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THE RELATIONSHIP OF SELECTED COW AND MANAGEMENT FACTORS TO SOMATIC CELL COUNT OF DHI COMPOSITE MILK SAMPLES

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THE RELATIONSHIP OF SELECTED COW AND MANAGEMENT FACTORS TO SOMATIC CELL COUNT OF DHI COMPOSITE MILK SAMPLES

By

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ABSTRACT

THE RELATIONSHIP OF SELECTED COW AND MANAGEMENT FACTORS TO SOMATIC CELL COUNT OF DHI COMPOSITE MILK SAMPLES

By

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This study was designed to examine effect of cow related, farm, and managerial factors on SCC.

SCC increased as age, parity, stage of lactation, percent milk fat, and herd size increased. Daily milk production decreased as SCC increased. A seasonal peak in SCC occurred in June, with secondary peak in October.

Analysis of milking system found lowest SCC associated with high pipeline parlors, vacuum levels of 11.5 and 13.5 inches Hg for low and high pipelines, respectively, complete loop vacuum line design, single pulsation, and 50:50 pulsation ratio.

Milking practices associated with lowest SCC were: washing teats with individual paper towel using water containing sanitizer, drying with separate paper towel, prepping 20-30 seconds with machine attachment 30-60 seconds later, teat dipping, and rinsing teat cups between cows.

The effects of housing system, mastitic and dry cow treatment policies, herd replacement practices, and maternity facilities on SCC were also examined.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

INTRODUCTION

Mastitis, inflammation of the mammary gland, is one of the most critical health and economic problems facing the dairy industry. Mastitis affects approximately forty percent of the cows in an average herd causing a decrease in milk production, an increase in labor, veterinary, drug and herd replacement costs and, in some cases, even death of the dairy cow. Compared to normal milk, mastitic milk is higher in whey proteins, chlorides and somatic cells and lower in casein proteins, lactose and total solids. This difference in composition decreases processed product yield of mastitic milk by over three percent per hundred pounds milk (Everson, 1980), which in turn directly affects the profitability of a processing plant. Thus, reduction of the incidence of mastitis through detection of infected animals and implementation of mastitis control programs has the potential to increase incomes of both dairy farmers and processors while simultaneously improving milk quality for the consumer.

There are two major forms of mastitis. Clinical mastitis which affects two to three percent of the cows in an average herd and is visibly detectable and subclinical mastitis which affects thirty-seven to thirty-nine percent of the cows in an average herd and is not visibly detectable. Subclinical mastitis accounts for approximately sixty percent of costs or losses attributed to mastitis. Since clinical symptoms are only present in approximately fifteen

percent of the total population of infected cows, detection of subclinically infected cows is critical to the control and reduction of mastitis.

In recent years electronic counting of somatic cells has been shown to be a reliable and accurate measure of subclinical mastitis. Electronic cell counting as done by several dairy cooperatives on bulk tank milk samples and milk testing associations, such as DHIA, for each lactating cow in the herd gives each participating dairy farmer a somatic cell count at the time the monthly or bi-monthly sample was taken. This information gives dairy farmers a clearer picture of herd udder health.

In addition to subclinical mastitis, somatic cell count could be affected or influenced by age of cow, parity, stage of lactation, daily milk yield (production), weather factors, various dairy management factors (milking hygiene, milking equipment, housing, etc.) and genetics. Accounting for the various factors affecting somatic cell count makes it possible to compare the somatic cell count of one cow or herd to any other cow or herd.

The purpose of this study was to 1) determine the effect of a cow's age, parity, stage of lactation, milk production, percent fat of milk produced, herd size and time (season) of sampling on somatic cell count, and 2) to determine the effect of various farm conditions and managerial practices on somatic cell count.

REVIEW OF LITERATURE

Economic impact and prevalence of mastitis

Mastitis, inflammation of mammary tissue, continues to be one of the most critical health and economic problems confronting the dairy industry. Blosser (1979) estimated that in the United States mastitis reduced milk yields by 386 kg/cow/year and resulted in 62 kg milk discarded/cow/year. Additional economic losses due to mastitis included fees for veterinary services, drugs, increased labor, decreased sale value and increased replacement costs. In 1981, annual losses averaged $223.24 per cow (Kubik, 1982).

Mastitis also causes economic losses in dairy product processing plants. High cell count milk (1,000,000 or more cells/ml) causes significantly ($p < .01$) longer cheese manufacturing setting time, an 8% increase in fat loss in whey, a 1.5% increase in protein loss in whey and a 4% cheese yield decrease (Richter, 1976). Everson (1980) reported high cell count milk yielded approximately three percent less cheese per hundred pounds milk. Additionally fluid milk and processed products had an undesirable rancid flavor.

In an average herd there are three levels of mammary health. The uninfected or normal mammary gland, the inflamed mammary gland without clinical symptoms or subclinical mastitis and the inflamed infected mammary gland with clinical symptoms or clinical mastitis.

It is generally accepted that 37-39% of cows in an average herd are subclinically infected, with an additional 1-3% displaying clinical symptoms (Philpot, 1978; NMC, 1978). Both forms of mastitis result in significant production losses from affected quarters (Forster, 1964; Philpot, 1967; Meijering et al., 1978).

Characteristics of normal and infected mammary glands

Before a discussion of bovine mastitis begins, one must define the characteristics of the normal mammary gland, subclinically infected mammary gland and clinically infected mammary gland. The most important aspect of this discussion is determination of the point at which the normal mammary gland becomes inflamed and significant milk production losses begin to occur.

Normal or uninfected mammary gland

When physically palpated the normal mammary gland is soft and pliable having an even surface temperature with no swelling or hard masses within the gland. The secretion is usually a whitish fluid, but is not watery and contains no flakes, clots, blood or other abnormal particles. Chemically, normal milk is a complex mixture of fats, carbohydrates, proteins, minerals, vitamins and other miscellaneous constituents dispersed in water (Bath et al., 1978). Table 1 lists these constituents along with the normal variation present.

Normal milk also contains a low level of epithelial cells and leukocytes as a result of normal breakdown and repair of mammary tissue. Somatic cell count (SCC), total cell count or cell count

TABLE 1.--Gross Composition of Mixed Herd Milk.

Constituent	% Average Content	% Normal Variation
Water	87.2	82.4-90.7
Fat (Milk Fat)	3.7	2.5- 6.0
Solids-Not-Fat	9.1	6.8-11.6
Protein	3.5	2.7- 4.8
Casein	2.8	2.3- 4.0
Lactalbumins & lactoglobulins	0.7	0.4- 0.8
Lactose	4.9	3.5- 6.0
Minerals	0.7	0.6- 0.8
TOTAL SOLIDS	12.8	9.3-17.6

Source: Bath et al. Dairy Cattle: Principles, Practices, Problems Profits, 2nd ed., 1978.

used to define normal milk includes epithelial cells plus all types of leukocytes. Leukocyte count refers only to neutrophils or polymorphonuclear (PMN) leukocyte counts. The term somatic cell count (SCC) is currently preferred (Schalm and Lasmanis, 1968; Schalm et al., 1971).

Schalm et al. (1968) found foremilk somatic cell counts from healthy bovine quarters to be less than 100,000 cells/ml. During a three year study involving composite milk samples, Natzke et al. (1972) reported an average somatic cell count of 148,000 ± 16,167 cells/ml, 112,000 ± 40,345 cells/ml and 153,000 ± 62,619 cells/ml for uninfected first, second plus third and fourth lactation cows, respectively. Based on bucket samples from each milking over a one month period Schultz (1977) calculated a mean somatic cell for cows with no udder infection of 169,000 cells/ml. Most reports estimate 35 to 82% of somatic cells in normal milk are sloughed mammary epithelial cells (Blackburn, 1966; Schalm et al., 1968; Schalm et al., 1971; Duitschaever and Ashton, 1972; Schultz, 1977).

Physical changes associated with mammary infection or injury

The subclinically inflamed mammary gland cannot be distinguished from the uninfected or normal mammary gland by either physical palpation or gross examination of the milk. The only difference is the presence of bacteria in the subclinically infected mammary gland. Since mammary tissue has or is being damaged, changes in milk composition occur (Schalm et al., 1971; NMC, 1978; Philpot, 1978). The nature of these compositional changes and tests used to detect

them will be discussed more fully later. The somatic cell count of the subclinically infected quarter can range from 50,000 to 5,000,000 somatic cells or more/ml/milk. However at 400,000 cells/ml the probability of infection is greater than 80%. The majority of these cases are cows with subclinical mastitis (Eberhart et al., 1979). Over half of the economic loss attributed to mastitis is due to decreased milk production from subclinically inflamed mammary tissue or subclinical mastitis (NMC, 1978; Philpot, 1978; Blosser, 1979; Kubik, 1982).

When extensive damage to mammary tissue has occurred due to injury or bacterial infection, phagocytosis and degranulation reactions cause swelling and edema of the mammary gland which is characteristic of clinical mastitis. Clinically involved region of the mammary gland is red, hot, hard and sensitive to the touch when physically palpated (Schalm and Lasmanis, 1968; Schalm et al., 1971; Schultz, 1977; Bath et al., 1978; NMC, 1978; Philpot, 1978). The secretion is visibly altered due to destruction of secretory cells and presence of leukocytes and blood elements (Wheelock et al., 1966; Schalm and Lasmanis, 1968; Schalm et al., 1971; Schultz, 1977; NMC, 1978). The secretion is watery, yellow or pinkish in color containing flakes, clots and other abnormal particles (Schalm et al., 1971; Bath et al., 1978 NMC, 1978; Philpot, 1978). Clinically infected cows can be detected visually by the abnormal appearance of milk and udder.

Acute mastitis is characterized by rapid onset of clinical symptoms. In acute cases, the cow is bacteriologically positive and may be physically sick. Symptoms may include increased respiration,

rapid pulse, fever, anorexia, depression, lethargy and toxemia. The secretion, if any, can resemble either a thick pus or watery serum-like fluid (Schalm et al., 1971; Berrie, 1976; NMC, 1978).

Economic losses attributed to clinical mastitis include decreased production, discarded milk, drugs, veterinary fees, increased labor, decreased sale value and increased replacement costs. These costs account for approximately 40% of the total economic loss attributed to mastitis (Blosser, 1979; Kubik, 1982).

Milk yield from infected quarters has been shown to decrease 3-100% depending on severity of infection (Wheelock et al., 1966; Ward and Schultz, 1973; Meijering et al., 1978; Moxley et al., 1978; Philpot, 1978; Jones et al., 1982). This loss is not compensated for by increased production from uninfected quarters (Meijering et al., 1978). Wheelock et al. (1966) reported yield from infected quarters remained depressed throughout the remainder of the lactation even if the infection was eliminated spontaneously or as a result of antibiotic therapy. Ward and Schultz (1973) reported milk production losses increased as somatic cell count increased. Bodoh et al. (1976) found the correlation between cell count and test day milk yield was -.165. Moxley et al. (1978) reported a significant ($p < .01$) regression constant of milk yield (measured in kg) on somatic cell count (measured in 1,000 cells/ml) of $-.59 \pm .15$, or for each 100,000 cell/ml increase in cell count milk yield declines 59 kg (129.8 lbs).

Production losses have been shown to occur even at very low cell counts. Ward and Schultz (1973) found detectable production losses occurred before cell count was 500,000 cells/ml. Meijering

et al. (1978) and Philpot (1978) reported a three percent decrease in production when cell count was 100-225,000 cells/ml and a five percent decrease before cell count reached 500,000 cells/ml. Jones et al. (1982) reported that dramatic milk production losses occurred between 10,000 and 300,000 cells/ml. Cows with cell counts of 50,000 and 100,000 produced 4.1 and 2.8 pounds more milk per day respectively than cows with 300,000 cells/ml. Additionally they determined that lactation milk yield decreased seven to ten percent for cows with somatic cell counts up to 400,000 cells/ml.

Milk composition changes associated with mammary infection or injury

Changes in milk composition are mainly the result of decreases in total amount of milk components into milk (Schalm et al., 1971; NMC, 1978). Milk composition changes, their cause and percent change from normal are summarized in Table 2.

The major effects of mastitis, as defined by a somatic cell count of 1,000,000 cells/ml or greater, is an 18% decrease in casein synthesis and a 15% depression in lactose content (Everson, 1980). Despite the major decrease in casein synthesis, total protein remains relatively constant during mastitis as increased diffusion of whey protein (α - lactalbumin, β - lactoglobulin and serum albumins) and immunoglobulins from the blood compensates for decreased casein synthesis (Waite and Blackburn, 1957; Wheelock et al., 1966; Schalm et al., 1971; Anderson and Andrews, 1977; Schultz, 1977; NMC, 1978). The change in milk protein composition during mastitis results in an inferior milk protein product (Everson, 1980).

TABLE 2.--General Changes in Milk Composition Associated with Mammary Tissue Injury.

Constituent	% Average Content	% Percent of Normal	Cause
Solids-Not-Fat	8.5	93	↓ synthesis
Protein	3.6	100	
Casein	2.3	82	↓ synthesis
Whey	1.3	162	leakage from blood
Lactose	4.0	85	↓ synthesis or ↑ reabsorbsion
Chloride	.147	161	leakage from blood
Sodium	.060	136	leakage from blood
Potassium	.157	91	↓ synthesis or ↑ reabsorbsion
Total Solids	12.0	92	↓ synthesis

Source: Schultz, 1977, J. Food Protection.

In general, milk fat percentage remains relatively constant during subclinical mastitis, but is depressed during clinical mastitis. As mammary damage due to mastitis increases, total fat yield declines proportionally to milk yield (Waite and Blackburn, 1957; Wheelock et al., 1966; Schalm et al., 1971; Gard and Watkins, 1977; Schultz, 1977; Bath et al., 1978; Everson, 1980).

Sodium chloride leaks into milk from the blood during mastitis as a result of altered permeability due to tissue damage. This increase in sodium chloride levels in milk gives mastitic milk a salty flavor. Milk concentrations of potassium, calcium and phosphorus are also reduced from infected quarters (Wheelock et al., 1966; Schalm et al., 1971; Schultz, 1977; NMC, 1978).

During acute mastitis, a watery or serum-like secretion is produced. This secretion is very close to the composition of blood and no further normal milk secretion is produced within 24-48 hours of onset of acute mastitis (Berrie, 1976).

Role of leukocytes in mammary tissue

There are five distinct types of leukocytes: neutrophils, also called polymorphonuclear (PMN) leukocytes, eosinophils, basophils, lymphocytes and monocytes (Blackburn and McAdam, 1954; Waite and Blackburn, 1957; Schalm and Lasmanis, 1968; Paape, 1979; Paape et al., 1979). Each type has a different and specific function designed to protect the body against disease and injury. Neutrophils or PMN-leukocytes are the most important in protection and repair of the lactating mammary gland (Waite and Blackburn, 1957;

Schalm and Lasmanis, 1968; Schultz, 1977; Paape, 1979; Paape et al., 1979).

Neutrophils are produced in bone marrow by the process of extra vascular granulopoiesis and, in the bovine, mature in bone marrow for four to six days before being released into the blood stream by diapedesis (Schalm and Lasmanis, 1968; Schalm et al., 1971; Giesecke, 1979). In many species, PMN-leukocytes make up most of the leukocytes circulating in blood (Table 3). In the bovine, PMN-leukocytes make up only 25% of total leukocyte count in circulation. However, the mature lactating bovine has a potential pool of 100-500 billion circulating mature PMN-leukocytes (Schalm and Lasmanis, 1968; Paape, 1979). In addition to circulating PMN-leukocytes, a marginal pool of mature PMN leukocytes adheres to the walls of blood vessels and can increase circulating PMN-leukocyte level by 60% (Paape et al., 1979). There is also a large reserve pool of immature and mature PMN-leukocytes in bone marrow (Schalm and Lasmanis, 1968). The number of PMN-leukocyte storage pools that respond to an irritation in the mammary gland depends on the severity of irritation and the strength of the biochemical agent drawing PMN-leukocytes from the blood stream into the injured area (Paape et al., 1979). The supply of PMN-leukocytes for protection of the body is almost limitless (Schalm and Lasmanis, 1968).

The function of the PMN-leukocyte is to engulf and destroy bacteria, tissue debris, and any other "non-self" material (Schalm and Lasmanis, 1968; Schalm et al., 1971; Schultz, 1977; Paape, 1979; Paape et al., 1979; Harmon and Heald, 1979). "Non-self" material,

TABLE 3.--Comparative Blood Leukocyte Counts for Different Species.

Species	Total leukocyte count (cells/mm^3)	Mature PMN leukocyte %	Total No. Mature PMN leukocytes (cells/mm^3)
Man	7,780	47	3,600
Dog	11,500	61	7,000
Cat	12,500	60	7,500
Horse	9,000	52	4,700
Pig	16,000	29	5,500
Cow	8,000	25	2,000

Source: O. W. Schalm, N. C. Jain and E. J. Carroll. 1975. Veterinary Hematology. Pub. by Lea and Febiger, Philadelphia, PA.

is engulfed by phagocytosis, a process that can be enhanced by immunoglobulins acting as opsonins (Paape, 1979; Paape et al., 1979; Guidry et al., 1980). Phagocytosis begins when the PMN-leukocyte makes contact with the foreign particle. The foreign particle adheres to the PMN-leukocyte. A pseudopodia surrounds the particle and draws it into the cell's interior creating a phagosome. Inside the cell, cytoplasmic granules migrate toward and fuse their membranes with those of the phagosome to form a phagolysosome. The particle is then digested. No catabolic products leak out into the cytoplasm, which protect the leukocyte from self destruction (Paape, 1979; Paape et al., 1979). During this process, PMN-leukocytes become degranulated, releasing chemical substances that increase the permeability of capillary and secretory cells allowing escape of fluids and proteins of the blood into mammary tissues (Schalm and Lasmanis, 1968; Schultz, 1977).

When mammary tissue is damaged as a result of injury or bacterial infection, dead cells release chemicals which in turn attract PMN-leukocytes. The drawing of PMN-leukocytes from the blood stream into an area of injury is called "Chemotaxis" (Schalm and Lasmanis, 1968; Schalm et al., 1971; Schultz, 1977; NMC, 1978; Giesecke, 1979). The various chemotactic agents increase adherence of free circulating PMN-leukocytes onto capillary walls and may cause changes in both blood vessel cells and PMN-leukocytes to aid adherence and migration. PMN-leukocytes pass through capillary barriers of endothelium, periendothelium, and basement membrane by a mechanism that opens cellular junctions, accumulating in the inter

alveolar-capillary spaces (Harmon and Heald, 1979; Harmon, 1980). Harmon and Heald (1979) showed large numbers of PMN leukocytes lined up along alveolar basement membrane. If little or no damage had occurred, no PMN-leukocytes were found in the alveolar lumen. They concluded no transepithelial crossing occurred. In other areas of the udder where damage to milk synthesizing cells had occurred, PMN-leukocytes were found in various stages of crossing the alveolar basement membrane and epithelial layer into the lumen. They were never found transversing secretory cell tight junctions. Migrating PMN-leukocytes often aligned in an end to end fashion, as if following one another. When secretory cells were completely destroyed creating gaps in alveolar tissue, PMN-leukocytes migrated into the alveolar lumen en masse through the gap created. During experimental staphlococcus aureus mastitis, Harmon and Heald (1978) found evidence of PMN-leukocyte invasion in both teat and gland cistern and ductal epithelium. However, they were unable to determine to what extent these PMN-leukocytes contributed to elevated cell counts in milk.

Rapid elevation of somatic cell count as a first sign of mammary infection, occurring before other changes are evident, has been well defined and documented (Waite and Blackburn, 1957; Schalm and Lasmanis, 1968; Schalm et al., 1971; Reichmuth et al., 1974; Reichmuth, 1975; Westgarth, 1975; Bodoh et al., 1976; Schultz, 1977; NMC, 1978; Eberhart, 1979; McDermott et al., 1981; Jones et al., 1982). Quantification of cell types when somatic cell count increased showed the majority of increase was due to increased numbers of polymorphonuclear (PMN) leukocytes. There was practically no

increase in non-polymorphonuclear cell count (Waite and Blackburn, 1957; Blackburn, 1966). Reichmuth et al. (1974) found the range of cell count values between secretions from healthy and diseased quarters showed the greatest difference. The increase in cell count has been shown to be strongly influenced by type of bacteria causing infection (Postle et al., 1971; Ward and Schultz, 1973; Pearson and Greer, 1974; Booth, 1975; Heald et al., 1977). Westgarth (1975) reported bulk tank milk somatic cell count generally reflected the relationship of infected quarters and severity of infection in that herd. Eberhart et al. (1979) also found a significant difference between mean somatic cell counts of infected and uninfected cows, but the distribution of cell counts within categories substantially overlapped. They concluded that while it was not possible to define cell count levels that accurately discriminated between infected and uninfected cows, it was possible to determine probability of infection by either a major or minor pathogen based on a single cell count.

Reichmuth (1975), using communication engineering theory, showed somatic cell count to be an effective diagnostic tool allowing for a greater range of responses and thus providing more information. Ladaal (1976) stated somatic cell count could be used as a quality test for raw milk since physiological variation in counts as freshening and drying-off were insignificant to high counts found during mastitis. McDermott et al. (1981) showed somatic cell count to be highly sensitive with good specifity and predictive value. These authors reported a normal somatic cell count ranged from 0 to

199,000 cells/ml; 81% of infected cows had counts above this threshold, 74% of infection-free cows were below it, and predictive value of a negative test was .93.

Techniques for determination of somatic cell count

Somatic cell count can be determined using several different methods. Comprehensive reviews have been done by Giesecki and Van den Heever (1974) and Gordon et al. (1980). This discussion will be limited to Direct Microscopic Cell Count (DMSCC), California Mastitis Test (CMT), Wisconsin Mastitis Test (WMT), electronic somatic cell count (Coulter Counter®) and optical somatic cell count (Foss-O-Matic®) methods of cell counting.

Direct microscopic cell count

The direct microscopic cell count (DMSCC) was first described by Prescott and Breed in 1910, but has since undergone modifications and refinements to meet specific needs of investigators. DMSCC involves fixing 0.01 ml of milk to a glass slide, staining the sample and then counting viable nucleated cells. The DMSCC is usually considered the reference standard for other cell count methods for detecting abnormal milk. As a confirmatory test, it is relatively rapid permitting microscopic determination of somatic cells in 10 to 15 minutes (Ward and Postle, 1970; Schultze et al., 1971a; Schultze et al., 1971b; Ginn et al., 1973).

The DMSCC is not an absolute count, but an estimate of milk cell count (Read et al., 1971; Schalm et al., 1971). Care must be taken in collecting, storing and preparing milk films in order to

obtain an accurate estimate of somatic cell content (Brazis et al., 1968; Smith, 1969; Schalm et al., 1971; Gordon et al., 1980). Improper counting technique also can lead to erroneous results (Gordon et al., 1980).

California Mastitis Test

The California Mastitis Test (CMT) was derived from the modified Whiteside Test. The CMT is a quick, simple, efficient and inexpensive method of estimating somatic cell count (Pearson et al., 1970; Schalm et al., 1970; Gordon et al., 1980). CMT reagent contains 3% alkyl arylsulfonate and an indictor dye, bromcresol purple. The reagent causes cells to rupture, releasing deoxyribonucleic acid (DNA), which then reacts with the reagent to form a gel. The more somatic cells present in milk the greater the gel reaction. Gel formation can range from no change in appearance of 1 to 1 ratio milk-reagent mixture (few somatic cells) to slight slime to thick viscous gel (many somatic cells). If the indicator dye turns deep purple, the milk is distinctly alkaline. This may occur either as a result of inflammation or drying-off of gland. Acid milk causes the indicator to turn yellow, indicating fermentation of lactose by bacterial action within the gland (Schalm et al., 1971; Gordon et al., 1980).

Degree of reaction is scored negative, trace, 1, 2, or 3 for gel formation and + (deep purple) or y (yellow) if any indicator change occurs. Gel formation is not a precise indicator of somatic cell count since scoring is subjective and dependent on test operator's training and ability (Schalm et al., 1971) and CMT scores have overlapping somatic cell count ranges (Table 4).

TABLE 4.--Grading and interpretation of the California Mastitis Test*

Symbol	Suggested Meaning	Description of Visible Reaction	Interpretation**
-	Negative	Mixture remains liquid.	0-200,000 cells/ml. 0-25% PMN.
T	Trace	A slight slime forms and is seen to best advantage by tipping the paddle back and forth and observing the mixture as it flows over the bottom of the cup. Trace reactions tend to disappear with continued movement of the fluid.	150,000-500,000 cells/ml. 30-40% PMN.
1	Weak	A distinct slime but with no tendency toward gel formation. With some milks the reaction is reversible, for with continued movement of the paddle the slime may disappear.	400,000-1,500,000 cells/ml. 40-60% PMN.
2	Distinct positive	The mixture thickens immediately with gel formation. As the mixture is caused to swirl, it tends to move as a mass around the periphery of the cup, leaving the bottom of the cup exposed. When the motion is stopped, the mixture levels out again and covers the bottom of the cup.	800,000-5,000,000 cells/ml. 60-70% PMN.
3	Strong positive	A gel is formed which causes the surface of the mixture to become convex. Usually there is a central peak which remains projecting above the main mass after the motion of the paddle has been stopped. Viscosity is greatly increased so that there is a tendency for the mass to adhere to the bottom of the cup.	Cell number generally over over 5,000,000/ml. 70-90% PMN.
+	Alkaline milk pH 7.0 or over	This symbol should be added to the CMT score whenever the reaction is distinctly alkaline, as indicated by a contrasting deeper purple color.	An alkaline reaction reflects depression of secretory activity. This may occur either as a result of inflammation or in drying-off of the gland.
y	Acid milk	Bromcresol purple is distinctly yellow at pH 5.2. This symbol should be added to the score when the mixture is yellow.	Distinctly acid milk in the udder is rare. When encountered, it indicates fermentation of lactose by bacterial action within the gland.

* When in doubt as to the correct score of a reaction, always assign the lesser score thus indicating the weaker reaction between two choices.
**PMN = Polymorphonuclear leukocyte.

SOURCE: Schalm, O.W., E. J. Carroll and N. C. Jain, 1971. Bovine Mastitis. Lea and Febiger, Philadelphia.

Jensen and Beck (1969) reported correlations of .603 to .801 between CMT and DMSCC. Schultze et al. (1972) studying utility and costs of various screening tests found 98% of samples with cell counts greater than 1.5 million cells/ml were identified correctly as positive by CMT. However 68% of milk samples with cell counts less than 1.5 million were misidentified as being positive by CMT when they were actually negative by DMSCC. Collaborative studies have reported high variability of results between laboratories using CMT on a standard sample (Schultze et al., 1972; Gordon et al., 1980). CMT results are affected by type of sample taken (foremilk, composite or stripping) and age of sample (Wesen et al., 1968; Schalm et al., 1971). Tucker and Paape (1966) found a progressive decrease in CMT scores two days after collection.

The CMT is a good screening test to determine whether or not milk might have an unsatisfactory cell count, but due to its subjectivity and difficulty in standardization, regulatory laboratories and regulatory agencies must use other tests, such as DMSCC, to confirm suspicious or positive CMT results.

Wisconsin Mastitis Test

The Wisconsin Mastitis Test (WMT) uses CMT reagent diluted 1:1 with distilled water. It was designed as an objective test for laboratory use on bulk tank milk. Equal amounts of milk and reagent are added to a 12.5 x 125 mm test tube. Tubes are capped, shaken, allowed to stand for 30 seconds and then inverted for 15 seconds. The mixture flows through a hole in the cap. The amount of gel remaining after a 15 second inversion is measured (Schalm et al.,

1971; Gordon et al., 1980). Researchers have found correlations of .714 to .89 between WMT and DMSCC results (Read et al., 1969; Ward and Berman, 1971; Thompson et al., 1976). Schultze et al. (1972) found 89% of samples greater than 20 mm were identified correctly as having cell counts greater than 1.5 million cells/ml while only 17% of samples having cell counts less than 1.5 million cells/ml were misidentified as having cell counts above 1.5 million cells/ml.

However, WMT does have several disadvantages. WMT must be done on fresh milk samples and results can be affected by other factors, such as sanitizers, heat and storage time greater than 24 hours (Richter et al., 1968; Marshall and Brechbeehler, 1977; Gordon et al., 1980).

Electronic Cell Count - Coulter Counter®

Electronic cell counting, as performed by the Coulter Counter® (Coulter Electronics, Inc., Hialeah, FL), is based on the principle that as particles or cells pass through an aperture and displace an equal volume of electrolyte, electrical resistance is changed. The corresponding changes in current or voltage is directly proportional to the volumetric size of particle or cell. The number of voltage changes within a specific length of time is proportional to number of particles or cells within the suspension (Gordon et al., 1980). The Coulter Counter® thus measures size and number of particles simultaneously.

There are many models of Coulter Counters® available, each with specific operating and sample requirements. However regardless of model used, milk samples must be fixed prior to further processing or counting. Correlation of various counting methods with DMSCC range from .807 to .997 (Read et al., 1966; Read et al., 1967; Pearson et al., 1970; Philpot and Pankey, 1973; Newbould, 1974; Ward and Berman, 1971). Coulter Counter® results are 1/2 to 1/3 less variable than single strip DMSCC (Ginn et al., 1977). While the Coulter Counter® is faster, easier and just as accurate as DMSCC, its accuracy depends on proper sample preparation and handling. Any errors in sample preparation or handling will cause erroneous somatic cell counts to be obtained (Read et al., 1967; Sweetsur and Phillips, 1976; Newbould, 1974; Pearson et al., 1974; Greer and Pearson, 1976; Sheldrake et al., 1977; Dijkman et al., 1979).

Optical Cell Count - Foss-O-Matic®

Optical somatic cell counts, as determined by the "Foss-O-Matic®" (A/S N. Foss Electric, Hillerd, Denmark), determines numbers of somatic cells in milk by automated and continuously operating fluoresence microscopy. Ethidium bromide, a fluorescent dye, combines with DNA of somatic cells. When excited by light from a Xenon lamp, the complex emits fluoresence of a certain wave length. Somatic cells are automatically counted by a photomultiplier tube attached to the microscope. While other particles may also fluoresce, these particles will be of a different wave length. Thus, particles other than somatic cells can be filtered out (Grappin and Jeunet, 1974; Madsen, 1975; Andrews, 1977; Gordon et al., 1980).

The correlation between Foss-O-Matic results and DMSCC range between .922 to .996 (Grappin and Jeunet, 1974; Heeschen, 1975; Madsen, 1975; Heald et al., 1976; Heald et al., 1977; Mochrie and Monroe, 1978). Repeatability of somatic cell counts on a Foss-O-Matic ranged from .95 to above .99 (Madsen, 1975; Wilcox et al., 1976; Mochrie and Monroe, 1978). Foss-O-Matic coefficient of variation of 2.5 to 4.7 (Grappin and Jeunet, 1974; Mochrie and Monroe, 1978) is superior to coefficient variation obtained with Coulter Counter centrifugation method (8%) (Read et al., 1971), Coulter Counter chemical method (13.3%) (Thompson et al., 1976) and DMSCC (14 to 29%) (Mochrie and Monroe, 1978). However, Heeschen (1975) found precision of DMSCC, Coulter Counter and Foss-O-Matic were not significantly different.

Unlike Coulter Counter samples that can only be stored two days before cell counts begin to increase, reliable results can still be obtained up to fourteen days with the Foss-O-Matic when milk samples are preserved with 0.2% potassium dichromate ($K_2Cr_2O_7$) at collection and stored at 5°C (Andrews, 1977). The correlation between Foss-O-Matic results on milk samples after seven days storage at 5°C and DMSCC was .96 (Heald et al., 1977). Madsen (1975) showed addition of 0.05% potassium dichromate to milk samples did not influence cell counts. Røn (1976) using 0.025% potassium dichromate, found cell counts decreased 1-2% after four weeks of storage at 4-6°C. Cell counts had decreased 6-7% from initial values by eight weeks of storage at 4-6°C. Madsen (1979) studied the changes in cell count from 3 to 80 hours after collection in both unpreserved and preserved

samples. He found the final cell counts of preserved samples were increased 10% or less over non-preserved samples. The rise in cell counts was believed to be the result of potassium dichromate preservative diminishing the viability of the cells thus enhancing stainability and countability of cells. Despite the increased cell count in preserved samples during the first 24 hours after collection, Madsen concluded the increase was "of relatively little significance in the evaluation of individual samples."

Non-somatic cell count techniques for detection of subclinical mastitis

Regardless of the infection status of mammary gland, total protein remains approximately 3.6% and therefore, cannot be used to accurately detect any form of mastitis (Waite and Blackburn, 1957; Wheelock et al., 1966; Schalm et al., 1971; Anderson and Andrews, 1977; Schultz, 1977; NMC, 1978). However protein composition does change during mastitis: casein decreases and whey proteins (α - lactalbumin, β - lactoglobulin and serum albumin) increase. Casein levels do not change until the somatic cell count reaches 800,000 cells/ml (Waite and Blackburn, 1957) to 1,000,000 cells/ml (Schalm et al., 1971). This is in excess of the less than 200,000 cells/ml somatic cell count of normal uninfected mammary glands (Schalm et al., 1968; Natzke et al., 1972; Schultz, 1977) and the 500,000 cells/ml established by International Dairy Federation as dividing point between normal and subclinically infected glands. In addition, the range of the casein:total protein ratio ($X = \frac{100 \text{ (casein \%)}}{\text{total protein \%}}$) used to quantitate the compositional change has a

large overlap between normal and mastitic milk (Schalm et al., 1971; Schultz, 1977). Therefore casein levels cannot be used to detect subclinical mastitis because of 1) delayed response time from onset of inflammation and 2) lack of a clear demarcation line between normal and altered casein levels.

Investigators have tried to correlate increased whey proteins, especially bovine serium albumin (BSA), with severity of mastitis (Schalm et al., 1971; Dawson et al., 1974; Anderson and Andrews, 1977; Weaver and Kroger, 1977; Smith et al., 1979; Verhoeff and Smit, 1981). However many of the techniques used to detect whey proteins, such as immunodifussion and polyacrylamidegel or paper electrophoresis, have problems with quantification and resolution (Schalm et al., 1971; Anderson and Andrews, 1977; Smith et al., 1979; Weaver and Kroger, 1977; Verhoeff and Smit, 1981). Smith et al. (1979) using Laurell electrophoresis and Verhoeff and Smit (1981) using radial immunodiffusion tried to correlate milk BSA values and subclinical mastitis. These investigators found uninfected cows had low somatic cell counts and BSA levels of 0.20 mg/ml or less. Infected cows had significantly ($p = .015$) higher somatic cell counts but no significant ($p = .064$) BSA increase.

Lactose levels begin to decline when somatic cell counts rise above 100,000 cells/ml (Waite and Blackburn, 1957). Waite and Blackburn (1957) reported a 16% decrease in lactose levels as somatic cell count levels increased from 50,000 to 500,000 cells/ml. Detection of subclinical mastitis through changes in lactose concentration has several serious deficiencies. First the range

of values for normal and abnormal milk are too close (Reichmuth et al., 1974). Mayer (1979) found 251 of 714 cows were misdiagnosed based on lactose content. Secondly, lactose content is affected by factors other than infection or injury such as underfeeding, low protein rations and duration of lactation (Dawson et al., 1974; Mayer, 1979). Thirdly, natural changes in lactose content caused by age and stage of lactation are irregular and cannot be quantified into a regular or normal pattern (Dawson et al., 1974; Mayer, 1979). Finally, lactose content of milk increases slowly after infection or injury is cured or healed. However differences in lactose concentrations are very small (Bozhkova and Stoyanov, 1980). Thus lactose alone cannot be used to detect subclinical mastitis.

There is also a difference in mineral content between normal and mastitic milk. The most rapid and dramatic change is a 61% increase in chloride content (Schultz, 1977). Accurate detection of chloride change in milk is difficult since analytic techniques are not precise (Schalm et al., 1971). Additionally changes in sodium and potassium concentration are not precise enough to warrant use as indicators of mastitis.

Closely related to the change in mineral concentration is a change in electrical conductivity of milk. Research results on use of electrical conductivity as an indicator of mastitis have been mixed. Conductivity measurements have been reported to be of little diagnostic use (Little et al., 1968), less effective in detecting infected quarters as compared to normal quarters (Greatrix et al., 1968; Gebre-Egziabher et al., 1979) and an accurate method of

detecting infected quarters (Linzell et al., 1974; Linzell and Peaker, 1975; Duirs, 1980; Fernando et al., 1982). Conductivity measurements have been shown to be influenced by breed, temperature, diet, milking interval, stage of lactation, milk fat concentration, type of sample (foremilk, strippings or composite of quarter or udder) and whether or not the cow is in estrus (Greatrix et al., 1968; Linzell and Peaker, 1975; Fernando et al., 1981; Fernando et al., 1982). At this point, electrical conductivity is a promising indicator of mastitis but further work is needed to determine its practical feasibility.

Somatic cell count has been shown to be a more reliable indicator of mammary status than casein (Waite and Blackburn, 1957; Schalm et al., 1971), whey proteins, especially bovine serum albumin (BSA) (Smith, 1979; Verhoeff and Smit, 1981), lactose (Bozhikova and Stoyanov, 1980) and mineral content (Schalm et al., 1971).

Factors affecting somatic cell count

Somatic cell count has been shown to be affected by factors other than infection. These factors can be divided into three broad categories; cow related factors, environmental factors and managerial-farm condition factors.

Cow related factors affecting somatic cell count

Cow age, stage of lactation, milk production (yield) and genetic make-up have been shown to affect somatic cell count.

Most researchers report increased cell numbers as age increased (Blackburn, 1966; Schalm et al., 1968; Duitschaeven and Ashton, 1972; Natzke et al., 1972; Schultz, 1977; Eberhart et al., 1979; Rindsig et al., 1979). These authors hypothesized that the cell count increase related to age was a result of current mammary inflammation or previous infection. Duitschaeven and Ashton (1972) and Natzke et al. (1972) observed that mean somatic cell counts of cows which remained uninfected during lactation and dry period were only slightly but not significantly ($p < .05$) increased in the subsequent lactation.

Ruffo et al. (1978) studied a 170 head commercial Holstein dairy herd for two years. They found average somatic cell count for first and second lactation cows was less than 100,000 cells/ml but cows with more than two lactations had an average cell count of 270,000 cells/ml. Kennedy et al. (1978) studying 29,629 Holstein cows in 728 commercial Canadian dairy herds reported a linear increase in somatic cell count with age of approximately 92,000 cells/ml/year. Syrstad et al. (1979) studied approximately 700 cows in 70 herds (2,570 samples). They found cell counts were strongly influenced by age, increasing approximately linearly with increasing cow age. Age effect on cell count was not related to infection status at time of sampling. However, Syrstad et al. (1979) report did not study infection status throughout the entire lactation. Short term infections could have influenced cell counts related to age. Titterton and Oliver (1979a, 1979b) studied 481 commercial dairy herds in Zimbabwe (approximately 50,000 cows). They found a significant ($p < .01$ for linear model; $p < .05$ for quadratic model) increase in somatic cell

count with increasing age. These authors attributed the cell count increase to exposure to infections and subclinical mastitis. Syrstad and Røn (1979) studied sources of variation in 2,570 milk samples from 765 cows collected bimonthly over a seven month period. They found differences among age groups to be highly significant ($p < .01$). They also found adjusting cell count for yield increased variation due to age.

A rise in somatic cell count during late lactation has been attributed to increased mammary tissue breakdown as the gland prepares to become nonfunctional (Schultz, 1977), a concentration effect due to decreased milk production with a stable leukocyte count; especially, from the third lactation onward (Schalm et al., 1968), or increased infection rate (Blackburn, 1966; Natzke et al., 1972; Eberhart et al., 1979).

Blackburn (1966), studying one herd over a twelve year period, found the increase in somatic cell count during late lactation was primarily, but not exclusively, due to an increase in polymorphs-leukocytes that respond to injury or infection. Ruffo et al. (1978) also found a rise in somatic cell count after the fourth month of lactation. Research done by Natzke et al. (1972) and Eberhart et al. (1979) supported the hypothesis that the somatic cell count rise during late lactation resulted from increased infection rate. Natzke et al. (1972) reported 225 cows with no history of infection did not show a significant ($p < .05$) rise in somatic cell count during late lactation. Eberhart et al. (1979) found cell count varied irregularly until after day 300 of lactation, when it began to rise. The rise

was directly associated with increased infection rate at that time.

The hypothesis that increased somatic cell count during late lactation is due to increased sloughing of epithelial cells and involution of mammary tissue as well as possible infection was supported by Schultz (1977). Blackburn (1966) does lend some support to Schultz's position, since he demonstrated cell types other than polymorphs also increased during late lactation. However, Natzke et al. (1972) has shown uninfected cows do not have a significant ($p < .05$) rise in somatic cell count in late lactation as would be expected if sloughed epithelial cells and mammary involution were major factors in the late lactation rise of somatic cell count.

Schalm et al. (1968) and Schalm et al. (1971) did not statistically support their hypothesis that the late lactation rise was the result of a concentration affect due to declining milk production (yield). Kennedy et al. (1978) observed peak production corresponded to lowest cell count. However, Syrstad et al. (1979) did the required statistical analyses. They adjusted somatic cell count for the effects of age of cow and level of production (yield). They found the adjusted somatic cell count decreased with advancing lactation.

There are investigations indicating heredity may have some influence on somatic cell count in milk and incidence of mastitis. Lush (1950) studied the mastitis susceptibility of daughters of 214 dams classified as susceptible (mastitis prior to eight years of age) and 280 dams classified as resistant (no mastitis prior to

eight years of age) which were distributed in 27 herds. Lush (1950) using intra herd regression of daughters on dams determined heritability of mastitis equal to .38 with a 95% confidence interval range of .06-.07. He concluded inheritance played a part in susceptibility to mastitis but his data set was too small to provide an accurate estimate that part within narrow limits. Afifi (1967; 1968b) using 1,491 paternal daughters of 35 Dutch Friesian sires estimated heritability of clinical mastitis to be 0.12 ± 0.06 by half-sib correlation. He also found no obvious relationship between somatic cell count and milking ease. In a different study, Afifi (1968a) using 799 first calf-daughters of 20 Dutch Friesian sires estimated heritability of somatic cell count to be 0.14 with a repeatability of 0.28 by parental half-sib correlation and intraclass correlation coefficients, respectively. These values are in agreement with previous findings (Afifi, 1967; Afifi, 1968b). However when studying 692 fourth lactation daughters of 15 sires, Afifi found heritability to be 0.37 with a repeatability of 0.40. These values are in agreement with Lush's (1950) estimate which was obtained by a different method from Afifi. However, it must be noted that fourth lactation heritability may be inflated due to selection against high somatic cell count cows during the first three lactations.

Wilton et al. (1972) determined heritability of infection by daughter-dam regression and sire. Heritability using daughter-dam regression for 605 pairs of first lactation cows in 186 herds was determined to be 0.14 ± 0.10 and 0.16 ± 0.08 for 854 pair of late lactation cows in 289 herds. These values are similar to values

obtained by Afifi (1967, 1968a, 1968b). However Wilton et al. (1972) did not find an increase in heritability with advanced lactation number as Afifi (1968a) had. Heritability of infection by sire groups was estimated as 0.00 for first lactation cows (9,177 records, 620 herds, 379 sires) and 0.11 for later lactation cows (15,292 records, 638 herds, 398 sires). Regression of later lactation infections on first lactation infections (2,672 cows in 364 herds) gave a coefficient of 0.18, indicating infection may be repeatable, but cause may not be genetic (Wilton et al., 1972).

Environmental factors affecting somatic cell count

The "comfort zone", environmental temperature interval during which no demands are made on an animal's temperature regulation mechanism(s) is -1.1 to 15.6°C (30-60°F) for European dairy cattle. A rise in environmental temperature above 15.6°C (60°F) increases respiration and vaporization rates. Thermoregulation mechanisms begin to fail at 26.7°C (80°F) which results in an increase in rectal temperature, decrease in feed consumption, decline in milk production and weight loss. Temperatures of 42.2°C (108°F) or above can be lethal (Brody, 1956; Bath et al., 1978).

Effect of thermal stress

Effects of thermal stress on milk somatic cell count has been studied by several investigators. Two main methods are used to study the effect of thermal stress on somatic cell count; environmental chamber and actual seasonal environmental stress.

Effect of thermal stress using environmental chambers

Using ten Holstein cows Roussel et al. (1969) studied the effect of various temperatures between 15.5 and 29.5°C (59.9-85.1°F) at 65% humidity. Cows were housed in environmental chambers at each of the selected temperatures for seven days. Roussel et al. (1969) found no evidence of increased somatic cell count with increased thermal stress. An environmental chamber study by Paape et al. (1973) used five or six cows in the control and each treatment group. Cows were either bacteriologically negative or positive for Corynebacterium bovis. No distinction was made between the two cow populations during analysis of data. The inclusion of Corynebacterium bovis infected cows with non-infected cows was considered justified by the authors since 1) Corynebacterium bovis infected quarters repond like non-infected quarters and 2) Corynebacterium bovis, in the authors' view, was not a mammary pathogen. Treatment groups were exposed for two or four weeks to either a constant 32°C (89.6°F) or fluctuating 32°C (89.6°F) day or 21°C (69.8°F) night and then compared to a control group held at 20°C (68°F). Humidity was 65% for all groups. Paape et al. (1973) found no significant increase ($p < .05$) in milk somatic cell count with thermal stress. However it should be noted that the control group used for comparison was also stressed (20°C or 68°F) vs maximum comfort zone limit of 15.6°C (60°F).

Wegner et al. (1976) stressed four aged cows with a history of mastitis in an environmental chamber at 40-48°C (104-118.4°F) and 70-80% humidity for 24 hours. No significant change in milk somatic

cell count was noted. However Wegner's study had two major flaws. First, temperatures used were near lethal limit of 42.2°C (108°F), so thermal regulation mechanisms were at or very near complete collapse. Second, sampling was done for only 48 hours after the stress. Simensen (1974) showed milk somatic cell counts did not fully respond until three to four days after initial stress. Brown et al. (1977) found variable milk somatic cell counts inconsistent in four dairy cows housed in environmental chambers and exposed to a series of temperature changes. Temperature pattern was 21-28°C (69.8-82.4°F), -16°C (3.2°F), 21-28°C again, 36-37°C (96.8-98.6°F) and 21-28°C again. Each temperature was maintained for five days. Brown et al. (1977) believed inconsistencies observed (somatic cell count up in some cows and down in others at same temperature) were the result of the organism present in the udder before the trial began, since all four cows carried a different pathogen. Also, the author's control temperature (21-28°C or 69.8-82.4°F) to which treatment temperatures were compared were heat stressed.

In summary, environmental chamber studies to data cannot be used to determine the effect of heat stress on milk somatic cell count since most studies, except Roussel et al. (1969), used heat stressed control groups.

Effect of seasonal temperature stress

European descended dairy cattle are rather tolerant of cold temperatures. However more extensive protection is needed during

hot weather. Methods to protect dairy animals from heat include conduction (water), convection (air) and vaporization (increasing air velocity from .5 to 5 miles per hour). These methods decreased heat stress in the range of 23.9-35°C (75-95°F) (Brody, 1956).

Nelson et al. (1969) studying 64 dairy cows from June to November found highest somatic cell counts occurred during periods of thermal heat stress. However, individual quarters responded differently which may indicate the manifestation of a stress condition may be determined by factors operative in individual quarters of cows. Paape et al. (1973) observed an increase in somatic cell counts in a dairy herd from May to November in two consecutive years. These researchers also observed an increase in the number of clinical cases treated during this period. They concluded that the rise in somatic cell counts due to heat stress resulted from subclinical infections becoming clinical. However Paape et al. did not speculate as to why the rate of clinical mastitis increased during May to November.

Wilton et al. (1972) had previously shown in a seven year study that season-year had no effect on udder infection. Simensen (1974) conducted a four year study using approximately 3,500 dairy cows in a cold, relatively dry region of Norway having a short summer pasture season. September to July cows were housed indoors. During this period there was no correlation between udder infection and any environmental parameter studied. However during July to September summer pasture season, there was a significant ($p < .05$) positive correlation of 0.4 between udder infection and air temperature. In a later study Simensen (1976) found somatic cell counts during

three periods of the summer (June 30th, July 21st, July 29th) were significantly ($p < .05$, .001 and .005, respectively) higher for cows on pasture than cows housed indoors during those same periods. The number of quarters infected in pasture groups decreased but somatic cell counts of uninfected quarters increased. Simensen observed diurnal variations in environmental temperatures were considerably greater for pasture cows, which were also subject to radiant heat, than for housed cows. He concluded that the majority of somatic cell count increases in the pasture group was due to increased cell counts of uninfected quarters which may not be directly related to air temperature *per se*. However Pearson and Mackie (1979) reported clinical mastitis cases occurred three times more frequently in winter, when cows were housed, than in summer, when cows were on pasture.

Wegner et al. (1975) studied 64 cows in Arizona from June to November. They found that as the temperature-humidity index increased; somatic cell counts significantly ($p < .05$) increased. Kennedy et al. (1978) studying 29,629 cows in 728 herds found herd-season and age-season interactions to be highly significant ($p < .01$). Syrstad and Røn (1979) found season was a highly significant ($p < .01$) source of somatic cell count variation and variation increased if somatic cell count was adjusted for yield.

In summary, thermal stress does cause elevation of somatic cell counts under field conditions. However thermal stress may not be the only environmental factor to affect somatic cell count.

Effect of other environmental factors

Limited work has been done correlating environmental factors other than thermal stress and somatic cell counts. Simensen (1974) looked at relationship of barometric pressure, humidity and precipitation to SCC during a four year study of dairy cows in Norway. He found no correlation between udder infection and either barometric pressure or humidity. However there was a significant ($p < .05$) positive correlation of 0.3 between udder infection and precipitation. Titterton and Oliver (1979) also observed a seasonal trend possibly related to precipitation. They observed somatic cell counts were higher during the rainy season (November to March) than during the dry season (June to October). Thus, precipitation appears to influence somatic cell count, however more work is needed.

Effect of various farm conditions

Farm conditions include herd size, type of housing, bedding material used and type of milking equipment. The effect of each condition on somatic cell count will now be discussed.

Effect of herd size

Only limited work has been done on effect of herd size on somatic cell count. Afifi (1967) found effect of herd size on somatic cell count was inconsistent. Afifi concluded herd size seemed to have little effect on somatic cell count. In a later study, Afifi (1968d) found herd size had no effect on somatic cell count. Schultz (1977) reviewing the results of several studies in

which herd size ranged from 11 to 220 cows also concluded herd size had no effect on somatic cell count. Syrstad and Røn (1979) found only 13% of total variation in somatic cell count could be ascribed to variation among herd size.

Effect of type of housing

Dairy animals are housed in many types of housing systems; stanchion, tie stall, enclosed warm or cold free stall, partially enclosed loose housing, open lots, etc. Adequate housing implies 1) light, airy draft free buildings, 2) stalls of adequate size, 3) adequate bedding, 4) potentially daily removal of manure, and 5) exercise lots maintained free of wire, stones, sharp objects, mud, standing water, etc. However limited work has been done in this country on how various housing systems affect somatic cell count.

Poor lighting conditions have been shown to adversely affect milking hygiene practices and increase udder and teat injuries (Saloniemi, 1980). Poor ventilation has been shown to increase surface humidity and drafts. These factors contribute in an increase in mastitis (Simensen, 1974; Saloniemi, 1980). These conditions also seem to be associated with occurrence and prevalence of teat lesions. Teat lesions are frequently colonized by staphylococcus and Streptococcus dysgalactiae. When teat lesions are prevalent, high new infection rates and frequent clinical cases of mastitis occur (Carroll, 1977; Farnsworth and Sieber, 1980).

Adequate stall size is an important factor in reducing teat and udder injury (Carroll, 1977; Bakken, 1980; Saloniemi, 1980;

Klastrup, 1981). Bakken (1980) reported incidence of clinical mastitis significantly ($p < .05$) decreased as stall length increased, but there was no significant effect on subclinical mastitis. Klastrup (1981) found incidence of subclinical mastitis was significantly ($p < .01$) greater with stall lengths less than 170 cm. Width of stall and presence or absences of partitions has, in general, been shown to have no effect on incidence of either subclinical or clinical mastitis (Carroll, 1977; Saloniemi, 1980). However Klastrup (1981) reported significantly ($p < .01$) more subclinical mastitis with stalls less than 100 cm wide.

Widespread use of loose housing systems has lead to research on effect of crowding on cell count. Arave et al. (1974) studied the effect of crowding on SCC using 17 Holstein cows. Cows were first housed in a 20 stall freestall shed with 9.3 m^2/cow lot space. Cows were observed and milk samples taken for two weeks. After two weeks, lot space was reduced to 2.3 m^2/cow for an additional two weeks. Arave et al. (1974) found cows restricted to 2.3 m^2 space/cow compared to 9.3 m^2 space/cow, had lower leucocyte counts and fewer herdmate encounters. These authors concluded that crowding did not stress cows since there were stable social groups.

Farm sanitation is designed to reduce probability of cow's teat coming into contact with high concentration of mastitis causing organisms that could penetrate past streak canal, gain access to the gland and may cause mastitis.

Very little work has been done comparing housing types Ekesbo (1966) found lower incidence of mastitis with soft bedded

loose-housed cows than either hard bedded loose-housed cows or tie stall cows. However Kingwell et al. (1977) found variation within a housing system was greater than variation between housing systems. Therefore, they concluded researchers needed to be careful when condemning one type of housing and favoring another.

Effect of bedding type

Many types of bedding are currently used on dairy farms including chopped straw, corn cobs, corn stalks, hay, long straw, separated manure solids, sand, sawdust, wood chips (shavings) and various combinations. Stall bases include cement, cement plus rubber mats, crushed limestone, packed clay, etc.

Sawdust bedding has been incriminated as a source of Klebsiella pneumoniae mastitis in high producing dairy cows. During 1972, Klebsiella pneumoniae was isolated in 40% of samples obtained from Michigan sawmills and storage bins (Newman, 1973). Apparently, green sawdust bedding is contaminated with klebsiella organisms when logs are skidded through the woods prior to debarking.

Bramley (1974) reported Klebsiella pneumoniae infection occurred when its population in bedding was greater than 10^6 organisms per gram wet bedding but not when population was less than 10^5 per gram wet bedding. Bramley (1974) also reported a direct relationship between bedding population, teat apex contamination and new infection rate. Most sawdust bedding samples contain 10^4 to 10^5 coliforms per gram wet bedding (Carroll and Jasper, 1980). Coliform counts in fresh sawdust bedding can rapidly increase from 10 to 10^7 coliforms

per gram wet bedding within one week when added to stalls (Carroll and Jasper, 1980).

Eberhart (1975) and Rendos et al. (1975) have compared sawdust to sand, straw and wood chip beddings. Rendos et al. (1975) found significantly ($p < .05$) more Klebsiella pneumoniae in sawdust bedding than in sand. Straw beddings and wood shavings or chips have also been shown to contain significantly ($p < .05$) less Klebsiella pneumoniae than sawdust bedding. The increased bacteria in sawdust was attributed to smaller particle size, greater surface area and greater capacity for water retention. Additionally, more organisms were recovered from sawdust bedded cows than from either straw or wood chip bedded cows (Eberhart, 1975; Rendos et al., 1975). Eberhart (1975) and Rendos et al. (1975) believed this was influenced by the tendency of small sawdust particles to adhere to the teat skin; especially, in the teat orifice. Newman et al. (1973) observed the incidence of klebsiella mastitis decreased from 54% to 9% when sawdust bedding was replaced with either sand or straw. Newman (1975) later observed incidence of klebsiella mastitis increased after inclement weather when bedding was changed from one type of sawdust to another or from old sawdust to fresh sawdust.

Wood chips are also contaminated with klebsiella and other coliforms species but to a lesser extent than sawdust (Eberhart, 1975; Rendos et al., 1975; Carroll, 1977). However staphylococci organisms are significantly ($p < .05$) lower in bedding and on teat skin with wood chips than either sawdust or straw bedding (Rendos et al., 1975). Carroll (1977) suggested use of kiln-dried wood chips may be practical,

but warned against using any wood chips that might be mechanically irritating to udders, teats or teat ends due to hardness or sharp corners.

Straw when amply (> 1 kg/day) used has been shown to reduce incidence of clinical mastitis and facilitate maintenance of clean dry stalls (Saloniemi, 1980). However Rendos et al. (1975) had shown straw contained a large population of streptococci and staphylococci per gram of straw.

Use of recycled manure solids as bedding material is of special interest to western states because of expense of traditional (sawdust, straw, wood chips, etc.) bedding materials. Carroll and Jasper (1980) reported dried or composted-dried manure was an adequate bedding material if it was kept dry and not damp with urine and feces. However good composting temperatures were only reached in the interior of the manure pile (Carroll and Jasper, 1980). Allen et al. (1980) conducted two separate studies on the practicality of recycled manure solid bedding. During the first studies, freestall bedding was changed from sawdust to recycled manure solids at 20% dry matter. Over the next year, CMT scores increased, incidence of clinical mastitis increased and coliform mastitis increased from 7 to 46 percent of total cases. However, occurring simultaneously with bedding change was a change in milking equipment, milking hygiene and dry cow treatment. Due to extent of mammary health problems, cows were rebedded on sawdust. Two years later Allen et al. (1980) conducted a more controlled study using recycled manure solid as freestall bedding. In this study, manure solids were composted for six weeks

prior to use. This procedure increased dry matter from 20 to 60 percent and eliminated all coliform organisms from outer 48 inches of compost pile. Allen et al. (1980) found no significant differences between sawdust and recycled manure solid bedding.

Effect of milking system

The milking machine comes in direct contact with the cow's teats two or more times daily. Improper functioning or use of the milking system has been suggested as preconditioning the cow to teat injury, to increased incidence and spread of mastitis and to higher somatic cell counts.

There are three general types of milking systems; bucket, barn pipeline and parlor pipeline. Saloniemi (1980) found incidence of mastitis was significantly ($p \leq .05$) higher with bucket systems than pipeline systems. Downey et al. (1977) found the average somatic cell count for bucket systems to be highest (393,000 ± 17,700 cells/ml) and parlor systems in general to be lowest (359,000 ± 23,500 cells/ml). Barn pipeline milking systems which were the most numerous type of system (76.4% of total) were associated with intermediate cell counts (385,000 ± 8,300 cells/ml). Schultz (1977) also found that parlor milking systems were associated with lower cell counts than other milking systems. Cows milked in a low line (pipeline located below udder) milking parlor had the lowest cell count compared to other parlors. Cows milked with barn pipeline systems were found to have highest somatic cell counts. Schultz believed the higher somatic cell counts for around the barn pipeline and high line parlor systems were due to the required elevation of milk in these systems.

McDonald (1971) had shown high milk pipelines (greater than five feet above udder) resulted in excessive vacuum fluctuations at teat end. High pipelines also require a higher preset vacuum level to maintain good milk flow rate. The higher vacuum level could increase probability of teat and streak canal injury at end of milking and, thus, increase probability of infection.

Downey et al. (1978) reported milking systems less than five years old had lower somatic cell counts than systems of greater age. Saloniemi (1980) found a significant ($p \leq .05$) correlation of 0.41 between milking machine age and incidence of mastitis. He calculated an increase of 0.012 cases/cow/year for each yearly increase in milking machine age.

Expansion of a milking system seems to increase somatic cell count. Downey et al. (1977) studied 26 progressive Canadian dairy herds. They found average somatic cell counts of expanded milking systems to be 394,000 ± 14,500 cells/ml and 381,000 ± 8,300 cells/ml for systems still operating at their original size. In a larger study, it was found expanded systems had a higher mean somatic cell count (530,000 cells/ml) than systems still operating at their original size (430,000 cells/ml) (Downey et al., 1978).

An increasing number of milking systems are being automated. Philpot (1973) reported CMT and microbe content of milk decreased and physical condition of teat ends improved with addition of Surge QTO® automatic take-off devices. By preventing overmilking, milking after milk flow is less than ½ pounds milk per minute, teat injuries and incidence of new infection are reduced (Mochrie et al., 1953a;

Mochrie et al., 1953b; Petersen, 1964; McDonald, 1971; Natzke et al., 1976). However Natzke et al. (1978) found no significant ($p > .05$) increase in new infections with overmilking.

Various milking system conditions influence teat injury and incidence of mastitis, including status of vacuum pump, vacuum level vacuum controller, vacuum line, pulsator, claw and inflation (liner). Saloniemi (1980) reported a highly significant ($p < .001$) increase in teat injury and incidence of mastitis as an increasing number of these conditions varied from manufacturer's specifications.

Vacuum level and vacuum stability have been associated with incidence of mastitis. As vacuum level increases the amount of teat damage increases resulting in an increased infection rate and elevated somatic cell count (Afifi, 1968c; McDonald, 1975; Nicolai et al., 1977; Noorlander, 1977; Galton and Mahle, 1980; Saloniemi, 1980). Static vacuum between 25.4 and 33.0 cm Hg (10-13 inches Hg) is best for minimizing teat trauma during milking (McDonald, 1971; Galton and Mahle, 1980). McDonald (1971, 1975) reported increased injury to both teat end and streak canal and teat congestion when vacuum at teat end exceeded 33 cm Hg (13 in Hg). Nicolai et al. (1977) reported no significant ($p = .01$) differences between cows milked at 10 and 12.5 inches Hg vacuum. However cows milked at 15 inches Hg vacuum had significantly poorer teat condition and higher CMT and DMSCC scores. Afifi (1968c) reported a significant ($p < .01$) increase in somatic cell count when vacuum levels exceeded 40 cm Hg (15.75 in Hg). Highest somatic cell counts occurred when vacuum levels were 55 cm Hg (21.65 in Hg). Saloniemi (1980) also reported a significant ($p < .05$)

increase in incidence of clinical mastitis when vacuum levels were greater than 50.7 kPa (14.91 inches Hg). However Mochrie et al. (1953a) and Mochrie et al. (1953b) reported no significant effect of vacuum levels of 10, 13.5 and 17 inches Hg on leukocyte count, presence of mastitis organisms or milk production. The effects of high vacuum level are more pronounced when cows are simultaneously overmilked (Afifi, 1968c; McDonald, 1975). Vacuum levels below recommended levels have also been shown to significantly ($p \leq .001$) increase teat injury (Saloniemi, 1980).

Any interference with free air flow in a milking system between the teat end and vacuum pump can cause vacuum fluctuation at the teat end during milking. Thiel et al. (1973) found vacuum fluctuation either at teat end or between shell and inflation (liner) of teat cups or large cyclic fluctuations did not cause a significant ($p > .05$) increase in infection, but vacuum fluctuation at teat end or between shell and inflation combined with large cyclic fluctuations significantly increased infections ($p = .01$ with constant pulsation rate, $p = .001$ with fluctuating pulsation rate).

Pressure changes within the milking system can cause bacterial laden milk droplets to be introduced into the teat during milking and thus increase risk of mastitis (Thompson, 1980; Noorlander, 1977; O'Callaghan and O'Shea, 1979). To prevent teat or streak canal injury and related increased udder infection, vacuum levels should not fluctuate more than 7.6 cm H (3 in. Hg) at teat end during maximum milk flow when the system is fully loaded (McDonald, 1971).

The vacuum controller (regulator) is closely associated with vacuum level and fluctuation. The function of the vacuum controller is to maintain preset vacuum level at a designated location in the milking system throughout almost the entire range of the vacuum pump's capacity to remove air. Smith and Fairbank (1975) compared spring loaded diaphragm, weighted sleeve value and weighted lever controllers. The latter two controllers were found to be adequate if maximum load changes were of low magnitude (± 10 cfm or less). Spring loaded diaphragm controllers were found to be more sensitive and able to handle load changes of large magnitude (± 20 cfm or more). However, increasing pump capacity does not compensate for a poor controller, nor does a more sensitive controller reduce pump requirement (Smith and Fairbank, 1975). Poor vacuum controller performance significantly ($p \leq .05$) increases clinical mastitis (Saloniemi, 1980).

Vacuum reserve capacity is recommended to be at least 50% above actual measured air requirements (McDonald, 1971). Inadequate effective vacuum reserve leads to irregular vacuum fluctuations and increased mammary infection (McDonald, 1975). Klastrup (1969) reported infection level decreased from 49 to 16 percent as vacuum reserve per unit increased from 0 to greater than 5.4 CFM free air. However Maatje and Rossing (1971) and Saloniemi (1980) found no relationship between cell count or incidence of clinical mastitis and vacuum reserve capacity.

Pulsation rate and ratio also effect somatic cell count and incidence of mastitis. Milne (1977) reported milking systems with operational faults, but with properly functioning pulsators, had

bulk tank milk cell counts 14% higher than completely efficient milking systems. Milking systems with faulty pulsators had 30% higher bulk tank milk cell counts than completely efficient milking machines. Afifi (1967, 1968c) showed pulsation rates greater than 50 cycles/minute or less than 44 cycles/minute significantly ($p < .05$) increased somatic cell count. McDonald (1971, 1975) reported pulsation rates of 40-60 cycles/minute were adequate with 60 cycles/minute being the most economical. However Saloniemi (1980) found pulsation rate had no effect on incidence of mastitis.

Galton and Mahle (1980) examined the interaction of pulsation ratio and vacuum level. Pulsation ratio of 60:40 (60% milking phase to 40% massage phase) was associated with lowest somatic cell count across all vacuum levels (10 inches (25.4 cm) Hg, 12.5 inches (31.75 cm) Hg and 15 inches (38.1 cm) Hg). A 50:50 ratio resulted in increased incidence of teat injury but not infection rate.

Pulsation ratios of 70:30 (70% milking phase to 30% massage phase) or greater increased teat injury and congestion, infection rate and somatic cell count (McDonald, 1971; McDonald, 1975; Galton and Mahle, 1980). These conditions are more severe at vacuum levels greater than 13 inches (33 cm) Hg. Galton and Mahle (1980) hypothesized the detrimental effects of 70:30 pulsation ratio were due to inadequate length of massage phase causing incomplete filling of the test cistern, undue stress on teat tissue and forcing of pathogens through the streak canal. Hoare et al. (1979) reported somatic cell count significantly ($p < .05$) increased as proportion of massage phase of cycle decreased and milking phase increased. Somatic cell count

increase was attributed to increased injury and spread of infection during long milking phase. Saloniemi (1980) found as milking phase increased there was a significant ($p \leq .05$) increase in teat injury.

Recent reports have implicated inflation (liner) design, material of construction, speed of closure and opening, and admission of air between inflation and teat (slip) as major causes of intramammary infections. Improper inflation design influences teat damage and erosion, teat massage, flooding of milk, milking efficiency and contamination of teat orifice (McDonald, 1971; Noorlander, 1977). There are two basic inflation types, narrow bore and wide bore. Narrow bore inflations (3/4 inch or less in diameter) have been shown to significantly ($p < .01$) reduce udder irritation, lower bacterial infection, significantly ($p < .05$) reduce clinical mastitis and amount of machine stripping and milking time when compared to wide bore inflations (Dillion et al., 1969; McDonald, 1971). However inflation size must match shell size; i.e. wide bore inflations used with wide shells, narrow bore inflations used with narrow shells.

Scanning electron microscopy has revealed the material from which the inflation is constructed is important. At 1,000 milkings the inner surface of rubber inflations contain deep cracks and caverns. By 5,000 milkings bacteria were common in these openings. However Silastic® inflations remained smooth after 1,000 milkings and only began to show signs of wear, but no bacterial contamination, at 5,000 milkings (Heckman and Noorlander, 1980; Noorlander and Heckman, 1980).

The speed of inflation closure and opening has been shown to be partly responsible for impinging of milk backward against the teat (Noorlander, 1977). Rapid closing and opening may also accentuate large cyclic vacuum fluctuations increasing incidence of infection (Thiel et al., 1973).

Admission of air between inflation and teat (liner slip) is associated with 50-60% of new infections (O'Shea et al., 1979). O'Shea et al. (1979) compared Gascoigne single stretch inflations with Alfa-Laval Liner 960000-1. They reported significantly ($p < .05$) more infection with Gascoigne single stretch. They concluded the higher infection rate with those inflations was due to greater liner slippage causing milk droplet impact on teat ends. Westgarth (1977) had been able to reduce new infections by 96% by using shields within short milk tubes during a short duration trial. However, new infections were only reduced 10% during a six month field trial.

O'Callaghan and O'Shea (1979) and O'Shea et al. (1979) reported impacts arising from slip were not prevented by shields, large claws or changes in pulsation characteristics. Thompson (1980) also found no significant effect of claw size on impacts. However venting of claw decreased impacts due to improved emptying of claw and constant out flow toward milk line (Thompson, 1980).

Effect of milking hygiene practices

Milking hygiene practices have been associated with both new infection rate and somatic cell count. Early reports advocated full milking hygiene which included washing the udder with either separate

paper towels or separate boiled cloth plus disinfectant solution, milkers wearing rubber gloves, pasteurization of teat cup clusters between cows and teat dipping immediately after milking. Full milking hygiene has been shown to reduce new infection rate 58%, infections due to Staphylococcus aureus 62% and infections due to streptococci 70% compared to limited hygiene (washing udders with common cloth using only water) (Neave et al., 1969). Previously Neave and Oliver (1962) reported washing udders with a common cloth significantly increased the chance of recovering organisms from teat skin. Schultz (1977) found use of individual paper towels during udder washing lowered somatic cell count. However Moxley et al. (1978) reported use of individual paper towels had no effect on somatic cell count.

Washing the udder with soap or sanitizer and water has been shown to significantly ($p < .05$) lower bulk milk somatic cell count (Hoare et al., 1979), but later work showed pre-milking washing with either water or water with 2% chlorhexidine solution did not significantly ($p > .05$) reduced new mammary infections (Sheldrake and Hoare, 1980a; Sheldrake and Hoare, 1980b).

Drying of teats and udders after washing has been recommended, but the effect of this practice has not been clearly determined. Moxley et al. (1978) reported udder drying with separate paper towel had the second greatest effect on lowering cell counts (-44,000 cells/ml). Hoare et al. (1979) found udder drying increased somatic cell count. However in this study drying was done with a cloth or sponge in a weak disinfectant, not individual paper towels.

Neave et al. (1969) and Moxley et al. (1978) reported pasteurization or disinfection of teat cup clusters between cows did not affect new infection rate or somatic cell count. Bushnell et al. (1978) reported addition of backflushing in a commercial dairy gave a rapid decrease in clinical mastitis and eliminated mycoplasma mastitis, but over a two year period subclinical mastitis increased from 20% to 40% due in part to failure of the backflush system to maintain 25 ppm iodine concentration in the sanitation cycle.

Machine stripping has not been shown to significantly increase somatic cell counts (Goff and Schmidt, 1967; Afifi, 1968c).

Teat dipping has been shown to have the greatest effect on reducing new infection rate and somatic cell count. Neave et al. (1969) reported teat dipping resulted in a seven-fold decrease in new streptococcus infections during a nine week period. Langlois and Pyles (1975) reported use of the commercial teat dip Bovadine® and Chlorox Liquid Bleach® (4% chlorine) showed Chlorox® treated group had 8% fewer clinical mastitis cases and fewer Staphylococcus aureus infections (Grant et al., 1976). Teat dipping with 5,000 mg available iodine/liter has been shown to significantly ($p < .05$) reduce new mammary infections and reduce both Staphylococcus aureus population on teat ends and new infections but was not effective against Streptococcus dysgalactiae (Sheldrake and Hoare, 1980a; Sheldrake and Hoare, 1980b). Moxley et al. (1978) showed teat dipping significantly ($p = .01$) lowered somatic cell count by

70,300 cells/ml. Hoare et al. (1979) also showed teat dipping significantly ($p < .05$) lowered bulk milk somatic cell count.

The bovine udder is most susceptible to infection at the beginning of the dry period mainly because bacteria are able to penetrate the streak canal more easily (Cousins et al., 1980). Dry treating, infusion of oil-based antibiotics into mammary gland at cessation of daily milking, has been shown to reduce new infection rate and cure existing infections. Oliver et al. (1962) reported dry treating resulted in very nearly complete protection against staphylococcus and streptococcus infections. Rindsig et al. (1978) reported a 3.1% new infection rate for complete dry treatment and 6.5% for selective dry treatment program. This compares to 10 to 15% new infection rate without dry therapy (Natzke, 1971). Dry treatment has been shown to eliminate 85.4% to 100% of existing infections depending on type of drug, type of infectious organism and whether therapy is complete or selective (Meaney and Nash, 1977; Rindsig et al., 1978).

Use of a lactating cow product for dry treatment is not as beneficial. In comparing lactating and dry cow products used at drying-off, Philpot (1973) found dry cow products had a 2.24 times greater cure rate against staphylococcus and 1.09 times greater against streptococcus than lactating cow products.

Use of teat dipping and dry cow treatment has been shown to be more beneficial than either practice alone. Cows which were both teat dipped and dry treated had 20.2% fewer Streptococcus agalactiae infected quarters, 6.6% fewer Staphylococcus aureus infected quarters

and a 1.03 new infections per cow per year compared to 1.50 for the control group which were dry treated only (Eberhart and Buckalew, 1972). The former group also had lower WMT scores after freshening. Schultz (1977) reported herds that dry treated only had higher somatic cell counts than any other combination of dry treatment and teat dipping. Schultz (1977) concluded that dairy farmers were attempting to control mastitis with dry treatment alone, without other good management practices. Hoare et al. (1979) reported bulk milk somatic cell counts were significantly ($p < .05$) lower for farms teat dipping and dry treating all cows than farms using any other combination of teat dipping and dry cow treatment.

MATERIALS AND METHODS

A series of models were constructed to determine effect of various bovine age, parity, stage of lactation, milk production, and milk fat production (measured in percent), environmental, and managerial factors on somatic cell counts (SCC).

The first model constructed determined the effect of various bovine and seasonal factors on somatic cell count (SCC). Cow age, parity, stage of lactation, and test day milk production, percent fat, and somatic cell count were obtained in cooperation with Michigan Dairy Herd Improvement Association (DHIA) for all cows in Michigan enrolled in the somatic cell count option from November, 1978 through January, 1981. SCC was determined using a Foss-O-Matic® manufactured by A/S N. Foss Electric, Denmark. The model, based on this information, was:

$$Y = \mu + Herd + Cow + Milk + Lac + Dim + Mo + Fat + Age + Hsize + e$$

where:

Y is natural log of reported somatic cell count

μ is population mean

Herd is herd effect

Cow is cow effect

Milk is milk production effect

Lac is parity effect

Dim is stage of lactation (days in milk) effect

Mo is seasonal (month of sample) effect

Fat is percent fat effect

Age is cow age effect

Hsize is size of herd effect

e is random error effect

All factors except error were fixed.

The second series of models were constructed to determine the effects of various managerial practices and farm conditions on somatic cell counts (SCC). It was determined that the most cost efficient method of obtaining information concerning managerial practices and farm conditions would be by a series of survey questionnaires. A series of survey questionnaires were developed concerning milking procedures and practices, milking equipment, milking herd management, dry cow practices, and calving practices (Appendix A). The questionnaires were checked for clarity, accuracy, lack of bias in either questions and/or responses and ability to code and analyze data obtained from the survey. Necessary modifications were made prior to distribution of any of the questionnaires. Due to increased postal rates and budgetary constraints, it was not possible to mail survey questionnaires and a preaddressed stamped return envelope directly to dairy farmers enrolled in the SCC option. After consultation with representatives of parties involved, it was decided to have DHIA technicians distribute the questionnaires to dairy farmers enrolled in the SCC option on test day. Completed questionnaires were picked-up and returned either that day or during the subsequent

test period. Individual technicians were given an information packet explaining the research program, a copy of each questionnaire, and a copy of an explanatory letter to be mailed to dairy farmers enrolled in SCC option (Appendix B). This information packet was given to them during a meeting held during March, 1980. Prior to distribution of survey questionnaires by DHIA technicians, dairy farmers enrolled in the SCC option were directly mailed a one-page letter briefly explaining the questionnaires and requesting the dairy farmer to complete and return questionnaires as soon as possible (Appendix C). The first set of questionnaires also contained a release form to permit use of the dairy farmer's DHIA records (Appendix D). Questionnaires were distributed and returned between April and June, 1980.

Questionnaire response rates were milking practices 43.7%, milking systems 43.4%, milking herd management policy 35.2%, dry cow and calving practices 35.1%, and housing 35.0%. Despite the decline in response rate, it is still much higher than accepted range of 10-15% for questionnaires of this type. As questionnaires were returned, they were coded onto computer sheets and later keyed into the main computer.

Prior to analysis, it was necessary to transcribe the data because of computer coding differences between DHIA's Honeywell system and Michigan State University's Control Data Corporation's 1600. Due to a DHIA computer operator error, addition of protein to reports, and a change in DHIA report forms, DHIA data was found to be written in three distinct formats. To facilitate analysis, a single format was developed and data reformated.

Actual recorded somatic cell counts, in 100,000 cells/ml, plus one were transformed into natural logarithms (Log_e). This transformation was used because of its proven linearity, equality of mean and median, normal distribution, uniform variance, and mean in midscale characteristics. Detailed discussions of somatic cell count transformations are found in Ali and Shook (1980) and Shook (1982).

Analysis of all models was done by Statistical Analysis System (SAS) GLM (General Linear Models) with absorption of herd and cow within herd effects. Absorption removed effect of these factors from the analysis. Since all farm condition and management practice models were based on results of a single survey conducted over a three month period, it was decided to use mean somatic cell counts, mean daily milk production, and mean days in milk during analysis based on these models. All models were checked for normality of errors and variance. Intrafactor comparisons within each model were done using Bonferroni-t test.

Due to the broad scope of information gathered, it was not possible to construct a single model including all farm conditions and managerial practices of interest to determine their effect on somatic cell count. Thus a series of models were used. The first series of models looked at effect of farm conditions on somatic cell count. Farm conditions were divided into two categories; milking system and housing system. In the milking system models, the symbols were defined as follows:

Y = estimated effect on log of mean somatic cell count

Mlk = mean daily milk production effect

Mdim = mean days in milk effect

Lac = parity effect

Sys = milking system effect

Equip = milking system equipment brand effect

Lineht = milk pipeline height effect

Vaclev = line vacuum level effect

Vaccon = brand of vacuum controller effect

Ldsgn = vacuum line design effect

Lmat = material of vacuum line construction effect

Unit = number of milking units effect

Pipedi = milk pipeline diameter effect

CFM = CFM per milking unit effect

Pultyp = pulsation type effect

Pulrat = pulsation ratio effect

Infla = inflation brand effect

Auto = type of milking system automation effect

e = random error effect

The milking system models were:

$$Y = Mlk + Mdim + Lac + Sys + Equip + e$$

$$Y = Mlk + Mdim + Lac + Sys + Lineht + Sys(Lineht) + e$$

$$Y = Mlk + Mdim + Lac + Sys + Lineht + Vaclev + Lineht(Vaclev) + e$$

$$Y = Mlk + Mdim + Lac + Sys + Lineht + Vaccon + Sys(Lineht) + Sys(Vaccon) + Lineht(Vaccon) + e$$

$$Y = Mlk + Mdim + Lac + Sys + Ldsgn + Lmat + Ldsgn(Lmat) + e$$

$$Y = Mlk + Mdim + Lac + Sys + Unit + Pipedi + Unit(Pipedi) + e$$

$$Y = Mlk + Mdim + Lac + Sys + CFM + e$$

$$Y = Mlk + Mdim + Lac + Sys + Pultyp + Pulrat + Pultyp(Pulrat) + e$$

$$Y = Mlk + Mdim + Lac + Sys + Pulrat + Vaclev + Pulrat(Vaclev) + e$$

$$Y = Mlk + Mdim + Lac + Sys + Infla + e$$

$$Y = Mlk + Mdim + Lac + Sys + Auto + e$$

In each of these milking system models all factors except error were fixed.

In housing system models, the symbols were defined as follows:

Y = estimated effect on log of mean somatic cell count

Mlk = mean daily milk production effect

Mdim = mean days in milk effect

Lac = parity effect

House = housing system effect

Bed = bedding material effect

Mat = maternity facility effect

Matbed = maternity facility bedding effect

Wcalf = fall to spring calving conditions effect

Scalf = spring to fall calving conditions effect

e = random error effect

Housing system models were:

$$Y = Mlk + Mdim + Lac + House + e$$

$$Y = Mlk + Mdim + Lac + House + Bed + House(Bed) + e$$

$$Y = Mlk + Mdim + Lac + House + Mat + Matbed + Mat(Matbed) + e$$

$$Y = Mlk + Mdim + Lac + House + Wcalf + Scalf + Mat + Matbed + Wcalf(Mat + Matbed) + Scalf(Mat + Matbed) + e$$

In each of these housing system models all factors except error were fixed.

Management practices were divided into five categories; milking hygiene practices, treatment of mastitic cows, dry cow treatment policy, free stall maintenance, and purchase of replacement animals. Symbols used in the management practices models were defined as follows:

Y = estimated log of mean somatic cell count

Mlk = mean daily milk production effect

Mdim = mean days in milk effect

Lac = parity effect

Sys = milking system effect

House = housing system effect

Prep = prep time effect

Lag = prep-lag time effect

Wash = udder washing method effect

Dry = udder drying effect

Dip = teat dipping effect

Spray = teat spraying effect

Bdip = brand name of teat dip effect

Rinse = teat cup liner rinsing between cows effect

Masmlk = when mastitic cows milked effect

Lactrt = type of treatment for mastitic lactating cows effect

Trtad = site of treatment administration effect

Numtrt = percent of total herd dry treated effect

Btrt = brand dry treatment product used effect

Bed = bedding effect

Bedadd = frequency free stall raking effect

Bedclean = frequency free stall cleaning effect

Ovsc = open vs. closed herd policy effect

e = random error effect

Management practices models were:

Y = Mlk + Mdim + Lac + Sys + Prep + Lag + Prep(Lag) + e

Y = Mlk + Mdim + Lac + Sys + Wash + Dry + Dip + Spray +
Wash(Dry) + Wash(Dip) + Wash(Spray) + Dip(Wash + Dry)
+ Spray(Wash + Dry) + e

Y = Mlk + Mdim + Lac + Sys + Wash + Dry + Bdip + Wash(Dry)
+ Wash(Bdip) + Bdip(Wash + Dry) + e

Y = Mlk + Mdim + Lac + Sys + Wash + Dry + Dip + Spray +
Rinse + Wash(Rinse) + Dip(Rinse) + Spray(Rinse) +
Rinse(Wash + Dry) + Rinse(Wash + Dry + Dip) +
Rinse(Wash + Dry + Spray) + e

Y = Mlk + Mdim + Lac + Sys + Masmlk

Y = Mlk + Mdim + Lac + Sys + Lactrt + Trtad + Lactrt(Trtad)
+ e

Y = Mlk + Mdim + Lac + Sys + Numtrt + e

Y = Mlk + Mdim + Lac + Sys + Btrt + Numtrt + Dip +
Dip(Btrt + Numtrt) + e

Y = Mlk + Mdim + Lac + Sys + Btrt + Numtrt + Bdip +
Bdip(Btrt + Numtrt) + e

$$Y = Mlk + Mdim + Lac + Sys + Wash + Dry + Dip + Rinse + Numtrt + Wash(Numtrt) + Dip(Numtrt) + Numtrt(Wash + Dry) + Numtrt(Wash + Dip) + Numtrt(Wash + Dry + Dip) + e$$

$$Y = Mlk + Mdim + Lac + House + Bed + Bedadd + Bed(Bedadd) + House(Bed + Bedadd) + e$$

$$Y = Mlk + Mdim + Lac + House + Bed + Bedrake + Bed(Bedrake) + House(Bed + Bedrake) + e$$

$$Y = Mlk + Mdim + Lac + House + Bed + Bedclean + Bed(Bedclean) + House(Bed + Bedclean) + e$$

$$Y = Mlk + Mdim + Lac + House + Bed + Bedadd + Bedrake + Bedclean + e$$

$$Y = Mlk + Mdim + Lac + House + Ovsc + e$$

All factors except error were fixed in each of these management practice models.

RESULTS AND DISCUSSION

Effect of various cow factors on somatic cell count

A model containing herd, cow within herd, daily milk production (lbs/day), parity, cow age, stage of lactation, percent milk fat, herd size, and month of year was constructed to examine how cow related factors affected somatic cell count (SCC). Actual SCC divided by 100,000 plus one was translated into natural log (ln SCC). Analysis was based on 1,073,587 cow records in 1,109 herds. The model accounted for 43.35% of variation in ln SCC. The resulting analysis of correlation summary table is presented in Table 5. All variables in the model had a statistically significant impact.

Cow within herd accounted for 64.01% of model variation, herd 21.06%, percent milk fat 6.36%, month of year 4.38%, parity 3.71%, daily milk production 0.23%, stage of lactation 0.23%, age of cow 0.11% and herd size .00074%. The large percentage for cow within herd variation in this analysis is primarily due to the large variation in farm conditions and managerial practices between herds.

Further analysis of individual effect of above listed factors on somatic cell count was performed. Variance due to herd and cow within herd was removed. As daily milk production increased, ln SCC decreased, and as parity, age, stage of lactation, percent fat and herd size increased, ln SCC increased.

TABLE 5.--Cow Factors Table of Analysis of Correlation Summary.

Source	d.f.	SS	Significance (p <)
Model	117,290	368,446.850	
Herd	1,108	77,609.117	.0001
Herd*Cow	116,156	235,850.623	.0001
Milk Prod.	1	863.715	.0001
Parity	12	13,673.748	.0001
Stage LAC.	1	831.982	.0001
Month	11	16,138.490	.0001
% Milk Fat	1	23,434.252	.0001
Cow Age	1	42.184	.0001
Herd Size	1	2.740	.01
Error	956,143	481,556.341	
Corrected Total	1,073,587	850.003.191	

Daily milk production had a negative effect on somatic cell count; an increase of ten pounds in milk production was associated with a 5,549.30 cells per ml decrease in SCC. Effect of milk production on SCC we observed was similar to the observations of Kennedy et al. (1978) and Moxley et al. (1978), but not as great as reported by Jones et al. (1982). However, Jones et al. (1982) used a slightly different method for determining the regression equation than Kennedy et al. (1978), Moxley et al. (1978), and our current study. These results suggest even relatively small increases in SCC because of injury, infection, or increased sloughing of mammary cells can be associated with a decrease in milk production.

Since most cows are between two and three years old when first lactation begins and the average interval between beginning of subsequent lactations is thirteen months, cow age and parity are highly correlated. This study found somatic cell count increased with advancing age and parity. These findings agree with other reports that showed a significant increase in SCC with advancing age (Blackburn, 1966; Schalm et al., 1968; Duitschaeven and Ashton, 1972; Natzke et al., 1972; Schultz, 1977; Eberhart et al., 1979; Rindsig et al., 1979). However, we found significant ($p < .10$) increases in SCC occurred only when comparing lactations that were separated by two or more lactations; for example, first and fourth lactations, second and fifth lactations, first and fifth lactations, second and sixth lactations, etc. These results call into question the conclusions of authors who only compared subsequent lactations and based their conclusions on the lack of significance between the

two lactations (Duitschaeven and Ashton, 1972; Natzke et al., 1972). This increase in SCC with age/parity is probably the result of increased exposure to infection and/or increased incidence of subclinical mastitis. To determine if this hypothesis is correct, studies similar to those of Duitschaeven and Ashton (1972) and Natzke et al. (1972) over four or more subsequent lactations would be needed.

Within a lactation, as days in milk (stage of lactation) increased, somatic cell count which was adjusted for effects of milk production and lactation number increased at a rate of 7.92 cells per ml per day or 2,414.75 cells per ml for a 305 day lactation. A rise in SCC with advancing stage of lactation has been reported by other researchers (Blackburn, 1966; Schalm et al., 1968; Schalm et al., 1971; Natzke et al., 1972; Schultz, 1977; Kennedy et al., 1978; Ruffo et al., 1978; Eberhart et al., 1979; Syrstad et al., 1979). The increase in SCC was not eliminated when stage of lactation was adjusted for effects of other variables in the model. This is contrary to the hypothesis of Schalm et al. (1968), Schalm et al. (1971) and Kennedy et al. (1978) and the statistically based conclusions of Syrstad et al. (1979) who felt the SCC increase was a concentration effect. Thus, the SCC increase is probably the result of increased infection rate with advancing lactation as proposed by Natzke et al. (1972) and Eberhart et al. (1979).

There was a highly significant ($p < .0001$) positive association between percent milk fat and somatic cell count. The increase in percent milk fat with increasing SCC is probably a concentration

effect. It has been previously observed by Kennedy et al. (1978), Moxley et al. (1978), Jones et al. (1982), and this study that as SCC increased, milk production decreased. It has also been reported that as SCC increased, fat production remained virtually unchanged (Waite and Blackburn, 1957; Wheelock et al., 1966; Schalm et al., 1971; Gard and Watkins, 1977; Schultz, 1977; Bath et al., 1978; Everson, 1980). Therefore, approximately the same quantity of fat is present in a smaller volume of fluid which explains the increase in milk fat percent.

Previous reports on the effect of herd size on somatic cell count have been either inconclusive (Afifi, 1967) or found no effect (Afifi, 1968d; Schultz, 1977). The current study found herd size to have a statistically significant ($p < .01$) effect on SCC. However, this relationship is probably not biologically significant since the increase is only 1.07 cells/ml per cow.

Somatic cell counts significantly ($p < .01$) increased from January to a yearly peak in June, declined from June to August, rose from August to a secondary significant ($p < .01$) peak in October, and then decreased to below January levels by December (Figure 1). This seasonal pattern of SCC is unlike anything previously reported (Nelson et al., 1969; Wilton et al., 1972; Paape et al., 1973; Simensen, 1974; Wegner et al., 1976; Simensen, 1976; Kennedy et al., 1978; Pearson and Mackie, 1979; Syrstad and Røn, 1979). This pattern was the same for all age groups. Since previous workers had found weather conditions affected somatic cell counts, information on atmospheric temperatures (Figure 2) and precipitation (Figure 3)

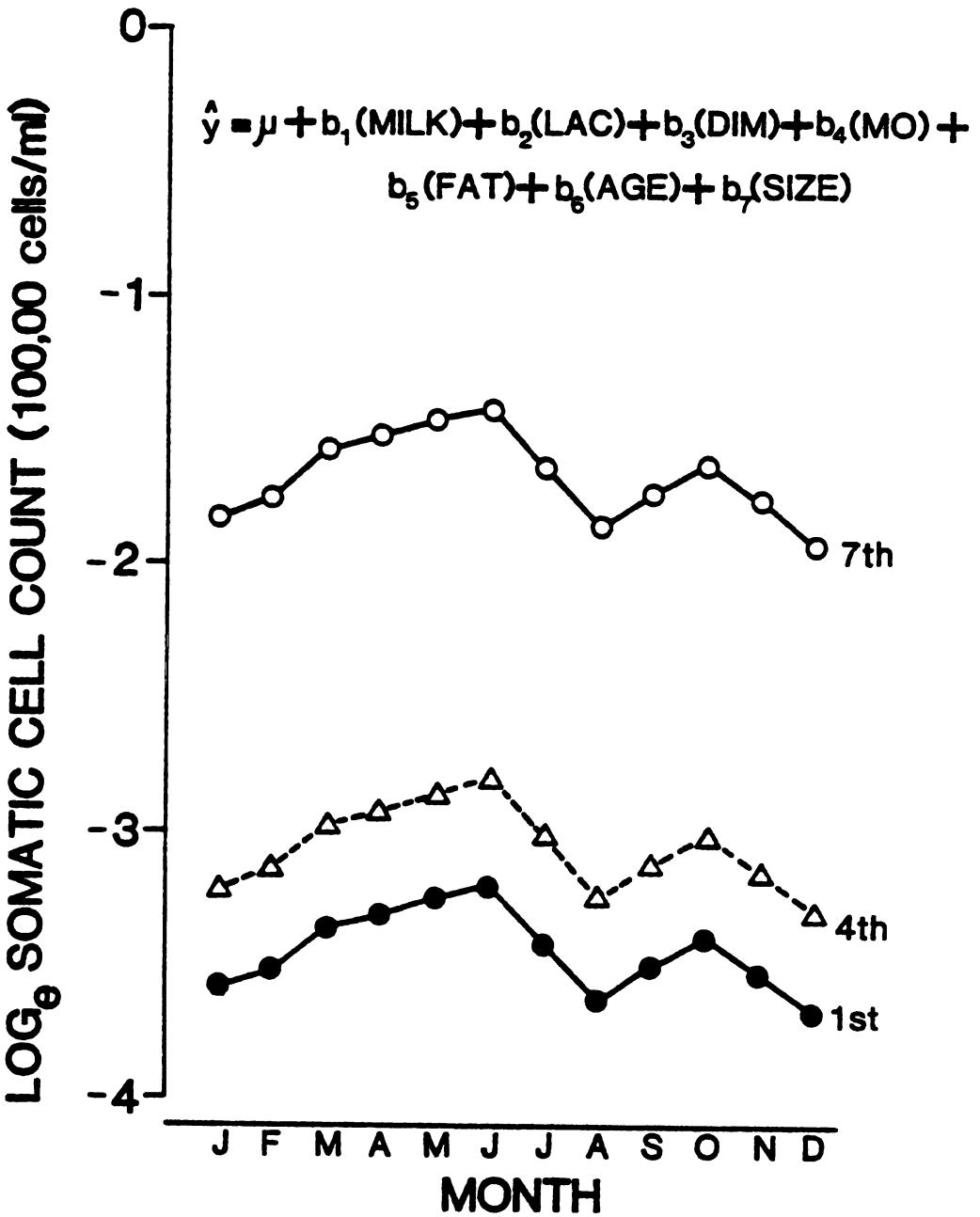


Figure 1.--Seasonal pattern of somatic cell count.

Figure 2.--Average minimum, average, and maximum daily temperature per month.

SOURCE: National Oceanic and Atmospheric Administration, Environmental Data. 1979-1981.

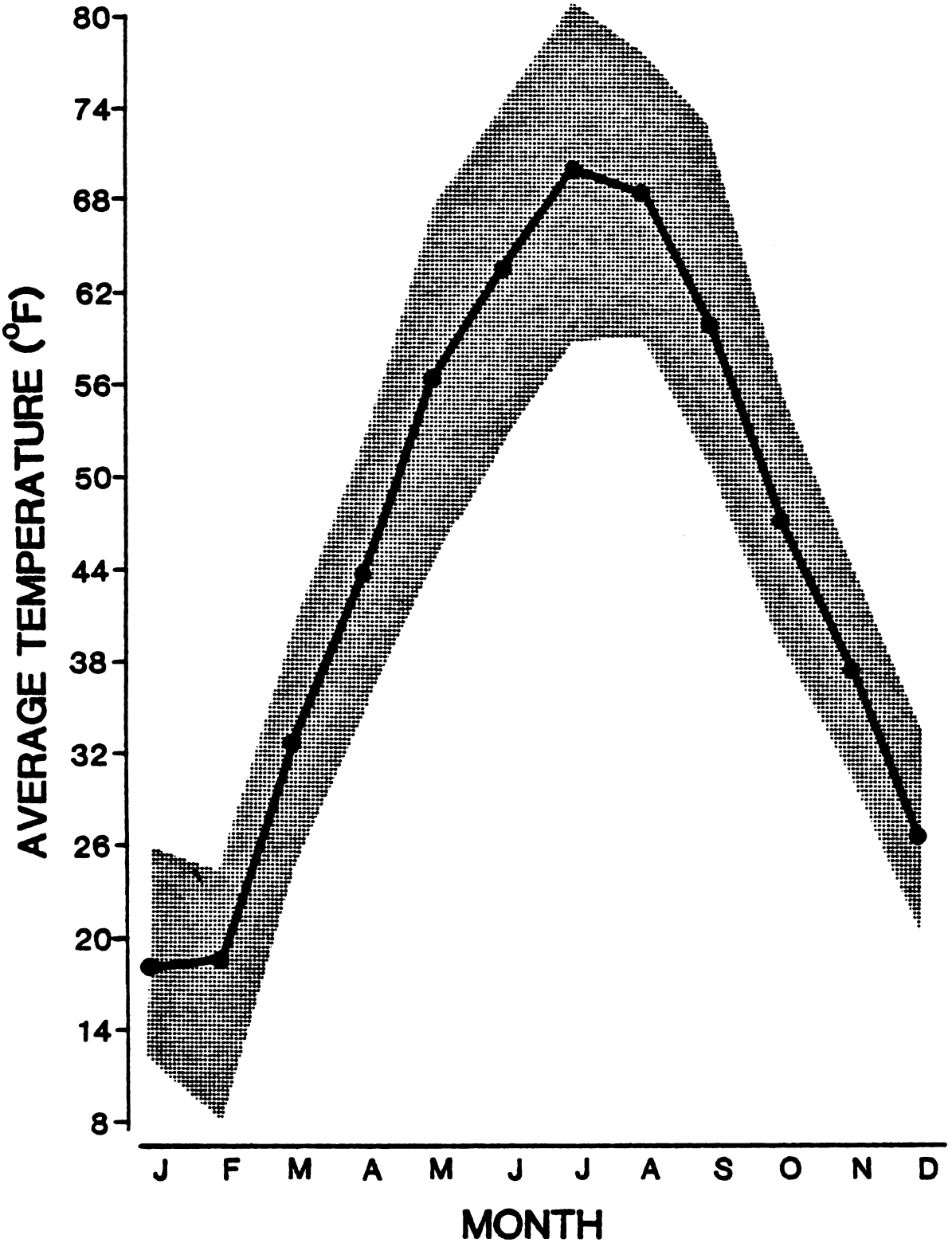
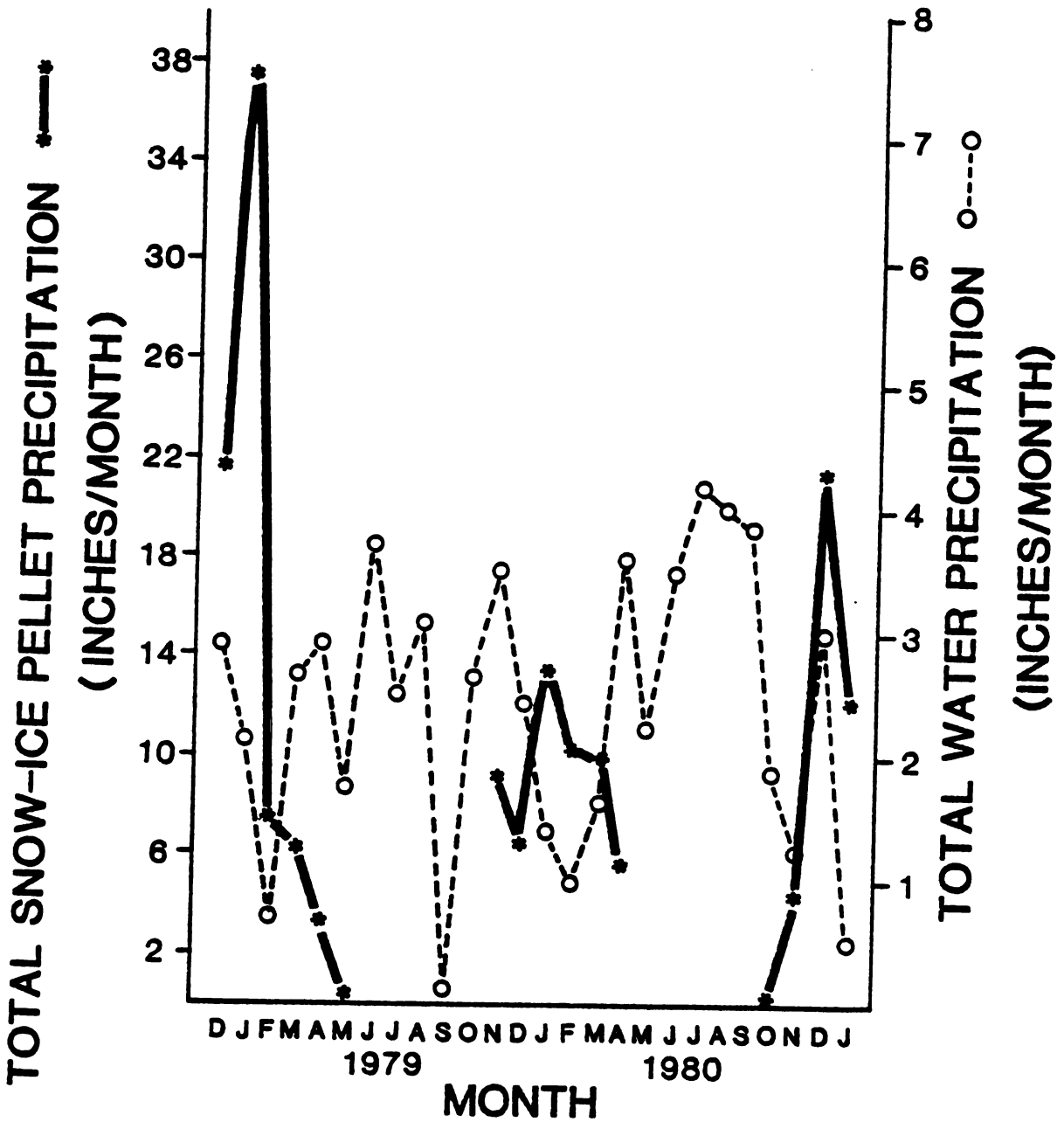

AVERAGE TEMPERATURE (°F)
80
74
68
62
56
50
44
38
32
26
20
14
8
J
F
M
A
M
J
J
A
S
O
N
D
MONTH

Figure 3.--Total snow-ice pellet precipitation and total water precipitation for each month of study.

SOURCE: National Oceanic and Atmospheric Administration, Environmental Data. 1979-1981.


TOTAL SNOW-ICE PELLET PRECIPITATION
(INCHES/MONTH)
38
34
30
26
22
18
14
10
6
2
8
7
6
5
4
3
2
1
TOTAL WATER PRECIPITATION
(INCHES/MONTH)
D J F M A M J J A S O N D J F M A M J J A S O N D J
1979
1980
MONTH

were obtained from five Michigan National Oceanic and Atmospheric Administration stations. In general, peaks in amount of precipitation corresponded to seasonal peaks in SCC. Simensen (1974) and Titterton and Oliver (1979) reported increased SCC with increased precipitation. The greater elevation of June SCC compared to October SCC may be an additive effect of higher environmental temperatures. Nelson et al. (1967), Wegner et al. (1976), Nelson et al. (1969), Whittlestone et al. (1970), and Paape et al. (1973) have shown elevated SCC during periods of warm environmental temperatures. Additionally, a change in cow's ration may also have contributed to SCC increase (Carroll, 1977; Ruffo and Sangiorgi, 1980).

The secondary SCC peak in October may be the result of an interaction among atmospheric temperature, precipitation, and housing. Amount of precipitation declined from June to September, then increased (Figure 3). Increased precipitation could have increased SCC (Simensen, 1974; Titterton and Oliver, 1979). The combination of cooler temperatures and precipitation has been shown to have a chilling effect, different from cold temperatures alone, on the udder. The chilling effect has been associated with elevated SCC (Simensen, 1974). A housing and weather interaction has been shown to affect incidence of mastitis. Vasil (1980) reported increased clinical mastitis in cows housed in barns with poor ventilation and barn humidity of 80% or more during summer-fall weather transition. Frances et al. (1981) found that as environmental temperatures decreased cows spent 50% more time lying in free stalls which was associated with an increased probability of udder infection. In

addition, a shift in cows ration to more ensiled feeds may have affected mean SCC (Carroll, 1977; Ruffo and Sangiorgi, 1980).

In addition to factors discussed here, other factors may affect somatic cell counts. These factors include, but are not exclusively limited to, date of freshening, heredity, wind chill, relative and absolute humidity and radiant heat (Lush, 1950; Afifi, 1967; Nelson et al., 1967; Afifi, 1968a; Nelson et al., 1969; Wilton et al., 1972; Simensen, 1976).

Effect of milking and housing systems on somatic cell count

Milking System.--Improper function or use of the milking system has been suggested as preconditioning the cow to teat injury, to increased incidence and spread of mastitis, and to higher somatic cell counts. It was the goal of this portion of the study to determine how various milking system factors affected SCC under field conditions. Mean values used for analysis of effect of milking system on SCC are presented in Table 6.

Compared to herds with milking systems containing more than one brand of equipment Conde®, Bodmin®, and Boumatic® milking systems were associated with significantly ($p < .002$, .04, and .005, respectively) higher mean somatic cell counts. Germania® and Zero® milking systems had a negative association with mean SCC, but it was not significant ($p < .40$). There was no significant ($p < .20$) differences in mean SCC among other brands of milking equipment. Since none of the systems surveyed were inspected to assure correct installation and operation, it is not possible to definitely state why only the

TABLE 6.--Mean Values Used for Analysis of Effect of Milking System on SCC.

Milk System	Mean ln SCC					Mean Milk Production					Mean Days in Milk				
	Mean	S.D.	Min.	Max.	S.E.	Mean	S.D.	Min.	Max.	S.E.	Mean	S.D.	Min.	Max.	S.E.
Bucket															
LAC = 1	.839	.280	.325	1.963	.039	39.5	6.9	23.5	62.7	1.0	168.8	30.4	113	296	4.3
LAC = 2	1.027	.407	.359	2.347	.057	46.5	8.8	24.1	75.1	1.2	159.4	29.6	107	295	4.1
LAC = 3	1.148	.370	.691	2.364	.052	48.6	9.3	26.3	69.8	1.3	162.3	36.0	76	297	5.1
LAC = 4	1.276	.440	.555	2.349	.062	49.4	11.2	17.8	79.7	1.6	153.7	33.6	60	212	4.8
LAC = 5	1.313	.375	.613	2.206	.055	49.5	11.4	26.1	78.7	1.7	158.9	40.7	46	250	6.0
LAC = 6	1.505	.665	.347	3.497	.099	48.4	9.0	29.0	69.4	1.3	143.3	49.4	8	243	7.4
LAC = 7	1.416	.551	.260	2.330	.088	46.3	12.9	18.9	76.1	2.1	156.4	63.1	47	352	10.1
LAC = 8	1.439	.634	.000	2.562	.112	46.1	18.8	0.0	91.8	3.3	154.0	76.8	24	328	13.6
LAC $\geq$ 9	1.349	.694	.000	2.583	.142	45.9	15.6	0.0	71.6	3.2	130.2	50.9	28	214	10.4
Barn pipeline, Low Line															
LAC = 1	.993	--	.993	.993	--	40.6	--	40.6	40.6	--	219.8	--	220	220	--
LAC = 2	1.171	--	1.171	1.171	--	51.5	--	51.5	51.5	--	227.9	--	228	228	--
LAC = 3	.987	--	.987	.987	--	54.1	--	54.1	54.1	--	213.5	--	214	214	--
LAC = 4	1.453	--	1.453	1.453	--	58.1	--	58.1	58.1	--	185.4	--	185	185	--
LAC = 5	1.727	--	1.727	1.727	--	73.7	--	73.7	73.7	--	118.4	--	118	118	--
LAC = 6	2.172	--	2.172	2.172	--	48.9	--	48.9	48.9	--	201.7	--	202	202	--
LAC = 8	1.025	--	1.025	1.025	--	76.1	--	76.1	76.1	--	98.7	--	99	99	--
Barn pipeline, High Line															
LAC = 1	.819	.215	.377	1.355	.024	40.9	7.9	23.4	60.6	0.9	173.1	27.7	87	269	3.1
LAC = 2	.965	.272	.412	1.542	.031	47.4	9.2	24.2	66.9	1.0	164.9	28.0	76	268	3.2
LAC = 3	1.120	.318	.540	1.834	.036	51.0	9.6	27.6	71.5	1.1	161.3	35.1	34	293	4.0
LAC = 4	1.233	.354	.645	2.214	.041	51.7	9.5	26.0	72.1	1.1	161.9	38.0	59	259	4.4
LAC = 5	1.336	.357	.506	2.409	.043	51.2	11.0	21.8	73.8	1.3	159.6	40.0	19	241	4.8
LAC = 6	1.369	.390	.514	2.255	.048	50.0	12.1	18.9	87.8	1.5	170.9	49.0	20	307	6.0
LAC = 7	1.442	.614	0.0	3.090	.078	48.6	14.5	8.7	91.2	1.8	156.1	69.7	12	408	8.9
LAC = 8	1.482	.639	0.0	2.829	.088	47.1	14.7	17.2	79.3	2.0	174.9	70.0	11	416	9.6
LAC = 9	1.491	.755	.358	4.605	.110	47.5	15.7	4.5	75.9	2.3	158.2	69.0	15	456	10.1
Parlor, Low Line															
LAC = 1	.813	.205	.407	1.543	.022	42.8	6.2	19.0	55.3	0.7	176.2	24.5	82	271	2.7
LAC = 2	.953	.251	.518	1.750	.027	48.5	7.3	27.9	61.9	0.8	166.8	19.3	120	219	2.1
LAC = 3	1.080	.290	.549	1.905	.032	51.2	6.9	34.2	66.9	0.8	168.3	19.3	102	220	2.1
LAC = 4	1.193	.321	.394	2.002	.035	51.9	8.0	32.4	70.7	0.9	164.0	27.6	24	237	3.0
LAC = 5	1.250	.376	.508	2.352	.042	51.8	8.0	28.7	70.9	0.9	172.3	29.3	59	265	3.2
LAC = 6	1.369	.396	.508	2.388	.045	51.7	9.2	31.6	79.5	1.0	168.8	31.8	29	233	3.6

TABLE 6 (Continued)

Milk System	Mean ln SCC					Mean Milk Production					Mean Days in Milk				
	Mean	S.D.	Min.	Max.	S.E.	Mean	S.D.	Min.	Max.	S.E.	Mean	S.D.	Min.	Max.	S.E.
LAC = 7	1.418	.575	.099	3.246	.066	52.4	14.2	18.8	100.3	1.6	150.0	51.1	28	345	5.8
LAC = 8	1.466	.628	0.0	2.876	.078	49.9	15.6	0.0	109.5	1.9	157.5	62.7	40	452	7.8
LAC = 9	1.644	.705	0.0	2.904	.097	43.8	13.7	0.0	68.7	1.9	168.8	68.9	30	398	9.5
Parlor, High Line															
LAC = 1	.804	.173	.455	1.250	.024	40.8	4.5	30.8	50.2	0.6	174.3	16.4	144	213	2.3
LAC = 2	.914	.236	.496	1.543	.033	45.8	5.3	34.3	60.8	2.7	161.3	24.0	48	210	3.4
LAC = 3	1.034	.277	.308	1.720	.039	47.9	5.9	32.5	62.4	0.8	170.8	26.6	58	239	3.8
LAC = 4	1.132	.276	.424	1.712	.039	49.4	7.2	35.7	70.3	1.0	169.8	25.9	91	235	3.7
LAC = 5	1.189	.348	0.0	1.813	.050	49.9	7.2	37.0	65.3	1.0	169.4	39.1	24	250	5.6
LAC = 6	1.294	.469	.347	2.917	.069	49.3	7.8	26.9	65.3	1.1	167.5	30.9	115	245	4.6
LAC = 7	1.362	.494	.347	2.343	.075	48.2	15.0	8.2	110.5	2.3	176.4	59.6	13	280	9.1
LAC = 8	1.477	.646	0.0	2.679	.102	46.0	13.4	0.0	71.4	2.1	157.9	67.3	27	325	10.6
LAC = 9	1.477	.592	.299	2.876	.112	42.0	12.4	7.9	62.8	2.3	170.2	71.0	14	327	13.4
Jars															
LAC = 1	.797	.208	.471	1.301	.036	43.9	5.9	30.6	55.0	1.0	179.2	19.3	137	218	3.4
LAC = 2	.950	.274	.439	1.716	.048	48.8	7.4	31.8	59.9	1.3	168.3	16.1	133	211	2.8
LAC = 3	1.108	3.12	.582	2.025	.054	51.0	7.8	33.1	63.5	1.4	172.3	18.5	145	230	3.2
LAC = 4	1.189	.340	.584	2.174	.059	51.6	8.0	30.6	67.2	1.4	169.2	30.1	118	284	5.2
LAC = 5	1.373	.501	.544	3.159	.087	51.2	6.9	31.5	62.1	1.2	165.3	30.4	99	242	5.3
LAC = 6	1.508	.654	.524	3.584	.116	50.1	8.7	31.0	66.3	1.5	170.0	42.5	74	276	7.5
LAC = 7	1.466	.465	.900	3.236	.085	48.9	10.1	18.2	67.3	1.9	171.5	44.9	105	297	8.2
LAC = 8	1.474	.621	.139	2.963	.122	51.4	17.0	2.1	82.7	3.3	161.0	74.2	16	412	14.6
LAC = 9	1.682	.797	0.0	2.983	.183	44.3	17.4	0.0	72.0	4.0	142.9	59.6	41	288	13.7

above named systems affected mean SCC. Saloniemi (1980) reported a highly significant ($p < .001$) increase in incidence of mastitis as equipment varied from manufacturer's specifications. Additionally, we do not know whether these systems were older and/or had been expanded more than other systems. Downey et al. (1977; 1978) and Saloniemi (1980) found increasing system age and expansion increased SCC.

A study of the effect of type of milking system and milk pipeline height on mean somatic cell counts was made. Parlor milking systems had significantly ($p < .05$) lower mean SCC than any other system. This finding is in agreement with the reports of Downey et al. (1977) and Schultz (1977). In our study, there were no significant ($p < .20$) differences among bucket, barn pipeline, and weigh jar milking systems. However, in general, bucket systems had numerically lower mean SCC than barn pipeline systems, which had lower mean SCC than systems with weigh jars. This is contrary to the reports of Downey et al. (1977) and Saloniemi (1980) who found SCC was significantly ($p < .05$) higher with bucket milking systems than barn pipeline systems. Neither had considered systems with weigh jars. The variation in results could be due to differences in age of milking systems, whether or not systems were expanded, or both (Downey et al., 1977; Downey et al., 1978; Saloniemi, 1980).

When milk pipeline height was considered in addition to type of system, the ranking was, from lowest mean SCC to highest SCC, high milk pipeline (3 or more feet above cow's udder) parlor, low milk pipeline (below cow's udder) parlor, bucket system, high milk

pipeline barn pipeline, and systems with weigh jars. Only high milk pipeline parlors were significantly ($p < .01$) different from the other systems. Without further study, it is not possible to determine why high milk pipeline parlors had the lowest mean SCC. However, LeDu (1980) reported more vacuum fluctuations occurred with low milk pipeline milking systems during milking, especially at end of milking. Additionally, transmission of vacuum fluctuations from a cluster to the nearest cluster occurred more easily with a low milk pipeline than high milk pipeline.

Only three line vacuum levels (11, 12.3 and 12.8 inches Hg) had significantly ($p < .02$, .08 and .01, respectively) lower mean SCC. The greatest reduction in mean SCC occurred at 12.8 inches Hg. Interaction of vacuum level and milk pipeline height had a significant ($p < .0001$) effect on mean SCC. Significantly ($p < .01$) lower mean SCC for systems with a low milk pipeline occurred between 11 and 12 inches Hg, with optimum reduction at 11.5 inches Hg. For systems with a high milk pipeline lowest mean SCC occurred between 13.2 and 14.5 inches Hg, with optimum reduction at 13.5 inches Hg. These findings refine previous reports that vacuum levels between 10 and 13 inches Hg were best in terms of reducing teat trauma (McDonald, 1971, 1975; Nicolai et al., 1977; Galton and Mahle, 1980). However these authors did not determine interrelationships of vacuum level and milk pipeline height. Vacuum levels of 15 inches Hg or greater at teat end have been associated with increased injury to both teat end and streak canal, teat congestion, higher SCC scores, and increased

incidence of clinical mastitis (Afifi, 1968c; McDonald, 1971, 1975; Nicolai et al., 1977; Saloniemi, 1980).

The vacuum controller (regulator) is closely associated with vacuum level and fluctuations. Saloniemi (1980) found poor vacuum controller performance significantly ($p \leq .05$) increased clinical mastitis. In this study, interaction between milking system and vacuum controller on mean somatic cell count was examined. There were no significant ($p < .20$) interactions between any brand of vacuum controller studied in bucket milking systems and mean SCC. In barn pipeline systems, ranking from greatest to least negative effect on mean SCC, Surge Equalizer®; Sentinel®; Westfalia® (Vac-U-Rex); Boumatic®, Universal®, Sta-Rite®, or Zero® weighted lever controllers; and Surge Oil Weight® vacuum controllers had a highly significant ($p < .005$) effect on mean SCC. There were no significant ($p < .20$) differences among these brands of vacuum controllers. All barn pipeline milking systems, except one, had milk pipelines three or more feet above the cow's udder. Higher mean SCC were associated with Sentinel® and Delaval Senior® vacuum controllers ($p < .16$ and .19) in parlor milking systems. Delaval Senior® vacuum controllers have a set vacuum level of 15 inches Hg and also are subject to load change related problems. Smith and Fairbanks (1975) found weighted sleeve value and weighted lever controllers were adequate if maximum load changes were ±10 CFM or less, while spring loaded diaphragm controllers (Sentinel®) were found to be more sensitive and able to handle load changes of greater than ±20 CFM or more without a change in vacuum level. Sentinel® vacuum controllers, which are more

sensitive and able to maintain a more stable vacuum level when properly installed and maintained, may be affected more by moisture, dust, location, maintenance practices, etc. than other types of controllers (LeDu, 1980).

Interference with free air flow has been shown to cause vacuum level fluctuations at the teat end during milking. The effect of factors that may interfer with free air flow on mean somatic cell count were examined.

Vacuum line design had a significant ($p < .002$) effect on mean somatic cell count. Complete loop vacuum line design, which has the best free air flow was associated with significantly ($p < .02$) lower mean SCC. While not statistically different ($p < .20$) dead end vacuum line designs were associated with numerically higher mean SCC than looped-T vacuum line designs. These results lend further support to the free air flow hypothesis, since dead end vacuum line designs have poorer free air flow than looped-T vacuum line designs and both have poorer free air flow than complete loop vacuum line design. There was no significant ($p < .50$) effect of type of material the vacuum line was made of on mean SCC. Vacuum line size also did not have a significant ($p < .20$) effect on mean SCC.

In an ideal milking system during milking, half the diameter of the milk pipeline contains air (Bath et al., 1978). Thus milk pipeline flooding could have an effect on air flow and somatic cell counts. To study this relationship, effect of the interaction of milk pipeline diameter and number of milking units on mean SCC was used. There was a highly significant ($p < .0001$) interaction between

number of milking units and milk pipeline diameter and mean somatic cell count. Milking systems have 1½ inch diameter milk pipelines and two or three milking units were associated with significantly ($p < .05$) lower mean SCC than milking systems having 2 inch diameter milk pipeline and two or three milking units. There were no significant ($p < .20$) differences in mean SCC among milking systems having 1½, 2, 2½ or 3 inch diameter milk pipelines and four milking units. However, milking systems with four milking units and either 2½ or 3 inch diameter milk pipelines had numerically higher mean SCC.

These results indirectly support a suggestion by LeDu (1980) that oversized milking pipelines lead to unstable vacuum levels which can result in increased teat trauma, infection, and SCC (Thiel et al., 1973; Noorlander, 1977; O'Callaghan and O'Shea, 1979; Thompson et al., 1980). There were no significant ($p < .20$) differences in mean SCC among milking systems with five milking units and 1½, 2, or 2½ inch diameter milk pipeline, but systems with 3 inch diameter milk pipelines were significantly ($p < .01$) higher. A comparison of milking systems having six milking units and various sizes of milk pipelines found systems with 2 inch diameter milk pipelines were associated with significantly ($p < .07$) lower mean SCC than 3 inch diameter milk pipelines, which were significantly ($p < .04$) lower than 2½ inch diameter milk pipelines. The use of eight or more milking units in a milking system resulted in significantly ($p < .07$) higher mean SCC in systems with 2½ inch diameter milk pipelines when compared with 3 inch diameter milk pipelines. The optimum number of milking units in terms of lowest mean SCC for milking systems having

1½, 2, or 2½ inch diameter milk pipelines was five and eight milking units for 3 inch diameter milk pipelines. A possible explanation is that air flow is relatively free until six milking units in milking systems with 1½, 2, or 2½ inch diameter milk pipelines or nine milking units in milking systems with 3 inch diameter milk pipelines are in operation. With the operation of six or nine milking units, the milk pipeline becomes flooded, slowing transport of milk from teat, teat cup and claw, interference with pulsation, and increasing milking time possibly leading to teat injury and/or overmilking.

Like Maatje and Rossing (1971) and Saloniemi (1980), we found pump capacity, measured in CFM/milking unit, had no significant ($p < .20$) effect on mean somatic cell count. However, reserve air flow is the critical measurement, and this parameter was not measured in this study.

Pulsation rate and ratio and the interaction of pulsation ratio and vacuum level have been shown to affect somatic cell count and incidence of mastitis (Afifi, 1967, 1968c; McDonald, 1971, 1975; Milne, 1977; Hoare et al., 1979; Galton and Mahle, 1980). Milking systems with single pulsation had significantly ($p < .10$) lower mean somatic cell counts when compared to milking systems with alternating pulsation. There were no significant ($p < .20$) differences in mean SCC among the six pulsation ratios studied or among possible interactions between alternating or single pulsation and the pulsation ratios. However there were significant ($p < .0001$) differences in mean SCC when effect of pulsation ratio and vacuum level were considered. The pulsation ratio of 50:50 was associated with lower mean SCC

over the widest range of line vacuum levels, 12.5 to 15 inches Hg. The optimum line vacuum level was 13.5 inches Hg. The wide range of vacuum levels associated with lower SCC is probably because the 50:50 pulsation ratio provides for adequate massage (Hoare et al., 1979). At pulsation ratios of 55:45 and 60:40 lowest mean SCC were associated with line vacuum levels of 12.5 and 14 inches Hg and 12.5 and 15 inches Hg, respectively. Lowest single mean SCC was associated with a 70:30 pulsation ratio at 11 inches Hg line vacuum level. Possibly the low line vacuum level (11 inches Hg) used in conjunction with the 70:30 pulsation ratio prevents or lessens severity or incidence of teat injury, congestion, and/or infection rate described by McDonald (1971, 1975) and Galton and Mahle (1980). Galton and Mahle (1980) had observed teat injury, congestion, and infection rate were more severe when the vacuum level exceeded 13 inches Hg. The effect of pulsation ratio and line vacuum level on mean SCC may be influenced by the type of inflation in use. Speed of inflation closure and opening has been shown to affect cyclic vacuum level fluctuation and teat cup liner slippage, and; thus, infection rate and SCC (Thiel et al., 1973; Noorlander, 1977; O'Callaghan and O'Shea, 1979; O'Shea et al., 1979).

Teat cup liners (inflations) were found to have a highly significant ($p < .0001$) positive effect on mean somatic cell count. However due to the large number of different styles of inflations in commercial use, it was not possible to isolate the effect of each style on mean SCC. O'Shea et al. (1979) conducted a series of controlled experiments comparing Gascoigne® single stretch inflations

with Alfa-Laval® Liner 960000-1. They found significantly ($p < .05$) more infection with use of the Gascoigne® single stretch inflations.

A general examination of the effect of automation on mean somatic cell count found no significant ($p < .20$) difference in mean SCC among non-automated and various types of automation with the exception of Surge VSO®. Use of Surge VSO® take-offs were associated with significantly ($p < .005$) higher mean SCC. While Philpot (1973) reported lower CMT scores with addition of Surge QTO®, we did not find a significant effect of Surge QTO® on mean SCC. Significantly ($p < .005$) higher mean SCC associated with Surge VSO® take-offs are probably associated with one or more of the following: 1) claw weight, 2) lack of support for claw, and/or 3) a possible interaction of inflations being used and Surge VSO®. O'Shea et al. (1979) reported 50-60% of new infections were due to liner slip. However, more research is needed to determine the validity of each of these hypothese.

Housing System.--Limited work has been done on how housing system affects milk somatic cell counts. The first part of this study examined the effect of milking herd housing system, bedding, and possible housing-bedding interactions on milk mean SCC. Mean values used for analysis of effect of housing system on SCC are presented in Table 7. Sand based free stall housing for the milking herd was associated with significantly (range $p < .02$ - $.0008$) lower mean SCC than any other housing system. There were no significant ($p < .20$) differences in mean SCC among the other housing systems. However, when ranking housing systems from smallest to largest

TABLE 7.--Mean Values Used for Analysis of Housing System on SCC.

Housing System	Mean ln SCC					Mean Milk Production					Mean Days in Milk				
	Mean	S.D.	Min.	Max.	S.E.	Mean	S.D.	Min.	Max.	S.E.	Mean	S.D.	Min.	Max.	S.E.
Stanchion															
LAC = 1	.810	.223	.377	1.234	.031	40.3	7.0	23.4	60.6	1.0	170.3	21.8	126	219	3.1
LAC = 2	.945	.311	.359	1.795	.044	47.0	8.1	24.2	66.9	1.1	165.0	25.3	108	228	3.5
LAC = 3	1.109	.329	.654	2.100	.047	49.9	8.7	28.4	66.7	1.2	166.7	28.8	107	246	4.1
LAC = 4	1.232	.404	.424	2.311	.057	49.1	9.2	26.0	72.1	1.3	168.1	31.3	88	251	4.5
LAC = 5	1.309	3.91	.613	2.206	.056	50.4	11.1	21.8	78.1	1.6	162.6	37.2	63	245	5.3
LAC = 6	1.490	.616	.701	3.584	.089	48.2	10.7	29.0	87.8	1.5	163.0	52.4	8	307	7.6
LAC = 7	1.442	.565	.693	2.436	.084	45.0	12.6	18.9	76.1	1.9	159.1	71.1	47	408	11.1
LAC = 8	1.533	.532	.693	2.765	.084	46.9	12.6	23.9	79.3	2.0	158.3	70.1	24	305	11.1
LAC = 9	1.354	.591	.358	2.778	.112	45.8	13.4	5.5	71.6	2.5	152.9	71.8	70	456	13.6
Comfort Stalls															
LAC = 1	.868	.256	.533	1.334	.047	43.3	6.7	25.6	54.2	1.3	171.7	28.4	87	204	5.7
LAC = 2	1.026	.361	.492	1.843	.072	48.8	8.6	25.6	61.1	1.7	158.6	25.7	76	195	5.1
LAC = 3	1.166	.329	.588	1.712	.066	52.1	8.0	37.6	69.8	1.6	162.2	28.0	76	198	5.6
LAC = 4	1.192	.373	.555	1.838	.075	54.6	8.3	40.3	79.7	1.7	152.7	38.1	59	197	7.6
LAC = 5	1.355	.412	.506	2.149	.086	52.9	9.5	35.3	65.2	2.0	152.8	38.9	46	238	8.1
LAC = 6	1.421	.477	.514	2.094	.104	53.1	12.6	26.9	80.1	2.8	163.1	31.8	105	240	7.0
LAC = 7	1.447	.725	0.0	3.090	.162	51.5	16.9	8.7	91.0	3.8	136.7	82.8	12	306	18.5
LAC = 8	1.506	.726	0.0	2.876	.171	48.2	19.0	10.1	74.4	4.5	161.1	59.3	40	261	14.0
LAC = 9	1.675	.633	.693	2.827	.158	51.8	15.9	15.8	75.9	4.0	130.6	46.9	30	183	11.7
Half Cement Free Stalls															
LAC = 1	.936	2.16	.563	1.250	.065	40.0	5.0	31.3	47.8	1.5	171.8	26.2	131	213	7.9
LAC = 2	1.085	.284	.642	1.567	.086	44.5	5.5	36.4	52.2	1.6	160.2	25.6	124	210	7.7
LAC = 3	1.229	.349	.624	1.905	.105	47.5	5.1	40.2	58.1	1.5	183.9	30.1	83	188	9.1
LAC = 4	1.302	.408	.645	1.950	.123	48.7	7.8	32.3	60.5	2.4	173.9	42.9	111	249	12.9
LAC = 5	1.593	.445	1.070	2.409	.148	51.8	7.1	39.8	63.2	2.4	145.9	37.8	76	201	12.6
LAC = 6	1.542	.543	.627	2.192	.205	47.8	6.7	39.4	55.3	2.5	183.2	27.8	134	215	10.5
LAC = 7	1.864	.806	.597	2.826	.305	45.8	11.2	28.1	58.2	4.2	205.1	73.8	95	245	27.9
LAC = 8	1.350	.797	0.0	2.233	.325	41.8	23.9	0.0	62.6	9.3	144.7	45.5	88	221	18.6
LAC = 9	1.980	.946	1.572	2.600	.248	45.5	7.4	36.6	54.4	3.7	185.1	30.3	158	226	15.1
Totally Cemented Free Stalls															
LAC = 1	.773	.286	.325	1.964	.048	42.1	6.2	27.4	53.7	1.0	179.1	26.6	126	260	4.4
LAC = 2	.897	.334	.518	2.347	.056	47.5	7.6	24.1	60.2	1.3	168.3	26.6	119	295	4.4
LAC = 3	1.034	.349	.550	2.364	.058	48.8	7.0	29.1	59.4	1.2	173.3	27.7	111	297	4.6
LAC = 4	1.140	.348	.669	2.349	.058	49.2	9.3	17.8	67.6	1.6	164.7	28.3	69	236	4.7
LAC = 5	1.146	.355	.508	2.227	.061	50.3	8.2	28.7	64.2	1.4	168.5	28.3	87	250	4.9
LAC = 6	1.228	.376	.347	2.388	.065	50.3	10.0	31.5	79.5	1.7	159.9	26.0	88	198	4.5
LAC = 7	1.264	.413	.497	2.333	.073	50.9	15.0	18.8	87.7	2.7	152.4	61.4	45	328	10.9
LAC = 8	1.223	.606	0.0	2.426	.119	50.3	20.9	2.1	109.5	4.1	156.9	93.4	29	412	18.3
LAC = 9	1.447	.782	.173	2.708	.190	44.7	13.6	11.0	68.7	3.3	154.6	71.9	55	341	17.4

TABLE 7 (Continued)

System	Mean In SCC					Mean Milk Production					Mean Days in Milk				
	Mean	S.D.	Min.	Max.	S.E.	Mean	S.D.	Min.	Max.	S.E.	Mean	S.D.	Min.	Max.	S.E.
Group Pens															
LAC = 1	.800	.192	.400	1.029	.058	37.0	5.6	28.1	42.7	1.7	171.6	27.3	126	212	8.2
LAC = 2	.983	.221	.583	1.394	.067	42.7	6.5	31.1	52.9	2.0	157.6	23.0	120	192	6.9
LAC = 3	1.065	.205	.691	1.338	.062	46.4	8.2	32.1	61.9	2.5	160.8	29.5	104	205	8.9
LAC = 4	1.201	.434	.684	2.214	.137	47.3	8.4	34.0	60.2	2.7	152.2	35.1	99	203	11.1
LAC = 5	1.246	.502	0.0	1.646	.159	47.1	8.1	35.0	65.2	2.6	148.2	64.0	24	237	20.2
LAC = 6	1.538	.547	1.072	2.917	.173	43.4	8.1	29.3	58.2	2.5	155.8	46.3	56	217	14.6
LAC = 7	1.535	.639	.801	2.595	.213	41.0	8.6	31.0	56.4	2.9	172.7	43.6	105	242	14.5
LAC = 8	1.582	.834	.439	2.649	.341	42.0	7.8	31.8	49.7	3.2	163.7	23.3	127	195	9.5
LAC = 9	1.787	1.524	.347	4.605	.622	47.9	13.5	31.1	70.0	5.5	117.9	100.2	15	292	40.9
Clay Based Free Stalls															
LAC = 1	.858	.192	.501	1.543	.023	42.1	5.4	28.6	55.0	0.6	180.4	24.8	137	296	2.9
LAC = 2	1.019	.260	.609	1.750	.031	47.4	6.8	27.9	61.9	0.8	167.5	22.6	48	228	2.7
LAC = 3	1.180	.318	.540	2.025	.038	50.1	6.7	33.1	63.6	0.8	169.6	27.6	76	239	3.3
LAC = 4	1.283	.313	.730	2.174	.038	51.0	8.6	30.5	78.8	1.0	164.3	29.6	24	210	3.6
LAC = 5	1.400	.420	.724	3.160	.052	50.7	8.8	30.5	73.7	1.1	171.3	29.7	59	239	3.7
LAC = 6	1.453	.490	.524	3.279	.062	50.4	7.6	31.6	70.1	1.0	170.4	32.1	74	248	4.0
LAC = 7	1.458	.608	.099	3.246	.079	49.8	13.1	8.2	110.5	1.7	156.4	49.5	13	280	6.4
LAC = 8	1.478	.577	0.0	2.600	.082	46.6	13.9	0.0	82.7	2.0	163.4	64.7	16	452	9.2
LAC = 9	1.625	.702	0.0	2.909	.111	43.8	16.7	0.0	63.7	2.6	168.9	64.7	33	347	10.2
Pasture															
LAC = 1	.829	.223	.583	1.023	.129	37.2	13.2	23.5	47.8	7.6	186.7	7.3	179	193	4.1
LAC = 2	.902	.317	.691	1.266	.183	44.6	8.8	35.3	52.8	5.1	162.6	26.1	143	192	15.1
LAC = 3	.972	.170	.861	1.168	.098	44.0	16.8	26.3	59.7	9.7	159.7	27.7	133	188	15.9
LAC = 4	1.291	.366	.869	1.529	.211	50.8	9.8	45.1	62.1	5.6	158.6	9.7	148	167	5.6
LAC = 5	1.442	.497	.877	1.812	.287	43.1	14.8	26.1	52.5	8.5	167.7	21.7	144	186	12.5
LAC = 6	1.466	.394	1.192	1.918	.228	48.1	8.9	38.4	56.0	5.1	155.6	20.8	142	180	12.0
LAC = 7	1.430	.054	1.392	1.468	.038	67.9	11.6	59.7	76.1	8.2	213.1	89.3	150	276	63.2
LAC = 8	1.202	1.129	0.0	2.241	.652	42.2	34.9	3.6	71.4	20.2	223.1	91.4	160	328	52.8
LAC = 9	1.962	.796	1.421	2.876	.460	46.8	14.7	31.4	60.6	8.5	122.4	73.1	39	173	42.2
Sand Based Free Stalls															
LAC = 1	.426	--	.426	.426	--	51.8	--	51.8	51.8	--	159.0	--	159	159	--
LAC = 2	.586	--	.556	.586	--	58.5	--	58.5	58.5	--	156.0	--	156	156	--
LAC = 3	.643	--	.643	.643	--	64.7	--	64.7	64.7	--	161.0	--	161	161	--
LAC = 4	.629	--	.629	.629	--	56.3	--	56.3	56.3	--	169.0	--	169	169	--
LAC = 5	.924	--	.924	.924	--	59.0	--	59.0	59.0	--	177.0	--	177	177	--
LAC = 6	.508	--	.508	.508	--	72.1	--	72.1	72.1	--	162.0	--	162	162	--
LAC = 7	.893	--	.893	.893	--	69.3	--	69.3	69.3	--	148.0	--	148	148	--
LAC = 8	1.208	--	1.208	1.208	--	63.4	--	63.4	63.4	--	153.0	--	153	153	--
LAC = 9	.994	--	.994	.994	--	63.6	--	63.6	63.6	--	209.0	--	209	209	--

effect on increasing mean SCC, totally cemented free stalls had the smallest effect, followed by stanchions, group pens, year round pasture, comfort stalls, clay based free stalls, and half cemented free stalls. Bedding type by itself did not have a significant ($p < .20$) on mean SCC. However there were several significant housing system-bedding type interactions. In comfort stall housing systems, use of both long straw and sawdust as bedding was associated with significantly (range $p < .06 - .0001$) lower mean SCC than any other bedding materials. Though not statistically different, the ranking of other comfort stall beddings from smallest to largest numerical effect on mean SCC was sand, chopped straw, long straw and sawdust. In half cement free stalls (front half cemented, rear half dirt base), bedding with a combination of long straw and sand was associated with significantly ($p < .05$) lower mean SCC than bedding with sawdust alone. This finding is not surprising for two reasons. First, sawdust bedding has been directly linked to an increased coliform infection rate and clinical mastitis (Newman, 1973; Bramley, 1974; Newman, 1975). Second, due to their design, half cement free stalls would tend to collect moisture and feces in the back half of the stall which is conducive to the growth of microorganisms. Use of both long straw and sand in half cement free stalls probably leads to formation of a relatively dry manure pack and thus lower mean SCC. In totally cemented free stall housing systems, lower mean SCC was associated with use of corn cobs/stalks, sand and sawdust, hay and sand, or chopped straw and sawdust. There were no significant ($p < .20$) differences in mean SCC among these beddings.

However, mean SCC in housing systems with cemented free stalls using sand bedding alone or long straw bedding alone was significantly ($p < .05$ and .002 respectively) higher. These results may be related more to management factors; such as depth of bedding, frequency of bedding addition, raking, or stall cleaning and weather conditions rather than actual bedding material (Ekesbo, 1966; Francis et al., 1981). In group pen housing systems, mean SCC was significantly ($p < .002$) higher when long straw bedding was used than when the combination of long straw and corn cobs/corn stalks was used as bedding. There was no significant ($p < .20$) difference in mean SCC between use of chopped straw and either long straw or corn cobs/corn stalks plus long straw in group pens. Again these differences in mean SCC may be the result of management practices or weather conditions and not the bedding material per se.

The second part of this study examined maternity facilities; type, number of stalls if applicable, type of bedding, type of facility-bedding type interaction, and seasonal interactions. A significant decrease in mean somatic cell count was associated with the presence of maternity (box) stalls. Herds with four to seven maternity stalls had significantly ($p < .10$) lower mean SCC than herds with one or two maternity stalls. Herds with three maternity stalls were not significantly ($p < .20$) different from herds with either one or two, or four or more maternity stalls. Lowest numeric mean SCC was associated with five maternity stalls.

When type of bedding in maternity stall(s) was considered, a significant ($p < .01$) reduction in mean SCC was associated from

greatest to least, with two maternity stalls bedded with chopped straw, two maternity stalls bedded with long straw, three maternity stalls bedded with long straw, and one maternity stall bedded with long straw. There were no significant ($p < .20$) differences among these four types of calving facilities.

Effect of season at calving and location of majority of calvings on mean SCC was examined. Significantly lower mean SCC were found when, during fall to spring calving period, a majority of calvings occurred either in out-of-door pens with no shelter or in pasture ($p < .0002$ and $.03$). Out-of-door pens with no shelter were associated with significantly ($p < .05$) lower mean SCC than pasture. Significantly ($p < .01$) higher mean SCC were associated with herds where the majority of fall to spring calvings occurred in an out-of-door pen with shelter. During spring to fall calving period, numerically lower mean SCC were associated with herds where majority of calvings occurred in either box stalls or group pens. Mean SCC was significantly ($p < .05$) higher in herds where the majority of calvings occurred either in the cow's normal stall or where ever the cow was at time of parturition.

Further analysis was performed to determine the interaction effect, if any, of season of calving, location of calving, number of maternity stalls, if applicable, and maternity stall bedding on mean somatic cell count. Those conditions associated with significantly lower mean SCC during fall to spring calving period are listed in Table 8 ($p < .05$), while those associated with the spring to fall calving period are listed in Table 9 ($p < .10$). Only significantly

TABLE 8.--Effect of Fall to Spring Calving Conditions on Mean SCC.

Facility	Number Maternity Stalls	Bedding Material	$\hat{y}$ (in 100,000 cells/ml) (est. effect on mean SCC)
group pen	1	long straw	-3.453
box stall	1	saw dust	-3.324
box stall	3	long straw	-3.290
out-of-door pen	-	chopped straw	-3.214
group pen	2	long straw	-3.211
box stall	1	long straw	-3.204
group pen	-	hay	-3.203
group pen	-	long straw	-3.165
box stall	2	long straw	-3.140
box stall	2	chopped straw	-3.123
out-of-door pen with shelter	-	corn cobs/stalks	-3.094
box stall	1	long straw	-3.044
out-of-door-pen with shelter	-	long straw	-2.929
group pen	-	chopped straw	-2.856

TABLE 9.--Effect of Spring to Fall Calving Conditions on Mean SCC.

Facility	Number Maternity Stalls	Bedding Material	$\hat{y}$ (in 100,000 cells/ml) (est. effect on mean SCC)
box stall	1	sawdust	-3.893
out-of-door pen with shelter	-	none	-3.547
out-of-door pen with shelter	2	sawdust	-2.926
group pen	-	none	-2.778
box stall	1	long straw	-2.323
box stall	2	long straw	-2.159
group pen	1	long straw	-2.074
box stall	3	long straw	-1.961

higher mean SCC occurred when a majority of cows calved either in a pasture, where ever the cow was at parturition, or in the herd's only box stall when bedded with hay during fall to spring calving period ($p < .01$, .08, and .09, respectively).

In light of the warning issued by Kingwell et al. (1977) that variability within housing systems can be greater than variability between housing systems and since this was not a controlled study, it is not appropriate at this point to recommend one housing system over another. However, it would appear certain bedding materials are best in terms of lower mean somatic cell counts in certain housing systems. The examination of maternity conditions (facilities, bedding material, and season of calving) suggest two box stalls bedded with long straw may be best on a year-round basis. Further detailed studies on maternity conditions are needed, especially in the area of management practices, such as frequency, method, and thoroughness of cleaning.

Effect of management practices on somatic cell count

Five areas directly controlled by the herd manager were considered; milking hygiene practices, treatment of mastitic cows, dry cow treatment policy, free stall maintenance, and purchase of replacement animals.

Milking Hygiene Practices.--In a study of milking practices, it was found length of time prepping the cow prior to milking and length of time between completion of prepping and attachment of milking machine (prep-lag time) had a highly significant ($p < .003$)

effect on mean somatic cell count. Prepping cows for 10-20 seconds or 20-30 seconds significantly ($p < .0001$) lowered mean SCC when compared to prepping less than 10 seconds or more than 30 seconds. Prepping 10-20 seconds and 20-30 seconds were not significantly ($p < .20$) different. There was no significant ($p < .01$) effect of time between completion of prepping and attachment of milking machine (prep-lag time) on mean SCC until pre-lag time exceeded two minutes. After two minutes a highly significant ($p < .0001$) increase in mean SCC occurred. The interaction of prepping and prep-lag times on mean SCC was highly significant ($p < .002$). The greatest negative effect on mean SCC was associated with a prep time of 20-30 seconds and a prep-lag time of 30-60 seconds. The least negative effect on mean SCC was associated with a prep time of less than 10 seconds and a prep-lag time of less than 30 seconds. Roark et al. (1952) reported prepping for 5-50 seconds and fore milking each teat four full hand squeezes resulted in maximum milk let-down. Jurco and Frtus (1974b), using Slovak spotted cattle, found the optimum prep time was 40 seconds. Prepping less than 40 seconds increased milking time by two minutes and decreased milk yield 4-15%. Prep-lag times of less than 60 seconds resulted in shorter average milking times and no reduction in milk yield (Fryman and Albright, 1962; Jurco and Frtus, 1974a). Prep-lag times greater than four minutes resulted in significantly ($p < .01$) longer average milk time, decreased milk yield, and decreased butterfat as measured in both pounds and percent (Miller and Petersen, 1941; Roark et al., 1952; Elliott, 1961; Fryman and Albright, 1962; Jurco and Frtus, 1974a).

In our analysis of milking hygiene practices, the effects of various methods of udder washing, udder drying, teat dipping or spraying, and rinsing of teat cups between cows on mean somatic cell count were examined. Washing the udder prior to milking had a significant ($p < .0002$) effect on mean SCC. Significantly ($p < .01$) lower mean SCC were associated with herds that washed the udder with a paper towel using water containing a sanitizer before milking. Spraying the udder with running water and washing the udder with a common rag or sponge with water containing a sanitizer had a non-significant ($p < .20$), but numerically negative effect on mean SCC. Significantly ($p < .006$) higher positive mean SCC were associated with herds washing the udder with a common rag or sponge using water alone. These findings agree with Schultz (1977), who found use of individual paper towels for udder washing lowered SCC, and Neave and Oliver (1962), who reported washing udders with a common cloth significantly increased the chance of infection.

Drying the udder after washing, in general, did not have a significant ($p < .30$) effect on mean somatic cell count. Drying the udder after washing only had a significant ($p < .05$) effect on mean somatic cell count when used in combination with either washing the udder with individual paper towels using water containing a sanitizer, or spraying the udder with running water. In these two cases, mean SCC was lower when udders were dried than when udders were not dried. The effect was greater with the latter udder washing procedure. These findings suggest that, while udder drying is not as harmful as some reports have concluded (Hoare et al., 1979), it may not be

as widely beneficial as others have concluded (Moxley et al., 1978).

Dipping or spraying teats with a sanitizer or disinfectant after milking was associated with significantly ($p < .10$ and .03) lower mean somatic cell counts. These results support previous studies on effectiveness of teat dipping in reducing new infection rate and SCC (Neave et al., 1969; Langlois and Pyles, 1975; Grant et al., 1976; Moxley et al., 1978; Hoare et al., 1979; Sheldrake and Hoare, 1980a, 1980b; Hoare and Huchenson, 1980). Consideration of specific teat dip brand names increased amount of variation in mean SCC accounted for by the model 9.98%, indicating an effect of specific brands on mean SCC. In this survey, herds using Bio-gard®, Dairy Mate®, or Klenzade® were associated with significantly ($p < .05$) lower mean SCC, while herds using either Blu-kote® or Chapless® were associated with significantly ($p < .05$) positive mean SCC. No other brand of teat dip was found to have a significant ($p < .20$) effect on mean SCC. However, due to the nature of this study and its lack of controlled conditions, these findings cannot and should not be interpreted as either recommending or condemning the brands of teat dip mentioned. Before one brand of teat dip can be recommended over another, carefully controlled experiments would be required.

As Neave et al. (1969) and Moxley et al. (1978) had previously found, we likewise found rinsing of teat cup liners between cows had no significant ($p < .20$) effect on mean somatic cell count as a single practice. However, rinsing of teat cup liners between cows was associated with numerically lower mean SCC when used in combination

with certain udder washing methods. The greatest negative effect of rinsing teat cup liners on mean SCC occurred when udders were washed with a common rag or sponge plus water alone or water containing a sanitizer. This suggests rinsing of teat cup liners between cows may be able to counteract some of the negative effects of these two udder washing methods on mean SCC; probably, by reducing number of pathogens transferred between cows (Neave, 1971; Bushnell et al., 1978; Ruffo and Sangiorgi, 1980).

The practices of washing the udder with an individual paper towel and water containing a sanitizer or spraying the udder with water containing a sanitizer reduced mean SCC when teat cup liners were rinsed between cows as compared to the same washing procedures when teat cup liners were not rinsed between cows. A possible explanation for these results is that there is an additive effect between washing the udder with a sanitizing solution and rinsing teat cup liners between cows. Both udder washing methods probably reduce number of bacterial organisms on teat skin and addition of rinsing teat cup liners between cows further reduces incidence of infection by eliminating or reducing infections caused by Streptococcus agalactiae, beta hemolytic Staphylococci, Coliforms, and Mycoplasma depending on rinsing method and type and concentration of sanitizer used (Neave, 1971; Bushnell et al., 1978; Ruffo and Sangiorgi, 1980).

As a result of this study, a modified full milking hygiene program should be followed to achieve maximum decrease in somatic cell counts. This program would include washing teats with a paper

towel using a sanitizing solution (minimum one paper towel per cow), followed by drying teats with another paper towel. The washing and drying procedure should last 20-30 seconds, and the milking unit should be attached 30-60 seconds later. At the completion of milking and removal of teat cups, teats should be dipped in an effective teat dip. Though this study found spraying of teats to be equally effective as dipping, dipping is preferred since there is less chance of "missing" one side of the teat. Provided the sanitizing solution is changed frequently enough to maintain effective germicidal levels, teat cup liners should be rinsed between cows.

Treatment of Mastitic Cows.--The management and treatment of mastitic lactating cows was examined. Herds where mastitic cows were milked before the rest of the herd were associated with significantly ($p < .05$) lower mean somatic cell count. However, only 0.72% of the herds studied were in this category and were evenly divided between bucket milking systems and milking systems with weigh jars making it possible for the portion of the system that came into contact with mastitic milk to be cleaned prior to milking of the rest of the herd. There were no other significant ($p < .20$) differences in mean SCC among the other practices concerning milking of mastitic cows.

While there were no significant ($p < .20$) differences among the twenty one types of treatments used for lactating cows with mastitis, how treatment was administered and interaction of treatment type and administration did have a significant effect on mean somatic cell count. Only herds that regularly treated clinically infected cows intravenously had significantly ($p < .10$) higher mean SCC than

herds using other routes of administration. This is not surprising considering successful treatment depends on effective passage of drug from blood to foci of infection to milk (MacDiarmid, 1978; Ziv, 1980a; Ziv, 1980b). Ziv (1980a) reported after intravenous injection with 20 mg/kg of penicillin G, tetracycline, or. spiramycin in lactating ewes less than .0003%, .006%, and 4.6% respectively, of the total amount of drug injected was recovered in milk during 36 hours after treatment. There were no significant ($p < .20$) differences among other methods of treatment administration. Two treatment type-method of treatment administration interactions had a significant effect on mean SCC. The practice of intramuscular administration of individually packaged commercial antibiotic or individually packaged antibiotic prepared by a veterinarian to clinically infected cows was associated with significantly ($p < .08$ and .02) higher mean SCC. Again, these results are not surprising due to the previous work of MacDiarmid (1978) and Ziv (1980a, 1980b). Additionally, Ziv (1980a) reported after intramuscular injection (20 mg/kg) of one of twenty-eight commonly used antibiotics in lactating cattle, recovery of drug in milk ranged from .001% for cloxacillin, cephaloridine, streptomycin, and polymixin B plus colistin to 2.40% for spiramycin.

Dry Treatment Policy.--Effect of dry treatment policy on mean somatic cell count was examined since the bovine udder is most susceptible to infection at the beginning of the dry period because bacteria are able to penetrate the streak canal more easily (Cousins et al., 1980) and administration of dry treatment has been shown to reduce new infection rate and cure existing infections (Oliver et al.,

1962; Natzke, 1971; Meaney and Nash, 1977; Rindsig et al., 1978). Significantly ($p < .0001$) lower mean SCC occurred in herds that dry treated all cows compared to any other dry treatment program. Dry treating either less than half of the herd of more than half of the herd had a numerically negative, but non-significant ($p < .20$) mean SCC. Previous reports found new infection rate increased as number of cows dry treated decreased from all cows to selective dry treatment programs to no cows dry treated (Natzke, 1971; Rindsig et al., 1978) while cure rate of existing infections among dry treated cows ranged from 85.4% to 100% (Meaney and Nash, 1977; Rindsig et al., 1978). Reduction and elimination of infection result in lower SCC. However, the current study found significant differences in mean SCC when dry cow treatment product was considered. Herds using Biodry®, Biodry® and Masterdry®, Dry Clox® and Tomorrow®, Orbenin-DC®, Orbenin-DC® and Quartermaster®, Quartermaster®, or Tomorrow® had numerically ($p < .20$) lower mean SCC. Significantly ($p < .01$) higher mean SCC were associated with use of Biogard®, Dairy Clox®, Impro®, and Oxytet 50®. Since it has been shown effectiveness of dry treatment is influenced not only by number of cows treated, but by teat dipping practices, the effect of the interaction of number of cows dry treated, the dry cow treatment product used, and whether or not teat dip was used after milking on mean SCC was examined (Natzke, 1971; Eberhart and Buckalew, 1972; Schultz, 1977; Rindsig et al., 1978; Hoare et al., 1979). Significantly ($p < .01$) lower mean SCC were associated with teat dipping and dry treatment of all cows with Biodry®, Biodry and Dry Clox®,

Orbenin-DC®, or Quartermaster®. Consideration of specific teat dip brands increased amount of variation in mean SCC accounted for, by the model 7.14%, indicating an effect of specific brands on mean SCC. In this analysis, significantly lower mean SCC were associated with teat dipping with Blu-gard® and dry treating all cows with either Biodry®, Orbenin-DC®, Quartermaster® or Tomorrow® (all $p < .01$), or Biodry® and Dry Clox® ($p < .05$), teat dipping with Uddergard® and dry treating all cows with Biodry® ($p < .03$), teat dipping with Bovadine® and dry treating all cows with Biodry® ($p < .08$), and teat dipping with Nolvasan® and dry treating all cows with either Biodry® or Orbenin-DC® ($p < .15$). These findings are similar to those of Eberhart and Buckalew (1972) and Hoare et al. (1979) in that lowest SCC were found in herds that were both teat dipping and dry treating all cows, except in the current study specific brands of teat dip and dry cow treatment product were considered. However, this should not be interpreted as an evaluation or recommendation of specific products since the analysis was not based on a scientifically designed and controlled study. It should also be noted the type and prevalence of organism(s) in each herd involved in this study is unknown. Thus, the effectiveness or appropriateness of a given product in a given herd cannot be determined.

Maintenance of Free Stalls.--No previous reports were found regarding how the maintenance of free stalls affected somatic cell count. Maintenance of free stalls can be divided into three categories; addition of bedding, raking, and cleaning down to the

base and then leveling the base. In this discussion, only the effect of the interaction of type of free stall, bedding material, and the maintenance practice on mean SCC will be considered. When considering this interaction, it is important to remember the primary concern is the bacterial number established in the bedding. If the stall contains standing water, excessive organic matter, or bedded with fine textured organic bedding, there is an increased risk of the bacterial numbers established in bedding to exceed a critical value and; thus, increase infection rate.

In the analysis of each of the three free stall maintenance categories, totally cemented free stalls were superior in terms of lower mean somatic cell counts. Totally cemented free stalls facilitate removal of contaminated bedding because of their slope. Bedding, such as chopped straw, sand, or sawdust, and manure on top of it slide into the gutter or alley whenever a cow exits or shifts around in a stall.

Seven combinations of type of free stall, bedding material, and addition of bedding had a significant ($p < .05$) effect on mean somatic cell count (Table 10). The first six interactions involved totally cemented free stalls, and six of seven interactions involved a fine textured bedding. Optimal bedding addition frequency appeared to be dependent on fineness of bedding. Very fine (i.e. sand) bedding, which is removed more easily, needs to be replaced more frequently.

Three combinations of type of free stall, bedding material, and raking had a significant ($p < .05$) positive effect on mean

TABLE 10.--Effect of Free Stall Housing System, Bedding Material, and Addition of Bedding on Mean SCC.

Free Stall Housing	Bedding Material	Addition of Bedding	$\hat{y}$ (in 100,000 cells/ml) (est. effect on mean SCC)
Totally cemented	chopped straw	every other week	-3.676
Totally cemented	sand	once a week	-2.065
Totally cemented	sawdust	every other week	-1.805
Totally cemented	sawdust	once a month	-1.673
Totally cemented	long straw	once a week	-1.559
Totally cemented	sawdust	every other month	-1.537
Half cemented	chopped straw	every other week	-1.126

somatic cell count. In numerical order, least positive effect on mean SCC to greatest, these were totally cemented free stalls bedded with chopped straw and raked every day, totally cemented free stalls bedded with sawdust and raked every day, and half cemented free stalls bedded with sawdust and raked every other week. It is not possible to satisfactorily explain these results at present. Perhaps raking is influenced by other factor(s) not included in the current model, such as addition of bedding, thoroughness of raking, etc.

There were two significant ($p < .05$) free stall, bedding material, and cleaning free stall down to base and leveling interactions on mean somatic cell count. Totally cemented free stalls bedded with sawdust and cleaned and leveled once a month were associated with significantly ($p < .05$) lower mean SCC. Significantly ($p < .05$) higher mean SCC was associated with totally cemented free stalls bedded with sawdust and cleaned and leveled once a year. Francis et al. (1981) reported increased levels of E. coli in sawdust bedded free stalls as environmental temperatures decreased and cows spent more time lying in free stalls. Additionally, E. coli levels were higher in free stalls of high producing cows as compared to low producing cows. Thus, more frequent cleaning probably reduces the number of pathogens below critical levels. Due to the size of the data bank, it was not possible to analyze for interaction effects of addition of bedding, raking, and/or cleaning-leveling of free stalls on mean somatic cell count.

Purchase of Replacement Animals.--Herds that had purchased any lactating cows in the past two years had significantly ($p < .0009$) higher mean somatic cell counts than herds that had not made any purchases of lactating cows. Considering the high probability of purchasing an infected lactating cow; especially, if the purchased cow does not have a complete health record or is not tested for mastitis at purchase, the above results are as expected.

GENERAL DISCUSSION

Somatic cell count has been determined to be a reliable indicator of mastitis (Waite and Blackburn, 1957; Schalm and Lasmanis, 1968; Schalm et al., 1971; Reichmuth et al., 1974; Reichmuth, 1975; Westgarth, 1975; Bodoh et al., 1976; Schultz, 1977; NMC, 1978; Eberhart, 1979; McDermott et al., 1981; Jones et al., 1982). However, SCC has been shown to be influenced by factors other than infection. This study was designed to examine the effect of cow related, farm, and managerial factors on SCC.

Cow factors affecting somatic cell count can be divided into three categories: 1) physiological factors, 2) lactation product factors, and 3) environmental factors. SCC increased as physiological factors of age, parity, and stage of lactation increased. This increase is probably due to increased exposure to infection and/or increased incidence of subclinical mastitis as each of the physiological factors increase. Examination of lactation product factors found as SCC increased, daily milk production (yield) decreased and percent fat increased. The decrease in daily milk production is due to damage of mammary cells, the stimuli which is causing increased SCC, while increase in percent fat is a concentration effect. While SCC increased as environmental factor of herd size increased, this increase was not biologically significant. Analysis of seasonal trends in SCC found a yearly peak in SCC occurred in June and a

secondary peak occurred in October. While these peaks are probably the result of interaction of several factors, environmental temperature and precipitation most likely account for majority of the observed phenomena. However more detailed and controlled research is needed.

Farm factors possibly affecting somatic cell count were divided into two categories: 1) milking system and 2) housing system. Only high milk pipeline milking parlor milking systems were associated with significantly lower mean SCC. This result is different from previous reports of low milk pipeline parlors having lowest SCC (Downey et al., 1977; Schultz, 1977). However, LeDu (1980) reported that with a low milk pipeline milking system, more vacuum fluctuations occurred during milking, especially, at end of milking, and transmission of vacuum fluctuations from a cluster to the nearest cluster occurred more easily. Lower mean SCC were also associated with line vacuum levels of 11.5 inches Hg and 13.5 inches Hg for low and high milk pipeline systems, respectively, complete loop vacuum line design, and single pulsation. Pulsation ratio of 50:50 was associated with lowest mean SCC over widest vacuum level range. This is probably because 50:50 ratio provides adequate massage and thus, there is minimal teat injury and potentially less spread of infection. Pulsation ratios of 55:45 and 60:40 were also associated with low cell counts over a wide range of vacuum levels, but not as wide a range as 50:50 pulsation ratio. Investigation of interaction of milking system and vacuum controller on mean SCC found significant interactions of brand of controller and barn pipeline and parlor milking systems. Why only certain brands of vacuum controller had

an effect is probably due to load change in weighted sleeve value and weighted level controllers which are less sensitive to vacuum fluctuations, while installation, lactation, and maintenance practices have a greater effect on the more sensitive spring loaded diaphragm controllers than load charge. Lowest SCC were associated with five milking units on 1½, 2, or 2½ inch milk pipelines and eight milking units on 3 inch milk pipelines. Brand of inflation being used probably has an effect on SCC, but controlled studies are needed to determine the effect of each brand of inflation on mean SCC. Mean SCC was not affected by the presence or absence of automation, unless Surge VSO® take-offs were in use. Higher mean SCC associated with Surge VSO® may be because of one or more of the following: 1) claw weight, 2) lack of support for claw, and/or 3) possible interaction of inflations being used and Surge VSO®. Material of vacuum line construction, vacuum line size, vacuum pump capacity, and interactions among various pulsation ratios and single or alternate pulsation did not have a significant effect on mean SCC.

How milking cow housing system and maternity facilities affected somatic cell count was examined. Lowest mean SCC were associated with sand based free stalls. There were significant interactions between certain housing systems and bedding materials. This indicates some housing system-bedding type interactions may keep bacterial numbers below critical infection levels, while others either enhance or promote bacteria growth. However more detailed controlled research is needed to support or refute this possibility. A general examination of maternity facilities found herds using

maternity or box stalls bedded with either chopped or long straw had lower mean SCC. When season of calving was also considered, different facilities were better during different seasons, but multiple calving facilities, one for each season, are not practical. Therefore, best year round calving facility appeared to be two maternity or box stalls bedded with long straw. These results confirm the importance of proper calving facilities as a mastitis control measure.

Five areas directly controlled by the herd manager are milking hygiene practices, treatment of mastitic cows, dry cow treatment policy, free stall maintenance, and purchase of replacement animals. Lowest mean SCC were associated with the following milking hygiene practices: washing teats with individual paper towel using water containing a sanitizer, drying with a separate paper towel, teat dipping with an effective teat dip immediately after removal of milking machine, and rinsing or backflushing of teat cups between cows. Prepping should last 20-30 seconds and milking machine attached 30-60 seconds after completion of prepping. While we found herds milking mastitic cows first were associated with lowest mean SCC compared to other milking practices, these herds had either bucket milking systems or milking systems with jars, making it possible to totally clean the portion of the system that was in contact with infected milk, and only accounted for 0.72% of total population studied. In general, herds where mastitis was primarily treated by intravenous or intramuscular injection of antibiotics had higher mean SCC. These results are not surprising considering

successful treatment depends on effective passage of drug from site of injection to blood to foci of infection to milk (MacDiarmid, 1978; Ziv, 1980a; Ziv, 1980b). Dry treatment policy is important since the bovine udder is most susceptible to infection at the beginning of the dry period because bacteria are able to penetrate the streak canal more easily (Cousins et al., 1980) and administration of dry treatment has been shown to reduce new infection rate and cure existing infections (Oliver et al., 1962; Natzke, 1971; Meaney and Nash, 1977; Rindsig et al., 1978). Lower mean SCC occurred in herds that dry treated all cows compared to any other dry treatment program. However, effectiveness of dry cow treatment policy was influenced not only by number of cows in herd treated, but by dry cow treatment product used, whether or not teat dipping was done during lactation, and brand of teat dipping product used, if any.

The prime managerial concern with free stall maintenance is keeping bacterial numbers in bedding below critical infection level. Lowest mean SCC was associated with totally cemented free stalls, bedded with a fine textured bedding; i.e. sand, sawdust, chopped straw, etc., raked every day and cleaned down to the base once a month. Totally cemented free stalls facilitate removal of contaminated bedding, especially fine textured bedding, because of its slope. Thus, reducing bacterial numbers. However Francis et al. (1981) has indicated weather may influence bacterial numbers in free stall bedding. Therefore more research should be done in the

area of free stall maintenance and factors affecting bacterial numbers in bedding.

Since many of the lactating cows sold for dairy purposes, i.e. non-slaughter, are sold because of low production and/or mastitis, it is not surprising herds purchasing lactating cows have higher mean SCC than herds not making such purchases. Purchase of lactating cows probably involves unknowingly purchasing a new pathogen in a majority of cases. The new pathogen is then spread to other cows in the herd, elevating their cell counts.

In conclusion, this study has shown somatic cell counts to be influenced by many other factors besides infection and/or injury. Fortunately, many of these factors can and should be controlled by the dairy farmer. These factors include proper operation and maintenance of all components of the milking system, following proper milking hygiene practices, proper treatment of lactating cows with mastitis, dry treating all cows with an effective product at end of lactation, providing and maintaining adequate calving facilities and housing for lactating cows, and limited, if any, purchase of lactating cows as herd replacements. Additional practically oriented research is needed in many of these areas, especially in area of brand name comparisons. Further research is also needed in how environmental parameters; i.e. air temperature, precipitation, radiant heat, wind chill, etc., affect not only SCC, but other farm conditions, i.e. housing, bedding, etc.

APPENDICES

APPENDIX A

SURVEY QUESTIONNAIRES

Herd No. _____

MILKING SYSTEM

1. What type of milking system are you presently using? (please check the appropriate boxes).

_____ bucket
_____ barn pipeline
_____ parlor

_____	herringbone	_____	No. stalls
_____	side opening	_____	No. stalls
_____	rotary	_____	No. stalls
_____	trigon	_____	No. stalls
_____	polygon	_____	No. stalls

2. What make of equipment do you have?

_____	Surge	_____	ChoreBoy	_____ Other
_____	DeLaval	_____	Universial-StaRite	
_____	BouMatic	_____	Zero	
_____	Germania	_____	Conde	
_____	Westfalia	_____	Bodmin	

3. How many milking units does this system have? _____

4. Do you have weigh jars?

_____ yes
_____ no

5. Do you have any of the following automation?

_____	DeLaval "300"	_____	BouMatic "Take-offs"
_____	DeLaval "200"	_____	Germania "Take-offs"
_____	DeLaval "Trade-offs"	_____	Universal-StaRite "Take-offs"
_____	Surge QTO	_____	Other, Specify_____
_____	Surge VSO	_____	None

6. IF YOU HAVE ANY AUTOMATION IN YOUR SYSTEM, is it powered by

_____ the milking system's vacuum pump.
_____ a separate vacuum pump.
_____ air compressor
_____ other, please specify _____

7. How many inlets are there to the receiver jar?

_____ one
_____ two
_____ three or more

8. In order to move milk to the bulk tank, does your system have a

_____ milk pump on the receiver?
_____ releaser between the receiver and bulk tank?
_____ vacuumized bulk tank?
_____ milk transfer station (veyor)?
_____ other, please specify _____.

9. What is the horsepower of the vacuum pump(s)? _____ _____ _____

10. What is the vacuum level of your system on the vacuum line or by the trap or receiver?

_____ inches

11. What make of vacuum regulator(s) do you have?

_____ Surge Oil Weight
_____ Surge (Equalizer)
_____ DeLaval (Senior)
_____ Alfa Laval (Servo)
_____ Germania
_____ Other, please specify _____
_____ Westphalia (Vac-U-Rex)
_____ Sentinel
_____ BouMatic, Universal, StaRite, or Zero Balance Arm
_____ ChoreBoy

12. How many vacuum regulators does your system have? _____

13. Where are the vacuum regulators located? (check more than one if more than one location)

_____ on the balance tank
_____ on the line between the balance tank and the receiver
_____ on the pulsation line
_____ by the moisture trapon the receiver
_____ by the wash trap
_____ other, please specify _____

14. What is the size (diameter) of the pulsation or vacuum line?

_____ 3/4 inch
_____ 1 1/4 inches
_____ 1 1/2 inches
_____ 2 inches
_____ 3 inches

15. The pulsation or vacuum line is

_____ galvanized pipe.
_____ plastic (PVC).

16. The pulsation or vacuum line is a

_____ dead end.
_____ looped T.
_____ complete loop back to the balance tank.

17. What is the pulsation ratio of your equipment?

_____ 50:50
_____ 55:45
_____ 60:40
_____ 65:35
_____ 70:30
_____ 50:50 front, 60:40 rear

18. Is the pulsation alternating (two teats at a time) or single (all four teats at the same time)?

_____ alternating
_____ single

19. How often is the pulsation or vacuum line cleaned?

_____ after each milking
_____ once a month
_____ once every three months
_____ other, please specify _____
_____ once every six months
_____ once a year
_____ has never been cleaned

20. What is the height of the milk pipeline?

_____ below the cow's udder
_____ 3 to 5 feet above the cow's udder
_____ 5 to 8 feet above the cow's udder
_____ more than 8 feet above the cow's udder

21. What is the size (diameter) of the milk line?

_____ 1 1/2 inches
_____ 2 inches
_____ 2 1/2 inches
_____ 3 inches

22. What make of claw do you have?

_____ DeLaval
_____ BouMatic
_____ Universal
_____ Zero
_____ Sta-Rite
_____ Bodmin
_____ Surge
_____ Germania
_____ ChoreBoy
_____ Other, please specify _____

23. What is the claw made of?

_____ plastic
_____ stainless steel

24. What style of inflation do you have? (for example; DeLaval 01, BouMatic R1)

_____

25. Who is the manufacturer or source of your inflations? (for example, MMPA, Maes, DeLaval)

_____

26. How many days are inflations used before they are discarded? _____ days.

27. Are sets of inflations

_____ washed, rested, and reused?
_____ used continuously before discarding?

28. How often is the system serviced by a qualified equipment specialist or representative?

_____ more than twice a year
_____ twice a year
_____ once a year
_____ never
_____ other, please specify _____

29. Are these calls mainly

_____ service contract?
_____ emergency service?

30. Who do you consult when you want information on milking systems? If more than one, please rank in order of importance with 1 = first.

_____ county agent
_____ dairy extension specialist
_____ dairy fieldman
_____ equipment company representative
_____ veterinarian
_____ other
_____ equipment company advertising
_____ friends and neighbors
_____ regulatory officials

31. Has the milking system been changed in the last year?

_____ yes
_____ no

32. IF YES, what was the nature of this renovation?

_____
_____
_____

Herd No. _____

MILKING PRACTICES

1. How many persons are involved in milking the herd? _____

2. What is the position of the milker? (If more than one milker, please indicate how many persons are at each position.)

_____ owner-operator
_____ herdsman
_____ full time hired employee
_____ part time hired employee
_____ family member of owner-operator
_____ other, please specify _____

3. If the milker(s) are employees, does that person have any additional non-milking related responsibilities?

_____ yes
_____ no

4. Do you (please check)

_____ use a prep stall
_____ prep at cowside
_____ no prepping

5. Which of the following items are part of your normal milking procedure? (please check the items which apply)

NORMAL PREPPING PROCEDURE

_____ wash udder with common sponge or rag before milking
_____ wash udder with paper towel(s) before milking
_____ spray udder with running water before milking
_____ include a sanitizer in udder wash or spray
NAME SANITIZER _____
_____ dry udder after washing

NORMAL PRE-MILKING PROCEDURE

_____ use a strip cup before machine attachment
_____ do a California Mastitis Test (CMT) on each cow before milking
_____ do a CMT on cows with abnormal milk before milking

NORMAL POST-MILKING PROCEDURE

_____ machine strip less than 1 minute before removing milkers
_____ machine strip more than 1 minute before removing milkers
_____ dip teats after milking
NAME OF TEAT DIP _____
_____ spray teats after milking
NAME OF TEAT SPRAY _____
_____ rinse teat cups in sanitizer between cows

6. Total time spent in prepping each cow is:

_____ less than 10 sec.
_____ 10 to 20 sec.
_____ 20 to 30 sec.
_____ more than 30 sec.

7. Amount of time between prepping and attachment of milking unit is:

_____ less than 30 sec.
_____ 30 to 60 sec.
_____ 1 to 2 minutes
_____ more than 2 minutes

8. Total milking time for herd: _____ hours.

9. In your herd, cows with mastitis and treated cows are milked (please check one).

_____ before the rest of the herd
_____ with the rest of the herd
_____ after the rest of the herd
_____ on special equipment not part of the regular milking system
_____ by hand
_____ not at all

10. How would you rate your herd's teat end condition?

_____ acceptable
_____ fair
_____ eroded

11. Who do you consult when you want information on milking practices? If more than one, please rank in order of consultation, 1 = first.

_____ county agent
_____ commercial companies
_____ dairy extension specialist
_____ dairy fieldman
_____ friends, neighbors
_____ state sanitarian
_____ veterinarian
_____ other

Herd No. _____

MILKING HERD MANAGEMENT POLICY

1. What is the dominate breed of dairy cattle on your farm?

   _____ Holstein
   _____ Jersey
   _____ Brown Swiss
   _____ Ayshire
   _____ Other, please specify _____

2. How many total cows (milking and dry) do you have? _____cows

3. How many cows are you currently milking? _____ cows

4. As of your last DHIA report, what was your rolling herd average? _____ lbs.

5. Is your herd split according to production levels?

   _____ Yes
   _____ No

   IF YES, HOW IS THE HERD SPLIT? _____

   _____

6. Milking cows are housed in

   _____ stanchions with dimensions of _____.
   _____ comfort stalls with dimensions of _____.
   _____ half cement floor free stalls with dimensions of _____.
   _____ totally cemented floor free stalls with dimensions of _____.
   _____ group pen(s), manure pack, with _____square feet per cow.
   _____ pasture in summer.

IF YOU HAVE FREE STALLS, PLEASE ANSWER THE FOLLOWING QUESTIONS. IF NOT, PLEASE GO TO QUESTION 10.

7. How often is bedding added to the free stalls?

   _____ once a week
   _____ once every other week
   _____ once a month
   _____ once every other month
   _____ once every three to four months
   _____ other, please specify _____

8. How often are the free stalls cleaned down to the base of the stall and the base then leveled off?

   _____ once a month
   _____ once every other month
   _____ once every three months
   _____ once every four months
   _____ once every six months
   _____ once a year
   _____ other, please specify _____

9. How often are the free stalls raked?

_____ every day
_____ every other day
_____ every three days
_____ once a week
_____ once every other week
_____ other, please specify _____

10. What type of bedding are the milking cows on?

_____ sand
_____ sawdust
_____ chopped straw
_____ long straw
_____ corn cobs or corn stalks
_____ hay
_____ other, please specify _____

11. Have you purchased any lactating cows in the past two years?

_____ Yes
_____ No

12. IF YES, were they checked for mastitis?

_____ Yes
_____ No

IF YOU CHECKED A NEWLY PURCHASED COW FOR MASTITIS, HOW DID YOU DO IT? _____

_____

13. When a cow has clinical mastitis (abnormal udder and/or milk), when is she treated?

_____ immediately after milking
_____ after consultation with a veterinarian
_____ after the results of a milk culture sample are received
_____ other time, please specify _____

14. Who treats a cow if she has clinical mastitis?

_____ owner-operator
_____ family member
_____ herdsman
_____ employee (milker)
_____ veterinarian
_____ other, please specify _____

15. When a cow has clinical mastitis, what is used to treat the cow?

_____ individually packaged commercial syringes or canulas.
_____ individually packaged syringes or canulas prepared by a veterinarian.
_____ individually packaged syringes or canulas containing an autogenous vaccine developed especially for your herd by a veterinarian.
_____ common syringe and multiple dose commercial drug.
_____ common syringe and multiple dose drug mixed by a veterinarian.
_____ common syringe and multiple dose autogenous vaccine developed especially for your herd by a veterinarian.
_____ other, please specify _____.

16. Where is the treatment for a clinically infected cow administered?

_____ intramammary
_____ intramuscularly
_____ sub-cutaneously
_____ intravenously
_____ intraperitoneal

17. I dry treat

_____ no cows.
_____ all cows.
_____ less than half the herd.
_____ more than half the herd.
_____ only cows that had mastitis that lactation.

18. IF YOU ARE DOING ANY DRY TREATING, what product are you using?_____
_____

19. Do you keep a record of mastitis cases for

_____ all cows?
_____ some cows?
_____ only problem cows?
_____ no cows?

20. Do you keep a record of the types of mastitis treatment used?

_____ Yes
_____ No
_____ Sometimes

21. How many cows were treated for mastitis during the last twelve months? _____ cows.

22. How many cows were culled primarily because of mastitis during the last twelve months? _____ cows.

23. Have you made any changes in your herd management policy, herd housing facilities or herd size in the last two years?

_____ Yes
_____ No

IF THERE HAVE BEEN CHANGES, PLEASE BRIEFLY OUTLINE THOSE CHANGES?

_____

_____

Herd No. _____

DRY COW AND CALVING PRACTICES

1. Are the dry cows separated from the milking herd?

_____ Yes
_____ No

2. Dry cows are housed in

_____ stanchions with dimensions of _____.
_____ comfort stalls with dimension of _____.
_____ half cement floor free stalls with dimensions of _____.
_____ totally cemented floor free stalls with dimensions of _____.
_____ group pen, manure pack, with _____ square feet per cow.
_____ pasture in summer.

3. What type of bedding are dry cows on?

_____ sand
_____ sawdust
_____ long straw
_____ chopped straw
_____ corn cobs or corn stalks
_____ hay
_____ other, please specify _____

4. Do 2 year old cows calve in same maternity stalls or area as mature cows?

_____ Yes
_____ No

5. During the fall to spring period of the year, most calvings occur (please check one)

_____ in comfort or box stalls in a barn.
_____ in the cow's normal stall.
_____ in the cow's group pen.
_____ in an out-of-door pen with shelter.
_____ in an out-of-door pen with no shelter.
_____ in a pasture or field.
_____ where ever the cow is a parturition.
_____ other, please specify _____

6. During the spring to fall period of the year, most calvings occur (please check one)

_____ in comfort or box stalls in a barn.
_____ in the cow's normal stall.
_____ in the cow's group pen.
_____ in an out-of-door pen with shelter.
_____ in an out-of-door pen with no shelter.
_____ in a pasture or field.
_____ where ever the cow is at parturition.
_____ other, please specify

7. If you use box stalls for calving purposes, how many box stalls do you have?_____

8. The bedding in the confined maternity area is

_____ corn cobs or corn stalks.
_____ hay.
_____ chopped straw.
_____ long straw.
_____ sand.
_____ sawdust.
_____ wood chips.
_____ none.
_____ other, please specify _____.

9. When are calves removed from the cow?

_____ immediately after birth
_____ within 2 hours after birth
_____ 2 to 6 hours after birth
_____ 6 to 12 hours after birth
_____ 12 to 24 hours after birth
_____ 24 to 36 hours after birth
_____ other, please specify _____

10. Are the calves fed

_____ fresh milk from mastitic cows?
_____ fermented milk from mastitic cows?
_____ no milk from mastitic cows?

11. Calves (birth to 3 weeks) are housed in

_____ individual calf hutches.
_____ individual pens or stalls in a barn.
_____ group pens in a cold barn.
_____ group pens in warm barn.
_____ group pens out-of-doors with shelter.

11. Who do you consult when you want information on dry cow and calving practices? If more than one, please rank in order of consultation, 1 = first.

	calving	dry cow
county agent	_____	_____
commercial companies	_____	_____
dairy extension specialist	_____	_____
dairy fieldman	_____	_____
friends/neighbors	_____	_____
veterinarian	_____	_____
other	_____	_____

12. Have there been any changes in either dry cow or calving facilities or practices in the last year?

_____ No
_____ Yes, please list the changes

_____

_____

APPENDIX B

PACKAGE GIVEN TO DHI TECHNICIANS

CONTENT: Cover Letter

Copy of Letter Being Mailed to Dairy Farmers

Survey Questionnaires (See Appendix A)

COOPERATIVE EXTENSION SERVICE
MICHIGAN STATE UNIVERSITY and
U.S. DEPARTMENT OF AGRICULTURE COOPERATING

DEPARTMENT OF DAIRY SCIENCE EAST LANSING • MICHIGAN • 48824

Dear DHIA Tester:

WE NEED YOUR HELP....

We are trying to put together a comprehensive picture of the mastitis problem in Michigan. A part of this picture involves a survey of dairymen's management practices and overall farm conditions. Using this information and DHIA production records, we will be able to determine what practices and on-farm conditions exist in Michigan and how they are related to somatic cell count, a reliable indicator of mastitis. We will then be better able to lower the level of mastitis in Michigan dairy herds by identifying those practices and conditions that lower the somatic cell count.

In order to obtain as complete a picture as possible of Michigan dairy farms, we have developed four questionnaires covering different aspects of farm operation. However to obtain any meaningful results, we need a response rate of at least 70% of the questionnaires. This is where your help is so critically needed.

In the next three weeks, you will be receiving the first set of two questionnairs and a cover letter explaining them. We ask that you take five to ten minutes with each dairyman you visit who is on the somatic cell count program, and help him complete the questionnaires as accurately as possible. After the questionnaires are completed, just mail them back to MSU in the envelope that will be provided. The second set of questionnaires will arrive approximately three weeks after the first set, and we ask that you again help the dairyman complete the questionnaires as accurately as possible.

The questionnaires will <u>not</u> be a "pop-quiz" in the dairyman's eyes. In approximately two weeks, Michigan dairymen will be receiving a letter explaining the questionnaires and how they will be used. You will also be receiving a copy of this letter.

Questionaire answers are <u>totally confidential</u>. The herd number is only recorded so production and somatic cell data can be correlated with questionnaire responses. An individual farm or group of farms will <u>never at any time</u> be referred to by either name or herd number.

We are very grateful for your help with these questionnaires.

Sincerely,

Patricia Potter

Roger Mellenberger

MICHIGAN STATE UNIVERSITY

COLLEGE OF AGRICULTURE AND NATURAL RESOURCES
DEPARTMENT OF DAIRY SCIENCE · 240 ANTHONY HALL
TELEPHONE (517) [illegible]

EAST LANSING · MICHIGAN · 48824

Dear Michigan Dairyman:

WE NEED YOUR HELP TO LOWER THE INCIDENCE OF MASTITIS IN MICHIGAN.

We are studying management practices and on-farm conditions in relation to the somatic cell count, a reliable indicator of mastitis. Since this information cannot be obtained from campus research, we need your help to determine what the current management practices and farm conditions are in Michigan. We have developed a four part survey questionnaire on milking systems, milking practices, milking herd management policy, and dry cow and calving practices. Using this information and DHIA production records, we will be able to determine what practices and on-farm conditions exist in Michigan and how they relate to somatic cell count.

Beginning in May, your DHIA tester will have the first two parts of the survey questionnaire and a release form to allow us to use your DHIA production and somatic cell records. The first two questionnaires concern the milking system and milking practices. Most of the questions only require that you check the response that applies to your own operation. The DHIA tester will be there to help you during the five to ten minutes it will take you to complete the questionnaires. We do ask that you complete the questionnaire and give it back to the tester during his or her visit in May. You will receive the second set of questionnaires on milking herd management policy and dry cow and calving practices from your DHIA tester in June.

During the next few months, questionnaires about the somatic cell count program will be directly mailed to you. These questionnaires ask how you use the somatic cell count and give you a chance to say what you like and dislike about the form and what changes you would like to see in the form.

All questionnaire answers are totally confidential. The herd number is used only to correlate the questionnaire answers with production and somatic cell information. An individual farm will not at any time be referred to by either name or herd number. Your responses will be combined with all other farms completing the questionnaires.

As conclusions are drawn from the analyzed information, we will be sending you Extension Service Fact Sheets so you can begin to consider and make use of the new information you helped develop.

We are deeply grateful for your help with this project to reduce the level of mastitis in Michigan.

Sincerely,

Patricia Potter

Patricia Potter
Research Assistant

Roger Mellenberger

Roger Mellenberger
Extension Specialist

APPENDIX C

LETTER TO MICHIGAN DAIRY FARMERS

MICHIGAN STATE UNIVERSITY

COLLEGE OF AGRICULTURE AND NATURAL RESOURCES
DEPARTMENT OF DAIRY SCIENCE · 230 ANTHONY HALL
TELEPHONE (517) 353-6[illegible]

EAST LANSING · MICHIGAN · 48824

Dear Michigan Dairyman:

WE NEED YOUR HELP TO LOWER THE INCIDENCE OF MASTITIS IN MICHIGAN.

We are studying management practices and on-farm conditions in relation to the somatic cell count, a reliable indicator of mastitis. Since this information cannot be obtained from campus research, we need your help to determine what the current management practices and farm conditions are in Michigan. We have developed a four part survey questionnaire on milking systems, milking practices, milking herd management policy, and dry cow and calving practices. Using this information and DHIA production records, we will be able to determine what practices and on-farm conditions exist in Michigan and how they relate to somatic cell count.

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We are deeply grateful for your help with this project to reduce the level of mastitis in Michigan.

Sincerely,

Patricia Potter

Patricia Potter
Research Assistant

Roger Mellenberger

Roger Mellenberger
Extension Specialist

APPENDIX D

RELEASE FORM

AGREEMENT

The undersigned does hereby warrant ownership and control of a certain herd of dairy cattle, and gives permission to the Dairy Herd Improvement Association (DHIA) to allow Patricia Potter and Roger Mellenberger of Michigan State University Department of Dairy Science to examine the herd's production and somatic cell count records with the provision the records will remain confidential between the Dairy Herd Improvement Association and Patricia Potter and Roger Mellenberger and no reference by either name or herd number will be permitted, published, or otherwise released to the general public at any time. However, the material referred to may be used and otherwise utilized for scientific purposes, including publication, provided said herd's identity is protected from public disclosure.

_____ _____

Date

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BIBLIOGRAPHY

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