Mastitis Management for the Future Milking Herd

S. C. Nickerson Ph.D., Professor of Dairy Science
Animal and Dairy Science Department
University of Georgia, Athens, GA
Email: scn@uga.edu

INTRODUCTION

Replacement heifers, whether they are raised on the farm, purchased from other dairies, or raised by contract growers, are critical to herd productivity because they represent the future milking and breeding stock in all dairy operations. The goal should be to provide an environment for heifers to develop full lactation potential at the desired age with minimal expense. Animal health and well-being play vital roles in achieving this potential, and the most important disease that can influence future productivity is mastitis. Unfortunately, most producers regard young dairy heifers as uninfected, and the presence of mastitis is not observed until freshening or until the first clinical flare-up in early lactation. Thus, animals may carry intramammary infections for a year or more before they are diagnosed with mastitis.

The greatest development of milk-producing tissue in the udder occurs during the first pregnancy, so it is important to protect the heifer mammary gland from pathogenic microorganisms to ensure maximum milk production during the first and future lactations. Louisiana researchers found that if bred heifers infected with *Staphylococcus aureus* were left untreated, they produced 10 % less milk in early lactation than those receiving therapy (Owens et al., 1991; Trinidad et al., 1990c). Likewise, research in New Zealand has shown that *Staph. aureus* mastitis in heifers results in significant production losses during the first lactation, which carries over into the subsequent lactation, even if infected quarters are successfully treated, because of damage to milk-producing tissues of the udder (Meaney, 1981). Thus, it is critical that the mammary glands of heifers develop and grow normally, and remain free of infection and the associated inflammation.

MAMMARY GLAND DEVELOPMENT AND GROWTH

The mammary gland of the cow begins to develop *in utero* as an embryo at a crown-rump length of only 1.4 cm, the diameter of a fingertip. At this stage, the immature gland is nothing more than a thickening of the ectoderm (skin) between the fore and rear limb buds of the embryo; this thickening is classified as the *band* stage of mammary development. In the embryonic stage, the gland goes through 6 distinct stages of development, terminating at the *bud* stage, at which time the teats have begun to form and the embryo has increased in size to 2.5 cm in length at 6 wk of age. The embryo matures into a fetus when its length is 8 cm, and it again progresses through 6 stages of mammary development. During this time, the teat and gland cisterns of each mammary quarter form, followed by the development of future milk-producing tissues, and terminating with the formation of the median suspensory ligament, when the fetus is approximately 48 cm in length. At this stage, the bulk of the mammary gland is fatty tissue, known as the fat pad.

During the first 4-6 mo after birth of the fetus, the mammary gland is stimulated to grow isometrically, or at the same rate as the

rest of the body; the major increase in total udder size is due to growth of the fat pad. With the onset of puberty and establishment of 21-d estrous cycles, estrogen and small levels of progesterone are released from the ovary. This hormonal profile stimulates mammary duct growth from the gland cistern into the fat pad, and with each successive estrous cycle, there is a stepwise increase in ductal growth, dilation, and elongation into the fat pad, but limited alveolar development. After the first 3-4 estrous cycles, there is a plateau in mammary gland growth until the time of conception.

After conception, the ductal system continues to grow into the fat pad under the influence of estrogen, while progesterone, produced by the corpus luteum, induces the proliferation of lobular-alveolar tissues; which will mature into milk-producing cells at the end of gestation. By the fifth month of the first pregnancy, lobules of alveoli have replaced much of the fat pad, and after this time, development accelerates rapidly, which coincides with the period of rapid fetal growth. Thus, by the end of gestation, the udder is fully developed and ready to provide nourishment to the new born calf, or to provide copious volumes of milk for human consumption.

Because future milk production is dependent on udder growth during the first pregnancy of the dairy heifer, it is vital that mammary tissue develop in an optimum fashion, and the major deterrent to normal milk-producing potential is the development of mastitis. Heifers are exposed to mastitis-causing bacteria as calves, during puberty, and during gestation; thus, a plan should be in place to cure intramammary infections once they are established, but more importantly, to prevent udder disease in the first place.

PREVALENCE OF HEIFER MASTITIS AND SOMATIC CELL COUNTS

Researchers became interested in heifer mastitis in the mid-1980s after several dairy producers complained that a large percentage of their heifers were freshening with clinical mastitis. Subsequent study of breeding age and younger animals revealed that intramammary infections could be diagnosed as early as 6 mo of age, and that infections persisted throughout pregnancy and into lactation (Boddie et al., 1987). Other studies demonstrated that greater than 90 % of breeding age and bred heifers (12 to 24 mo of age) may be infected (Trinidad et al., 1990b). Most of the infections were shown to be caused by the coagulase-negative staphylococci (**CNS**) followed by *Staph. aureus* (20 %). Mixed isolates of CNS and *Streptococcus* species were also found.

Somatic cell counts (**SCC**) are used to assess udder health status of mature, lactating cows, and this parameter has been examined in heifer mammary secretions. In secretions from uninfected quarters, SCC are approximately 5×10^6/ml. The volume of mammary secretion is very low in breeding-age animals; thus, somatic cells become concentrated, resulting in high SCC. However, SCC may be 20×10^6/ml in quarters infected with *Staph. aureus* and over 10×10^6/ml in those infected with the CNS and *Streptococcus* species. Such elevated SCC in infected quarters over a long period of time demonstrate that these mammary glands are in a state of chronic inflammation, which would adversely affect development of milk-producing tissues. In fact, histological analysis of mammary tissues obtained from bred heifers chronically infected with *Staph. aureus*

demonstrated that the potential for milk production was significantly reduced compared with tissues from uninfected quarters (Trinidad et al., 1990a).

EFFICACY OF NONLACTATING DRY COW ANTIBIOTIC TREATMENT

Because of the high level of infection commonly found in heifers at some dairies, especially mastitis caused by *Staph. aureus*, infected quarters should be treated with local intramammary infusion. Prior to treatment, heifers should be restrained in a squeeze chute equipped with a head gate or restrained on a hoof trimming table. Teat ends should be sanitized with cotton balls soaked in 70 % alcohol or with the pledgets accompanying mastitis tubes. While administering the antibiotic, the partial insertion technique must be used to avoid stretching the teat canal and the sphincter muscle as well as to avoid the introduction of bacterial contaminants. All quarters of each animal should be treated to cure existing *Staph. aureus* infections and to prevent new ones. After infusion, teats may be infused with a teat sealant product and should be immersed in a germicidal barrier teat dip to kill any contaminating bacteria at the teat opening and seal the teat orifice from the environment.

The cure rate for *Staph. aureus* mastitis after use of nonlactating cow therapy in heifers is usually greater than 90 %. Therapies evaluated have included:

1) A product containing 1 million units of penicillin and 1 gram of streptomycin;
2) A product containing 300 mg cephapirin benzathine; and
3) A product containing 400 mg novobiocin with 200,000 international units (IU) of penicillin (Owens et al., 2001).

This cure rate is far greater than the 25 % rate observed after mature cows are treated for this disease during lactation using lactating cow therapy. The relatively small secretory tissue area of heifer mammary glands compared with mature cows might allow for greater drug concentrations in the udder of the heifer. Similarly, histological studies have demonstrated less scar tissue and abscess formation in the mammary glands of heifers compared with older cows, a condition which would allow for better drug distribution and greater contact with colonized bacteria.

In one study, an economic analysis was performed to justify use of a heifer treatment program (Trinidad et al., 1990c). Milk yield data collected over the first 2 mo of lactation demonstrated that *Staph. aureus*-infected heifers receiving nonlactating cow therapy during pregnancy produced an average of 5.5 lb more milk per day than infected herd mates that did not receive treatment. At the milk price received at that time, the greater milk yield translated to a $42 increase for treated heifers, which was well worth the $5.00 cost of treatment.

In another trial, treatment of heifers 8 to 12 wk prior to expected calving date with 300 mg of cephapirin benzathine resulted in a cure rate of greater than 95 % (Owens et al., 1991). An examination of SCC showed that at the time of treatment, average SCC were 15 x 10^6/ml but decreased to 4 x 10^6/ml 1 wk later, and to 700,000/ml on the day of calving. If infected quarters were left untreated, heifers freshened with *Staph. aureus*-infected quarters having an average SCC of 5 x 10^6/ml. When these later animals were treated with lactating cow products immediately after calving, cure rate was only 56 %. Thus, cure rates are much

greater when nonlactating cow products are administered 2 to 3 mo prepartum than when a lactating cow product is given shortly after calving.

EFFICACY OF LACTATING COW PRODUCTS

Lactating cow products also have been used successfully in heifers when treating infections caused by the CNS immediately prior to calving. In one study (Oliver et al., 1992), quarters of infected heifers were infused one time at approximately 1 wk prepartum with either 200 mg sodium cloxacillin, 200 mg cephapirin sodium, or left untreated. At the time of infusion, approximately 90 % of heifers were infected in one or more quarters, and if left untreated, 78 % of animals remained infected at time of calving. However, only 18 % of the heifers remained infected at calving if they were treated prepartum, regardless of the treatment used. This study also examined the influence of prepartum antibiotic treatment on subsequent lactational performance and demonstrated that heifers receiving treatment produced approximately 1,000 lb more milk per lactation than untreated controls.

Prepartum treatment with lactating cow therapy has been shown to be effective for quarters infected with CNS (Oliver et al., 2004), but waiting until this time to treat chronic *Staph. aureus* mastitis might be too late. A mammary gland that has been infected with *Staph. aureus* for several months to a year will not develop normally, and treatment during the immediate prepartum period would most likely be of little benefit in curing infections or salvaging mammary tissue. At this point, the tissue damage would have already been done, and affected quarters should have been treated earlier in gestation to:

1) Cure existing infections;
2) Reduce chronic inflammation; and
3) Allow mammary tissue to develop normally during the later stages of pregnancy.

THE OPTIMUM TREATMENT SCHEDULE

The question arises as to when is the best time to treat bred heifers for optimizing cures against *Staph. aureus* mastitis. A 2-yr study involving 175 Jersey heifers was designed to answer this question (Owens et al., 1994). In the trial, heifers were sampled shortly after they were confirmed pregnant and at 4-wk intervals thereafter. After the initial sampling, animals were treated with a one-time infusion of one of three nonlactating cow infusion products during the first (0 to 90 d), second (91 to 180 d), or third (181 to 270 d) trimester of pregnancy. Products evaluated were:

1) A combination of 1 million units of penicillin and 1 gram streptomycin;
2) 300 mg cephapirin benzathine; and
3) A combination of 400 mg novobiocin and 200,000 units of penicillin G.

Cure rates among treatments indicated that all antibiotics were equally effective in curing infections, and there were no apparent effects of the timing of therapy on cure rate. Treatment efficacy ranged from 83.3 to 100 %.

Because therapy during the first, second, or third trimester of gestation had no effect on treatment efficacy, the timing of treatment is best determined by what is most convenient for the management practices of a particular dairy. For example, heifers could be treated:

1) At time of artificial insemination;
2) During routine rectal palpation to determine pregnancy status; or

3) When moved to a close-up pen.

Treatment should be administered no less than 45 d prior to expected calving date to prevent antibiotic residues at calving.

The treatment of heifers during pregnancy with a nonlactating cow product is advantageous because:

1) The cure rate is higher than during lactation, especially against *Staph. aureus*;
2) There are no milk losses during therapy;
3) The risk of antibiotic residues is minimal;
4) New infections caused by the environmental streptococci are prevented;
5) SCC at calving is reduced; and
6) Milk production is increased by approximately 10 % in successfully treated animals.

The potential for residues at calving should be considered, especially if heifers calve early, and testing should be done before mixing milk from treated animals with herd milk.

ROLE OF VACCINATION IN MASTITIS CONTROL

Research has demonstrated that several experimental *Staph. aureus* vaccines, as well as one commercial vaccine, can increase antistaphylococcal antibody titers and reduce the new infection rate in heifers. Reductions in *Staph. aureus* infection rates for experimental vaccines have ranged from approximately 45 to 65 % (Giraudo et al., 1997; Nordhaug et al., 1994; Sears et al., 1990). In view of these successes, researchers in Louisiana evaluated a commercially available *Staph. aureus* vaccine in young dairy animals (Nickerson et al., 1999). The vaccine was a lysed culture of polyvalent *Staph. aureus* somatic antigens representing 5 phage types in an aluminum hydroxide adjuvant base, including serotypes 5, 8, and 336; the most common *Staph. aureus* serotypes associated with clinical mastitis (Lysigin®, Boehringer Ingelheim Vetmedica, Inc., St. Joseph, MO). At 6 mo of age, 35 Jersey heifers were vaccinated using a 5-ml dose intramuscularly in the semimembranosus muscle of the rear leg, and 14 d later, vaccinates received a booster dose; which was repeated at 6-mo intervals. Another 35 heifers served as unvaccinated controls. Results demonstrated that:

1) The number of quarters exhibiting chronic intramammary infection during pregnancy was reduced 43.1 % in vaccinates compared with controls;
2) The rate of new intramammary infection during pregnancy was reduced 44.8 %;
3) The rate of new intramammary infection at freshening was reduced 44.7 %; and
4) The SCC was reduced by 50 % in vaccinates compared with controls.

In a subsequent study using the same vaccine (Lysigin), 106 Holstein heifers from the James River Correctional Center dairy herd in Goochland, VA were evaluated (Nickerson et al., 2009). This herd had a 22,000-lb rolling herd average for milk production with an average SCC of 200,000/ml. Previous microbiological culture of heifer mammary secretions indicated that approximately 35 % of animals were infected with *Staph. aureus*. At approximately 6 to 18 mo of age, heifers were processed through a restraining chute to collect aseptic quarter mammary secretion samples for microbiological analysis. Fifty-three heifers were vaccinated using a dose of 5 ml intramuscularly that was administered as above, and the other 53 heifers served as

unvaccinated controls. Fourteen days after the initial processing, the vaccinated group was boosted with Lysigin. All animals were maintained on pasture and rotated by age group through calving. At 6-mo intervals after the initiation of the trial and through time of calving, the vaccinated group received booster injections per label instructions.

At 2-mo intervals after the trial initiation and through calving, mammary secretion samples were collected for bacteriological culture and for the determination of SCC. Microbiological examination of quarter samples collected from bred heifers over gestation demonstrated that 19.8 % of heifers were infected with *Staph. aureus*, 68.9 % of heifers were infected with CNS, 6.6 % of heifers were infected with environmental streptococci, and 1 % of heifers were infected with coliforms.

At time of calving, heifers were enrolled in the Dairy Herd Improvement Program (**DHIA**) and data were recorded for milk yield, percentages and actual pounds of fat and protein, days in milk (**DIM**), and SCC. Data on vaccine efficacy were examined in terms of mean percentage reduction in rate of new *Staph. aureus* or CNS intramammary infections achieved among immunized heifers compared with the rate among unimmunized controls at the time of calving.

Vaccine efficacy data showed that the percentage of heifers with *Staph. aureus* mastitis at freshening was lower in vaccinates (13.3 %) compared with controls (34.0 %); a reduction of 60.9 % ($P \leq 0.01$). Likewise, an examination of health records showed that the percentage of heifers that were culled or died during the trial was reduced by approximately one-third by vaccination: 16.9 % in vaccinates and 24.5 % in controls ($P > 0.05$). The vaccinated group also experienced a slight, insignificant reduction in mastitis caused by CNS; e.g., at freshening, intramammary infections with CNS were lower in vaccinates (64.2 %) compared with controls (69.8 %); a reduction of 8.1 %.

Somatic cell counts in samples collected during the prepartum period were highest for animals with *Staph. aureus* ($6,730 \times 10^3$), followed by the environmental streptococci ($3,850 \times 10^3$), and CNS ($3,510 \times 10^3$). Somatic cell counts in samples collected during the first week of lactation, irrespective of infection status, were 45 % lower in vaccinates compared with controls (287,317 vs. 522,345/ml). Somatic cell counts collected during the first week of lactation from uninfected heifers for vaccinates and controls were 66,095 and 132,754/ml; a 50.2 % reduction; and for infected heifers, SCC were 441,764 and 892,176/ml; a 50.5 % reduction.

An examination of 305-d lactation milk yields for the first lactation of both vaccinated and unvaccinated control heifers demonstrated an approximate 8 % increase in vaccinates vs. controls (24,729 vs. 22,778 lb) or a difference of 1,951 lb. Likewise, the percentage of 305-d lactation fat was higher in vaccinates than controls (3.64 vs. 3.27 %); however, the percentage of 305-d lactation protein was slightly higher in controls than vaccinates (3.06 vs. 2.95). Actual 305-d pounds of both fat and protein were higher in vaccinates than controls (fat: 899 vs. 747 lb; protein: 728 vs. 694 lb). An examination of the number of DIM for the first lactation demonstrated that vaccinates persisted 13 d longer than unvaccinated controls (349 vs. 336 d). In addition, average first lactation SCC were 11,000 cells/ml lower in vaccinates

compared with controls (49,000 vs. 60,000/ml).

Results of this Virginia investigation demonstrated that vaccinating dairy heifers reduced the number of new *Staph. aureus* intramammary infections at time of calving by over 60 %, lowered the SCC by 50 %, and decreased the culling rate by approximately one-third. In addition, overall milk yield, production of fat and protein, and number of DIM were greater in vaccinated heifers compared with controls.

The question becomes: Is it economically feasible to use this commercial vaccination protocol on young dairy heifers? Based on an average of 1,951 lb milk more per vaccinated heifer, which translates to 19.51 hundredweights (**cwt**) of milk (1,951/100), at the current price of $25.00/cwt, an increased income of $488.00/heifer would be realized (19.51 cwt x $25.00/cwt = $488.00). If each heifer was vaccinated beginning at 6 mo of age until calving, this would entail vaccinations at 1) 6 mo, 2) a booster 2-wk later, and subsequently at 3) 12 mo, 4)18 mo, and 5) 24 mo, or a total of 5 immunizations through calving. At $1.50/dose, this cost would total $7.50, which when subtracted from the increased income from milk production, would yield a net income of $480.50 per heifer ($488.00 - $7.50). This figure does not include the costs of labor involved in the immunization process nor any increased feed cost; however, it is evident that vaccination is well worth the cost of the vaccine. Not only does it reduce new infections in first calf heifers at parturition and lower SCC, it reduces the introduction of *Staph. aureus* to the milking herd.

FLY CONTROL

In the US, fly control is used to reduce these insect pests on farm premises, and subsequently reduce animal stress; but its application as an adjunct management practice for preventing new cases of mastitis and reducing SCC has not really been considered or embraced by producers. Surprisingly, very little research has been conducted on the relationship between mastitis in adult cows and fly control; most studies in this regard have been carried out in dairy heifers. An initial survey showed that prevalence of mastitis in bred heifers was lower in dairy herds that used some form of fly control for their lactating cows, dry cows, and heifers compared with herds applying no fly control. For example, in herds without fly control, 100 % of heifers were infected with some type of mastitis pathogen compared with 44.4 % for those with some form of fly control. Likewise, for the CNS, 41.4 % of heifers were infected in herds without fly control vs. 32.9 % for herds with fly control. For *Staph. aureus*, the figures were 55.2 % without and 5.6 % with fly control; for the environmental streptococci: 20.7 vs. 3.7 %; and for *Arcanobacterium pyogenes*: 3.4 vs. 0 %. The greatest reductions in infections in herds using fly control were in numbers of *Staph. aureus* and the environmental streptococci, both major mastitis pathogens in adult cows associated with elevations in SCC.

Results of this survey also demonstrated that bred heifers having teats with bite lesions and scabs caused by the blood-sucking horn fly (*Haematobia irritans*), exhibited a 70 % frequency of intramammary infection compared with a 40 % frequency in heifers with normal, healthy teats. Such infections are always associated with SCC in excess of 5×10^6/ml.

Since that first survey, researchers have proven through DNA studies that the horn fly is not only responsible for teat lesions on heifers, but is indeed a vector in the transmission of mastitis-causing bacteria, such as *Staph. aureus*, from heifer to heifer (Gillespie et al., 1999). Such mastitic heifers serve as sources of intramammary infections for transmission to the entire lactating and dry herd.

Once it was established that the horn fly was a vector in the transmission of mastitis-causing bacteria, the next step was to develop management practices to reduce flies and lower prevalence of infection. Insecticide-impregnated tags placed on the tail switch in close proximity to the udder during the spring and summer months were successful in reducing horn fly populations in beef heifers as well as the incidence of mastitis during the first 2 mo after placement (Nickerson et al., 2000). However, after 2 mo, tags fell off and replacing them was impractical from a management standpoint.

In a subsequent trial, the daily dietary supplementation of an insect growth regulator (methoprene) helped to suppress fly populations, but not sufficiently to prevent new cases of mastitis in dairy heifers (Owens et al., 2000). Lastly, the use of an insecticidal pour-on (Eprinex®) every 2 wk for 6 wk followed by treatment with insecticidal ear tags in both ears (Patriot®) markedly reduced fly populations and decreased the incidence of new *Staph. aureus* by 83 % during a 6-mo trial in heifers during the warm season in Louisiana (Owens et al., 2011). During the trial period, monthly sampling of mammary glands showed 21 new *Staph. aureus* IMI in control heifers (18 % of quarters) and only 2 new *Staph. aureus* IMI in treated heifers (3 % of quarters). The use of the pour-on for 6 wk followed by ear tag placement maintained fly numbers to less than 25 per animal; whereas in controls, fly populations ranged between 100 and 300 per animal.

These studies demonstrate that during the warm and humid months of the year, horn flies do serve as vectors in the transmission of *Staph. aureus* mastitis, which is associated with elevated SCC in these young dairy animals. A simple fly control program can serve as an important adjunct to a plan of mastitis control and assist dairy producers in lowering their bulk tank SCC and earn quality premiums for their product.

TEAT SEALANTS

Teat sealants are viscous, inert, paste-like products containing 65 % bismuth subnitrate in a paraffin base that are infused via the teat orifice into the teat canal. The sealant serves as a physical barrier to bacterial penetration and is designed to prevent new infections during the dry period in adult cows or during the prepartum period in heifers.

In one study (Parker et al., 2007), the infusion of a teat sealant approximately 4 wk prior to calving reduced the risk of clinical mastitis by 68 % and reduced the risk of *Strep. uberis* by 84 %. In a subsequent trial (Parker et al., 2008), the infusion of a teat sealant approximately 4 wk prior to calving reduced the risk of infection with any organism by 74 %; reduced the prevalence of post-calving intramammary infection by 65 %; reduced the risk of *Strep. uberis* infection in quarters with an intramammary infection pre-calving by 70 %; and reduced the incidence of bacterial clinical mastitis by 70 % in quarters diagnosed with mastitis prior to calving. Thus, use of teat sealants is an excellent management tool to prevent new

infections during the peripartum period in heifers. Teat sealants should be administered approximately 30 d prior to calving, and strict hygiene followed when administering these products.

CONCLUSIONS

Whether heifers are raised on the dairy or in grower operations, managers of these young dairy animals should be vigilant of udder health. Visual and manual examination of the developing udders, mammary fluid, and teat skin will help identify swollen quarters, abnormal secretions, and presence of teat scabs. Swollen quarters with abnormal secretions (clots and flakes) and those with teats exhibiting scabs and abrasions are likely infected and should be treated. Managers should be cautioned, however, that treatment of bred heifers may constitute extra-label drug use and should be carried out under the supervision of the herd veterinarian. Prevention strategies are best applied through vaccination, fly control programs, and use of teat sealants. With the proposed reduction in the SCC legal limit to 400,000/ml, and in light of the fact that milk buyers are imposing their own limits, it is imperative that dairy producers utilize all possible means to prevent new cases of mastitis and their associated SCC.

LITERATURE CITED

Boddie, R.L., S.C. Nickerson, W.E. Owens, and J.L. Watts. 1987. Udder microflora in nonlactating heifers. Agri-Practice 8:22-25.

Gillespie, B.E., W.E. Owens, S. C. Nickerson, and S.P. Oliver. 1999. Deoxyribonucleic acid fingerprinting of *Staphylococcus aureus* from heifer mammary secretions and from horn flies. J. Dairy Sci. 82:1581-1585.

Giraudo, J.A., A. Calzolari, H. Rampone, A. Rampone, T. Giraudo, C. Bogni, A. Larriestra, and R. Nagel. 1997. Field trials of a vaccine against bovine mastitis. 1. Evaluation in heifers. J. Dairy Sci. 80:845-853.

Meaney, W.J. 1981. Mastitis levels in spring-calving dairy heifers. Ir. Vet. J. 35:205.

Nickerson, S.C., W.E. Owens, G.M. Tomita, and P.W. Widel. 1999. Vaccinating dairy heifers with a *Staphylococcus aureus* bacterin reduces mastitis at calving. Large Anim. Pract. 20:16-20.

Nickerson S.C., W.E. Owens, and S.M. DeRouen. 2000. Mastitis prevalence in first calf beef heifers and effect on calf weaning weight. Large Anim. Pract. 21:20-23.

Nickerson, S.C., L.O. Ely, E.P. Hovingh, and P.W. Widel. 2009. Immunizing dairy heifers can reduce prevalence of *Staphylococcus aureus* and reduce herd somatic cell counts. *In:* Dairy Cattle Mastitis and Milking Management. DAIReXNET. http://www.extension.org/pages/Dairy_Cattle_Mastitis_and_Milking_Management

Nordhaug, M.L., L.L. Neese, N.L. Norcross, and R. Gudding. 1994. A field trial with an experimental vaccine against *Staphylococcus aureus* mastitis in cattle. 1. Clinical parameters. J. Dairy Sci. 77:1267-1275.

Oliver, S.P., M.J. Lewis, B.E. Gillespie, and H.H. Dowlen. 1992. Influence of prepartum antibiotic therapy on intramammary infections in primigravid heifers during early lactation. J. Dairy Sci. 75:406-414.

Oliver, S.P., S.J. Ivey, B.E. Gillespie, M.J. Lewis, D.L. Johnson, K.C. Lamar, H. Moorehead, H.H. Dowlen, S.T. Chester, and J.W. Hallberg. 2004. Influence of prepartum intramammary infusion of pirlimycin hydrochloride or penicillin-novobiocin on mastitis in heifers during early lactation. J. Dairy Sci. 87:1727-1731.

Owens, W.E., S.C. Nickerson, R.L. Boddie, G.M. Tomita, and C.H. Ray. 2001. Prevalence of mastitis in dairy heifers and effectiveness of antibiotic therapy. J. Dairy Sci. 84:814-817.

Owens, W.E., S.C. Nickerson, P.J. Washburn, and C.H. Ray. 1991. Efficacy of a cephapirin dry cow product for treatment of experimentally induced Staph. aureus mastitis in heifers. J. Dairy Sci. 74:3376-3382.

Owens, W.E., S.C. Nickerson, P.J. Washburn, and C.H. Ray. 1994. Prepartum antibiotic therapy with a cephapirin dry cow product against naturally occurring intramammary infections in heifers. Vet. Med. B 41:90-100.

Owens, W.E., S.C. Nickerson, and R.L. Boddie. 2000. The effect of methoprene on horn fly numbers and mastitis in dairy heifers. Large Anim. Pract. 21:26-28.

Owens, W.E., C.H. Ray, and S.C. Nickerson. 2011. Effect of a pour-on and fly tag insecticide combination in controlling horn flies and *Staphylococcus aureus* mastitis in dairy heifers. *In:* Dairy Cattle Mastitis and Milking Management. DAIReXNET. http://www.extension.org/pages/Dairy_Cattle_Mastitis_and_Milking_Management

Parker, K.I., C. Compton, F.M. Anniss, A. Weir, C. Heuer, and S. McDougall. 2007. Subclinical and clinical mastitis in heifers following the use of a teat sealant precalving. J. Dairy Sci. 90:207-218.

Parker, K.I., C.W.R. Compton, F.M. Anniss, C. Heuer, and S. McDougall. 2008. Quarter level analysis of subclinical and clinical mastitis in primiparous heifers following the use of a teat sealant or an injectable antibiotic, or both, precalving. J. Dairy Sci. 91:169-181.

Sears, P.M., N.L. Norcross, K. Kenny, B. Smith, R.N. Gonzalez, and M.N. Romano. 1990. Resistance to *Staphylococcus aureus* infections in staphylococcal vaccinated heifers. *In:* Proc. Intl Symp. Bovine Mastitis. March 23-27, 1990. Indianapolis, IN, USA, pp. 69-74.

Trinidad, P., S.C. Nickerson, and R.W. Adkinson. 1990a. Histopathology of staphylococcal mastitis in unbred dairy heifers. J. Dairy Sci. 73:639-647.

Trinidad, P., S.C. Nickerson, and T.K. Alley. 1990b. Prevalence of intramammary infections and teat-canal colonizations in unbred and primigravid heifers. J. Dairy Sci. 73:107-114.

Trinidad, P., S.C. Nickerson, T.K. Alley, and R.W. Adkinson. 1990c. Efficacy of intramammary treatment in unbred and primigravid dairy heifers. J. Am. Vet. Med. Assoc. 197:465-470.