CHAPTER I

INTRODUCTION

Mastitis is the most common and costly disease in dairy herds (Calhoun, 1995). It causes the greatest economic losses through decreased milk production, reduced milk quality and lower quality premiums (Philpot and Nickerson, 2000). Investigations have shown that a quarter suffering from subclinical mastitis losses on average 10 % of possible milk yield (Gietema, 2001). In addition, losses from mastitis are twice as high as losses from infertility and reproductive disorders (Philpot and Nickerson, 2000).

Mastitis is generally initiated by udder pathogens penetrating into the teat canal. After intramammary infection, the pathogens pass through the teat canal and multiply inside the infected quarter. The severity of mastitis varies widely, from subclinical to various forms of clinical disease (Philpot and Nickerson, 2000).

After intramammary infection, phagocytic cells in milk serve as a first line of defense against pathogens. They attack the pathogens and release chemoattractants such as cytokine to induce polymorphonuclear cells (PMN) accumulated in infected udder. The accumulated PMN merge as somatic cells. Approximately 99% of all cells in milk from an infected quarter are white blood cells, while the remaining are milk secretory cells that originate from the mammary tissues. These two types of cells make up the somatic cell count of milk (Philpot and Nickerson, 2000).

The increased SCC in milk and bulk milk tank is used to monitor and evaluate the successful of mastitis control program. SCC higher than 200,000 cells/ml indicates the infected status of cow (Suriyasathaporn, et al., 2000). The first barrier to prevent intramammary infection is an integumentary system. Teat end shape plays an important role in the prevention of bacterial access to the teat canal.

The teat canal is a natural barrier, but it is not always able to resist invasion by bacteria especially after milking. Factors such as the condition of the milking machine and how it is used are associated with damage of teat canal orifice and teat canal. Subclinical mastitis and teat canal infections can be mediated by milking equipment (Gietema, 2001). The milking machine has direct and indirect effects on the udder health (Rasmussen and Madsen, 2000.). To maintain healthy teats, it is necessary to maintain recommended milking machine function and operation.

However, veterinarians or agricultural farm advisors often have a limited knowledge necessary to diagnose correctly a herd mastitis problem caused by malfunction of milking machine and/or error on milking technique (Hogeveen, et al., 1995). The cause and effect of poorer teat condition have not been fully established and are likely to be multifactorial (Hillerton, et al., 2000). The slow progress is partly due to the complexity of mastitis which is caused by different pathogens, with a different epidemiology and, is required a different approach for control (Dodd and Booth, 2000.).

To date, report of risk factors associated with teat structure for high somatic cell count is limited and there are some published reports of risk factors due to milking machine for high SCC. Furthermore, there are no published reports on the effect of milking machine on SCC. The aim of the present study is to investigate the effect of

milking machine performances and teat structures on quarter somatic cell count in dairy cows kept in small holder dairy farms in Chiang Mai and Lamphun provinces.



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OBJECTIVES

To determine the effect of milking machine performances and teat structures on quarter somatic cells.

EDUCATIONAL ADVANTAGES

To obtain the effect of milking machine performances and teat structures on quarter somatic cells.

To apply the ultrasonographic technique for monitoring and diagnosing udder health research.

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