 80(8):1755-1766.
- Gröhn, Y.T.; Eicker, S.W. and Hertl, J.A. (1995): The association between 305-day milk yield and disease in New York dairy cows. *J. dairy sci.* 78:1693-1702.
- Haile-Mariam, M.; Bowman, P.J. and Goddard, M.E. (2001): Genetic and environmental correlations between test-day somatic cell count and milk yield traits. *Livestock. Prod. Sci.* 73:1-13.
- Haile-Mariam, M.; Goddard, M.E. and Bowman, P.J. (2001): Estimates of genetic parameters for daily somatic cell count of Australian dairy cattle. *J. dairy sci.* 84,1255-1264.
- Hamann, J. (2001): Mastitis notes from members countries, Germany. *Bull. IDF* 367/2001. 18-21.
- Harmon, R.J. and Reneau, J.K. (1993): Factors affecting SCSs in milk. *Proc. Annu. Mtg. Natl. Mastitis Counc., Kansas City, MO. Natl. Mastitis Counc., Inc., Arlington, VA.* 48-54.
- Heringstad, B. G; Klemetsdal, G. and Ruane, J. (1999): Clinical mastitis in Norwegian cattle: Frequency, variance components and genetic correlation with protein yield. *J. dairy sci.* 82:1325-1330.
- Hill, A.W. and Shears, A.L. (1979): Recurrent coliform mastitis in the dairy cow. *Vet. Resci.* 105:299-301.
- Hill, A.W.; Shears, A.L. and Hibbit, K.G. (1978): The elimination of serum-resistant *Escherichia coli* from experimentally infected single mammary glands of healthy cows. *Res. Vet. Sci.* 25:89-93.
- Hillerton, J.E. (1999): Redefining mastitis based on somatic cell count. *Inter. Dairy Fed. Bullet. No.* 345/1999, 4-6.

- Hogan, J.S. and Smith, K.L. (1987): A practical look at environmental mastitis. *Comp. Cont. Educ. Pract. VET.* 9: F341.
- Hogan, J.S., Smith, K.L., Hoblet, K.H., Schoenbrger, P.S., Todhunter, D.A., Hueston, W.D., Pritchard, D.E., Bowman, G.L., Heider, L.E., Brockett, B.L. and Conrad, H.R. (1989a) : Field survey of clinical mastitis in low somatic cell count herds. *J. dairy sci.* 72:1547-1556.
- Hogan, J.S.; White, D.G. and Pankey, J.W. (1987): Effects of teat dipping on intramammary infections by staphylococci other than *S. aureus*. *J. dairy sci.* 70: 2520-2525.
- Hortet, P. and Seegers, H. (1998): Calculated milk production losses associated with elevated somatic cell counts in dairy cows: review and critical discussion. *Vet. Res.* 29(6):497-510.
- Hortet, P.; Beaudreau, F.; Seegers, H. and Fourichon, C.(1999):Reduction in milk yield associated with somatic cell counts up to 600×10^3 cells/ml in French Holstein cows without clinical mastitis. *Livestock prod. Sci.*61:33-42.
- IDF (1987): Bovine mastitis. Definition and guidelines for diagnosis. International Dairy Federation Bulletin 211, pp24.
- Jemeljanovs, A. and Bluzmanis, J. (2000): Somatic cells and microorganisms content in milk and it effecting factors. 51th Annual meeting of the European association for animal production, the Hague, the Netherlands 21-24 August 2000. Poster no. 530.
- Jemeljanovs, A.; Bluzmanis, J.; Mozgis, V. and Reine, A. (1999): The evaluation of mastitis pathogenic agents and its possible influence on consumers health. 50th Annual meeting of the European association for animal production, Zurich, Switzerland, 22-26.
- Jones, F.G., Ward, G.E. (1989): Cause, occurrence and clinical signs of mastitis and anorexia in cows in a Wisconsin, *JAVMA*.195(8) 1108-1113.
- Jones, G.M. and Bailey, Jr. (1998), Mastitis control in heifers and first lactation. Virginia cooperative extension, publication number: 404/281.
- Jones, G.M.; Pearson, R.E.; Clabaugh, G.A. and Heald, C.W. (1984): Relationships between somatic cell counts and milk production. *J. dairy sci.* 67(8):1823-1831.
- Jonsson, P.; Olsson, S.O.; Olofson, A.S.; Fälf, C.; Holmberg, O. and Funke, H. (1991): Bacteriological investigations of clinical mastitis in heifers in Sweden. *J. dairy Res.* 58:179-185.
- Kelly, A.L.; Tiernan, C.O'Sullivan and Joyce, P. (2000): correlation between bovine milk somatic cell count and polymorphnuclear leuckocyte level for samples of bulk milk and milk from individual cows. *J. dairy sci.* 83:300-304.
- Keown, J.F. (1997): Dairy 10-point quality control program-Mastitis treatment records. Institute of Agric. Nat. Resour. Uni. Nebraska-Lincoln. G92-1101-A.
- Kiiman, H. (2001): The analysis of the milk somatic cell count reducing possibilities. *J. agric. Sci.* 12(3):162-174.

- Kiiman, H. and Saveli, O. (2000): On the factors affecting milk somatic cell count in dairy cattle in Estonia. European Association for animal production 51st Meeting-The Hague-The Netherlands.
- Kirk, J.H.; Wright, J.C.; Berry, S.L.; Reynolds, J.P.; Maas, J.P. and Ahmadi, A. (1996): Relationships of milk culture status at calving with somatic cell counts and milk production of dairy heifers during early lactation on a California dairy. *Preventive Veterinary Medicine* 28(3):187-198.
- Kirk, J.H.; Wright, J.C.; Berry, S.L.; Reynolds, J.P.; Maas, J.P. and Ahmadi, A. (1996): Relationships of milk culture status at calving with somatic cell counts and milk production of dairy heifers during early lactation on a Californian dairy. *Prev. Vet. Med.* 28(3):187-198.
- Klaas, I.C. (2000): Untersuchungen zum Auftreten von Mastitiden und zur Tiergesundheit in 15 Milchviehbetrieben Schleswig-Holsteins. Dissertation, Free university-Berlin.
- Klei, L.; Yun, J.; Sapru, A.; Lyunch, J.; Barbano, D.; Sears, P. and Galton, D. (1998): Effects of milk somatic cell count on cottage cheese yield and quality. *J. Dairy sci.* 81:1205-1213.
- Koldewei, E.; Emanuelson, U. and Janson, L. (1999): Relation of milk production loss to milk somatic cell count. *Acta Veterinaria Scandinavica* 40(1):47-56.
- Korhonen, H. and Kaartinen, L. (1995): The bovine udder and mastitis: Changes in the composition of milk induced by mastitis. Univ. of Helsinki, Faculty of vet. Med. ISBN: 951-834-047-1: 76-82.
- Labohm, R. ; Götz, E.; Luhofer, G.; Hess, R.G. and Bostedt, H. (1998): factors influencing the somatic milk-cell count in dairy cows. 1- influence of bacteriological findings, stage and number of lactation. *Milchwissenschaft* 53(2):63-69.
- Laevens, H., Deluyker, H., Schukken, Y.H., De Meulemeester, L., Vandermeersch, R., DE Muelenaere, E. and De Kruif, A. (1997) : Influence of parity and stage of lactation on somatic cell count in bacteriologically negative dairy cows. *J. dairy sci.* 80,3219-3226.
- Lafi, -S.; Al-Rawashdeh, -O.; Na`was , -T. and Hailat, N. (1994): National cross-sectional study of mastitis in dairy cattle in Jordan. *Trop. Anim. Health. Prod.* 26(3):168-174.
- Lam, .J.G.M.; Lipman, L.J.A.; Schukken. Y.H.; Gaastra, W. and Brand, A. (1996): Epidemiological characteristics of bovine clinical mastitis caused by *Escherichia coli* and *Staphylococcus aureus* studied by DNA fingerprinting. *Am. J. Vet. Res.* 57:38-42.
- Lescourret, F. and Coulon, J.B. (1994): Modeling impact of mastitis on milk production by dairy cows. *J. dairy sci.* 77:2289-2301.
- Leslie, K.E. (1996): Somatic cell counts: Interpretation for individual cows. Ontario factsheet. Agdex: 410/662.
- Liebe, A.; Worstorff, H. and Schams, D. (1996) : Changes in somatic cell count and plasma cortisol concentration during three relocation trials in German Brown cows. *Milchwissenschaft* 51:423-426.

- Linde, C.; Holmberg, O. and Astrom, G. (1980): The interference between coagulase negative staphylococci and *Corynebacterium bovis* and the common udder pathogens in the lactating cow. *Nord. Veterinaermed.* 3:552-
- Lipman, L.J.A.; de Nijs, A.; Lam, T.J.G.M. and Gaastra, W. (1994): Identification of *Escherichia coli* strains from cows with clinical mastitis by serotyping and DNA polymorphism patterns with REP and ERIC primers. *Vet. Microbiol.* 43:13-19.
- Lucey, S. and Rowlands, G.J. (1984): The association between clinical mastitis and milk yield in dairy cows. *Anim. Prod.* 39:165-175.
- Lund, T.; Miglior, F.; Dekkers, J.C.M. and Burnside, E.B. (1994): Genetic relationship between clinical mastitis, somatic cell count, and udder conformation in Danish Holstein. *Livestock Prod. Sci.* 39(3):243-251.
- Ma, Y.; Ryan, C.; Barbano, D.M.; Galton, D.M.; Rudan, M.A. and Boor, K.J. (2000): Effects of somatic cell count on quality and shelf-life of pasteurized fluid milk. *J. dairy sci.* 83:264-274.
- Malinowski, E. (2000): The role of udder disinfection and sanitizer types. *Medycyna Weterynaryjna* 56(11):709-714.
- Malinowski, E. (2001): Somatic cells in milk. *Medycyna Weterynaryjna* 57(1):13-17.
- Mantere-Alhonen, S. (1995): The bovine udder and mastitis: Microbiology of normal milk. Univ. of Helsinki, Faculty of vet. Med. ISBN: 951-834-047-1 pp 115-141.
- Matthews, K.R., Harmon, R.J. and Langlois, B.E. (1992) : prevalence of *S. species* during the periparturient period in primiparous and multiparous cows, *J. dairy sci.* 75:1835-1839.
- Mazzucchelli, F.; Parrilla, G.; Blanco, F.J.; Martin, J.V. and Gonzalez, M. (2000): Bovine mastitis. An evaluation of problems on the farm. *Mundo-Ganadero.* 11(118):44-46.
- McDonald, J.S. (1982): Experimental infection of the bovine mammary glands during the dry period. *Proc. 21st Annu. Meet. Natl. Mastitis Coun., Louisville, KY. Natl. Mastitis Coun., Arlington, VA*, pp 112.
- Meaney, W. (1981): Mastitis level in spring-calving dairy heifers. *Ir. Vet. J.* 35:205-209.
- Michalcova, A.; Benczova, E. and Canigova, M. (1999): The influence of the manner of housing and milking on milk production and quality. *Acta Fytotechnica Zootechnica (Slovak republic).* 2(2):49-51.
- Milner, P.; Page, K.L. and Hillerton, J.E. (1997): The effects of early antibiotic treatment following diagnosis of mastitis detected by a change in the electrical conductivity of milk. *J. dairy sci.* 80:859-863.
- Moore, D.A. and O'Connor, M.L. (1993): Coliform mastitis: Its possible effect on reproduction in dairy cattle. *Proc. 32nd Annu. Meet. Natl. Mastitis Council; Kansas city, Mo. Natl. Mastitis council, Inc. Arlington, VA.* 162-166.
- Moore, D.A.; Cullor, J.S.; BonDurant, R.H. and Sischo, W.M. (1991): Preliminary field evidence for the association clinical mastitis with altered inter-estrus intervals in dairy cattle. *Theriogenology* 36:257-265.

- Morin, D.E. and Hurley, W.L. (1999): Mastitis lesson B. <http://classes.aces.uiuc.edu/AnSci308/mastitisb.html> (22.12. 1999).
- Mrode, R.A. and Swanson, G.J.T.(1996): genetic and statistical properties of somatic cell count and its suitability as an indicator means of reducing the incidence of mastitis in dairy cattle. *Anim. breed. Abstr.*64:847-857.
- Munch-Petersen, E.: mastitis in bovine primiparae. *Vet.res.*87:568-574.
- National mastitis council (1996): Current concepts of Bovine Mastitis. 4th ed. Natl. Mastitis Counc., Inc., Madison, WI.
- National mastitis council (1997a): A practical look at contagious mastitis. 1-4.
- National mastitis council (1997b): A practical look at environmental mastitis. 1-5.
- Natzke, R.P. (1981): Elements of mastitis control. *J. dairy sci.* 64:1431-1442.
- Neave; F.K., Dodd, F.H., Kingwill R.G., and Westgarth D.R. (1969): Control of mastitis in the dairy herd by hygiene and management. *J. dairy sci.* 52:696-707.
- Nickerson, S.C.; Owens, W.E. and Boddie, R.L. (1995): Mastitis in dairy heifers: initial studies on prevalence and control. *J. dairy sci.* 78:1607-1618.
- Norman, H.D.; Miller, R.H.; Wright, J.R. and Wiggans, G.R. (2000): Herd and state means for somatic cell count from dairy herd improvement. *J. dairy sci.* 83:2782-2788.
- Oleggini, G.H.; Ely, L.O. and Smith, J.W. (2001): Effects of region and herd size on dairy herd performance parameters. *J. dairy sci.* 84(5):1044-1050.
- Oliver, S.P. and Mitchell, B.A. (1983): Intra-mammary infection in primigravid heifers near parturition. *J. dairy sci.* 66: 1180 (quoted by Roberson et al.,1994. *J. dairy sci.*77:958-969).
- Oliver, S.P., Gillespie, B.E., Lewis, M.J., Ivey, S.J., Almeida, R.A., Luther, D.A., Johnson, D.L., Lamar, K.C., Moorehead, H.D. and Dowlen, H.H. (2001): Efficacy of a new premilking teat disinfectant containing phenolic combination for the prevention of mastitis. *J. dairy sci.* 84,1545-1549.
- Omoro, A.O.; McDermott, J.J.; Arimi, S.M. and Kyule, M.N. (1999): Impact of mastitis control measures on milk production and mastitis indicators in smallholder dairy farms in Kiambu district, Kenya. *Trop. Anim. Helth. Prod.* 31(6):347-361.
- Ostensson, K. (1993): trafficking of leukocytes and immunoglobulin isotypes in the bovine udder. Ph.D. Diss. Swedish Univ. Agric. Sci. Uppsala,Sweden. Cited by Schepers et al. (1997).*J. Dairy sci.* 80:1833-1840.
- Österås, O.; Edge, V.L. and Martin, S.W. (1999): Determinants of success or failure in the elimination of major mastitis organisms in selective dry cow therapy. *J. dairy sci.* 82:1221-1231.
- Owens, W.E.; Nickerson, S.C.; Boddie, R.L.; Tomita, G.M. and Ray, C.H. (2001): Prevalence of mastitis in dairy heifers and effectiveness of antibiotic therapy. *J. dairy sci.* 84(4):814-817.
- Pankey, J.W. (1989): Pre-milking udder hygiene. *J. dairy sci.* 72:1308-1312.

- Pankey, J.W.; Drechsler, P.A. and Wildman, E.E. (1991): Mastitis prevalence in primigravid heifers at parturition. *J. dairy sci.* 74:1550 (quoted by Roberson et al., 1994. *J. dairy sci.* 77:958-969).
- Pankey, J.W.; Nickerson, S.C.; Boddie, R.L. and Hogan, J.S. (1985): Effects of *Corynebacterium bovis* infections on susceptibility to major mastitis pathogens. *J. dairy sci.* 68:2684-2693.
- Pankey, J.W.; Pankey, P.B.; Barker, R.M.; Williamson, J.H. and Woolford, M.W. (1996): The prevalence of mastitis in primiparous heifers in eleven Waikato dairy herds. *New Zealand Veterinary J.* 44(2):41-44.
- Peeler, E.J., Green, M.J., Fitzpatrick, J.L., Morgen, K.L., and Green, L.E. (2000): Risk factors associated with clinical mastitis in low somatic cell count British dairy herds. *J. dairy sci.* 83: 2464-2472.
- Philipsson, J.; Ral, G. and Berglund, B. (1995): Somatic cell count as a selection criterion for mastitis resistance in dairy cattle. *Livestock Prod. Sci.* 41(3):195-200.
- Politis, I.; and Ng-Kwai-Hang, K.F. (1988a): Effects of somatic cell counts and milk composition on cheese composition and coagulating properties of milk. *J. Dairy sci.* 71:1711-1719.
- Politis, I.; and Ng-Kwai-Hang, K.F. (1988b): Association between somatic cell counts of milk and cheese yielding capacity. *J. Dairy sci.* 71:1720-1727.
- Pösö, J. and Mäntysaari, E.A. (1996): Relationships between clinical mastitis, somatic cell score and production for the first three lactations of Finnish Ayrshire. *J. dairy sci.* 79:1284-1291.
- Pyörälä, S. (1995): The bovine udder and mastitis: Mastitis caused by different microbes, Staphylococcal and Streptococcal mastitis. Univ. of Helsinki, Faculty of vet. Med. ISBN: 951-834-047-1 pp 143-160.
- Radostits, O.M., Leslie K.E. and Fetrow, J. (1994): Herd health: Food animal production medicine 2nd ed. W.B.Saunders co., Philadelphia, PA.
- Rainard, P.; Ducelliez, M. and Poutrel, B. (1990): The contribution of mammary infection by coagulase-negative Staphylococci to the herd bulk milk somatic cell count. *Vet. Res. Comm.* 14:193-198.
- Rajala-Schultz, P.J. and Gröhn, Y.T. (1999): Culling of dairy cows. Part 3. Effects of diseases, pregnancy status and milk yield on culling in Finnish Ayrshire cows. *Prev. Vet. Med.* 41(4):295-309.
- Rajala-Schultz, P.J.; Gröhn, Y.T.; McCulloch, C.E. and Guard, C.L. (1999): Effects of clinical mastitis on milk yield in dairy cows. *J. dairy sci.* 82:1213-1220.
- Reneau, J.K. and Packard, V.L. (1991): Monitoring mastitis, milk quality and economic losses in dairy fields. *Dairy, food, and Environ. Sanit.* 11:4-11.
- Risco, C.A.; Donovan, G.A. and Hernandez, J. (1999): Clinical mastitis associated with abortion in dairy cows. *J. dairy sci.* 82:1684-1689.

- Roberson, J.R.; Fox, L.K.; Hancock, D. D.; Gay, J.M. and Besser, T.E. (1994): Ecology of *Staphylococcus aureus* isolated from various sites on dairy farms. *J. Dairy sci.* 77:3354-3364.
- Rodenburg, J. (1990): Mastitis prevention: Environmental control: Ontario. Ministry of Agriculture and food Factsheet. AGDEX 410/662.
- Rodriguez, Zs.Sl.; Gianola, D. and Shook, GE. (2000): An approximate Bayesian analysis of somatic cell score curves in Holsteins. *Acta Agriculturae Scandinavica.-Section-A, Animal Science.* 50(4):291-299.
- Rodriguez, Zs.Sl.; Gianola, D. and Shook, GE. (2000): Evaluation of models for somatic cell score lactation patterns in Holsteins. *Livestock Production science* 67(1-2):19-30.
- Ruffo, G.; Sangiorgi, F.; Möller, F. and Gavazzi L. (1978): The influence of the animal's age and the period of lactation on the cell count of milk. *Arch. Vet. Ital.* 29:241.
- Rupp, R. and Boichard, D. (1999): Genetic parameters for clinical mastitis, somatic cell score, production, udder type traits, and milking ease in first lactation Holsteins. *J. dairy sci.* 82:2198-2204.
- Rupp, R.; Bertrand, C. and Bazin, S. (2000): Overview of milk somatic cell counts in French dairy cattle breeds. *Productions-Animales* 13(4):257-267.
- Saloniemi, H. and Kulkas, L. (2001): Mastitis control in Finland. *Inter. Dairy Fed. Bullet.* No. 367/2001, 13-17.
- Sandholm, M. and Korhonen, H. (1995): The bovine udder and mastitis: Antibacterial defence mechanisms of the udder. Univ. of Helsinki, Faculty of vet. Med. ISBN: 951-834-047-1 pp 37-48.
- Sargeant, J.M., Leslie, K.E., Shirley, J.E., Pulkrabek, B.J. and Lim, G.H. (2001): Sensitivity and specificity of somatic cell count and California mastitis test for identifying intramammary infection in early lactation. *J. dairy sci.* 84:2018-2024.
- SAS (1996): SAS user's guide: Statistics (version 6.12). SAS Inst. Inc., Cary, NC. USA.
- Schaeffer, L.R. and Solbu, H. (1987): Disease recording of dairy cows. *Holstein J.* (Sept., 1987).
- Schepers, A.J.; Lam, T.J.G.M.; Schukken, Y.H.; Wilmink, J.B.M. and Hanekamp, W.J.A. (1997): Estimation of variance components for somatic cell counts to determine thresholds for uninfected quarters. *J. dairy sci.* 80:1833-1840.
- Schrick, F.N.; Hockett, M.E.; Saxton, A.M.; Lewis, M.J.; Dowlen, H.H. and Oliver, S.P. (2001): Influence of sub-clinical mastitis during early lactation on reproductive parameters. *J. dairy sci.* 84:1407-1412.
- Schukken, Y.H.; Lam, T.J.G.M. and Barkema, H.W. (1997): Biological basis for selection on udder health traits. *INTERBULL Bull. No.15.Int.Bull Eval. Serv., Uppsala, Sweden.* 27-33.
- Schultze, W.D. (1985): Control of new intramammary infection at calving by prepartum teat dipping. *J. dairy sci.* 68:2094-2099.

- Seegers, J.; Fourichon, C.; Beaudeau, F. and Bareille, N. (1997a): Mastitis control programs and related costs in French dairy herds. Proc. 48th Annu. Meet. Europ. Assoc. Anim. Prod., Vienna, Austria. Wageningen Pers, Wageningen, The Netherlands. 143-146.
- Seegers, J.; Menard, J.L.; Dejean, O. and Weber, M. (1997b): Cell count evolution and clinical mastitis frequency in milk recording herds of the OPTILAIT area (South West). *Rencontres Rech. Ruminants* 4: 279.
- Seker, I.; Risvanli, A.; Kul, S.; Bayraktar, M. and Kaygusuzoglu, E. (2000): Relationship between California mastitis test (CMT) scores and udder traits and milk yield in Brown-Swiss cows. *Lalahan Hayvancilik Arastirma Enstitusu Dergisi* 40(1):29-38.
- Sheldrake, R.F. Hoare, R.J.T. and McGregor, G.D. (1983): Lactation stage, parity and infection affecting somatic cells, electric conductivity and serum albumin in milk. *J. dairy sci.* 66:542-547.
- Shook, G. and Ruegg, P. (1999): Geometric mean somatic cell count: what are they and what do they do? Proc. 38th Annu. Mtg. Natl. Mastitis Council, Arlington, VA. Natl. Mastitis Council, Inc., Madison, WI. 93-100.
- Shoshani, E. and Berman, A. (1998): Subclinical mastitis assessed by deviation in milk yield and electrical resistance. *J. dairy res.* 65(1):31-41.
- Shpigel, N.Y.; Winkler, M.; Ziv, G. and Saran, A. (1998): Clinical, bacteriological and epidemiological aspects of clinical mastitis in Israeli dairy herds. *Prev. Vet. Med.* 35(1):1-9.
- Smith, J.W. and Ely, L.O. (1997): The influence of feeding and housing systems on production, reproduction and somatic cell count scores of southern Holstein herds. *Prof. Anim. Sci.* 13:155-161.
- Smith, J.W.; Ely, L.O. and Chapa, A.M. (2000): Effect of region, herd size and milk production on reasons cows leave the herd. *J. dairy sci.* 83(12):2980-2987.
- Smith, K.L. and Hogan, J.S. (1995): Epidemiology of mastitis. Proc. 3th IDF. International mastitis seminar, Tel-Aviv, Israel, S6,3-13 (cited by Klaas, 2000: Untersuchungen zum Auftreten von Mastitiden und zur Tiergesundheit in 15 Milchviehbetrieben Schleswig-Holsteins. Dissertation, Free university-Berlin).
- Smith, K.L. and Hogan, J.S. (1998): Milk quality-a worldwide perspectives. Proc. 37th Annu. Mtg. Natl. Mastitis Council, St. Louis, MO. Natl. Mastitis Council, Inc., Madison, WI. 3-9.
- Smith, K.L., Todhunter, D.A. and Schoenberger (1985): Environmental pathogens and intra mammary infection during the dry period. *J. dairy sci.* 68:402-417.
- Solbu, H. (1983): Disease recording in Norwegian dairy cattle. 1. Disease incidence and non-genetic effects on mastitis, ketosis and milk fever. *Z.Tierz. Zuechtungsbiol.* 100:139-157.
- Suriyasathaporn, W.; Schukken, Y.H.; Nielsen, M. and Brand, A. (2000): Low somatic cell count: a risk for subsequent clinical mastitis in a dairy herd. *J. dairy sci.* 83:1248-1255.

- Thüringer Verband für Leistungs- und Qualitätsprüfung in der Tierzucht e.V. (2000): Jahres Bericht 2000.
- Todhunter, D.A., Smith, K.L., and Hogan, J.S. (1995): Environmental Streptococcal intra mammary infection of the bovine mammary gland. *J. dairy sci.* 78:2366-2374.
- Trifunovic, G.; Lazarevic, Lj.; Latinovic, D.; Stojic, P. and Stevanovic, Lj. (1998): Analysis of milk production and fertility of Black Pied cows in first three lactation in a free stall housing. *Savremena- poljoprivreda (Yugoslavia)*. No.1-2:49-54.
- Trinidad, P.; Nickerson, S.C. and Alley, T.K. (1990): Prevalence of intra-mammary infection and teat canal colonization in unbred and primigravid dairy heifers. *J. Dairy sci.* 73:107-114.
- Tucker, H.A. (1987): Quantitative estimates of mammary growth during various physiological states: a review. *J. dairy sci* 70:1958.
- Tyler, J.W.; Thumond, M.C. and Lasslo, L. (1989): Relationship between test-day measures of somatic cell count and milk production in California dairy cows. *Cand. J. Vet. Res.* 53(2):182-187.
- Van Asseldonk, M.A.; Huirne, R.B. and Dijkhuizen, A.A. (1998): Quantifying characteristics of information-technology applications based on expert knowledge for detection of oestrus and mastitis in dairy cows. *Prev.Vet. Med.* 36(4):273-286.
- Van Schaik, G.; Lotem, M. and Schukken, Y.H. (2002): Trends in somatic cell counts, bacterial counts and antibiotic residue violations in New York State during 1999-2000. *J. dairy sci.* 85(4):782-789.
- Vech, U.; Wisselink, H.J. and Defiz, P.R. (1989) : Dutch national mastitis survey. The effect of herd and animal factors on SCC. *Neth. Milk Dairy J.* 43:425-
- Waage, S., Mørk, A., Aasland, D., Hunshamar, A. and Ødegaard, S.A. (1999) : bacteria associated with clinical mastitis in dairy heifers. *J. dairy sci.* 82:712-719.
- Waage, S.; Sviland, S. and Odegaard, S.A. (1998): Identification of risk factors for clinical mastitis in dairy heifers. *J. dairy sci.* 81(5):1275-1284.
- Washburn, S.P.; White, S.L.; Green, J.T. and Benson, G.A. (2002): Reproduction, mastitis and body condition of seasonally calved Holstein and Jersey cows in confinement or pasture systems. *J. Dairy sci.* 85:105-111.
- Watts, J.L. (1988): Etiological agents of bovine mastitis. *Vet. Microbiol.* 16:41-66.
- Weller, J.L.; Saran, A and Zeliger, Y. (1992): Genetic and environmental relationships among somatic cell count, bacterial infection and clinical mastitis. *J. dairy sci.* 75:2532-2540.
- Wendt, K.; Bostedt, H.; Mielke, H. and Fuchs, H.W. (1994): Euter- und Gesäugekrankheiten. Gustav Fischer Verlag, Jena, Stuttgart (cited by Klaas, 2000: Untersuchungen zum Auftreten von Mastitiden und zur Tiergesundheit in 15 Milchviehbetrieben Schleswig-Holsteins. Dissertation, Free university-Berlin).
- Whitaker, D.A.; Kelly, J.M. and Smith, S. (2000): Disposal and disease rates in 340 British dairy herds. *Vet. Record.* 146(13):363-367.

- White, F. and McDonald, I. (1961): some observations on an outbreak of Staphylococcal mastitis in a dairy herd. *J. Comp. Pathol.* 71:159 (quoted by Roberson et al.,1994. *J.dairy sci.* 77:958-969).
- Wilesmith, J.W.; Francis, P.G. and Wilson, C.D. (1986): Incidence of clinical mastitis in a cohort of British dairy herds. *Vet. Record.* 118:199-204.
- Williams, D.J.; Marschke, R.J.; Nottingham, S.M. and Kitchen, B.J. (1991): Effects of stage of lactation, number of lactations and dry period on N-acetyl- β -D-glucosaminidase levels and somatic cell count in bovine milk. *Aust.J.dairy technol.* 46(1):43-45.
- Wilson, C.D. and Kingwill, R.G. (1975): A practical mastitis control routine. *Int. dairy Fed. Annual Bullet.* 85.
- Woodward, W.D.; Ward, A.C.S.; Fox, L.K. and Corbeil, L.A. (1988): Teat skin normal flora and colonization with mastitis pathogen inhibitors. *Vet. Microbio.* 17:357-365.
- Yalcin, C.; Stott, A.W.; Logue, D.N. and Gunn, J. (1999): The economic impact of mastitis-control procedures used in Scottish dairy herds with high bulk-tank somatic-cell counts. *Prev. Vet. Med.* 41(2-3):135-149.
- Zadoks, R.N.; Allore, H.G.; Barkema, H.W.; Sampimon, O.C.; Gröhn, Y.T. and Schukken, Y.H. (2001): Analysis of an outbreak of *Streptococcus uberis* mastitis. *J. dairy sci.* 84:590-599.
- Zepeda, L.; Buelow, K.L.; Nordlund, K.V.; Thomas, C.B.; Collins, M.T. and Goodger, W.J. (2000): Corrigendum to ‘‘A linear programming assessment of the profit from strategies to reduce the prevalence of *Staphylococcus aureus* mastitis’’. *Prev. Vet. Med.* 44:61-71.

Untersuchung zur Verbesserung der Eutergesundheit bei Färsen und Jungkühen

<p>1. Betriebsanschrift:-----</p> <p>EDV Betriebs-Nu. der MLP:-----</p> <p>3. B-Kühe (Stück 1998/99)-----</p> <p>5. Art der Bestandreproduktion</p> <table border="0" style="width: 100%;"> <tr> <td>Eigene Aufzucht</td> <td>ja/nein</td> <td></td> </tr> <tr> <td>Pensionstierhaltung</td> <td>ja/nein</td> <td></td> </tr> <tr> <td>Zukauf Färsen</td> <td>ja/nein</td> <td></td> </tr> </table> <p>6. Anzahl zugekaufter Färsen (1998/99):-----</p> <p>8. Hauptabgangsursache in % der abgegangenen Tiere aus MLP-Bericht (1998/99):</p> <p>-----</p> <p>9. Fütterungsverfahren (Zutreffendes ankreuzen):</p> <table border="0" style="width: 100%;"> <tr> <td>Mobile</td> <td><input type="checkbox"/></td> <td>stationäre</td> <td><input type="checkbox"/></td> </tr> <tr> <td>TMR</td> <td><input type="checkbox"/></td> <td>Grundration mit Kraftfutterautomat</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Ganzjährige Silage</td> <td><input type="checkbox"/></td> <td>Grünfütter im Sommer</td> <td><input type="checkbox"/></td> </tr> <tr> <td colspan="4">Weidegang</td> </tr> <tr> <td>Trockensteher</td> <td><input type="checkbox"/></td> <td></td> <td></td> </tr> <tr> <td>laktierende Tiere</td> <td><input type="checkbox"/></td> <td></td> <td></td> </tr> </table> <p>10. Stoffwechseluntersuchungen</p> <table border="0" style="width: 100%;"> <tr> <td>Regelmäßig</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Unregelmäßig</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Nein</td> <td><input type="checkbox"/></td> </tr> </table> <p>11. Haltungsform in Milchproduktion</p> <table border="0" style="width: 100%;"> <tr> <td>Anbindstall eingestreut</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Anbindstall Gummi/Gitterrost</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Laufstall-Spalten-Boxen eingestreut</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Laufstall-Spalten-Boxen Gummi/Asphalt</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Laufstall-Plan-Boxen eingestreut</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Laufstall-Plan-Boxen sonstiges</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Laufstall-tiefstreu</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Auslauf vorhanden</td> <td><input type="checkbox"/></td> </tr> </table> <p>13. Dauer der Melkzeit in Stunden</p> <table border="0" style="width: 100%;"> <tr> <td>Morgens-----</td> <td></td> </tr> <tr> <td>Abends-----</td> <td></td> </tr> </table> <p>16. Zwischendesinfektion des Melkzeuges:</p> <table border="0" style="width: 100%;"> <tr> <td>Back Flush</td> <td><input type="checkbox"/></td> <td>Sprühverfahren</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Air Wash</td> <td><input type="checkbox"/></td> <td>entfällt</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Wanne</td> <td><input type="checkbox"/></td> <td></td> <td></td> </tr> </table>	Eigene Aufzucht	ja/nein		Pensionstierhaltung	ja/nein		Zukauf Färsen	ja/nein		Mobile	<input type="checkbox"/>	stationäre	<input type="checkbox"/>	TMR	<input type="checkbox"/>	Grundration mit Kraftfutterautomat	<input type="checkbox"/>	Ganzjährige Silage	<input type="checkbox"/>	Grünfütter im Sommer	<input type="checkbox"/>	Weidegang				Trockensteher	<input type="checkbox"/>			laktierende Tiere	<input type="checkbox"/>			Regelmäßig	<input type="checkbox"/>	Unregelmäßig	<input type="checkbox"/>	Nein	<input type="checkbox"/>	Anbindstall eingestreut	<input type="checkbox"/>	Anbindstall Gummi/Gitterrost	<input type="checkbox"/>	Laufstall-Spalten-Boxen eingestreut	<input type="checkbox"/>	Laufstall-Spalten-Boxen Gummi/Asphalt	<input type="checkbox"/>	Laufstall-Plan-Boxen eingestreut	<input type="checkbox"/>	Laufstall-Plan-Boxen sonstiges	<input type="checkbox"/>	Laufstall-tiefstreu	<input type="checkbox"/>	Auslauf vorhanden	<input type="checkbox"/>	Morgens-----		Abends-----		Back Flush	<input type="checkbox"/>	Sprühverfahren	<input type="checkbox"/>	Air Wash	<input type="checkbox"/>	entfällt	<input type="checkbox"/>	Wanne	<input type="checkbox"/>			<p>2. A-Kühe (Stück 1998/99)-----</p> <p>4. Abkalbrate in % (1998/99)-----</p> <p>7. Anzahl abgegangener Kühe (1998/99)-----</p> <p>12. Melktechnik (Forms des Melkstands und Melkplätze):-----</p> <p>14. Anzahl Melker je Melkzeit:-----</p> <p>15. Art der Euterreinigung vor Melken-----</p> <p>17. Zitzendesinfektion:</p> <table border="0" style="width: 100%;"> <tr> <td>Dippbecher</td> <td><input type="checkbox"/></td> <td>Einsprühen im Melkstand</td> <td><input type="checkbox"/></td> </tr> <tr> <td>Sprühbalken</td> <td><input type="checkbox"/></td> <td>entfällt</td> <td><input type="checkbox"/></td> </tr> </table>	Dippbecher	<input type="checkbox"/>	Einsprühen im Melkstand	<input type="checkbox"/>	Sprühbalken	<input type="checkbox"/>	entfällt	<input type="checkbox"/>
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17. **Trockenstellen:**
- | | | |
|---------------------|------------------|--------------------------|
| Ohne Trockensteller | | <input type="checkbox"/> |
| Mit Trockensteller | alle Tiere | <input type="checkbox"/> |
| | BU positiv Tiere | <input type="checkbox"/> |
21. **Bewertung der Haltungsbedingungen in Ihrem Betrieb bezüglich des Auftretens subklinischer Masttiden:**
 Welcher Faktoren bereiten Probleme?-----

22. **Gibt es einen saisonalen Einfluss der Mastitiserkrankung in Ihrem Betrieb und welches sind die Problemmonate?**

19. **Euterbehandlungskartei;** ja/nein
20. **Art und Häufigkeit der Mastitiskontrolle:**
- | | |
|----------------------|--------------------------|
| Vormelkbecher | <input type="checkbox"/> |
| Mastitis-Schnelltest | <input type="checkbox"/> |

CURRICULUM VITAE

(2002)

Name: Abdelaziz A. **Fadlelmoula**

Birth date: 01.09.1963

Birth place: Barakat, Sudan

Nationality: Sudanese

Education:

1971-1977: Primary school, Umsunut, Sudan

1977-1980: Intermediate school, Umsunut, Sudan

1980-1983: Secondary school, Barakat, Sudan

1984-1989: Faculty of Veterinary science, University of Khartoum, Sudan

Qualifications:

B.V.Sc. (1989), University of Khartoum, Sudan

M.Sc. (Anim. Prod.) (1995), University of Khartoum, Sudan

Professional Records:

1989-1995: Vet. supervisor, private poultry farm, Shambat, Sudan

1990-1994: Teaching assistant, Dept. of Genetics and Breeding,
Faculty of animal production, University of Khartoum

1995-to date: Lecturer, Dept. of Genetics and Breeding,
Faculty of animal production, University of Khartoum

Publications:

Fadlelmoula, A.A.; Abunekhalia, A.M. and Yousif, I.A. (1998): Productive performance of the crossbred dairy cattle in Sudan I- Lactation curve and persistency. Proc. 8th Arab Vet. Conf. Khartoum-Sudan.

Yousif, I.A.; **Fadlelmoula, A.A.** and Abunekhalia, A.M. (1998): Productive performance of the crossbred dairy cattle in Sudan II- Lactional performance. Proc. 8th Arab Vet. Conf. Khartoum-Sudan.

ERKLÄRUNG

Hiermit erkläre ich, dass die vorliegende Arbeit selbständig und nur auf Grundlage der angegebenen Hilfsmittel und Literaturstellen verfasst zu haben.

Des weiteren erkläre ich, dass keine Strafverfahren gegen mich anhängig sind.

Halle/Saale, den 08.08.2002