INTRAMUSCULAR AND INTRAMAMMARY

TREATMENT OF MASTITIS IN BEEF COWS:

EFFECTS ON UDDER HEALTH AND

GROWTH OF CALVES

By

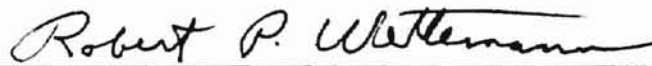CLAY A. LENTS

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INTRAMUSCULAR AND INTRAMAMMARY

TREATMENT OF MASTITIS IN BEEF COWS:

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GROWTH OF CALVES

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CHAPTER I

INTRODUCTION

Mastitis is not often thought of as a problem associated with beef cows. Mastitis is an inflammation of the mammary gland usually caused by an infection. The problem can afflict most mammals including sheep, pigs, and humans, however, mastitis is most commonly a problem in dairy cows. This affliction results in an increase in somatic cell counts with losses of milk production and composition (Janzen, 1970). Other losses include discarded milk, death, veterinary expense, drug expense, and herd replacement cost (Fetrow and Anderson, 1987). Economic losses attributable to mastitis in dairy cows have been estimated to range from $235 million to as great as $2 billion per year in the United States (see Janzen, 1970; Fetrow and Anderson, 1987).

For cow/calf producers, weaning weight of calves is one of the most important factors in maintaining profitability. Weaning weights of calves are influenced by the age and milk production of the cow, calf sex, and forage intake of the calf prior to weaning (Cundiff et al., 1966; Melton et al., 1967; Sowell et al., 1996). However, milk production of the cow is the most important factor influencing weaning weight (Neville, 1962; Rutledge et al., 1971). Therefore, factors that influence milk production of the cow should be of primary concern for beef cattle producers.

In beef cows, mastitis influences milk production of cows and weaning weight of calves. Mastitis increases somatic cell counts which results in less milk production (Watts et al., 1986; Simpson et al., 1995). Mastitis decreases 205 d weight of calves by 7 to 10% (Haggard et al., 1983; Watts et al., 1986). Between 4 and 54% of beef cows are infected with mastitis causing bacteria (Sobari et al., 1976; Watts et al., 1986; Newman et al., 1991; Simpson et al., 1995). Most commonly, infection rates range from 10 to 16% (Hunter and Jeffrey, 1975; Haggard et al., 1987; Duenas et al., 1994). The most recent estimate is that the nation's cow herd contains 103,819,000 head, of which 35,333,000 are beef cows that have calved (United States Department of Agriculture, 1996). If 10% of the cows had mastitis, this would result in a 7% decrease in the weaning weights of calves, and it is possible that 123,665,500 pounds less calf weight would be available for sale. At an average value of $75/cwt., that's a loss of $92,749,125 or $26.25 per cow with mastitis. Yet most beef cow producers demonstrate no concern as long as calf weights are acceptable. If a cow is unable to feed her calf so that it achieves a desirable weight, she is most often culled. If a single treatment of beef cows for mastitis could increase calf weights, substantial monetary losses could be avoided.

The objectives of this study were: to evaluate the effects of mammary health of beef cows on weight gains of calves, and to determine the effects of intramuscular or intramammary treatment of beef cows with antibiotics after calving or at drying-off on udder health and subsequent calf growth.

CHAPTER II

REVIEW OF LITERATURE

Weaning weight of calves is one of the most important factors influencing the profitability of beef cow producers. The cow is generally considered the most important factor that influences weaning weight of the calf. In the dairy industry, mammary health and factors that adversely effect it are known to alter milk production. However, in beef cattle, little attention has been paid to udder health. Therefore, the purpose of this review is to summarize factors influencing mammary health of dairy cows, and to evaluate studies that have evaluated udder health of beef cows.

Calf Growth

Age of Cow

Age of dam influences weaning weight of calves. Calves from older cows weigh more than calves from younger cows, and maximum production is at 6 years of age (Koch and Clark, 1955). A 22 kg increase in calf weaning weights was observed when age of dam increased from 2 to 4 years (Cundiff et al., 1966). Calves that were heavier at birth were heavier at eight months of age (Neville, 1962). However, when age of dam

differences were adjusted for cow weight and milk production, there was no significant influence of age of cow on calf weight at eight months of age (Neville, 1962). Some researchers have found that cow age does not influence calf weaning weight (Christian et al., 1965), however, others found that cow age affected milk production (Drewry et al., 1959), and cows five years of age or older produced more milk than younger cows (Melton et al., 1967). This indicates that the influence of dam age on calf weights could be due primarily to differing abilities of cows to produce milk. Young cows that are still growing would partition some of their nutrient intake for growth. Older cows may have health problems associated with age such as poor teeth. This would make it difficult for them to consume enough feed to meet the requirements for milk production. Milk production is usually decreased and calf weight gain is reduced in young and very old cows.

Sex of Calf

Sex influences calf weights (Christian et al., 1965). Bulls weighed more than heifers at both birth and weaning (2.55 and 12 kg more respectively; Koch and Clark, 1955). Steers weighed 6.64 kg more than heifers at eight months of age (Neville, 1962). Cundiff et al., (1966), found that bulls were 25 kg heavier than heifers and 20 kg heavier than steers at weaning. Bulls gained more rapidly than heifers, and during early lactation, cows nursing bull calves had a .58 kg per day advantage in milk production over cows nursing heifer calves (Melton et al., 1967). However, this advantage was not present during late lactation. Some of the weight advantage of males over heifers may be attributable to

increased milk consumption due to the larger initial size of bull calves, but as heifer calves grow, this advantage could be reduced.

Milk Production of the Cow

The effect of milk production on calf weight has been studied extensively (Koch and Clark, 1955; Drewry et al., 1959; Neville, 1962; Christian et al., 1965; Melton et al., 1967; Rutledge et al., 1971; Beal et al., 1990; Marston et al., 1992; Simpson et al., 1995). Milk production of beef cows accounts for 60 to 66 % of the variation in weaning weight of calves (Neville, 1962; Rutledge et al., 1971). Seventy five and 77 % of the variation in calf weight at one and three months of age was attributable to the milk production of the dam (Drewry et al., 1959). Correlation between milk production and weaning weight of beef calves range from .30 to .81 (Drewry et al., 1959; Neville, 1962; Melton et al., 1967; Clutter and Nielsen, 1987; Marston et al., 1992). Each additional kilogram of milk per day increased calf weaning weights by 7 to 14 kg (Jeffery and Berg, 1971; Boggs et al., 1980), and calves suckling high milk producing cows had larger total gains from the first to the sixth month of age (Drewry et al., 1959).

Peak milk production occurs in beef cattle at 50 to 70 days (Clutter and Nielsen, 1987; Marston et al., 1992). Milk production of the dam had its greatest influence on calf weight during the first 60 days of age (Neville, 1962). Drewry et al., (1959), noted a strong correlation of .43 between average daily milk production and weight of the calf during the first month of lactation, but the correlation decreased to .12 by the sixth month. Neville (1962) found that the correlation between milk production of the cow and weight of the calf decreased from .74 during the first 60 days of life, to .59 by the sixth month.

The relationship of average daily gain of the calf with average daily milk production of the cow decreased as lactation progressed (Melton et al., 1967). Furthermore, the influence of the dam's milk production on weight gain of the calf was decreased when calves had access to creep feed (Christian et al., 1965). This indicates that the calf is more dependent upon milk during the early stages of growth. However, as the calf gets older, and the rumen begins to develop, the calf may consume nutrients from sources other than milk.

Forage Intake

Total milk yield is not the only factor that influences weaning weights, and the change in milk production with relation to how soon the rumen develops may be important. Forage intake of single lambs was correlated (r = .71, and .82 respectively) with lamb weight in late lactation, and accounted for 37 and 66 % of the variation in lamb body weight (Ramsey et al., 1994). Others found that twin lambs had greater forage intake than single lambs (Gardner and Houge, 1964; Kleeman et al., 1984; Ramsey et al. 1994). In both cases, lambs may compensate for decreased milk availability either due to decrease milk production by the ewe as the lactation progresses or decreased milk availability due to twins (Langlands, 1972, 1973; Peart, 1982; Ramsey et al., 1994).

Calves increase forage intake prior to weaning (LeDu et al., 1976; LeDu and Baker, 1979; Broesder et al., 1990; Ansotegui et al., 1991; Sowell et al., 1996). Calves received more energy from forage than milk during late lactation (Maddox, 1965), and after 3 months of age, calves spend less time suckling than during early lactation (Drewry et al., 1959). Calves which were prevented from suckling had increased grazing time (Sowell et al., 1996). Forage intake of spring born calves increased from .3 kg more forage per kg

decrease in milk intake by 120 days, to .6 kg more forage intake per 1 kg decrease in milk intake by 160 to 180 days (Ansotegui et al., 1991). At four months, 5.5 kg of calf weight was due to nutrition other than milk, whereas at eight months, 24.2 kg of calf weight was attributable to nutrition other than milk (Neville, 1962). Spring born calves obtained only 32% of their energy from milk just prior to weaning, due to increased forage intake (Haggard et al. 1983). Calves with a 30 to 60 % reduction in milk replacer availability increased forage organic mater intake, but total organic matter intake was not different between restricted calves and controls (Broesder et al., 1990). This indicates that calves were able to make up for decreased milk replacer availability with increased forage intake. However, the ability of calves to compensate for decrease milk is eventually limited by bulk fill (Broesder et al., 1990; Sowell et al., 1996), and calf weights are compromised (Boggs et al., 1980; Sowell et al., 1996). However, as the calf gets older and is able to consume more forage, weight gains increase (Boggs et al., 1980, Ansotegui et al., 1991).

Mammary Health of the Dam

Udder infections in beef cows decrease weaning weights of calves (Haggard et al., 1983; Watts et al., 1986; Newman et al., 1991; Simpson et al., 1995). Udder infection causes a decrease in the milk production of dairy cows (Janzen, 1970; Blosser, 1979; Bartlett et al., 1991; Lescourret and Coulon, 1994), and weaning weight of beef calves is influenced by milk production of the cow. Intramammary infection is associated with increased somatic cell counts (SCC) in beef cows (Watts et al., 1986). Beef cows with minimal SCC had greater milk production (Simpson et al., 1995). Somatic cell counts of beef cows were negatively correlated with weaning weights of calves (Watts et al., 1986).

Udder infection of beef cows decreased weight gain of calves from 60 to 100 days of age, but not over the entire 205 day period (Newman et al. 1991). Other reports indicate that udder infections in beef cows decrease 205 day weaning weights 7 to 10% (Haggard et al., 1983; Watts et al., 1986). Beef cows treated for mastitis had calves with greater weaning weights (Kirkbride, 1977). Intramammary infection is associated with a decrease in milk production, and can have adverse effects on calf weaning weights.

Mastitis

Definitions

Mastitis is a descriptive term referring to an inflammation of the mammary gland. It is derived from the Greek word "mastos", which means breast, and the suffix "itis", which means inflammation (Little and Plastridge, 1946; Jain, 1979). This term refers to changes in udder tissues and secretions which are not normal. Mastitis is most commonly caused by infectious microorganisms (Bartlett et al., 1991), however it can also be a response to traumatic injury of the teat or udder (Plastridge, 1958). Infection occurs when microorganisms enter the udder through the teat canal, and have almost the perfect environment in which to multiply; a readily available energy source and an optimal temperature. The inflammation serves to neutralize invading microorganisms and assist in repairing damaged tissue (Philpot and Nickerson, 1991). Mastitis can be clinically classified as acute, subclinical, or chronic.

Acute mastitis is a severe reaction. It involves both the parenchyma and interstitial tissues (Little and Plastridge, 1946). It is easily detected as the teats are usually distended, swollen, and painful (Harmon , 1994). Symptoms include increased pulse and respiration, depression and loss of appetite, loss of or decreased milk production, loss of muscle coordination and reduced pupillary reflex, as well as dehydration and diarrhea (Lohuis et al., 1990; Philpot and Nickerson, 1991). Body temperature is increased and abscesses form deep within the udder (Lohuis et al., 1990; Harmon, 1994). It may terminate in gangrenous mastitis (Little and Plastridge, 1946), resulting in tissue sloughing or death of the animal (Houben et al., 1993).

Subclinical mastitis is less severe than acute mastitis. It is not visually apparent and even experienced dairymen may not notice it. Subclinical mastitis results in decreased milk production and changes in the secretions such as thickening of the milk, blood specs, flakes, or abnormal milk color (Plastridge, 1958; Philpot and Nickerson, 1991). The milk may contain chlorides resulting in alkalinity (Little and Plastridge, 1946; Timms and Schultz, 1987). Subclinical mastitis is 15 to 40 times more prevalent than acute cases, and is typically of longer duration (Philpot and Nickerson, 1991).

Chronic mastitis is persistent. Scar tissue develops and the udder changes size (Little and Plastridge, 1946). Intermittent incidences of acute mastitis will occur (Philpot and Nickerson, 1991).

Organisms That Cause Mastitis

Mastitis is most often the result of infection within the udder by microorganisms (Bartlett et al., 1991). Organisms that cause mastitis in dairy cows can be broadly

classified into four categories; contagious, environmental, opportunistic, and others (Philpot and Nickerson, 1991). Contagious organisms are transmitted from one cow to another and usually occurs at milking in dairy cattle. Environmental organisms would come from the soil, water, bedding, feces, and other matter that may come in contact with the cow's udder. The dairy cow is usually exposed to these during the time between milkings, and would be the organisms to which the beef cow is most often exposed. Opportunistic organisms would be those that are on the surface of the teat or udder, and may be transmitted into the udder during times of milking or nursing of the calf. Finally, other microorganisms would be those that are not as common, but can still cause mastitis.

Contagious organisms are *Staphylococcus aureus*, *Streptococcus agalactiae*, *Mycoplasma bovis*, and *Corynebacterium bovis* (Philpot and Nickerson, 1991). *Staphylococcus aureus* infection results in fibrotic tissue encapsulating the infection such that antibiotics cannot reach the site of infection (Nickerson and Owens, 1993). This makes it difficult to cure. *Streptococcus agalactiae* comes from contaminated milk and results in an extremely high SCC, however it can be eradicated from the herd (Philpot and Nickerson, 1991). Along with the coliforms and coagulase negative *Staphylococcus*; *Streptococcus agalactiae* and *Staphylococcus aureus* are two of the most prominent mastitis causing organisms in dairy cattle (Jain, 1979; Harmon, 1994).

Environmental organisms that cause mastitis would include *Streptococcus uberis*, *Streptococcus dysgalactiae*, and coliforms such as *Escherichia coli*, *Klebsiella pneumoniae*, *Klebsiella oxytoca*, and *Enterobacter aerogenes* (Philpot and Nickerson, 1991). About 1 to 5 % of infections result from these organisms, and duration of infection

is usually short. However, some of the coliforms will produce endotoxins that can have a systemic effect and result in death of the cow (Shuster et al., 1991).

Opportunistic microorganisms are staphylococci other than *Staphylococcus aureus*. These include *Staphylococcus capitis, chromogenes, cohnii, epidermidis, gallinarum, hominis, simulans, xylosus*, and others (Matthews et al., 1992). Infact, there are over 20 species (Philpot and Nickerson, 1991), which are commonly grouped together under the title coagulase negative *Staphylococcus*.

Other microorganisms that cause mastitis are less prevalent. These include *Pseudomonas aeruginosa, Actinomyces pyogenes, Nocardia spp., Candida spp., Bacillus spp., Seratia spp., Pasteurella spp., Prototheca spp.*, and other *Streptococcus spp.* (Philpot and Nickerson, 1991; Duenas et al., 1994).

In beef cows, data are limited as to which microorganisms are present. *Staphylococcus aureus* and coagulase negative *Staphylococcus* appear to be the predominant organisms (Kirkbride, 1977; Haggard et al., 1983; Watts et al, 1986; Newman et al., 1991; Duenas et al., 1994; Simpson et al., 1995). *Streptococcus dysgalactiae* and *Streptococcus agalactiae* have also been isolated from the milk of beef cows (Wilson et al., 1971; Hunter and Jeffrey, 1975; Kirkbride, 1977; Watts et al., 1986; Simpson et al., 1995). Others such as *Streptococcus uberis, Streptococcus faecalis*, and *Staphylococcus epidermidis* have been found (Wilson et al., 1971; Hunter and Jeffrey, 1975; Watts et al., 1986; Newman et al., 1991). *Staphylococcus spp.* that have been isolated from beef cows are *Staphylococcus hyicus, simulans, hominis, capitis, saprophyticus, cohnii*, and *xylosus* (Watts et al., 1986; Newman et al., 1991). *Corynebacterium pyogenes* and

Corynebacterium bovis are also present in beef cow udders (Sobari et al., 1976; Duenas et al., 1994; Newman et al., 1991), as well as *Actinomyces pyogenes* and *Bacillus spp.* (Duenas et al, 1994; Simpson et al., 1995).

Bovine Mastitis

Mastitis reduces milk production in dairy (Philpot, 1967; Carroll, 1977; Blosser, 1979; Lescourret and Coulon, 1994) and beef cows (Simpson et al., 1995), however, little information is available concerning mastitis in beef cows.

Infection Rates

Since a large number of microorganisms can cause mastitis in dairy cows, infections are not usually reported based on individual organisms. Instead, infections are reported based on two broad categories; major pathogens and minor pathogens. Major pathogens include organisms such as *Streptococcus spp.*, *Staphylococcus aureus* and coliforms (Dohoo and Meek, 1982). Minor pathogens include *Corynebacterium bovis* and coagulase negative staphylococci (Dohoo and Meek, 1982). Specific major pathogens such as *Streptococcus agalactiae*, *Staphylococcus aureus*, *Staphylococcus uberis* or *Escherichia coli*, and some specific minor pathogens such as *Corynebacterium bovis* and the general group of coagulase negative staphylococci sometimes have been reported. *Staphylococcus aureus* and coagulase negative staphylococci are major mastitis causing organisms in both dairy (Jain, 1979; Harmon, 1994) and beef cows (Kirkbride, 1977; Haggard et al., 1983; Watts et al, 1986; Newman et al., 1991; Duenas et al., 1994; Simpson et al., 1995).

Twenty-four percent of all health disorders in dairy cows were comprised of mastitis (Lescourret and Coulon, 1994). Most udder infections occur within the first 30 d of lactation (Bunch et al., 1984). The percentage of quarters infected at drying-off in dairy cows ranges from 13 to 39% (Boddie and Nickerson, 1986; Seymour et al., 1989; Soback et al., 1990). In 15 herds, 24% of quarters were infected with a mastitis causing pathogen during the course of one year (Hinckley et al., 1985). Major pathogens are of primary concern since they often result in clinical mastitis (Philpot, 1979; Timms and Schultz, 1984; Houben et al., 1993). As much as 95% of mastitis can be caused by major pathogens (Philpot, 1979), and major pathogens may infect from 23 to 42% of quarters (Hinckley et al., 1985; Bartlett et al., 1991; Guterbock et al., 1993). In an 80 herd survey, prevalence of infection with a major pathogen averaged 16% (Eberhart et al., 1982). Timms and Schultz (1984) found that 70% of quarters had bacterial isolates. Twenty two percent of quarters were infected with major pathogens and 46% were infected with minor pathogens (Timms and Schultz, 1984). Infection with a major pathogen resulted in 49% of clinical cases of mastitis, whereas, only 13% of nonclinical cases were attributable to major pathogens (Timms and Schultz, 1984). Infection with minor pathogens resulted in a similar number of clinical and nonclinical cases of mastitis. Minor pathogens comprise the greatest percentage of infections (Cummins and McCaskey, 1987), and become more prevalent as lactation progresses (Fox et al., 1987; Timms and Schultz, 1987). In primiparous dairy heifers, minor pathogens are more prevalent than major pathogens (Boddie et al., 1987; Miller et al., 1991).

Major pathogens infect a large percentage of dairy cows (Boddie and Nickerson, 1986). Coliforms comprise 21 to 31% of infections (Smith et al., 1985; Houben et al., 1993). Twenty-four percent of clinical mastitis cases were due to coliform infections, and 16% were the result of coagulase positive staphylococci, which includes *S. aureus* (Bartlett et al., 1991).

Staphylococcus aureus (SA) is a prominent major pathogen. The incidence of *Staphylococcus aureus* can be as low as 3 to 8% of infections (Smith et al., 1985; Boddie and Nickerson, 1986; Matthews et al., 1991; Houben et al., 1993). However, as many as 22 to 64% of quarters were infected with SA (Jarp et al., 1986; Sol et al., 1994; Enevoldsen et al., 1995). *Staphylococcus aureus* was present in 2 to 28% of quarters at drying off (Harmon et al., 1986; Erskine et al., 1994). In 76 herds, the prevalence of infection with SA ranged from 10 to 50% (Wilson et al., 1995).

Coagulase negative staphylococci (CNS) are considered minor pathogens, but are still implicated in the mastitis complex (Jarp et al., 1986; Bartlett et al., 1991). Coagulase negative staphylococci can cause as little as 9% of the clinical cases of mastitis (Smith et al., 1985; Bartlett et al., 1991), but has accounted for 84 to 89% of total infections throughout lactation (Timms and Schultz, 1987; Seymour et al., 1989). More quarters are infected with CNS at drying-off than during lactation (Harmon et al., 1986; Timms and Schultz, 1987).

Major pathogens are more contagious than minor pathogens. Infected cows are the reservoir for these organisms which are usually spread at the time of milking due to contamination of milkers, or the hands of workers. Beef cows are not exposed to this

practice, therefore the opportunity for cow to cow spreading of major pathogens is expected to be less than with dairy cows. Infections with major organisms in beef cows are about half as frequent as in dairy cows (Haggard et al., 1987). Ten to 17% of beef cows can be infected with mastitis causing organisms (Hunter and Jeffrey, 1975; Haggard et al., 1987), however, infection rates can be as great as 32 to 37% of cows (Watts et al., 1986; Simpson et al., 1995). Forty-two percent of beef cows had intramammary infection, however, only 3.5% of isolates were considered to be pathogens that cause mastitis (Sobari et al., 1976). Infection rates are greater during early and late lactation, but decrease during mid lactation (Hunter and Jeffrey, 1975; Newman et al., 1991). Younger cows have fewer infections than older cows (Haggard et al., 1983; Duenas et al., 1994).

Staphylococcus aureus is also a prominent mastitis causing organism in beef cattle (Haggard et al., 1987). In one study, SA comprised 13 and 38% of infections in young and older beef cows respectively (Duenas et al., 1994). Typically, 10% of beef cow udders are infected with SA (Kirkbride, 1977; Haggard et al., 1983). *Stapylococcus aureus* can comprise as few as 3% of infections throughout lactation (Sobari et al., 1976; Newman et al., 1991), but can be as great as 21 to 40% of infections (Watts et al., 1986; Simpson et al., 1995).

Coagulase negative *Staphylococcus* are some of the most frequently isolated organisms from beef cow udders, and may have a role in the mastitis complex (Haggard et al., 1987; Newman et al., 1991). Coagulase negative staphylococci accounted for 16 to 36% of intramammary infections in beef cows (Kirkbride, 1977; Watts et al., 1986; Simpson et al.,

1995). Coagulase negative staphylococci caused 74 and 40% of infections in young and older cows respectively (Duenas et al., 1994).

Other microorganisms are found in beef cows, but it is unclear how significant a role they have in udder health. We previously determined that *Corynebacterium spp.*, *Bacillus spp.*, and *Streptococcus spp.* are present in the udders of beef cows (Duenas et al., 1994). *Micrococcus spp.*, *Streptococcus uberis*, *Actinomyces pyogenes*, *Streptococcus dysgalactiae*, *uberis*, and *faecalis* have also been isolated (Watts et al., 1986; Newman et al., 1991; Simpson et al., 1995). *Streptococcus agalactiae*, a major pathogen in dairy cows, made up 2 and 12% of intramammary infections in two beef cow herds (Hunter and Jeffrey, 1975).

Somatic Cell Counts

Several methods have been tried to identify udder infections without direct culture for organisms. Chloride concentration and N-acetyl-β-D-glucosaminidase activity are reasonably useful to identify infections (Fox and Schultz, 1985; Timms and Schultz, 1987). However, with the help of the Dairy Herd Improvement Association's once a month sampling program, somatic cell counts (SCC) have been the most widely utilized tool for determining possible infection (Bramley and Dodd, 1984; Reneau, 1986; Harmon, 1994).

Somatic cell counts are commonly used to identify cows or quarters that may be infected with mastitis causing bacteria (Schukken et al., 1991). Somatic cells are composed primarily of Polymorphonuclear (PMN) leukocytes (see Paape et al., 1979;

Craven and Williams, 1985; Harmon, 1994). These PMN leukocytes consist of lymphocytes, monocytes, and eosinophils (Paape et al., 1979). Macrophages can also make up a part of the SCC (Nonnecke and Harp, 1985), however, epithelial cells rarely make up more than 14% of SCC (Miller et al., 1991).

Polymorphonuclear leukocytes function as a defense mechanism. They kill and digest the invading mastitis causing bacteria (Paape and Wergin, 1977; Paape et al., 1979; Craven and Williams, 1985). Damage to milk synthesizing cells results in an increase in PMN and thus SCC (Harmon, 1994). Experimentally induced SA resulted in an increase in SCC due to migration of PMN into the mammary gland (Harmon and Heald, 1982; Nickerson and Pankey, 1984). These PMN moved across five structural barriers (Capilary endothelium, periendothelial layer, basal lamina of the alveolus, alveolar stroma, and alveolar epithelium) to reach the point of experimentally induced infection (Harmon and Heald, 1982). Naturally occurring SA resulted in an increase in PMN compared with uninfected glands (Nonnecke and Harp, 1985). However, mitogenesis from infected glands was decreased, possible due to the greater numbers of PMN in the milk of infected udders. Abnormally increased numbers of SCC can persist until the gland is healed (Harmon, 1994).

Factors that Influence Somatic Cell Counts

Infection. Intramammary infection with mastitis-causing bacteria is the most important factor influencing SCC (Dohoo and Meek, 1982; Reneau, 1986; Harmon, 1994). Somatic cell counts of uninfected dairy cows are less than 165,000 cells/mL (Reneau, 1986). Somatic cell counts are greater in quarters infected with minor or major pathogens as

compared with uninfected quarters (Sheldrake et al., 1983; Fox and Schultz 1985). Uninfected quarters only had a slight increase in SCC to 80,000 cells/mL through 285 days postpartum, However quarters infected with SA had an increase to 800,000 cells/ mL (Sheldrake et al., 1983). There is a strong correlation between infection within the herd and bulk tank SCC (Eberhart et al., 1982). When bulk tank SCC were 200,000 cells/mL, 6% of quarters within the herd were infected, however at 1,500,000 cells/mL, 48% of quarters were infected with mastitis causing microorganisms (Eberhart et al., 1982). Bulk tank SCC decreased by almost half when cows infected with SA were removed from the milking herd (Wilson et al., 1995). Experimentally introduced infection with SA resulted in an increase in SCC from 181,000 cells/mL prechallenge to 758,600 cells/mL postchallenge (Schukken et al., 1994). In general, SCC of dairy cows range from 100,000 to 200,000 cells/mL for uninfected quarters, 200,000 to 500,000 cells/mL for quarters infected with minor pathogens, and greater than 500,000 cells/mL for quarters infected with major pathogens (Natzke et al., 1972; Schultz, 1977a; Sheldrake et al., 1983).

Somatic cell counts are also increased in beef cows infected with mastitis causing organisms. Infected quarters of beef cows had SCC greater than 500,000 cells/mL (Hunter and Jeffrey, 1975; Watts et al., 1986; Newman et al., 1991; Simpson et al., 1995).

Beef cows infected with SA had 3,827,000 somatic cells/mL compared with 555,000 cells/mL for uninfected cows, and infection with CNS also increased SCC (Watts et al., 1986). Beef cows infected with SA had 1,522,000, 344,000 and 509,000 somatic cells/mL in early, mid, and late lactation respectively (Newman et al., 1991). These

researchers also found that minor pathogens such as CNS and *C. bovis* increased SCC of beef cows. Simpson et al. (1995) also found that beef cows infected with SA and *Streptococcus dysgalactiae* had greater SCC than uninfected cows.

Cow Age. Age or lactation number greatly influences SCC in dairy cows (Natzke et al., 1972; Schultz, 1977b; Sheldrake et al., 1983). In 150 herds, older cows had greater SCC compared with younger cows (Bodoh et al., 1976). An increase in SCC of about 100,000 cells/mL occurred per lactation (Schultz, 1977b). Intramammary infections in older cows cause an increased response in SCC (Marshall and Edmondson, 1962). However, older cows have had more exposure to infectious organisms, and even may have had a prior infection which may increase their sensitivity (Reneau, 1986).

There is also an effect of age on SCC is in beef cows. More older cows are infected with mastitis causing bacteria than younger cows (Haggard et al., 1983; Duenas et al., 1994). Due to the close relationship between infection and SCC, SCC would also increase with age of the cow. However, Wilson et al. (1971) found that SCC in beef cows did not increase with lactation number.

Stage of Lactation. Milk SCC are greatest in dairy cows after freshening and in late lactation, and least in mid-lactation (Reneau, 1986). Somatic cell counts at freshening can be elevated for up to 2 weeks after parturition (Cullen, 1968; Natzke et al., 1972). This could cause false positive identification of infections in early lactation (Reneau, 1986). In late lactation, when milk production had decrease to less than 4 kg per day, there was an increase in SCC (Bodoh et al., 1976). However, the increase in SCC may be caused by decreased milk or a decrease in milk volume (Harmon, 1994).

Stage of lactation influences SCC in beef cows (Wilson et al., 1971). Somatic cell counts were greatest after calving, decreased through mid lactation, and increased in late lactation (Hunter and Jeffrey, 1975; Newman et al., 1991).

Stress. The roll of stress on SCC in milk has been evaluated (Dohoo and Meek, 1982; Reneau, 1986; Harmon, 1994). Kay et al. (1977) found that SCC increased from 175,000 to 420,000 cells/mL after mixing pens of dairy cows. However others found no change in SCC of uninfected cows after mixing (Arave and Albright, 1976). Stray voltage caused an increase in SCC (Appleman and Gustafson, 1985).

The effects of heat stress on SCC has been evaluated. Somatic cell counts were increased during the summer months (Paape et al., 1973a; Bodoh et al., 1976). Somatic cell counts increased in uninfected quarters as well as infected quarters of heat stress dairy cows as compared with cows maintained under a thermoneutral environment (Elvinger et al., 1991). Induction of heat stress with environmental chambers did not evoke an increase in SCC (Paape et al., 1973a; Wegner et al., 1976), rather differences may be a result of concentrating SCC due to decreased milk production associated with heat stress (Harmon, 1994). Infected dairy cows have an increased response to stressors compared with uninfected cows (Paape et al., 1973b).

Diethylstilbestrol treatment increases SCC, presumably due to increased permeability of the capillaries to PMN (Astrom, 1972). In contrast, Guidry et al. (1975) found that neither estrus or exogenous estradiol increased SCC.

Adrenocorticotropic hormone (ACTH) treatment may induce an increase in SCC (Wegner and Stott, 1968; Wegner et al, 1976). However, Convey et al. (1971) and Paape

et al. (1973b) found that administering ACTH, corticosteroids, or synthetic glucocorticoids increased blood leukocytes, but did not alter milk SCC. The effect of stress on SCC of beef cows has not been evaluated.

Diurnal Variations. Diurnal variation is the variation in SCC that occurs throughout the day (King, 1972; Dohoo and Meek, 1982). Somatic cell counts rise through milking and are greatest in strippings (White and Rattray, 1965). Somatic cell counts are greater when the milking interval is decreased (Cullen, 1967; Fernando and Spahr, 1983). This variation should be of concern for researchers due to the fact that a single sample, or sampling at different times of the day may introduce a bias into the data (Reneau, 1986). Therefore, composite samples are desirable to increase accuracy of classification of cows based on SCC (Smith and Schultze, 1967; Dohoo and Meek, 1982).

Use of Somatic Cell Counts to Estimate Lost Milk Production

Clinical mastitis resulted in an immediate decrease in milk production (Bartlett et al., 1991; Shuster et al., 1991). Somatic cell counts are negatively correlated with 305 day milk yield (Raubertas and Shook, 1982; Bartlett et al., 1990). Milk production decreases at SCC greater than 500,000 cells/mL (Schultz, 1977b; Fetrow et al., 1988; Miles et al., 1992). Cows with mastitis had an 80 kg/d decrease in milk production during the subsequent lactation (Fetrow et al., 1991).

A decrease in milk production due to increased SCC also occurs in beef cows, and greater SCC results in less milk production per day as compared to cows with less SCC (Simpson et al., 1995). Decreased weight gain of calves from infected cows is presumably

attributable to decreased milk production (Haggard et al., 1983; Watts et al., 1986; Newman et al., 1991).

California Mastitis Test

Due to the relationship of SCC with milk production, it was desirable to develop a cow side test to estimate SCC. Whiteside (1939) was one of the first to discover a possible solution to the problem, but the test was still limited to the laboratory. Schalm et al. (1955) modified the Whiteside test using test tubes prepared with 4% sodium hydroxide which could be used in the barn, however, this Field Whiteside Test still had some inconsistency (Schalm and Noorlander, 1957), and led to the development of the California Mastitis Test (CMT) which could be used effectively at cowside (Schalm and Noorlander, 1957). Although other test have been developed based on the concept of the CMT, the CMT remains the most widely used cowside test (Paape et al., 1962). Currently the use of the CMT has subsided considerably due to the wide use of the Dairy Herd Improvement Association's programs that monitor SCC on a monthly basis.

The CMT as described by Schalm and Noorlander (1957) utilizes a plastic paddle with four cups. A small sample of milk (2 to 3 mL) from each quarter of the udder is milked into its corresponding cup on the CMT paddle. An equal volume of CMT reagent is added by estimation. The CMT reagent consists of 1.5% sodium hydroxide, bromcresol purple, and a 3 to 5% concentration of a surface-active anionic component such as alkyl sulfates, alkyl sulfonates, alkyl arylsulfates, or alkyl arylsulfonates. The paddle is moved in

a gentle swirling motion and based on the amount of reaction a CMT score is assessed for each quarter.

The reaction observed is the formation of a gel or precipitate, and contrasting colors of purple (Schalm and Noorlander, 1957). Somatic cells rupture on contact with the CMT reagent causing a release of cellular protein. These proteins unfold due to bonds being broken, and combine with the reagent to form a gel or precipitate. The color change is caused by the pH of the milk.

The CMT scores ranges from negative to strong positive (Schalm and Noorlander, 1957). A negative score indicates that milk had no precipitate. A trace (T) is indicative of a transient precipitate that disappears quickly. Scores of weak positive (1), distinct positive (2), and strong positive (3) indicates progressive degrees of precipitate and gel formation.

Gray and Schalm (1960) found that 33 to 39% of quarters had a positive CMT score, and other reports confirm this (Forster et al., 1967; Pearson and Greer, 1974). Most quarters are in the CMT negative range, however, CMT scores may increase as cows get older (Gray and Schalm, 1962; Daniel et al., 1966a). California Mastitis Test scores are highly correlated with actual SCC (Schalm and Noorlander, 1957; Pearson and Greer, 1974; Philpot and Nickerson, 1991), and there is a strong relationship between CMT score and infection (Jackson, 1961; Marshall and Edmondson, 1962; Pearson and Greer, 1974). However, CMT scores trace and 1 are not as reliable as 2 and 3, and are usually considered only suspect (Gray and Schalm, 1960).

Milk production losses associated with CMT scores have been evaluated (Dobbins, 1977). A progressive decrease in milk production was observed as CMT scores increased (Gray and Schalm, 1960; Gray and Schalm, 1962). Monthly milk production decreased from 5 to 43% as CMT score increased from T to 3 (Daniel et al., 1966b; Forster et al., 1967). Milk production was decreased by 1.5 lb/d for quarters with a CMT score of 3 (Forster, 1964; Appleman et al., 1965; Natzke et al., 1965; Philpot, 1967).

Studies in beef cows using the CMT are extremely limited. Increasing age and stage of lactation results in a greater CMT score for beef cows (Wilson et al., 1971). The percent of infected quarters of beef cows increased from 8 to 71% as the CMT score increased from negative to 3 (Newman et al., 1991).

Treatment of Mastitis

Methods of eliminating mastitis are limited. The only absolute method to eliminate infections is culling chronically infected cows, which may not be practical. Antimicrobial therapy is an important component in mastitis control.

Dry Cow Therapy

The rate of new infections is greater in the dry period as compared with lactation (Neave et al., 1950; Oliver and Mitchell, 1983). However, infections with major pathogens such as SA were greater during lactation (Oliver and Mitchell, 1983). This would be consistent with the evidence that organisms are spread primarily during the milking process. During the dry period, cows would be exposed to pathogens of environmental origin. Due to the lack of milking hygiene, substantially greater numbers of

microorganisms may populate the antral surface of the teat. Teat dipping up to seven days after the cessation of milking decreased the incidence of udder infections (Thoreson, 1973; Sinkevich, 1974a). Thus, it is important to treat infections during the dry period.

The purpose of dry cow therapy is to eliminated existing infections and prevent new infections from occurring. Cure rates are dependent on dosage and solubility of the drug, along with type of base, and frequency of administration (Philpot, 1979). Properties of the drug should include absence of tissue irritation when administered, low minimal inhibitory concentrations (MIC) for common bacteria, high affinity for udder tissues, and slow, stable release of antibiotic in excess of the MIC for greater than three weeks (Prescott and Baggot, 1988). The advantages of dry cow treatment versus lactation therapy are better cure rates, use of greater doses, milk does not have to be withheld from the market, sustained concentrations for a greater length of time, and time for regeneration of damaged tissue (Philpot, 1979; Nickerson and Owens, 1993).

Intramammary Infusion. The efficacy of dry cow treatment has been studied extensively. Cure rates due to dry cow therapy range from 25 to 100% (Ziv et al., 1981). Dry cow treatment decreased infections during the dry period, and prevented new infections 4 to 10 days postpartum as compared with controls (Sinkevich et al., 1974b). Multiple infusions of dry cow treatment throughout the dry period did not decrease existing infections, but did provide more protection at the subsequent calving, presumably due to increased concentrations of the drug in the mammary tissue prior to freshening (Cummins and McCaskey, 1987). Typically, dry cow treatment decreases the prevalence

of infection during the dry period by 74 to 92% (Harmon et al., 1986; Batra, 1988; Davidson et al.; 1994).

Selective dry cow therapy has been discussed (see Eberhart, 1986). However, selective dry cow treatment based on SCC or clinical history could result in 30 to 60% of infected quarters not treated (Philpot, 1979). Somatic cells can persist for long periods of time after infection has been eliminated (Philpot, 1979). Thus it is possible that some negative quarters would also be treated. Selective treatment based on SCC could result in subclinical cases that are not treated, and less protection from infection at the subsequent calving. Therefore, current methodology is to treat all quarters of all dairy cows at drying off (Natzke, 1971).

Reasons for Treatment Failure. A wide range in cure rate of udder infections during the dry period may be associated with the differing antibiotics employed. Also, the types of organisms that make up the infections may play a role. The probability of a cure when streptococci was the infectious organism was greater than for staphylococcal infections (Philpot, 1979). Cephapirin decreased prevalence of infection at the subsequent lactation, but novobiocin and penicillin-streptomycin treatments were not effective (Harmon et al., 1986). Although penicillin-streptomycin significantly reduced CNS infections during the dry period, only cephapirin was effective in preventing CNS at the subsequent lactation. Sodium nafcillin-procaine benzylpenicillin dihydrostreptomycin had greater cure rates than products containing cloxacillin or cephalonium (Ziv et al., 1981). Full insertion of the syringe cannula during treatment at drying-off resulted in a greater infection rate than partial insertion (Boddie and Nickerson, 1986). Some researchers report that the

effectiveness of mammary leukocytes are decreased with treatment (Nickerson et al., 1986). Cure rate from dry cow therapy was reduced with increasing SCC, age, and number of infected quarters (Sol et al., 1990; Sol et al., 1994).

Most studies do not utilize untreated controls, rather they compare pre-treatment to post-treatment. Spontaneous recovery can occur in 20% of cases (Harmon et al., 1986; Philpot and Nickerson, 1991). This could introduce a bias in favor of dry cow treatment, in that it is unknown how many of the treated cows would have eliminated infections on their own due to spontaneous cure. Therefore, actual cure rates attributable to dry cow treatment may be substantially less than reported.

Systemic Therapy. The possibility for using systemic therapy as opposed to intramammary treatment for mastitis has been discussed (Giesecke, 1977; Ziv, 1980). Benzypenicillin, ampicillin, cloaxacillin, oxytetracycline, and erythromycin all maintain MIC in udder secretions (Giesecke, 1977). Norfloxacin resulted in greater cure rates than the oxytetracycline, cephapirin, or control groups (66.7 vs 25, 30.8 and 33.3% respectively; Soback et al., 1990). Although oxytetracycline did not differ from controls in cure rate of existing intramammary infections, it did provide greater protection against new infections (9.7 vs 29.2% respectively; Soback et al., 1990). Norfloxacin and oxytetracycline have good distribution in body fluids, and reach the site of infection (Soback et al., 1990). Cure rates for combination therapy of intramuscular oxytetracycline and intramammary cephapirin were greater than for intramammary therapy alone at 30 and 60 days after drying-off (Erskine et al., 1994). However, combination therapy was over

twice as effective in eliminating infection at 30 and 60 days if cows were in their first
lactation.

Therapy During Lactation

Intramammary Infusion. Susceptibility of cows to infection is increased just prior to
and immediately after freshening (Neave et al., 1950; Oliver and Mitchell, 1983). The
majority of infections occurring during lactation developed by 30 days postpartum (Fox et
al., 1987). Therefore, the early postpartum period may be a time when antibiotic
treatment could be utilized. Newbould (1974) found a 55% decrease in infections in cows
treated during lactation, however no control cows were utilized. In another study, there
was no difference in the clearance of infections from the udders of treated cows compared
with controls (Fox et al., 1987; Guterbock et al., 1993). Although 64% of infections were
eliminated from treated cows, 54% of infections were spontaneously cured in non-treated
controls (Fox et al., 1987). Cure rates between two different lactating cow treatments and
oxytocin treated controls were not different (Guterbock et al., 1993). Oxytocin was
administered because cooperating dairy producers objected to untreated controls.
However, cure rates of 49% for oxytocin treated cows compared with 44 and 55% for
antibiotic treated cows demonstrates the limited success of treatment during lactation.
Treatment during lactation was not as effective as dry cow therapy (Hinckley et al., 1985).
Heifers have been successfully treated during their first lactation, but lactational therapy
was still 30 to 50% less effective than dry cow treatment (Nickerson et al., 1994). Poor
distribution of intramammary antibiotics in the udders of lactating cows could be the
problem (Owens and Nickerson, 1990). When treating for mastitis during lactation, cure

rate, SCC, and milk production were not altered (Timms and Schultz, 1984). They concluded that the cost of treating cows during lactation was not justified based on the lack of increased milk yield. Thus treatment during lactation is of less benefit compared with dry cow therapy.

Intramuscular Therapy. Intracellular location of some organisms such as SA, and poor distribution of intramammary infusion products during lactation have raised interest in possible alternative methods of treatment. Limited reports are available on the use of systemic treatment during lactation. Intramuscular treatment with penicillin G and methicillin was just as effective as intramammary therapy in eliminating sensitive SA (Ziv and Storper, 1985). However, treatment was far less effective against resistant strains of SA. Duration of treatment has a greater impact on cure rate than method of treatment (Jarp et al., 1986). Intramuscular treatments were slightly better than intramammary treatment, however only cows with penicillin sensitive isolates were used, and untreated controls were not utilized (Jarp et al., 1986).

Combination Therapy. Reasons for treatment failures have been discussed (see Nickerson and Owens, 1993). Treatment failures can occur due to delayed treatment, inappropriate selection of treatment antibiotics, premature cessation of treatments, microbial resistance to treatment drugs, organisms metabolic state at the time of treatment, poor drug diffusion, inactivation of drugs by milk or plasma proteins, lack of contact of the drug with bacteria due to scar tissue and walling off of infection, and intracellular protection of some bacteria such as SA. Due to these factors, interest has increased in the prospect of utilizing a combination of intramuscular and intramammary

therapy. Combination therapy is more effective than intramammary alone due to increased distribution of antibiotics throughout the mammary gland and tissues (Owens et al., 1994). Combination therapy resulted in a greater bacteriologic cure rate as compared with intramammary controls (48 vs 30% of cows respectively; Owens et al., 1988).

Mastitis Therapy in Beef Cows

Few researchers (Kirkbride, 1977; Newman et al., 1991; Duenas et al., 1995) have examined possible therapeutic regimes for mastitis in the beef cow. Calves from beef cows treated with antibiotics at drying-off weighed 12% more at 60 days of age than calves from untreated controls (Kirkbride, 1977). Dry cow treatment resulted in a decreased percentage of cow infected at the subsequent calving as compared with controls (8 vs 22% of quarters respectively; Newman et al., 1991), due primarily to the elimination of infections present at drying-off. However, treatment had no effect on preventing new infections during the dry period, which is usually longer in beef cows than dairy cows (4 to 5 vs 2 months respectively). Thus it would be anticipated that dry cow treatment may not provide protection through at least the last one half of the dry period. It was not tested weather dry cow treatment increased gains of calves. Our laboratory has studied the effects of intramuscular therapy of mastitis in beef cows (Duenas et al., 1995). Multiparous range beef cows were randomly assigned to a 2 x 2 factorial design to receive a single injection of oxytetracycline both at drying-off and the subsequent calving, at drying-off only, at calving only, or untreated controls. Intramuscular treatment with oxytetracycline only at drying-off, or only at calving had no effect on udder infections as compared with controls. However, cows treated both at drying-off and at calving had an

11% decrease in infected quarters compared with controls. Treatment at both times also resulted in a decrease of 52% in SCC. Cows treated at both drying-off and calving had a reduced incidence of new infections with SA as compared with control cows (7 vs 13% respectively). However, treatment of cows did not influence weaning weights of calves (Lents et al., 1996). Thus intramuscular treatment of subclinical mastitis in beef cows was effective in reducing intramammary infections, but the effect of treating cows on the weight gain of calves has not been established.

Conclusions

Growth of calves is influenced by many factors. The most important factor associated with calf growth is milk production of the cow. Therefore, factors that influence milk production of the cow are of concern to producers and animal scientists everywhere.

Mastitis, an infection of the mammary gland, is caused by microorganisms. In dairy cows, infection rates with mastitis causing bacteria are usually very high. These infections cause an increase in somatic cell counts and a dramatic decrease in milk production. In beef cows, udder infections are also present, but at a substantially reduced frequency compared with dairy cows. Intramammary infections of beef cows result in increased somatic cell counts and decreased milk production. Intramammary infections of beef cows also decrease calf weights, presumably associated with decreased milk production of the cow.

To decrease infection rates and increase milk production of cows, the dairy industry has adopted the treatment of cows at the time of drying-off with intramammary antibiotics.

Many dairymen also treat cows during lactation. The beef industry has not adopted these methods. In-fact, few researchers have studied the effects of antibiotic treatment on the udder health of beef cows. Treatment of beef cows before the dry period as well as during lactation has resulted in decreased infection rates. The effects of treatment on milk production of the beef cow and weight gain of the calf is not established. Research should be conducted to better understand the relationship between udder health of the cow and growth of the calf. If treatment of beef cows for udder infections is beneficial to calf growth, this practice will be utilized by the industry. This could result in the ability of cattlemen to produce the same amount of beef with fewer cows, thus increasing the overall efficiency of production in the nations cow herd.

Table 1. Common mastitis-causing organisms in dairy and beef cows

Organism	Organism found in	
	Dairy cows	Beef cows
Staphylococcus aureus	Nickerson and Owens, 1993	Duenas et al., 1994
Streptococcus agalactiae	Harmon, 1994	Hunter and Jeffrey, 1975
Streptococcus dysgalactiae	Philpot and Nickerson, 1991	Simpson et al., 1995
Corynebacterium bovis	Philpot and Nickerson, 1991	Duenas et al., 1994
Escherichia coli	Philpot and Nickerson, 1991	---
coagulase negative staphylococci	Matthews et al., 1992	Watts et al., 1986

CHAPTER III

EFFICACY OF INTRAMUSCULAR TREATMENT OF BEEF COWS WITH OXYTETRACYCLINE TO REDUCE INTRAMAMMARY INFECTION AND TO INCREASE CALF GROWTH

ABSTRACT: Spring calving Hereford and Hereford x Angus multiparous cows were used to determine the efficacy of intramuscular treatment with oxytetracycline to reduce the incidence of mastitis causing bacteria, decrease milk somatic cell counts (SCC), and increase calf growth. Milk samples were collected from each quarter of 319 cows at 8 to 14 d after calving and at weaning, to determine the presence of bacteria and SCC. A California Mastitis Test (CMT) was performed on milk from each quarter of each cow at the initial sample collection. Cows with a CMT score of 1, 2, or 3 in at least one quarter, were randomly assigned to receive either an intramuscular injection of oxytetracycline (n = 63) or the control vehicle (n = 60), and cows with a CMT score of 0 or trace in all four quarters were not treated (n=196). Calf weights were determined at birth, 60 d of age, and weaning. The number of somatic cells in milk increased as CMT score increased (P < .01). The percentage of quarters that were infected increased from 11% to 44% as CMT

score increased from 0 to 3 (P < .01). Fifty-two percent of cows, and 38% of quarters that were infected post partum were still infected at weaning, whereas if cows were noninfected after calving, only 26% of cows and 12% of quarters were infected at weaning (P < .05). Treatment did not influence the percentage of cows or quarters infected with mastitis causing bacteria at weaning. Forty-one percent of treated cows and 28% of treated quarters were infected at weaning compared with 36% of control cows and 22% of control quarters (P > .1). The presence of mastitis causing bacteria at weaning was associated with increased SCC in cows and quarters (P < .01). Average SCC per cow increased as the number of infected quarters per cow increased (P < .05). Treatment did not alter SCC of cows or quarters at weaning (P > .1). Average SCC per cow was negatively correlated with 60 d (r = -.26; P < .05), but not adjusted 205 (ADJ205) d (r = -.11; P > .1) weights of calves. Infection status of the dam post partum did not alter 60 d or ADJ205 d calf weights (P > .1). There was an interaction between year and treatment on calf weights a 60 d (P < .07). Treatment did not influence 60 d calf weights in 1995, but in 1996, control cows had calves that were heavier than calves of treated cows (P < .05). Treatment did not influence ADJ205 d weights. Cows with one or more dry quarters after calving had calves that weighed less at 60 d, and 205 d than cows with four functional quarters (P < .01). Treatment of beef cows with intramuscular oxytetracycline after calving did not influence intramammay infection rates, SCC, or calf weights at weaning.

Introduction

Milk production is the most important factor influencing weaning weights of calves. As much as 60 to 66 % of the variation in 205 d weight of beef calves is due to the direct influence of the dam's milk production (Neville, 1962; Rutledge et al., 1971). Mastitis decreases milk production in both dairy (Crossman et al., 1950; Bartlett et al., 1991; Lescourret and Coulon, 1994) and beef cows (Simpson et al., 1995). Intramammary infections in beef cows resulted in decreased weight gain of calves (Haggard et al, 1983; Watts et al., 1986; Newman et al., 1991). Therefore, it is necessary to establish an effective treatment for mastitis in beef cows (Kirkbride, 1977; Newman et al., 1991; Duenas et al., 1995).

A standard procedure in the dairy industry is to treat cows at drying-off with antibiotics. Cure rates for intramammary infections by dry cow therapy are 25 to 100% (Ziv et al., 1981; Davidson et al, 1994; Erskine et al., 1994). Susceptibility of cows to mammary infection may be increased just prior to and immediately after calving (Neave et al., 1950; Oliver and Mitchell, 1983; Fox et al., 1987), so antibiotics are infused into the gland during lactation. Cure rate after treatment during lactation ranges from 30 to 50% (Hinckley et al. 1985; Fox et al., 1987; Guterbock et al., 1993).

Reasons for reduced or lack of efficacy of mastitis treatments in lactating cows are: delayed treatment, inappropriate selection of treatment antibiotics, premature cessation of treatments, microbial resistance to treatment drugs, poor drug diffusion, inactivation of drugs by milk or plasma proteins, lack of contact of drugs with bacteria due to scar tissue, and intracellular location of some bacteria such as *Staphylococcus aureus* (Nickerson and

Owens, 1993). Thus studies have been conducted to increase the efficacy with intramuscular and intramammary treatments for mastitis (Giesecke, 1977; Ziv, 1980). Giesecke (1977) determined that systemic drugs including erythromycin and oxytetracycline maintain minimal inhibitory concentrations in the udder. Intramuscular treatment of dairy cows with oxytetracycline was effective in preventing new udder infections during the dry period (Soback et al., 1990). Combination of intramuscular oxytetracycline and intramammary cephapirin treatment at drying-off was twice as effective in eliminating infection at 30 and 60 d after calving as intramammary treatment alone (Erskine et al., 1994), and the combination treatment was better than intramammary treatment alone in maintaining elevated antibiotic concentrations in the mammary gland (Owens et al., 1994). Combination therapy is also more effective than only intramammary treatment in eliminating udder infections during lactation (Owens et al., 1994). Ziv and Storper (1985) found that intramuscular treatment was just as effective as intramammary therapy in eliminating udder infections during lactation.

Few researchers have examined possible treatments of mastitis in beef cows. Beef cows which were treated with an intramammary antibiotic at drying-off had calves that weighed 12.5 % more at 60 d compared with untreated cows (Kirkbride, 1977). Drycow treatment of beef cows resulted in a decreased incidence of udder infections following the next calving (Newman et al., 1991), however, they did not determine if treatment of cows increased weight gains of calves. Intramuscular treatment of beef cows with oxytetracyline at calving and drying-off improved intramammary health (Duenas et al., 1995), but did not increase calf gain (Lents et al., 1996). Therefore, the objective of this

study was to determine if intramuscular oxytetracyline treatment of beef cows would decrease infection rate and increase calf growth.

Materials and Methods

Animals

Spring calving, multiparous Hereford and Hereford x Angus cows, with increased somatic cell counts (SCC) after calving, were used to determine the efficacy of intramuscular oxytetracycline to reduce the incidence of mastitis causing bacteria, decrease milk SCC, and increase calf growth. Three hundred and nineteen cows were sampled in two years (1995, n = 160; 1996, n = 159) to identify cows with increased SCC after calving. Cows grazed bermuda grass pastures and native range at the Oklahoma Agricultural Experiment Station Range Cow Research Center, 24 km west of Stillwater. Cows were fed 40% crude protein supplements during the winter to maintain a body condition score of 4 to 5.5 (Wagner et al., 1988) at calving. Weights of calves were determined at calving, mid-lactation, and at weaning.

Twenty control and 19 treated cows had their calves weaned early (50 ± 20 d). The remainder of the cows had calves weaned at approximately 205 d.

Milk Samples

Milk samples were collected from each quarter of cows at 8 to 14 d post partum and at weaning. Calves were removed from cows for approximately 2 h before sampling. Cows were restrained in a squeeze chute and administered 10 units of oxytocin (Vedco, Inc., St. Joseph, MO) to facilitate milk let-down. Teats were dipped in a .1 % iodine solution and

wiped dry with individual paper towels. The first two or three streams of milk were discarded and 10 mL of milk from each quarter were collected into plastic vials containing preservative (D & F Control Systems, Inc., San Ramon, CA). Teat ends were then individually disinfected with a cotton swab soaked in 70% ethyl alcohol. Two streams of milk from each quarter were discarded and 3 mL of milk were aseptically collected into sterile polypropylene snap cap tubes (Fisherbrand®, Pittsburgh, PA). After sampling, teats were dipped in .1 % iodine solution. Samples (10 mL) were sent to the DHIA laboratory, Manhattan, KS, within 24 h for analysis of SCC . Sterile samples were placed on ice and transported to the lab, stored at -20 °C until packaged in dry ice and transported to the Immunology and Disease Resistance Laboratory, USDA-ARS, Beltsville, MD, for bacteriological analyses.

Bacteriological analyses

Bacteriological analyses were performed at the Immunology and Disease Resistance laboratory USDA-ARS, Beltsville, MD. Sterile milk samples were allowed to thaw at room temperature and vortexed. Twenty µL of milk were plated on one quarter of an esculin blood agar plate (5 % red blood cells), and on P-agar plates supplemented with acriflavine. Plates were incubated at 37 °C and bacterial growth was determined at 24 and 48 h.

A quarter was considered to be infected if three or more colonies of the same organism were isolated from the esculin blood agar plate. Identification of organisms was based on colony morphology, hemolytic and hydrolytic patterns, gram stain (Bacto® Gam Stain Set, Difco Laboratories, Detroit, MI), catalase production (Hydrogen peroxide, Sigma®,

Sigma Chemical Co., St. Louis, MO), and tube coagulase test (Coagulase Plasma EDTA, Difco Laboratories, Detroit, MI).

California Mastitis Test

After collection of bacteriological and SCC samples, a California Mastitis Test (CMT) was performed for each quarter of each cow. A stream of milk from each quarter was discarded, and approximately 5 mL of milk were collected from each quarter into its corresponding cup on the CMT paddle (Dairy Research Products Inc., Spencerville, IND). The paddle was tilted to allow excess milk to drain from each cup without mixing with other samples, and approximately 2 mL of milk remained in each receptacle. Approximately 2 mL of CMT reagent was added to each cup and the paddle was gently swirled to mix the samples. A CMT score of 0, trace (T), 1, 2, or 3, as described by Schalm and Noorlander (1957), was assigned to each sample based on the amount of precipitant and gel formation. Cows with a CMT score of 0 or trace in all four quarters were not treated (n=196). Cows with a CMT score of 1, 2, or 3 in at least one quarter were randomly assigned to receive either an intramuscular injection of oxytetracycline (n = 63) or control vehicle (n = 60).

Treatment

Each mL of antibiotic contained 200 mg of oxytetracycline (Liquamycin® LA200®, Animal Health Division of Pfizer, Inc., New York), and on a weight to volume basis, 40% 2-pyrrolidone, and 5% poyvinlypyrrolidone. The control vehicle consisted of 40% 2-pyrrolidone (BASF Corp., Parisppany, NJ.) and 5% poyvinlypyrrolidone (Aldrich Chemical Comp., Inc., Milwaukee, Wiss.) in a sterile aqueous solution. Treated cows

received 1 mL of antibiotic per 10.1 kg of body weight, and control cows received 40 mL of the control vehicle. Intramuscular injections were administered along the lateral surfaces of the upper one third of the neck, and only 10 mL of solution was administered per injection site, with only three injection sites per side of the neck.

Statistical analyses

Somatic cell counts were analyzed using log transformed values, however, actual values are reported. Log transformed SCC greater of less than three standard deviations from the mean were considered outliers. The quarter sample with the greatest SCC for each cow was determined and used as the maximum SCC (MXSCC) value for that cow. Average SCC (AVSCC) of the four quarters for each cow were determined as a geometric mean and used as the SCC value for each cow. A cow was classified as infected if one or more of coagulase negative staphylococci (CNS), *Corynebacterium bovis* (CB), and *Staphylococcus aureus* (SA) were present in one or more quarters. Least squares analyses of variance were used to determine treatment effect on SCC and infection status at weaning. The model included treatment, post partum infection status, year, and all interactions. Separate statistical analyses were performed on early weaned cows (n = 30) and normal weaned cows.

Regression analyses were used to determine the relationships between SCC (independent variable) and CMT score (dependent variable). Due to a significant year effect, analyses were performed within year. California Mastitis Test score of T (trace) was assigned a value of .5 for statistical analysis. Dummy variables were used to

determine if regression lines for each year were different (Neter et al., 1989; Steel et al., 1997).

Mean calf age at the mid-lactation weight was 51 ± 20 d. Calves that were greater or less than one standard deviation from the mean age were excluded ($n = 33$) from the analysis of mid-lactation weight. Data from 66 calves were analyzed using least squares analysis of variance to test effects of post partum infection status and treatment on calf weights. The model included post partum infection status, treatment, calf age, birth weight, sex, cow age, and year. A significant year x treatment interaction was included, but all other nonsignificant interactions were dropped from the model. The relationship between calf weight and AVSCC post partum was determined by partial correlation adjusted for year. Analyses of 60 d weights included both early weaned and normal weaned cows.

The effects of post partum infection status and treatment on weaning weight of calves adjusted to 205 d were determined using a model that included year, post partum infection status, treatment, and all interactions, with sex and cow age as covariables. Relationship of AVSCC and adjusted 205 d weights was determined by partial correlations adjusted for year. Analysis of adjusted 205 d weights included only normal weaned cows.

The effect of dry quarters after calving on calf growth at 60 and 205 d was assessed. The model included dry quarters post partum, year, calf age, birth weight, sex, and cow age for 60 d weights, while the model for adjusted 205 d weights included year, dry quarters post partum, and the interactions, with sex and cow age as covariables.

Results

Quarter SCC post partum in 1995 were greater than in 1996 (P < .0001; data not shown). The intercepts for the regressions of SCC on CMT for 1995 and 1996 were different (P < .01), but the slopes were similar. In both years, SCC increased with increasing CMT score (P < .001; Figure 1). Nine hundred-thirty one, 80, 82, 55, and 47 quarters had CMT scores of 0, T, 1, 2, and 3 respectively. Somatic cell counts for CMT scores of 0, T, 1, 2, and 3 were 66×10^3, 194×10^3, 510×10^3, 1408×10^3, and 3035×10^3 cells/mL, respectively. The percentage of quarters infected increased with increasing CMT score (Figure 2). The percentage of infected quarters was not different for quarters with CMT scores 0 or T (11 vs 16% infected respectively; P > .1). More quarters with a CMT score of 1 were infected (25%) compared with quarters that had a CMT score of 0 (P < .01). The percentages of quarters with CMT scores of 2 or 3 that were infected (40 and 44%, respectively) were greater than for any other score (P < .01).

Treatment did not influence the percentage of cows or quarters infected at weaning (P > .1; Figure 3), and there was not a treatment x post partum infection status interaction. Fourty-one percent of cows and 28% of quarters that were treated post partum were infected at weaning, compared with 36% of control cows and 22% of control quarters (P > .1).

The presence of mastitis causing bacteria after calving increased the percentage of both infected cows and quarters at weaning (Figure 4). Fifty-two percent of cows that were infected post partum were infected at weaning, and 26% of cows noninfected post partum were infected at weaning (P < .05). Similarly, 38% of quarters that were infected post

partum were infected at weaning, and 12% of quarters that were noninfected post partum were infected at weaning ($P < .01$).

Treatment did not alter AVSCC or MXSCC per cow at weaning ($P > .1$), and there was not a treatment x post partum infection status interaction ($P > .1$). Average SCC at weaning were not different for control and treated cows (247 ± 60 x 10^3 vs 311 ± 59 x 10^3 cells/mL, respectively; $P > .1$; Figure 5a). Maximum SCC per cow at weaning were not different for control and treated cows (838 ± 247 x 10^3 vs 638 ± 242 x 10^3 cells/mL, respectively; $P > .1$; Figure 5b). Somatic cell counts per quarter at weaning were not different for control and treated quarters (501 ± 85 x 10^3 vs 524 ± 86 x 10^3 cells/mL, respectively; $P > .1$; Figure 6).

The presence of mastitis causing bacteria at weaning was associated with increased AVSCC at weaning ($P < .05$; Figure 7). Cows infected with mastitis causing bacteria at weaning had greater AVSCC than noninfected cows (288 ± 82 x 10^3 vs 54 ± 63 x 10^3 cells/mL, respectively).

There was an interaction between year and infection status of quarters at weaning on SCC per quarter at weaning ($P < .01$; Table 1). Somatic cell counts from noninfected quarters were not different between years. Infected quarters had greater SCC than noninfected quarters in both years, but in 1995, the increase in SCC with infection was greater ($P < .01$) than in 1996.

Cows with more quarters infected at weaning had greater AVSCC at weaning ($P < .01$). Cows with no infection, or only one infected quarter had similar AVSCC (54 ± 63 x 10^3 and 65 ± 116 x 10^3 cells/mL, respectively; Figure 8). However, AVSCC of cows with

two or more infected quarters were greater ($484 \pm 107 \times 10^3$ cells/mL; $P < .01$) than for cows with 0 or 1 infected quarters.

Twenty control and 19 treated cows had their calves weaned early, at approximately 60 d of lactation. Postpartum infection rate of cows tended ($P = .1$) to alter infection rate at early weaning. Thirty-eight percent of cows infected post partum were infected at 60 d compared with 10% of cows that were not infected post partum. Postpartum infection status of quarters affected infection status at early weaning ($P < .01$). Twenty-two percent of quarters that were infected post partum were infected at 60 d compared with 6% of noninfected quarters that were infected at 60 d. The percentage of cows and quarters infected at 60 d was similar for treated and controls ($P > .1$; data not shown). Treatment did not influence AVSCC or MXSCC per cow, or SCC per quarter at 60 d ($P > .1$; data not shown).

Calves in 1995 weighed less at 60 d of age than calves in 1996 (76 ± 3 vs 86 ± 2 kg, respectively; $P < .05$). Average SCC of all cows after calving were negatively correlated with 60 d weights of calves (adjusted for year; $r = -.26$; $P < .05$). In both years, cows with one or more dry quarters after calving had calves that weighed 15.6 kg less at 60 d than cows with no dry quarters ($P < .01$; Figure 9a).

There was an interaction between year and treatment for calf weights a 60 d ($P < .07$), however, there was not a treatment x infection status effect on calf weights at 60 d ($P > .1$). In 1995, calf weights were similar for treated and control cows at 60 d, but in 1996, control cows had calves that were heavier ($P < .05$) at 60 d compared with calves from treated cows (Figure 10).

Infection status of the dam post partum did not alter calf weights at 60 d (P > .1).
Cows noninfected post partum had calves with similar 60 d weights compared with cows
infected with mastitis causing organisms post partum (Figure 11a).

Adjusted 205 d (ADJ205 d) weights for treated and control calves were similar (232 ±
5 vs 223 ± 5 kg respectively; P > .1; Figure 12), and there were no interactions between
treatment, infection status post partum, and year. Calves weighed more at weaning in
1996 than in 1995 (240 ± 5 vs 215 ± 5 kg respectively; P < .01). Cows that were
noninfected post partum had calves with similar ADJ205 d weights compared with cows
that were infected post partum (231 ± 5 vs 223 ± 5 kg respectively; P > .1; Figure 11b).
In both years cows with one or more dry quarters (n = 13) had calves that weighed 31 kg
less at weaning compared with calves from cows with no dry quarters (n = 71; P < .01;
Figure 9b).

Discussion

Somatic cell counts of dairy cows are highly correlated with CMT scores (Pearson and
Greer, 1974). We observed a similar relationship between CMT scores and SCC in beef
cows, however, the values for SCC of beef cows (range of 66 to 3035 x 10^3 cells/mL) are
less than those for dairy cows (Schalm and Noorlander, 1957; Philpot and Nickerson,
1991). The use of the CMT for beef cattle has been limited. One study reported factors
in a beef herd that influenced CMT scores (Wilson et al., 1971), however they did not
provide a range of SCC for CMT scores. Another study utilized CMT to screen quarters
of beef cows for mastitis treatment, but mean SCC for CMT scores were not reported

(Newman et al., 1991). To our knowledge, this study is the first to summarize a range of SCC for CMT scores of beef cows.

The percentage of quarters infected with any organism post partum averaged 17% for CMT scores 0 thru 1, which was less than 42% for CMT scores 2 and 3. In dairy cows, only CMT scores of 2 and 3 are considered reliable to predict infection, with all others usually classified as suspect (Gray and Schalm, 1960). Percentage of infection in dairy cows ranges from 33 to 54% for quarters with a CMT score of 1, and up to 71 to 96% for quarters with a CMT score of 3 (Marshall and Edmondson, 1962; Pearson and Greer, 1974). In beef cows, 8, 21, 45, 60, and 71 % of quarters with CMT scores of 0, T, 1, 2, and 3 were infected (Newman et al., 1991), which is greater than the percentage of infected quarters in our experiment with similar CMT scores.

Somatic cell counts are usually greatest during early and late lactation in dairy (Bodoh et al., 1976; Reneau, 1986) and beef cows (Wilson et al., 1971; Hunter and Jeffrey, 1975; Newman et al., 1991), and SCC may be elevated for up to two weeks after parturition (Cullen, 1968; Natzke et al., 1972). Newman et al. (1991) took initial CMT samples much later post partum than we did (4 vs. 2 weeks respectively). In our experiment, CMT scores of 2 and 3 could be a result of greater SCC associated with a normal increase due to stage of lactation rather than infection. Newman et al. (1991) determined that sensitivity of the CMT test was greatest when any positive reaction was considered (78%) but decreased to 19% when only scores of 3 were evaluated. Furthermore, specificity was greatest when considering only CMT scores of 2 and 3, but decreased to unacceptable levels when considering any positive score. These researchers concluded that the CMT

could not discriminate effectively between infected quarters and noninfected quarters to select quarters or cows for treatment. In our study, the CMT scores was correlated with SCC, but not infection. The fact that SCC may have been greater than normal during the early post partum period may mean that CMT scores were more a function of SCC rather than the presence of mastitis causing bacteria. Thus the CMT was a better indicator of SCC than infection.

The presence of mastitis causing bacteria after calving was associated with an increased incidence of infection at weaning. Fourteen to 31% of quarters of dairy cows remained infected throughout lactation (Jackson, 1961). Intramammary infections in beef cows tend to persist throughout lactation (Newman et al., 1991), and Simpson et al. (1995) determined that 39% of infected quarters of beef cows remained infected.

Intracellular location of some mastitis causing organisms can result in decreased effectiveness of intramammary treatment (Nickerson and Owens, 1993). Thus, systemic treatment of mastitis has been evaluated (Giesecke, 1977; Ziv, 1980). Intramuscular treatment has been as effective as intramammary treatment in eliminating mastitis (Ziv and Storper, 1985; Jarp et al., 1986). A combination of intramuscular and intramammary treatment increases cure rates and maintains greater concentrations of antibiotics in the mammary tissue (Owens et al., 1988; Erskine et al., 1994; Owens et al., 1994). Oxytetracycline can maintain minimal inhibitory concentrations in udder secretions and provides protection against new infection (Giesecke, 1977; Soback et al., 1980). In a previous study with beef cows, we determined that intramuscular oxytetracyline treatment after calving did not decrease udder infection. However, no attempt was made to identify

infected cows before treatment, thus the inclusion of uninfected cows could have negated any possible benefit of treating infected cows. In our current study, cows were selected for treatment on the basis of CMT score, however treatment did not decrease the percentage of infected cows. Reasons for the lack of efficacy of oxytetracycline are not readily apparent. In dairy cows, duration of treatment has the greatest impact on cure rate (Jarp et al., 1986). It may be that a single injection after calving is too short a duration of treatment to decrease bacterial populations. Another possibility is that treatment may cure infections over a short term, but cows may become reinfected prior to the subsequent sampling period. However, no treatment effects were found on infection rates of cows at approximately 60 d of lactation. Furthermore, in this study, antibiotic sensitivity of isolated bacteria was not performed. Some resistant strains of bacteria may have been present, however we have previously found that oxytetracycline can decrease udder infections in this herd when cows were treated at drying-off and again after calving (Duenas et al., 1995). We defined a cow to be infected based on the presence of mastitis causing bacteria. No attempt was made to quantify bacterial populations for each quarter, and not all cows that were defined to be infected had clinical mastitis. Treatment may reduce bacterial populations and improve udder health, but not completely eradicate the organisms.

Intramammary infection is the major factor that contributes to an increase in SCC in dairy cows (Dohoo and Meek, 1982; Reneau, 1986; Harmon, 1994). Both experimentally induced and naturally occurring infections have resulted in a four-fold increase in SCC (Sheldrake et al., 1983; Fox and Schultz, 1985; Schukken et al., 1994). Our results with

beef cows revealed that AVSCC per cow were greater for infected cows compared with noninfected cows. This is in agreement with other reports for beef cows (Watts et al, 1986; Newman et al., 1991; Simpson et al., 1995). Hunter and Jeffrey (1975) determined that most infected quarters had SCC greater than 500×10^3 cells/mL, and 20% had SCC greater than $1,000 \times 10^3$ cells/mL. These investigators point out that while individual quarters that were infected had abnormal SCC, cows usually had SCC of less than 500×10^3 cells/mL. This is in contrast to other investigators who found that SCC of non-infected quarters were 555×10^3 cells/mL while infected quarters had SCC greater than 794×10^3 cells/mL (Watts et al., 1986). In our study, noninfected quarters had SCC of 91 $\times 10^3$ cells/mL while infected quarters had SCC of 1077×10^3 cells/mL. These findings would agree with Newman et al. (1991) who found that noninfected quarters of beef cows had SCC of about 20×10^3 cells/mL. In previous investigations, we found that SCC from noninfected quarters ranged from 58 to 153×10^3 cells/mL, while infected quarters had SCC of 400 to 533×10^3 cells/mL (Duenas et al, 1994). Wilson et al. (1971) observed that although mastitis causing bacteria may be present in beef cows, SCC usually were not elevated to an abnormal concentration.

Treatment of beef cows with systemic antibiotics at drying-off and after calving decreased SCC per quarter by 48% (Duenas et al., 1995). However, in our experiment, when cows were only treated post partum, it did not decrease MXSCC or AVSCC per cow, nor did it decrease SCC on a quarter basis. Treatment may have decreased SCC through mid-lactation, but we did not sample cows until weaning. However, when SCC

were analyzed for cows or quarters that had calves weaned early, at 60 d of lactation, a treatment effect was not found.

Milk loss of dairy cows is 7 to 9% when SCC are greater than 400×10^3 cells/mL (Schultz, 1977b; Miles et al., 1992). Average SCC per cow were negatively correlated with 60 d weights, but not 205 d weight of calves. The decreased weights at 60 d was presumably due to a decrease in milk production. Milk production of primiparous Simmental cows was less for cows with greater SCC (Simpson et al., 1995). Other investigators found that SA infection caused an increase in SCC which were associated with decreased weaning weights presumably through decreased milk production (Watts et al., 1986). In another study, increased SCC were associated with udder infection and decreased calf weight gain (Newman et al., 1991).

Intramammary infections of beef cows decreased 205 d weaning weights of calves by 7 to 9% (Haggard et al., 1983; Watts et al., 1986). Growth of calves was influenced the most by the infection status of dams from 60 to 100 d after calving (Newman et al., 1991). We found that post partum infection status of the cow did not alter 60 d or ADJ205 d weights of calves. We previously determined that infection status of the dam did not alter 205 d weaning weights of the calves (Lents et al., 1996). This lack of detrimental influence of udder infection on calf weight gain could be due to the fact that not all cows defined as infected had clinical mastitis, and milk production may not have been compromised severely enough to cause an effect on calf weights. Another possible explanation is that udder infections may decrease milk production during early lactation without a major influence on calf weight gain. A calf may not consume all milk that is

secreted early in lactation (Newman et al., 1991), thus reduced milk secretion of the dam early in the life of the calf may not adversely affect gain. As calves get older, they receive energy from sources other than milk (Neville, 1962; Haggard et al., 1983; Ansotegui et al., 1991). Therefore the effect of decreased milk production due to udder infections may not be apparent over the entire lactation. Furthermore, most cows were infected in only one quarter, and this may not produce a large enough decrease in milk production to adversely effect calf growth.

Newman et al. (1991) determined that treatment of beef cows for mastitis decreased udder infections, but did not evaluate the effects of treating cows with antibiotics on weight gains of calves. In another study, calves from beef cows treated for mastitis weighed 12.5% more at 60 d of age than calves from untreated controls (Kirkbride, 1977). We previously determined that intramuscular treatment of cows with antibiotics at drying-off and/or after calving did not alter 205 d weaning weights of calves (Lents et al., 1996). In the current study, treatment did not alter 60 d or 205 d weight of calves. This agrees with the observation that treatment did not alter infection status of cows, and that infection status did not adversely influence weight gain of calves. With a highly variable trait such as weaning weight, we may not have had enough observations to detect a treatment effect.

In conclusion, the CMT is a better indicator of SCC than an indicator of infection. Average SCC per cow is negatively correlated with calf weight at 60 d, but not at 205 d. Intramammary infections of beef cows causes increased SCC, but does not adversely influence weight gain of calves. Treatment of beef cows with intramuscular

oxytetracycline after calving did not alter SCC or infection rates, and did not increase calf weights at weaning.

Implications

Treatment of beef cows with intramuscular oxytetracycline after calving does not decrease somatic cell counts in milk, or decrease intramammary infection at weaning. Weight of calves at 60 and 205 d is not influenced by intramuscular oxytetracycline treatment of cows after calving, but weights are significantly reduced if cows have one or more dry quarters. Producers should use weaning weights of calves to help identify cows that have nonfunctional quarters, which should be culled.
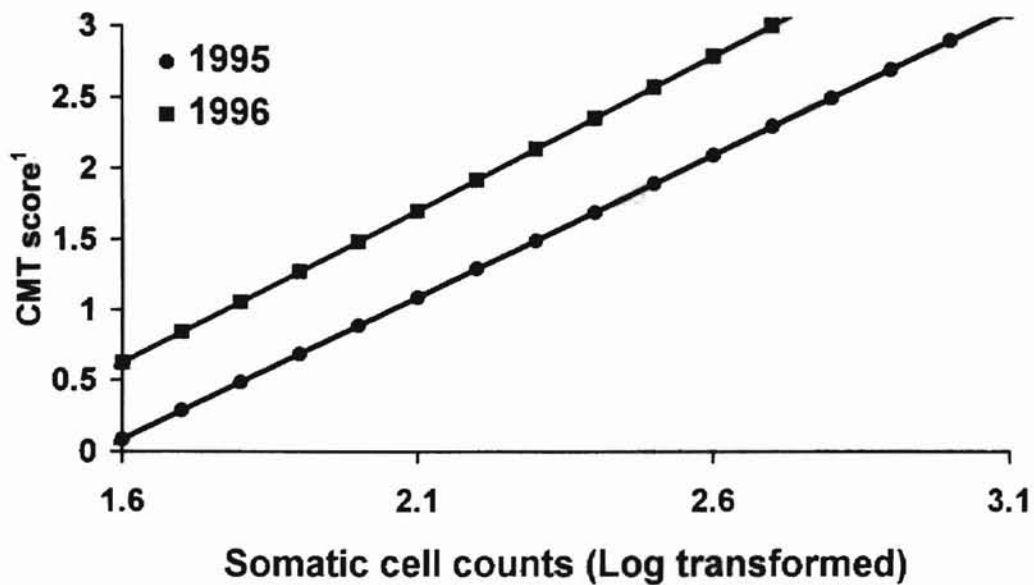
Figure 1. Least squares regression for quarter somatic cell counts after calving with California Mastitis Test (CMT) scores. [1]A CMT value of .5 represents the CMT score T.
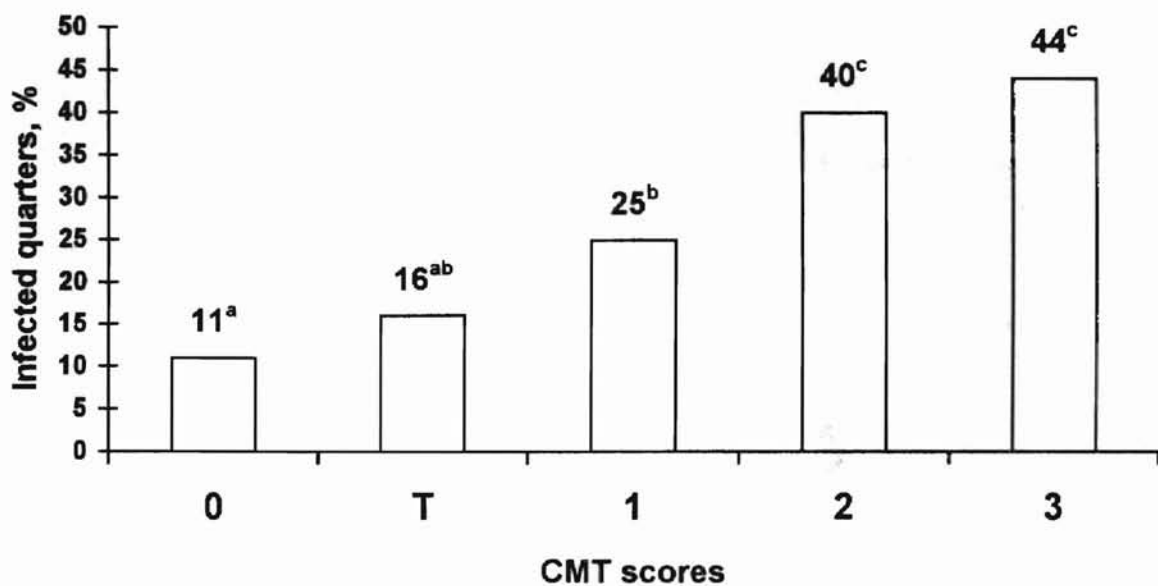
Figure 2. Least squares means for the percentage of quarters that were infected when quarters were classified as 0, T, 1, 2, or 3 by the California Mastitis Test (CMT).
[abc]Columns lacking common superscripts differ (P < .01).
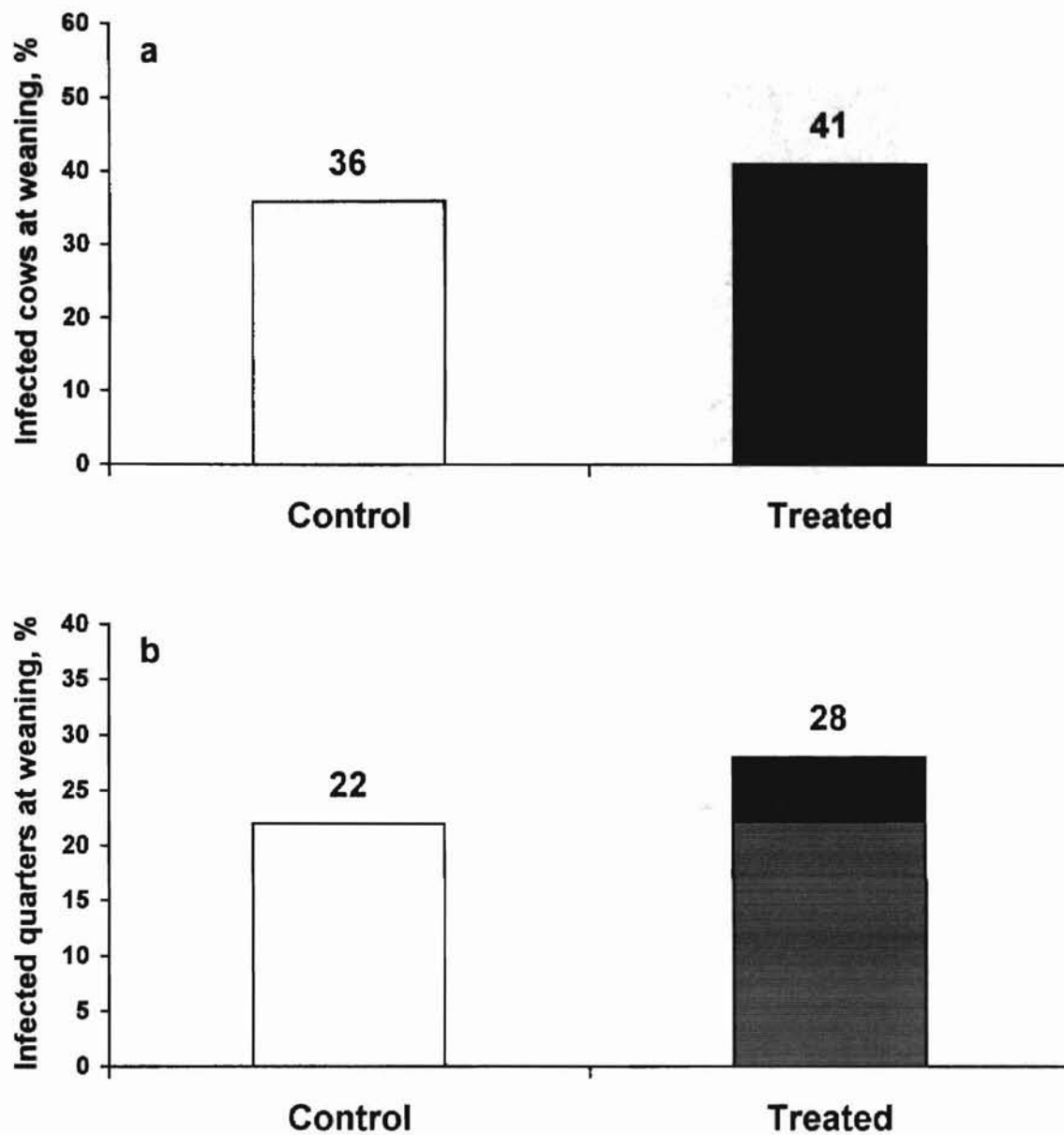
Figure 3. Least squares means for the percentage of control and treated (a) cows; (b) and quarters infected with any organism at weaning.
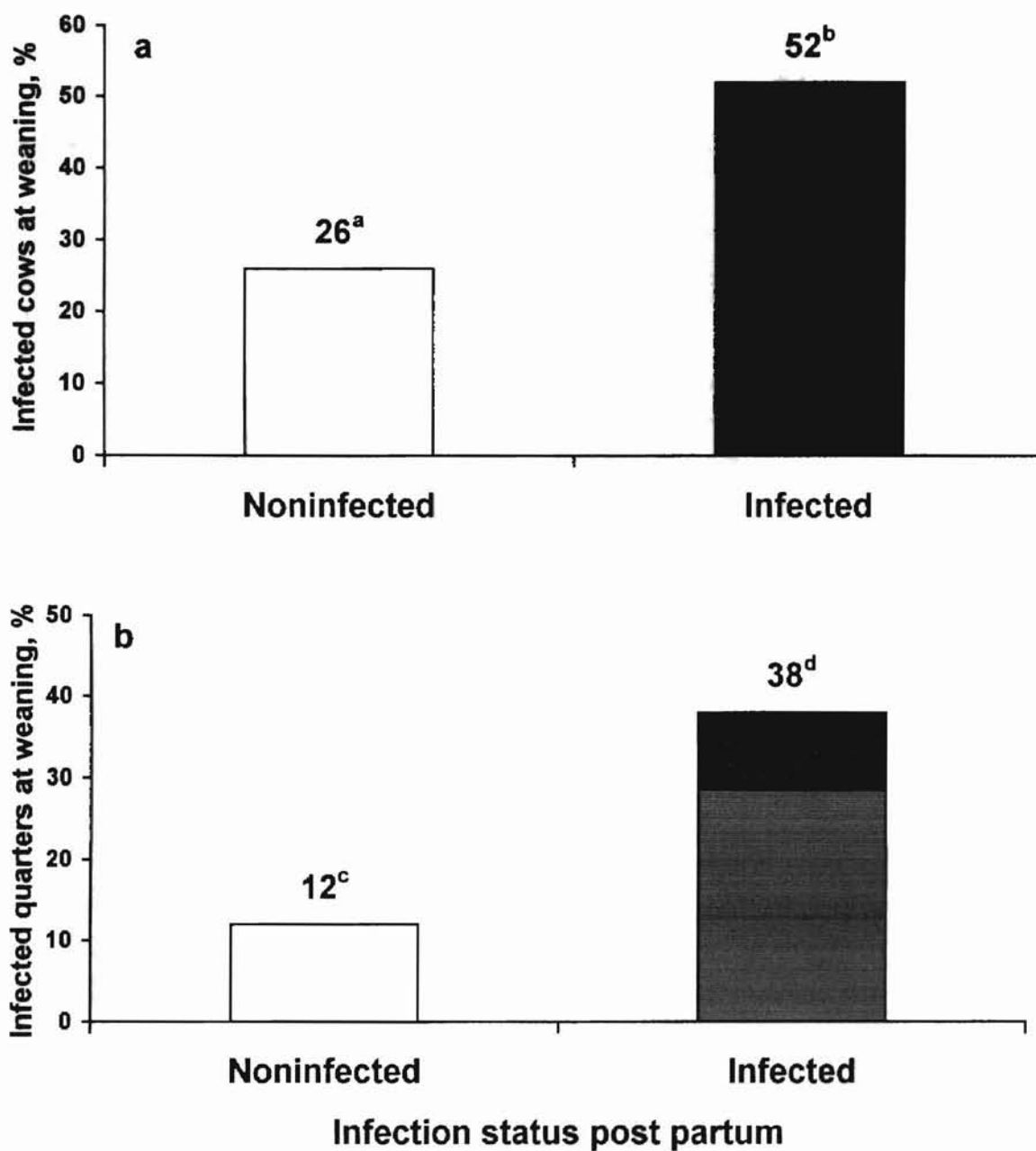
Figure 4. Least squares means for the percentage of (a) cows; (b) and quarters noninfected or infected with any organism after calving that were infected at weaning. [ab]Means lacking a common superscript letter differ (P < .05). [cd]Means lacking a common superscript letter differ (P < .01).
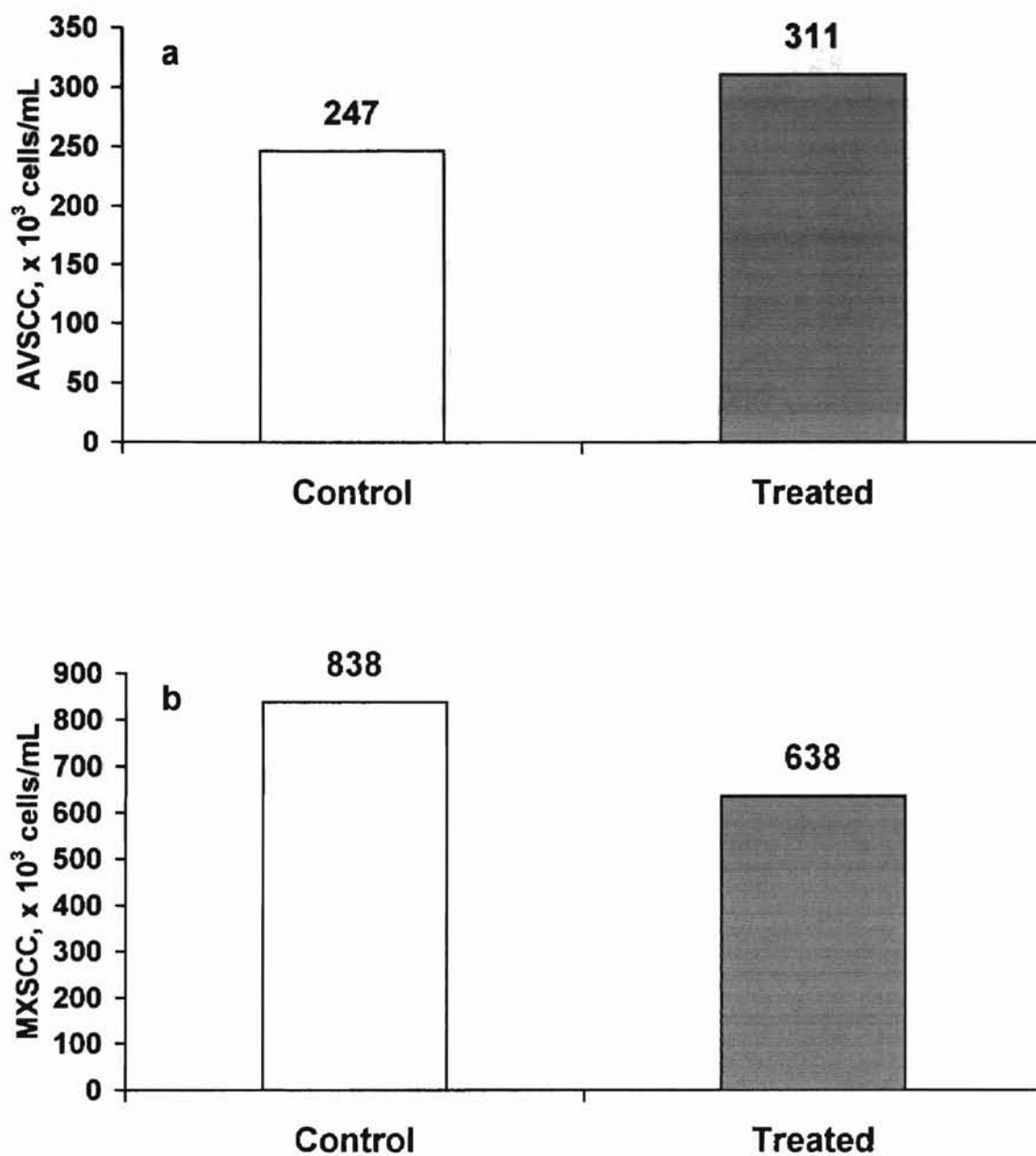
Figure 5. Least squares means for (a) average somatic cell counts (AVSCC; SEM = 60) and (b) maximum somatic cell counts (MXSCC; SEM = 245) per cow at weaning for treated and control cows.
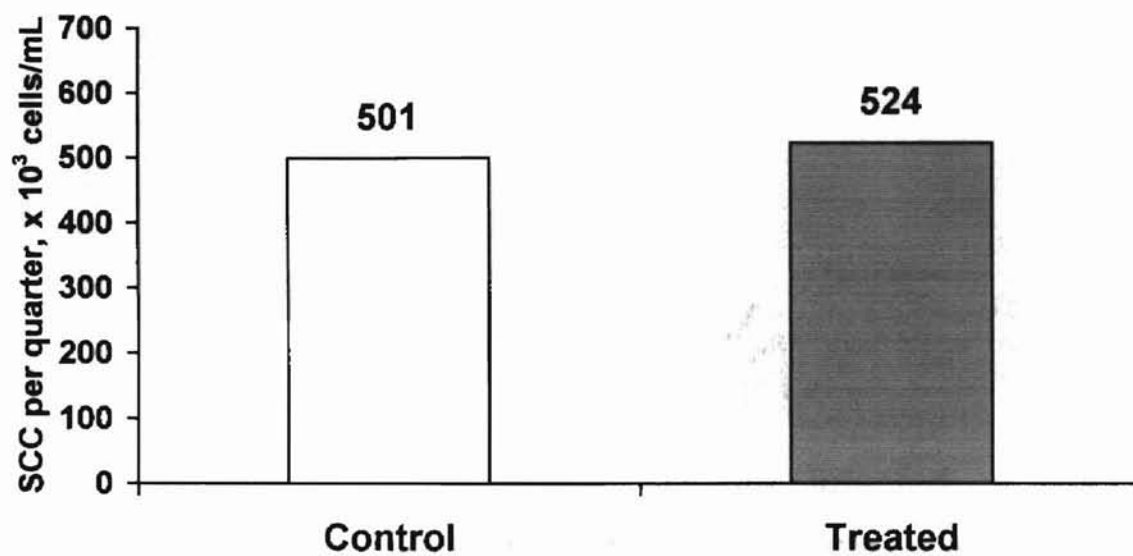
Figure 6. Least squares means for somatic cell counts (SCC) per quarter at weaning for treated and control cows (SEM = 86).
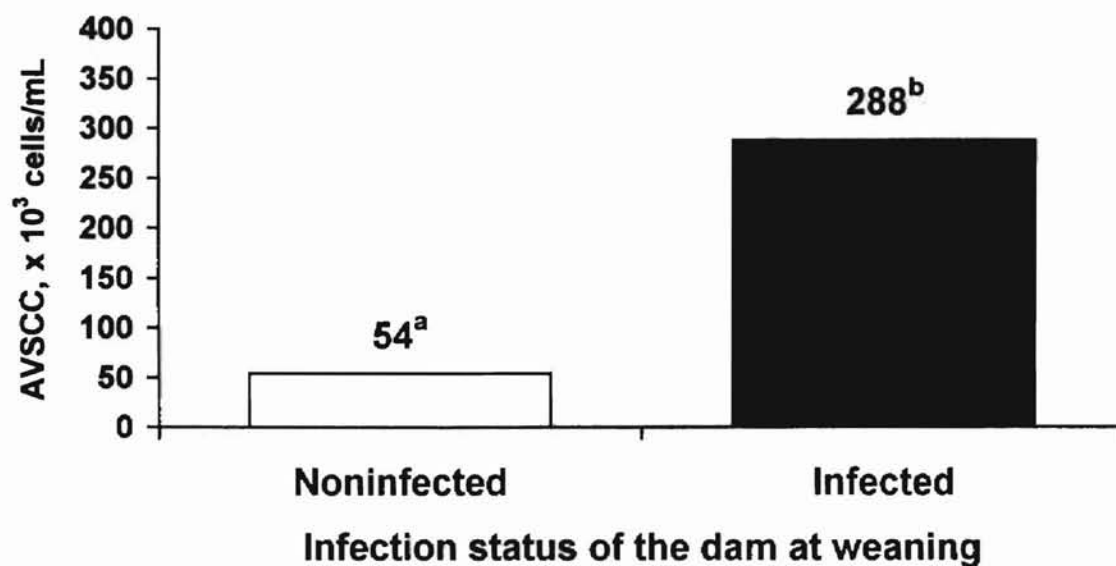
Figure 7. Least squares means for average somatic cell counts (AVSCC) at weaning for cows noninfected or infected with any organism at weaning (SEM = 73). [ab]Means with different superscript letters differ (P < .05).

Table 1. Least squares means for somatic cell counts (SCC)[1] of noninfected and infected quarters for each year (year x infection status; $P < .01$)

Infection status[3]	Year[2]	
	1995[4]	1996[4]
Noninfected	75(150)^{ax}	106(107)^{ax}
Infected	1571(34)^{bx}	583(20)^{by}

[1]SCC x 10^3 cells/mL.
[2]MSE = 606663.
[3]Infection status at weaning.
[4]Number in parentheses indicates number of quarters.
^{ab}Means within a column, with different superscript letters differ ($P < .05$).
^{xy}Means within a row, with different superscript letters differ ($P < .01$).
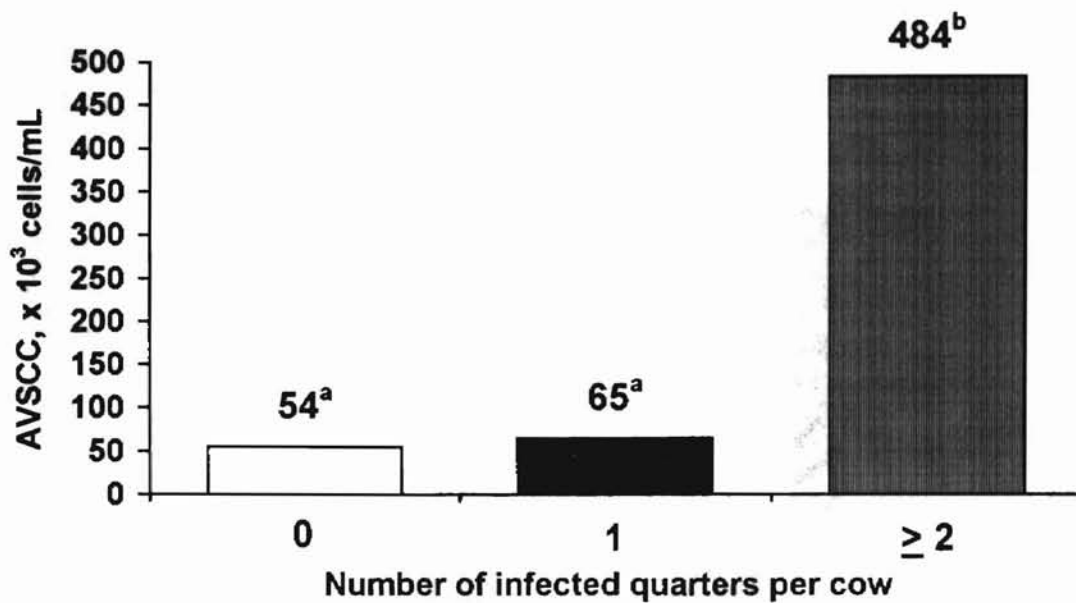
Figure 8. Average somatic cell counts (AVSCC; SEM = 96) at weaning for noninfected cows, and cows with one infected quarter (1), or two or more infected quarters (≥ 2). [a,b]Means with different superscript letters differ ($P < .05$).
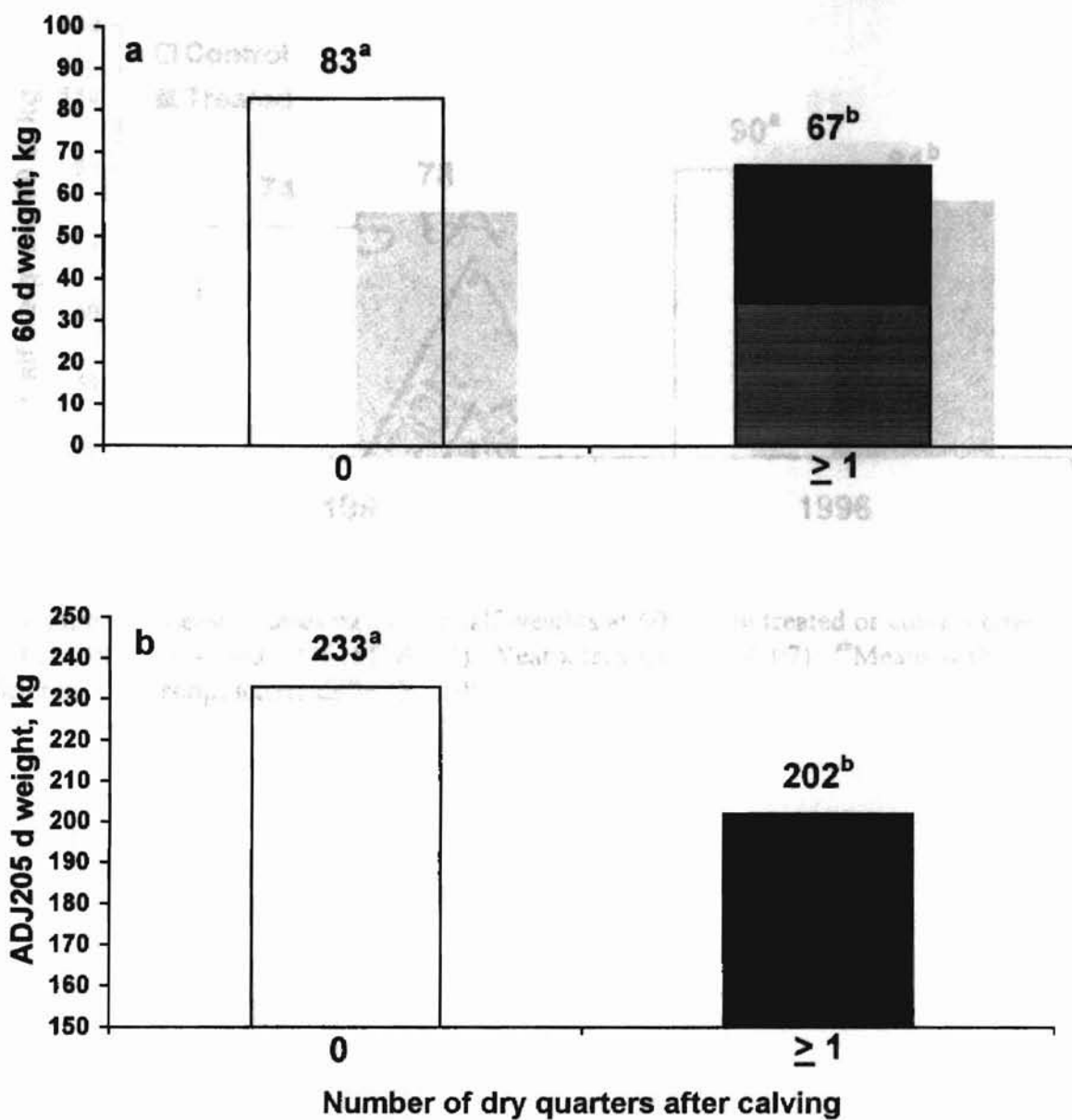
Figure 9. Least squares means for (a) 60 d weight (SEM = 3) and (b) adjusted 205 d weaning weight (ADJ205; SEM = 6) of calves from dams that had one or more dry quarters (≥ 1) or no dry quarters (0) after parturition. [ab]Means with different superscripts differ (P < .01).
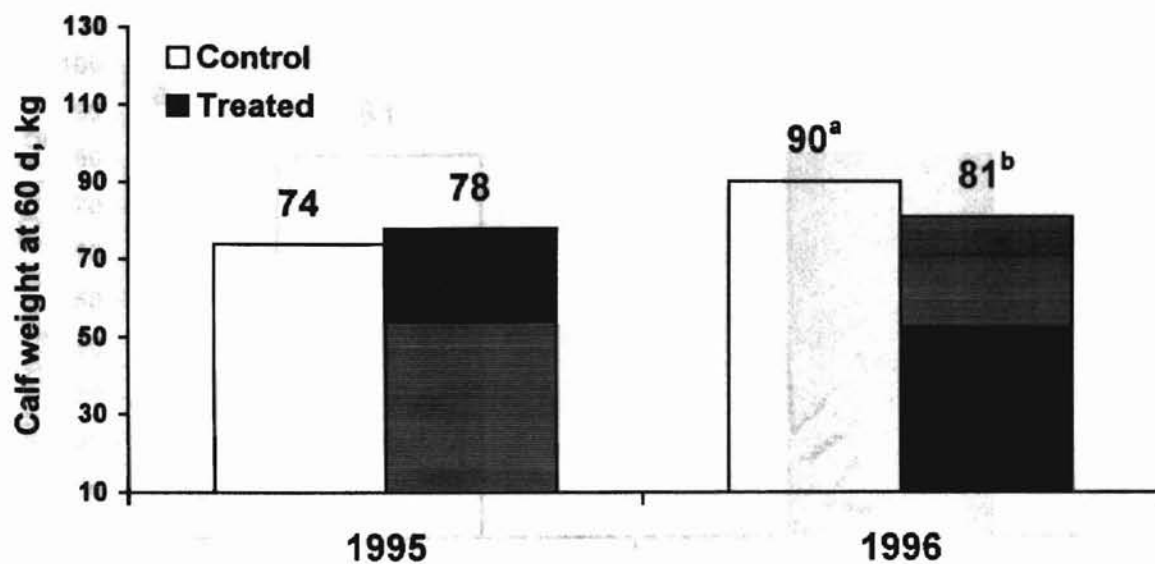
Figure 10. Least squares means for calf weights at 60 d from treated or control cows in 1995 (SEM = 4) and 1996 (SEM = 3). Year x treatment (P < .07). [ab]Means with different superscript letters differ (P < .05).
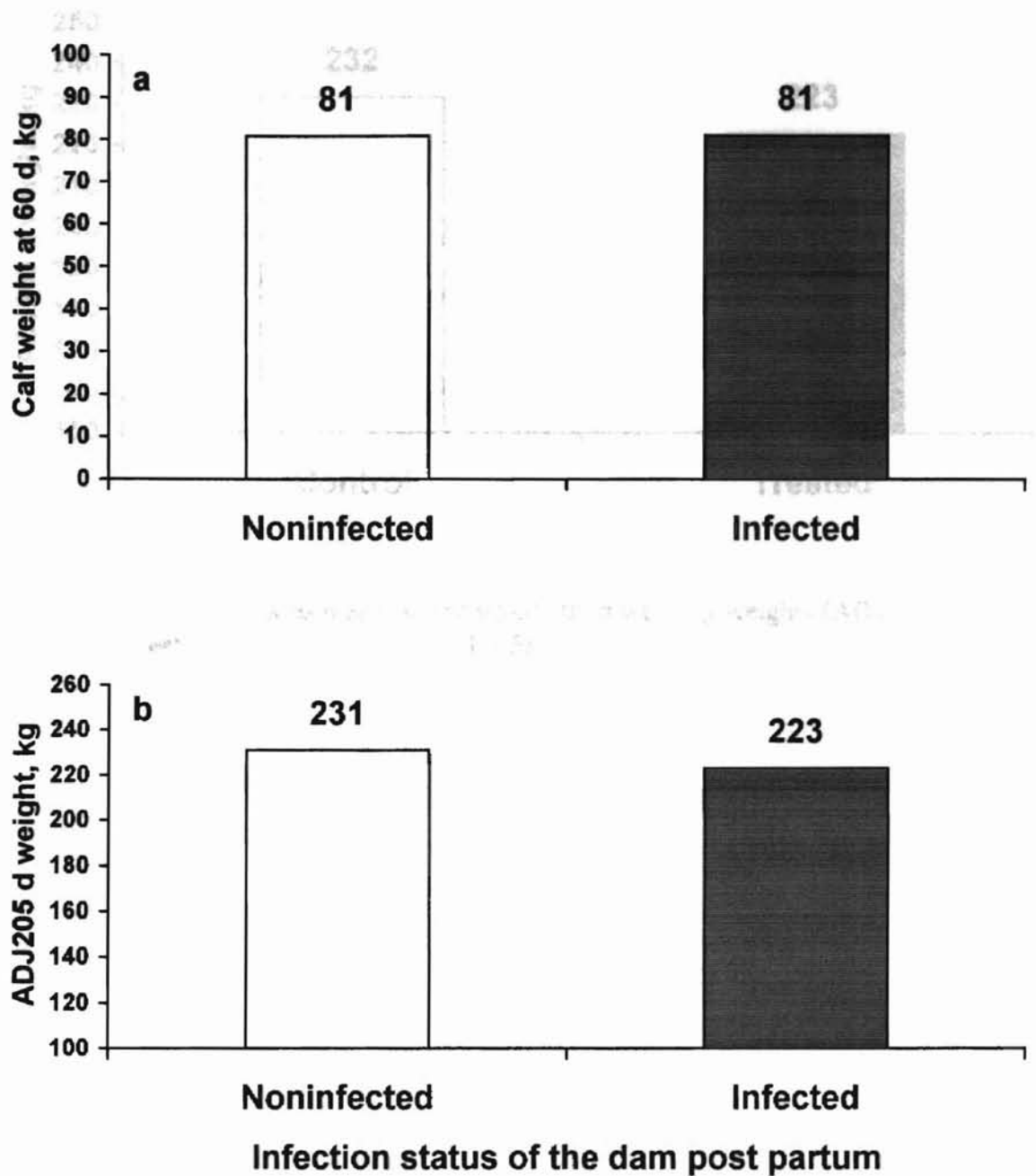
Figure 11. Least squares means for (a) 60 d weights of calves (SEM = 2); (b) adjusted 205 d weights of calves (ADJ205; SEM = 5) from dams that were noninfected or infected post partum.
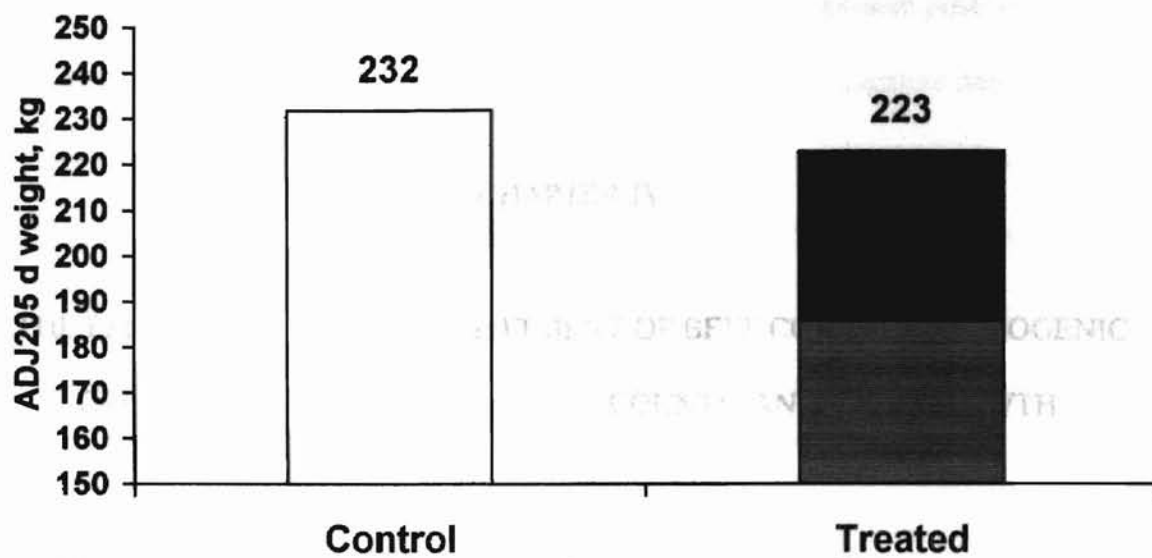
Figure 12. Least squares means for adjusted 205 d weaning weights (ADJ205) of calves from treated or control cows (SEM = 5).

CHAPTER IV

THE EFFECTS OF DRY COW TREATMENT OF BEEF COWS ON PATHOGENIC ORGANISMS, MILK SOMATIC CELL COUNTS, AND CALF GROWTH.

ABSTRACT: Spring calving Hereford and Hereford x Angus multiparous cows (n = 90) were utilized to determine the effects of intramammary treatment with Penicillin G procaine and novobiocin at the time of drying-off on udder health and calf growth following the subsequent calving. Cows were blocked by age and randomly assigned to receive intramammary treatment (n = 45) or untreated controls (n = 45). Quarter milk samples were collected at drying-off and at 8 to 14 d post partum. Milk samples were analyzed for somatic cell counts (SCC) and mastitis causing bacteria. Dry cow treatment decreased the number of cows and quarters infected post partum (P < .05). Treatment was effective in preventing (P < .05) the development of new infections during the dry period, but not in eliminating (P > .1) infections that were present at drying-off. Average SCC (AVSCC) per cow were similar for treated and control cows, however, maximum SCC were greater for control compared with treated cows. There was a significant interaction between infection status at drying-off and treatment on SCC per quarter post partum (P < .06). Somatic cell counts for treated and control quarters that were noninfected at drying-off were similar, but if quarters were infected at drying-off, treated

quarters had less SCC than controls (P < .05). Type of bacteria present post partum altered SCC per cow and quarter. Cows infected with coagulase negative staphylococci (CNS) had AVSCC similar to noninfected cows, however cows infected with Staphylococcus aureus (SA) had the greatest AVSCC (P < .05). Quarters infected with CNS had greater SCC than noninfected quarters, and SA infected quarters had the greatest SCC (P < .05). Treatment of cows at drying-off did not influence 110 d or 205 d weights of calves during the next lactation (P > .1). Post partum infection status of the cow did not influence 110 d or 205 d weights of calves (P > .1). In conclusion, treatment of beef cows at drying-off with intramammary antibiotics decreased intramammary infections post partum, and decreased SCC in quarters that were infected at drying-off. However, treatment did not alter 110 d or 205 d weights of calves during the subsequent lactation.

Introduction

Heavier weights of calves at weaning will increase profitability for cow-calf producers. Weaning weights of calves are effected by age of the dam, sex of calf, and forage intake (Neville, 1962; Cundiff et al., 1966; Ansotegui et al., 1991; Sowell et al., 1996). Milk production of cows is the most important factor influencing weight gain of calves, and accounts for about 60% of the variation in weaning weights (Neville, 1962; Rutledge et al., 1971). This influence of milk production on gain is greatest during the first 60 days of age (Drewry et al., 1959; Neville, 1962), which corresponds with peak milk production in mature beef cows (Clutter and Nielsen, 1987; Marston et al., 1992).

Mastitis increases somatic cell counts (SCC) and decreases milk production of dairy cows (Crossman et al., 1950; Bartlett et al., 1991; Lescourret and Coulon, 1994). Increased SCC are negatively correlated with milk production (Raubertas and Shook, 1982). Mastitis can reduce milk production of dairy cows 5 to 25% (Janzen, 1970), and the detrimental effect on milk production is evident in subsequent lactations (Fetrow et al., 1991). Mastitis causes increased SCC in beef cows (Watts et al., 1986; Newman et al., 1991; Simpson et al., 1995). Beef cows with greater SCC produced less milk (Simpson et al., 1995), and SCC were negatively correlated with weaning weights of calves (Watts et al., 1986). Mastitis in beef cows is also associated with decreased weight gain of calves (Haggard et al, 1983; Watts et al., 1986; Newman et al., 1991).

Most intramammary infections occur during the dry period (Neave et al., 1950; Oliver and Mitchell, 1983). Therefore, dairy producers have adopted the practice of treating all quarters of all cows at drying off (Natzke, 1971). Intramammary dry cow therapy eliminated 25 to 100 % of udder infections (Ziv et al., 1981; Davidson et al, 1994; Erskine et al., 1994). Treatment of quarters of beef cows that had increased SCC at weaning resulted in decreased infection rates at the subsequent calving (Newman et al., 1991). However, this method will result in some quarters with subclinical infection not being treated (Philpot, 1979). The objective of this experiment was to determine the effect of treating all quarters of beef cows with an intramammary infusion product at the time of weaning on udder health, somatic cell counts, and calf growth following the subsequent calving.

Materials and Methods.

Animals

Spring calving, multiparous Hereford and Hereford x Angus cows (n = 90) were

utilized to determine the effects of intramammary treatment with penicillin G procaine and

novobiocin (200,000 i.u. and 400 mg, respectively; Albadry Plus®, Upjohn Limited,

Flemingway, Crawley, West Sussex, England) at drying-off on udder infection and calf

growth following the subsequent calving. Cows grazed bermuda grass and native range

pastures at the Oklahoma Agricultural Experiment Station Range Cow Research Center,

24 km west of Stillwater. At drying-off in October, cows were blocked by age and

randomly assigned to intramammary treatment with antibiotic (n = 45), or untreated

control (n = 45). Calves were weaned and cows were supplemented with a 40% range

cube during the winter to maintain a body condition score of 4 to 5.5 (Wagner et al.,

1988). Following the subsequent calving (February thru May), calf weights were recorded

at birth and every thirty days until weaning in October.

Milk Samples.

Milk samples were collected from each quarter of cows at drying-off and at 8 to 14 d

following the subsequent calving. Calves were removed from cows for approximately two

hours before sampling. Cows were restrained in a squeeze chute and administered 10

units of oxytocin (Vedco, Inc., St. Joseph, MO) to facilitate milk let-down. Teats were

dipped in a .1 % iodine solution and wiped dry with individual paper towels. The first two

or three streams of milk were discarded and 10 mL of milk from each quarter were

collected into plastic vials containing Microtablets (D & F Control Systems, Inc., San

Ramon, CA) for preservation. Samples were sent to the DHIA laboratory, Manhattan, KS, within one day for analysis of somatic cell counts (SCC) . Teat ends were then individually disinfected with a cotton swab soaked in 70 % ethyl alcohol. Two streams of milk from each quarter were discarded and 3 mL of milk were aseptically collected into sterile polypropylene snap cap tubes (Fisherbrand®, Pittsburgh, PA). Sterile samples were immediately cooled to 4 °C, transported to the lab and stored at -20 °C, until packaged in dry ice and transported to the Immunology and Disease Resistance Laboratory, USDA-ARS, Beltsville, MD, for bacteriological analyses for coagulase negative staphylococci and *Staphylococcus aureus*.

Bacteriological analysis.

Sterile milk samples were allowed to thaw at room temperature and vortexed. Twenty µL of milk were plated on one quarter of an esculin blood agar plate (5 % red blood cells), and a P-agar plate supplemented with acriflavine. Plates were incubated at 37 °C and bacterial growth was determined at 24 and 48 h.

A quarter was considered to be infected if three or more colonies of the same organism were isolated from the esculin blood agar plate. Identification of microorganisms was based on colony morphology, hemolytic and hydrolytic patterns, gram stain (Bacto® Gram Stain Set, Difco Laboratories, Detroit, MI), catalase production (hydrogen peroxide, Sigma®, Sigma Chemical Co., St. Louis, MO) and tube coagulase test (Coagulase Plasma EDTA, Difco Laboratories, Detroit, MI).

Dry-cow Treatment

After milk samples were collected at drying-off, teat ends were individually wiped with alcohol pads. One tube of penicillin G procaine (200,000 i.u.) and novobiocin sodium (400 mg) was administered to each quarter. Teat canals were held closed with fingers to retain the infusion product in the udder, and teats were lightly massaged two or three times from the tip to the dorsal surface to facilitate the movement of the infusion product upward into the gland cistern. After treatment, each teat was dipped in .1 % iodine solution. Control cows were not treated, however each teat was dipped with .1 % iodine solution after sampling.

Statistical Analysis

Cows were considered to be infected if one or more of coagulase negative staphylococci or *Staphylococcus aureus* were present in one or more quarters. Chi-square analysis, using the FREQ procedure of SAS (1994), was used to determine the efficacy of treatment in eliminating infections or preventing new infections during the dry period.

Somatic cell counts were analyzed using log transformed values, however, actual SCC values are reported. The quarter with the greatest SCC for each cow was determined and used as the maximum SCC (MXSCC) value for that cow. Average SCC (AVSCC) of the four quarters for each cow were determined as a geometric mean and used as the SCC value for each cow. Least squares analysis of variance was used to determine treatment effects on SCC post partum. The model included treatment, infection status at drying-off, and the interaction. If the interaction was not significant, it was dropped from the model.

Calf weights were recorded at birth (February thru May) and every 30 d from May 5 to weaning. Multiple regression equations were developed to determine the weights of

individual calves at 110 d and 205 d of age. The model contained linear, quadratic, and cubic components. Calves with weight gains greater or less than three standard deviations from the mean were considered to be outliers. One calf died early in the experiment, and weights of two calves were less than the three standard deviations from the mean and were deleted from the analysis. The effect of treatment on 110 d and 205 d weights were determined using a model that included infection status at drying-off, treatment, and the interaction, with sex and cow age as covariates.

Results

Cure rates of infections present at drying-off were not different for treated cows or quarters compared with controls, but fewer infections developed over the dry period in treated quarters and cows compared with controls (Table 1). At drying-off, 15 treated quarters and 11 treated cows were infected. This was not different compared with the 21 control quarters and 16 control cows that were infected at drying-off (P > .1). Treatment did not influence the number of infected quarters or cows with infections cured during the dry period (P > .1). Twelve treated quarters and 7 treated cows had infections cured during the dry period compared with 18 control quarters and 8 control cows. However, treatment decreased the incidence of new infections during the dry period (P < .05). Only 13 treated quarters and 7 treated cows developed new infections during the dry period compared with 27 control quarters and 16 control cows. Thus, treatment decreased the number of quarters and cows infected post partum (P < .05). Only 15 treated quarters and

11 treated cows were infected post partum compared with 30 control quarters and 24 control cows.

Treatment increased the percentage of cows with noninfected quarters post partum (Table 2). Seventy-six percent of treated cows had no infection in any quarter post partum compared with only 51% of control cows (P < .05). Only 15% of treated cows were infected in one quarter compared with 31% of control cows (P < .1). Treatment did not affect the percentage of cows with ≥ 2 quarters infected post partum (P > .1). Nine percent of treated cows were infected in two or more quarters compared with 18% of control cows.

Dry cow treatment did not alter AVSCC for treated cows compared with controls (148 ± 41 x 10^3 vs 96 ± 39 x 10^3 cells/mL respectively; P > .1; Figure 1a). However, treated cows had less MXSCC per cow compared with control cows (400 ± 173 x10^3 vs 732 ± 165 x10^3 respectively; P < .05; Figure 1b).

There was a significant interaction between infection status at drying-off and treatment on SCC per quarter post partum (P < .06; Table 3). Post partum SCC were similar for treated and control quarters that were noninfected at drying-off. However, if quarters were infected at drying-off, treated quarters had less SCC post partum than controls (246 ± 175 x 103 vs 489 ± 137 x 103 cells/mL respectively; P < .05).

The type of bacterial infection post partum influenced AVSCC per cow (Figure 2a). Average SCC for noninfected cows were not different compared with CNS infected cows (58 ± 33 x 10^3 vs 109 ± 52 x 10^3 cells/mL respectively; P > .1). However AVSCC post partum for SA infected cows were greater (262 ± 84 x 103 cells/mL) than both CNS

infected or noninfected cows (P < .05). Type of bacteria present post partum also affected SCC per quarter (Figure 2b). Quarters infected with CNS had greater SCC than noninfected quarters ($347 \pm 98 \times 10^3$ vs $129 \pm 33 \times 10^3$ cells/mL respectively; P < .05), whereas quarters infected with SA had greater SCC ($851 \pm 190 \times 10^3$ cells/mL) than CNS infected or noninfected quarters (P < .05).

There was a significant interaction between infection status at drying-off and treatment on 110 d weights of calves during the next lactation (P < .05; Table 4). Calf weights at 110 d were similar for treated and control cows that were noninfected at drying-off, but if cows were infected at drying-off, treated cows had calves with greater 110 d weights than control cows (154 ± 5 vs 139 ± 3 kg respectively; P < .07). Treatment of cows at drying-off did not influence 110 d weights of calves during the next lactation (Figure 3a). Both treated and control cows had calves with similar 110 d weights (145 ± 3 vs 142 ± 2 respectively; P > .1). Treatment of cows at drying-off did not affect 205 d weights of calves following the subsequent lactation (Figure 3b). Both, treated and control cows had calves that weighed 243 ± 4 kg at weaning (P > .1).

Post partum infection status of the cow did not influence 110 d or 205 d weights of calves. Cows that were noninfected or infected post partum had calves with similar 110 d weights (143 ± 2 vs 143 ± 2 respectively; P > .1; Figure 4a). Post partum infection status of cows did not influence 205 d weights of calves (241 ± 3 vs 245 ± 5 kg for noninfected and infected cows respectively; P > .1; Figure 4b).

Discussion

In dairy cows the rate of new intramammary infections is greater in the dry period as compared with lactation (Neave et al., 1950; Oliver and Mitchell, 1983). The purpose of dry cow therapy is to eliminate existing infections and prevent new infections from occurring. In dairy cows, dry cow treatment is effective in decreasing infections at the next lactation (Harmon et al., 1986; Hogan et al., 1994). In this study, dry cow treatment decreased the number of infected cows and quarters post partum. This is in agreement with Newman et al. (1991) who determined that dry cow treatment of infected quarters of beef cows decreased intramammary infection following the subsequent calving.

In dairy cows, dry cow treatment increases cure rates of existing infections (Ziv et al., 1981; Harmon et al., 1986; Schukken et al., 1993). In our experiment, treatment did not cure infections present at drying-off. This is in contrast to Newman et al. (1991) who determined that dry cow treatment of infected quarters of beef cows decreased infection by eliminating infections present at drying-off. Reasons for this difference are not apparent. Not all our cows that had organisms present had clinical mastitis. In dairy cows that had subclinical symptoms of mastitis there was no difference in cure rates between treated and control cows (Seymour et al., 1989). Spontaneous elimination of infections in control cows was similar to treated cows in this study. Spontaneous cure rates of 13 to 20% of dairy cows have been reported (Harmon et al., 1986; Philpot and Nickerson, 1991), and occur in beef cows (Simpson et al., 1995).

Dry cow treatment of dairy cows is beneficial in preventing the development of new infections during the dry period (Cummins and McCaskey, 1987; Batra, 1988; Hogan et

al., 1994). Dry cow treatment of dairy cows prevented new intramammary infections at four to 10 d post partum (Sinkevich et al., 1974b). In our experiment, dry cow treatment decreased the number of new infections that developed during the dry period. Newman et al. (1991) found that treating infected quarters of beef cows at drying-off decreased udder infections during the dry period by eliminating infections present at drying-off, but did not influence the prevention of new infections during the dry period. In dairy cows, most new infections occur right after drying-off, or immediately before lactation begins (Neave et al., 1950; Oliver and Mitchell, 1983).

The antibiotic used will influence the effectiveness. In our experiment, we used a penicillin/novobiocin product while Newman et al. (1991) used cephapirin benzathine. Seymour et al. (1989) found that Cephapirin did not to influence infection status of dairy cows during the dry period, and cephalonium, a cephapirin analog, was less effective than other antibiotics in preventing the development of new infections in dairy cows (Ziv et al., 1981). In contrast, Harmon et al. (1986) determined that cephapirin was more effective than novobiocin in preventing new infections from developing during the dry period.

In dairy cows, the percentage of cows infected in one or two quarters is greater than cows infected in three or four quarters (Erskine et al., 1994). In our study, treatment increased the number of noninfected cows post partum, as well as decreased the percentage of cows with only one quarter infected, however, treatment did not alter the percentage of cows with two or more infected quarters. As previously discussed this is through preventing the development of new infections during the dry period. The majority of infected cows were infected in only one quarter.

Although Newman et al. (1991) found that dry cow treatment of beef cows decreased udder infections, they did not determine if SCC were affected. We have previously determined that intramuscular antibiotic treatment of beef cows at drying off and again after calving decreased SCC per quarter (Duenas et al., 1994). In this study, treatment did not decrease AVSCC per cow post partum compared with controls. However, treatment decreased MXSCC per cow post partum. Coupled with the fact that most cows were infected in only one quarter, this indicates that treatment improved the health of the quarter that had the greatest SCC per cow.

There was a significant interaction between infection status of the cow at drying-off and treatment on SCC per quarter post partum. Cows that were noninfected at drying-off had similar SCC per quarter post partum whether they were treated or not. However, if cows were infected at drying-off, treated cows had less SCC per quarter post partum compared with controls. This demonstrates that treatment decreased SCC per quarter post partum if cows were infected at the time of treatment.

Somatic cell counts are increased for cows and quarters infected with mastitis causing organisms in both dairy (Dohoo and Meek, 1982; Reneau, 1986; Harmon, 1994), and beef cows (Hunter and Jeffrey, 1975; Watts et al., 1986; Simpson et al., 1995). In dairy cows, quarters infected with major pathogens such as SA had greater SCC than quarters infected with minor pathogens such as CNS (Sheldrake et al., 1983; Fox and Schultz, 1985). In this study, CNS infections post partum caused an increase in SCC for quarters that was greater than that for noninfected quarters, and cows and quarters infected with SA post partum had greater SCC than CNS or noninfected cows or quarters. Infected quarters of

beef cows have more than 500 x 10^3 somatic cells/mL (Hunter and Jeffrey, 1975; Watts et al., 1986). However, Watts et al. (1986) determined that while infected quarters had SCC greater than 794 x 10^3 cell/mL, noninfected quarters had SCC of 555 x 10^3 cells/mL. We have previously determined that SCC from noninfected quarters ranged from 58 to 153 x 10^3 cell/mL, while infected quarters had 400 to 533 x 10^3 somatic cells/mL (Duenas et al., 1994). In the current study, we found that quarters infected with CNS post partum had 347 x 10^3 somatic cells/mL, whereas quarters infected with SA post partum had 851 x 10^3 somatic cells/mL. This minimal increase in SCC of CNS infected quarters may play a role in preventing the development of infections caused by major pathogens such as SA (Linde et al., 1975). In dairy cows, it is hypothesized that an increase in SCC of CNS infected quarters may provide a greater initial response to infection with major pathogens (Rainard and Poutrel, 1988; Matthew et al., 1990; Matthews et al., 1991).

Wilson et al. (1971) observed that although mastitis causing bacteria may be present in beef cows, SCC may not be elevated to abnormal concentrations. In our study, AVSCC per cow was not different for noninfected and CNS infected cows post partum, but AVSCC for cows infected with SA post partum were greater. These observations indicate that cows infected with CNS may not show clinical symptoms.

Although mastitis has been studied in beef cows, few researchers have attempted to evaluate the effects of treatment of cows on weight gain of calves. Newman et al. (1991) found that beef cows with udder infections had calves with decreased weight gains, and that intramammary dry cow treatment decreased udder infections during the subsequent lactation. However, these researcher did not evaluate the effects of dry cow therapy of

cows on performance of calves during the subsequent lactation. Other investigators report that calves from cows treated for mastitis at drying-off weighted 12.5% more at 60 d than calves from untreated controls (Kirkbride, 1977). However, only 20 cows were used. We previously determined that intramuscular treatment of cows with intramuscular antibiotics at drying-off and again post partum decreased udder infections (Duenas et al., 1994), but did not alter calf weights (Lents et al., 1996). In this study, there was a significant interaction between treatment and post partum infection status of the cow on 110 d calf weights. Weight of calves was similar for treated and control cows if they were noninfected at drying-off. However, if cows were infected at drying-off, 110 d weights of calves were greater for treated than control cows. Adjusted 205 d calf weights of treated and control cows were similar. This could be do to the fact that not all cow defined to be infected had clinical mastitis. Therefore, milk production of cows with subclinical infections may be adequate for normal calf growth. Furthermore, as calves get older, they receive energy from sources other than milk (Neville, 1962; Haggard et al., 1983; Ansotegui et al., 1991). Therefore, the effects of decreased milk production due to udder infections may not be apparent over the entire lactation. In addition, most cows were only infected in one quarter. This may not produce a large enough decrease in milk production to adversely effect calf growth.

Treatment of beef cows at drying-off with intramammary antibiotics decreased the incidence of udder infections following the subsequent calving, by preventing the development of new infections during the dry period. Calf weights at 110 d and 205 d

were not influenced by antibiotic treatment of cows at drying-off despite the reduced

infection rate.

Implications

Treatment of beef cows at drying-off with penicillin and novobiocin is an effective

method for decreasing udder infections following the subsequent calving. Many factors

can influence weaning weights of calves, and additional lactations should be studied to

further evaluate the effects of treating cows with antibiotics at drying-off on calf growth

during the next lactation.

Table 1. Number of quarters and cows with infections that were cured or with infections that developed during the dry period

Item		Number infected			
		Drying-off	Cured	Developed	Calving
Quarters	T	15	12	13[a]	16[a]
	C	21	18	27[b]	30[b]
Cows	T	11	7	7[a]	11[a]
	C	16	8	16[b]	24[b]

[ab]Within a column, within an item, numbers with different superscripts differ (P < .05).

Table 2. Percentage of cows with various numbers of infected quarters post partum

	Quarters infected[1]		
	0	1	≥ 2
Control	51^a	31^x	18
Treated	76^b	15^y	9

[1]Number of quarters infected per cow post partum.
[ab]Within a column, means with different superscript letters differ (P < .05).
[xy]Within a column, means with different superscript letters tend to differ (P < .1).

Figure 1. (a) Average somatic cell counts (AVSCC; SEM = 40); (b) and Maximum somatic cell counts (MXSCC; SEM = 169) for control and treated cows post partum. [ab]Means with different superscript letters differ (P < .05).

Table 3. Least squares means for somatic cell counts per quarter[1] post partum. Significant interaction between infection status at drying-off and treatment (P < .06)

| | Infection status at drying-off | |
Item	Noninfected	Infected
Control	196[a]	489[a]
Treated	137[a]	246[b]
SEM	50	156

[1]SCC x 10^3 cells/mL.
[ab]Means with different superscript letters differ (P < .05).
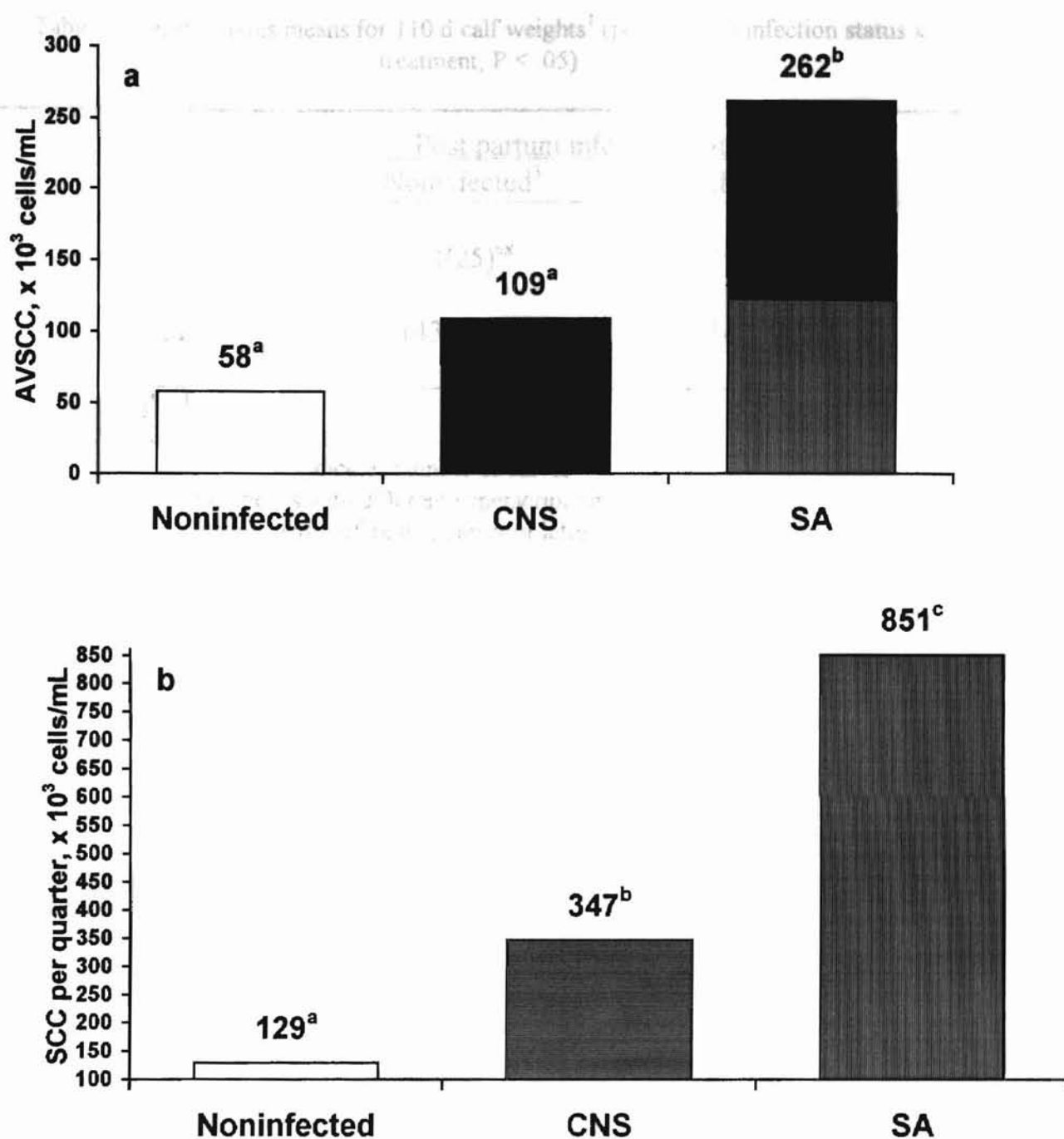
Figure 2. Least squares means for (a) average somatic cell counts (AVSCC; SEM = 56) of cows; (b) and quarters (SEM = 107) infected with coagulase negative staphylococci (CNS) or *Staphylococcus aureus* (SA) compared with noninfected cows post partum. [a,b]Means with different superscript letters differ (P < .05).

Table 4. Least squares means for 110 d calf weights[1] (post partum infection status x treatment; P < .05)

| Item[2] | Post partum infection status | |
	Noninfected[3]	Infected[3]
Control	144(25)[ax]	139(34)[ax]
Treated	143(21)[ax]	154(8)[by]

[1]Calf weight, kg
[2]MSE = 1104.685
[3]Number in parentheses indicates number of calves.
[ab]Within a column, means with different superscript letters differ (P < .05).
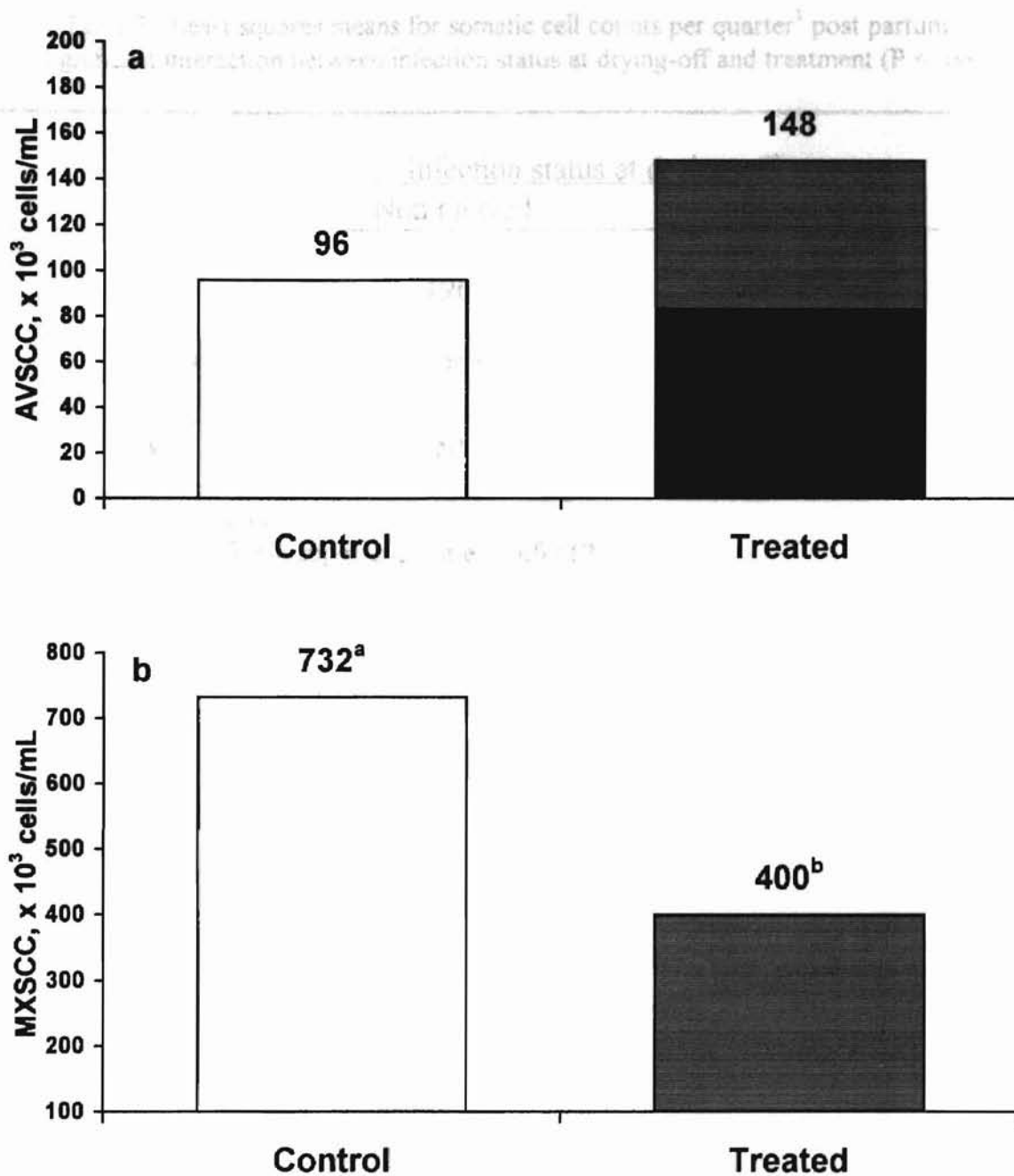[xy]Within a row, means with different superscript letters differ (P < .08).
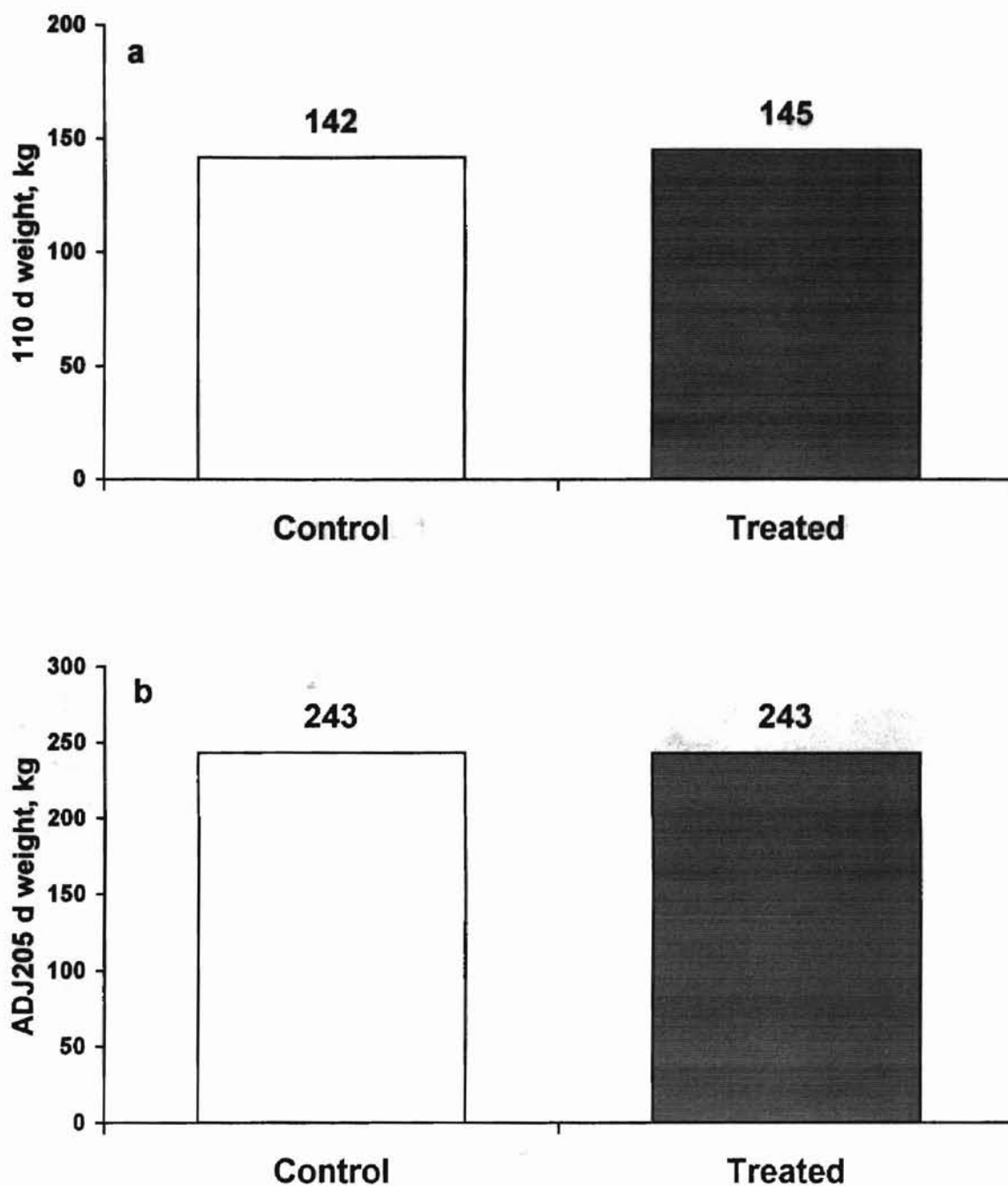
Figure 3. Least squares means for (a) 110 d weights (SEM = 3); (b) and ADJ205 d weights (ADJ205; SEM = 4) of calves from control and treated cows.
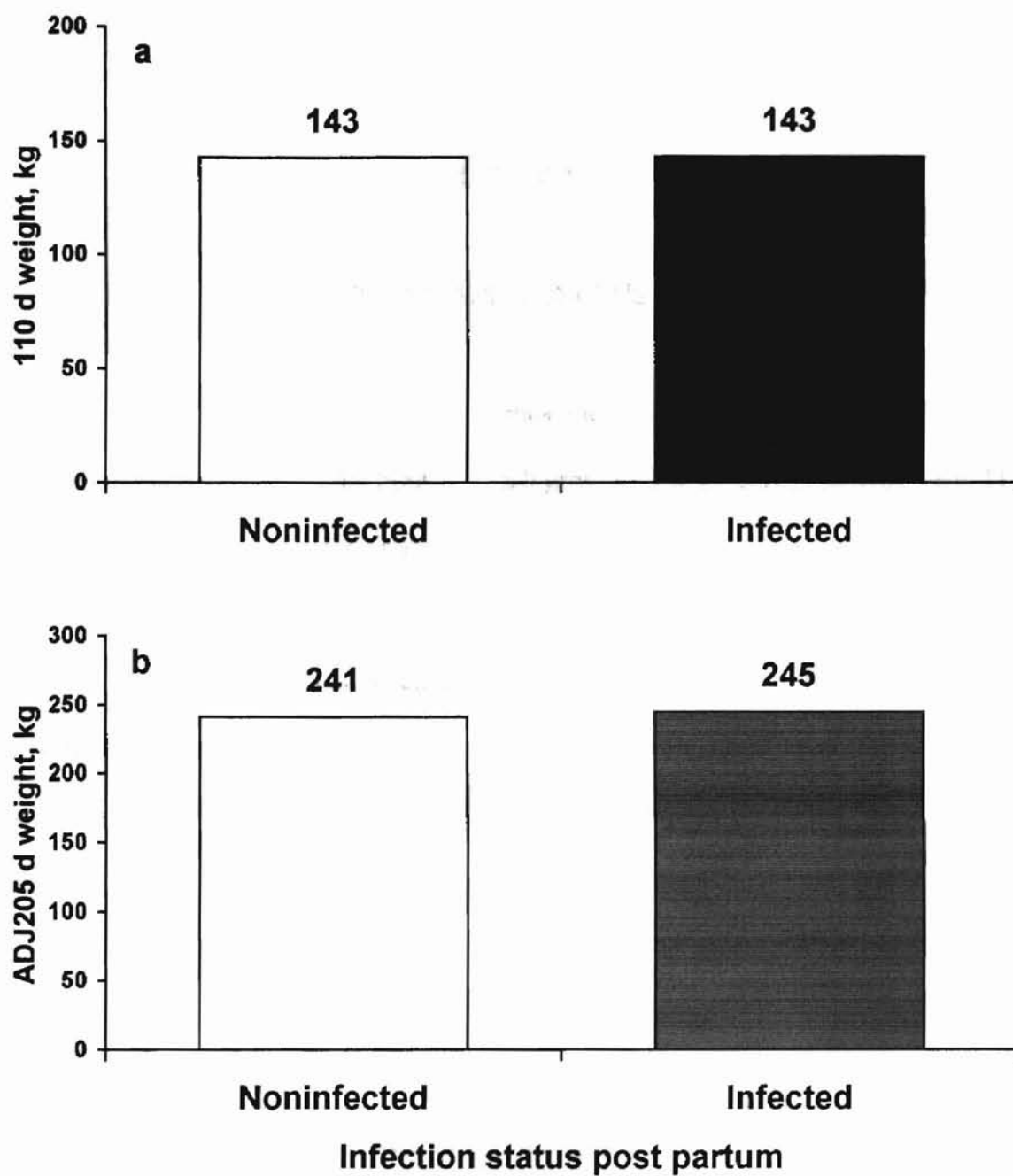
Figure 4. Least squares means for (a) 110 d weights (SEM = 2); (b) and ADJ205 d weights (SEM = 4) of calves from cows that were noninfected or infected post partum.

CHAPTER V

SUMMARY AND CONCLUSIONS

Two experiments were conducted to determine the effects of administration of
antibiotics to beef cows on udder health and calf growth. The specific objectives were: 1)
to evaluated the effects of intramuscular or intramammary antibiotics administered to beef
cows on intramammary infection and somatic cell counts (SCC) during lactation and the
dry period; 2) to determine if intramammary infections of beef cows have an adverse effect
on weight gain of calves; and 3) to evaluate the effect of treatment of cows with
antibiotics on calf growth.

In experiment 1, 319 spring calving Hereford and Hereford x Angus multiparous cows
were used, in two replications, to determine the efficacy of intramuscular treatment with
oxytetracycline to reduce the incidence of mastitis causing bacteria, decrease milk SCC,
and increase calf growth. At 8 to 14 d post partum, a California Mastitis Test (CMT) was
performed on milk from each quarter of each cow. Cows with a CMT score of 1, 2, or 3
in at least one quarter, were randomly assigned to receive either an intramuscular injection
of oxytetracycline (n = 63) or control (n = 60), and cows with a CMT score of 0 or trace
in all four quarters were not treated (n = 196). Milk samples were collected at 8 to 14 d
post partum and at weaning to determine the presence of bacteria and SCC. Calf weights
were determined at birth, 60 d of lactation, and weaning.

Nine hundred-thirty one, 80, 82, 55, and 47 quarters had CMT score of 0, trace, 1, 2, and 3, respectively. Means SCC increased from 66×10^3 cells/mL to 3035×10^3 cells/mL as CMT score increased from 0 to 3. The CMT score increased as SCC increased indicating the usefulness of this tool to predict SCC in milk.

The percentage of quarters infected increased as CMT score increased. When quarters had CMT scores of 0, trace, and 1, 17% were infected. However, when quarters had CMT scores of 2 and 3, 42% of quarters were infected. This indicates the usefulness of CMT scores of 2 and 3 to identify infected quarters. Since even with CMT scores of 2 and 3, a limited percentage of quarters were correctly identified as infected, the CMT is a better indicator of SCC than infection.

Treatment with oxytetracycline at calving did not alter the prevalence of infection or decrease SCC for cows or quarters at weaning. A single injection of oxytetracycline after calving may be to short a duration of treatment to decrease bacterial populations, or infections may have been eliminated and reinfection may have occurred. Furthermore, we did not quantify bacterial populations. Thus treatment may reduce populations of bacteria and improve udder health, but not completely eradicate the organisms.

The presence of mastitis causing bacteria post partum was associated with increased percentages of infected cows and quarters at weaning. Cows or quarters that were infected at weaning had greater SCC at weaning compared with noninfected cows or quarters.

Treatment of cows did not alter 60 d or adjusted 205 d (ADJ205) weights of calves. This agrees with the observation that treatment did not alter infection status of cows.

Somatic cell counts were negatively correlated with 60 d calf weights. Postpartum

infection status of the dam did not influence 60 d or ADJ205 weights of calves. Due to

the high variability in a trait such as weaning weight, more observations are needed to

evaluate the effect of mastitis on calf growth.

In experiment 2, ninety Hereford and Hereford x Angus multiparous cows were used to

determine the effects of intramammary treatment of beef cows with Penicillin G procaine

and novobiocin at the time of drying-off on udder health and calf growth following the

subsequent calving. Cows were blocked by age and randomly assigned to intramammary

treatment (n = 45) or untreated controls (n = 45). Quarter milk samples were collected at

drying-off and at 8 to 14 d post partum. Milk samples from each quarter were analyzed

for SCC and mastitis causing bacteria. Calf weights were determined at birth and every 30

d from May 5 until weaning in October.

Treatment at drying-off decreased the number of cows and quarters infected post

partum. This was due to the prevention of new infections from developing during the dry

period rather than the elimination of infections present at drying-off. This indicates that as

in dairy cows, intramammary antibiotic treatment of beef cows at drying-off improves

udder health following the subsequent calving.

More treated cows were noninfected post partum compared with controls. Treatment

also decreased the percentage of cows with only one quarter infected post partum, but did

not influence the percentage of cows with two or more infected quarters. This indicates

that most cows were infected in only one quarter, and that treatment was more effective in

those cows with only one quarter infected as compared to cows with two or more quarters infected.

Treatment did not decrease average SCC per cow, but decreased the maximum SCC per cow. This indicates that treatment was beneficial and improved the health of the quarter with the greatest SCC, but did not decrease overall SCC of the cow.

There was an interaction between infection status at drying-off and treatment on SCC per quarter post partum. If quarters were noninfected at drying-off, SCC were similar for treated and control quarters. However, it quarters were infected at drying-off, treated quarters had less SCC than controls. Treatment of infected quarters may have decreased bacterial populations, thereby decreasing of SCC.

Type of bacteria present after calving altered SCC for cows and quarters. Noninfected cows and quarters had the least SCC, CNS infected cows and quarters had intermediate SCC, and SA infected cows and quarters had the greatest SCC. Thus, major pathogens elicit a greater infectious response than minor pathogens. Furthermore, cows infected with CNS may not exhibit clinical symptoms of mastitis.

Treatment of cows with intramammary antibiotics at drying-off did not alter 110 d or 205 d weights of calves during the subsequent lactation. Furthermore, postpartum infection status of cows did not alter weight gain of calves. Even though treatment decreased infection post partum this may not have been great enough to increase calf weight gains. In addition, cows could become reinfected during lactation and negate any benefit that may have occurred due to dry cow treatment.

We conclude that administration of intramuscular oxytetracycline after calving did not improve udder health at weaning. Intramammary infusion of penicillin and novobiocin at drying-off decreased the incidence of mastitis-causing organisms at the subsequent calving. The presence of mastitis-causing bacteria increases SCC, and SA elicits a greater SCC response than CNS. Under the conditions in our research herd, postpartum infection status and treatment did not alter weights of calves at mid lactation, or weaning. Growth of calves is influenced by many factors, additional animals must be studied to determine if intramammary infusion of antibiotics at drying-off influences weight gain of calves during the subsequent lactation.

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