

Kansas Agricultural Experiment Station Research Reports

Volume 0
Issue 2 *Dairy Research (1984-2014)*

Article 356

1997

Biosecurity in the dairy

Gerald L. Stokka

Thomas R. Falkner

Patrick Bierman

Follow this and additional works at: <https://newprairiepress.org/kaesrr>



Part of the [Dairy Science Commons](#)

Recommended Citation

Stokka, Gerald L.; Falkner, Thomas R.; and Bierman, Patrick (1997) "Biosecurity in the dairy," *Kansas Agricultural Experiment Station Research Reports*: Vol. 0: Iss. 2. <https://doi.org/10.4148/2378-5977.3281>

This report is brought to you for free and open access by New Prairie Press. It has been accepted for inclusion in Kansas Agricultural Experiment Station Research Reports by an authorized administrator of New Prairie Press. Copyright 1997 the Author(s). Contents of this publication may be freely reproduced for educational purposes. All other rights reserved. Brand names appearing in this publication are for product identification purposes only. No endorsement is intended, nor is criticism implied of similar products not mentioned. K-State Research and Extension is an equal opportunity provider and employer.



BIOSECURITY IN THE DAIRY

G. L. Stokka, T. R. Falkner, and P. Bierman

Summary

Three strategies exist to control unwanted disease in a livestock operation: 1) prevent the introduction of infected cattle, 2) raise the overall level of resistance and specific resistance to infectious disease, and 3) minimize herd exposure to infectious disease. In addition, if unwanted disease exists in the herd, then a plan to eliminate the disease should be implemented. Maintenance of closed herds, testing procedures, vaccination schedules, sanitation, and good husbandry practices are integral parts of biosecurity procedures. The procedures in place should produce a benefit in terms of both economics and public perception that the quality and safety of our food supply is of the utmost importance to livestock producers.

Livestock units exist for the purpose of producing a nutritious food product, which is accomplished through the use of forages and cereal grains. This system benefits the producer by adding value to renewable resources. Society benefits through the availability of a wholesome, safe, food supply in addition to the creation of new wealth within our economic system. The time has come for the food production industry, especially the dairy and beef sectors, to recognize the benefits of biosecurity procedures. Those of us involved in the food production business must always keep in mind the importance of maintaining healthy animals and a healthy food supply.

(Key Words: Biosecurity, Disease, Management.)

Introduction

In the dairy industry, measurements of the cost of disease are quite sensitive. Each doubling of the somatic cell count above 50,000 is estimated to cost as much as 400 lb per lactation in mature cows. Any clinical disease or even subclinical disease will result in a cost to the operation. In addition, some disease incidents may pose a risk to herd mates as well as a zoonotic risk to producers and a foodborne risk to the public.

Three strategies can prevent disease from entering or occurring in a livestock production unit. The first is preventing the introduction of infected cattle. This approach begins by ensuring that cattle are purchased from uninfected herds or herds with known health status, which implies an effective vaccination program. Never purchase cattle from unknown sources or from commingled sources. Purchased cattle should be isolated and monitored for 30 days before entering the herd. If necessary, test new herd additions for infectious disease, such as brucellosis, Johne's, BVD, and bovine leukosis, before introduction to the herd. Recipients used for embryo transfer also can be sources of disease and should be tested as necessary. If young calves are purchased, they should be from a reputable source and tested as necessary, particularly for persistent infection with BVD viruses. Purchased animals should be transported in clean and disinfected trailers or trucks.

The second is raising the overall level of resistance and specific resistance to infectious disease. Reducing environmental stress can increase the overall level of resistance. Provide clean dry bedding and comfortable housing to all

animals and use shades during the summer and windbreaks during the winter. Reduce nutritional stress through proper transition diets and balanced lactation rations. Colostral management is the most important factor for increasing the overall resistance in newborn calves.

The third is minimizing herd exposure to infectious disease. Limit exposure of the dairy facilities to outside people. Require your veterinarian and other professionals to use sanitary practices (e.g., sanitize equipment, use clean boots and coveralls). Reduce manure contamination of feed bunks, water sources, feed, and feeding equipment. Utilize cattle loading facilities away from the main animal facilities to minimize exposure to buyers and transportation equipment. Raise calves in individual hutches that are disinfected between uses. Sick animals should be isolated in most instances, particularly in unusual cases or when the response to treatment is unfavorable. Animals that die should be necropsied, either to identify the cause of death or to confirm the diagnosis. Necropsied animals must be disposed of properly, either via the renderer or burning and burying.

Elimination of disease when present in a herd is almost always more expensive and difficult than prevention. For some diseases such as leptospirosis, eliminating or effectively keeping animals from exposure may be impossible. Other diseases require a long-term and disciplined plan for reduction and elimination. For some diseases, elimination may involve total depopulation. Elimination of certain diseases requires active monitoring and action plans to handle each case as it occurs.

The following sections provide short description of diseases of importance to dairy herds and suggestions for prevention, treatment, and elimination.

Bovine Virus Diarrhea

Bovine virus diarrhea (BVD) is one of the most significant viral infections of cattle. BVD was first recognized as a disease syndrome in 1946, and today 70-90% of the world's cattle population is seropositive for BVD. The BVD

virus (BVDV) has at least two genotypes, type 1 and type 2, and two biotypes, cytopathic and noncytopathic. Both type 1 and type 2 genotypes have cytopathic and noncytopathic biotypes as members, and both type 1 and type 2 genotypes have many different strains, some of which are more deadly than others. Recently, the type 2 genotype has caused many of the most severe cases of BVD.

Clinical Syndromes

Most BVDV infections are subclinical, but the clinical disease syndromes can be grouped into three categories: acute BVD, *in utero* infections, and diseases in persistently infected (PI) animals.

Acute BVD can vary greatly in presentation from fever, depression, and runny nose and eyes to diarrhea to respiratory disease and can end in complete recovery or death depending on several factors. These include the immune status of the animal, strain with which they are infected, and age of the animal. BVDV has a profound immunosuppressive effect on infected cattle. Infected cattle are more susceptible to many respiratory and intestinal pathogens. BVDV is also an important component of bovine respiratory disease complex (BRDC).

In utero infections with BVDV can result in abortion; PI animals; congenital defects; or normal, immune-tolerant calves, depending on the stage of gestation and the cow's immune status when she is infected with the virus. The noncytopathic biotype is responsible for all *in utero* infections. If a cow is infected with BVDV in the first trimester of pregnancy, the fetus will most likely die, and the cow may reabsorb the fetus, abort, or give birth to a mummified fetus. The abortions are usually sporadic and at a low rate, usually only 2-7% in an outbreak. If the cow is infected with BVDV between 60 and 120 days of gestation, the calf may be PI. These animals are lifelong carriers of BVDV and shed large quantities of virus in all secretions throughout their entire lives. The immune system in calves less than 120 days of gestation is not capable of responding properly to BVDV, so the virus simply multiplies in the

calf. When the immune system becomes competent, the virus is recognized as “self”, and the calf is “immune tolerant” to that strain of BVDV for life; that is, it never develops an immune response to that strain. Infection with BVDV between 100 and 180 days in gestation can result in congenital defects such as cerebellar hypoplasia, hydrocephaly, cataracts, and other similar defects. Infection of the dam in the last trimester of gestation, when the calf’s immune system is functional, will yield a normal, immunized calf.

Persistently infected animals can result from *in utero* infection as described above or by being born to a PI dam. The prevalence of these animals in cattle herds is low (.5-3%), but their potential to shed large quantities of virus and infect other animals in the herd is tremendous. Persistently infected cows always give birth to infected calves, and seronegative cows (cows that have not mounted an immune response to BVDV) are much more likely to give birth to infected calves. However, some seropositive cows can give birth to infected calves, if their circulating antibodies do not cross-react with the virus to which they are exposed. Persistently infected calves are often “poor doers” and are more susceptible to other calfhoo diseases because of the immunosuppressive effects of BVDV. Sometimes, however, infected calves may look perfectly normal and healthy. Persistently infected calves reportedly have death rates of 50% in the first 12 months of life. Some of these probably die from other calfhoo diseases as well, but many die from BVD-mucosal disease (BVD-MD). BVD-MD occurs when PI animals that harbor noncytopathic BVDV are exposed to a cytopathic variant probably through mutation of the noncytopathic strain to a cytopathic strain. BVD-MD is characterized by profuse diarrhea with severe erosions and ulcers on all mucosal surfaces. It occurs most often in cattle 6 to 24 months of age and is nearly 100% fatal.

Transmission

BVDV rapidly loses infectivity outside the host and is very susceptible to detergents, light, temperature changes, and other environmental conditions. It is mainly transmitted by close contact with PI or acutely infected cattle via the oral or nasal routes. Acutely infected animals shed the virus only for a short time (about 2 wk), whereas PI animals shed constantly in all bodily secretions for life. Acutely infected bulls shed virus in their semen for at least 2 wk, and PI bulls shed virus constantly in their semen. Thus, semen is another potential source of infection during natural mating. Reputable A.I. studs will check their bulls and semen for BVDV. Sheep, goats, and pigs can become infected from close contact with cattle, and sheep can transmit the virus to cattle in close contact. Needles, rectal sleeves, water troughs, feed bunks, nose tongs, and other equipment can aid the spread of virus. Experiments have shown that biting insects also spread the virus.

Diagnosis

Diagnosis of BVD is accomplished by observation of clinical signs, serology, virus isolation, fluorescent antibody, or polymerase chain reaction (PCR) tests. The virus can be isolated from nasal swabs, serum, or tissue depending on the disease syndrome present. Diagnosis of BVD-MD is very important, because if BVD-MD animals are found, the herd should be screened for more persistently infected animals.

Prevention

Adding PI animals to a herd should be avoided, because that is the most common way to introduce BVDV into a herd. Replacement animals should be purchased from herds with accurate records of disease and vaccination. All new animals, or at least any small group of new animals, such as bulls, should be isolated and tested for BVDV before entering the herd. Semen should be from tested bulls only. If embryo transfer work is performed, all recipients

should be isolated, tested for acute or persistently BVDV infection, and vaccinated against BVDV.

Vaccination programs are essential to decrease losses to BVD. The goal of any vaccination program is to prevent fetal infection and increase colostral immunity. This may not always work, depending on the strain of vaccine and the field strain, but it is the best weapon we have. Vaccination does not clear persistent infections from a herd, but the virus doesn't spread as quickly through a vaccinated herd.

Two types of vaccine available include the modified live and inactivated (killed) forms. Much controversy exists over which is better. Modified live vaccines (MLVs) offer more cross-protection against different strains, and the immunity conferred by them is longer lasting and stronger. Modified live vaccines should be used with caution, however, because they can cause immunosuppression or fetal infection or revert to virulence. Inactivated vaccines are not immunosuppressive, do not infect fetuses, and have minimal risk. However, the immune response they generate is weaker, of shorter duration, and may not cross-protect as well as MLVs. Cattle receiving inactivated vaccine also must have a booster 3 to 4 wk after the first vaccination. Neither MLVs nor inactivated vaccines give lifelong protection, and yearly boosters are required with both.

No one vaccination program works for all situations. Producers should consult their veterinarian for a program tailored for their herd. Here are a few options.

For replacement heifers (separated from pregnant cows), use an MLV at 6 months of age and again 60 days before breeding or use an inactivated vaccine 5 wk before breeding and again 2 wk before breeding.

For cows, use inactivated vaccine 2 wk before breeding or use MLV before breeding. Either of these options can be used with either option for heifers.

Vaccinate calves with a different strain of inactivated vaccine than used for cows 3 to 4 wk

before weaning or vaccinate with MLV before weaning.

Control

If a diagnosis of BVD is made in a herd and significant losses are occurring, control measures may need to be taken to decrease future economic losses. Vaccination will slow the spread through the herd but will not prevent PI animals from shedding virus. They must be removed from the herd. Several procedures have been outlined to screen the herd for PI animals. One option for complete herd screening is to collect serum from every animal in the herd and analyze it with the microplate virus isolation test, and then remove all animals that test positive. This test is very sensitive and specific for BVDV. Another option is to perform serology on every animal in the herd and cull those animals with very low or absent titers. This can be performed only in herds that are vaccinated or have active BVDV circulating and is not as sensitive for detecting PI animals as the microplate test. Some researchers recommend testing only the calves with microplate virus isolation and then testing only the dams of the calves that test positive. A common theme in these procedures is that all calves born for the next 9 months also must be tested to ensure that no new PI animals are born into the herd. With any screening procedure, biosecurity measures as outlined in the "Prevention" section must be implemented to prevent reintroduction of PI animals.

Neosporosis

Neospora caninum is a coccidian protozoa that originally was found in dogs but later found to be a major cause of abortion in cattle, sheep, goats, and horses. The complete life cycle is not known, which makes formulation of control programs difficult. We assume that some definitive host sheds oocysts in its feces; however, most cows are infected congenitally, which is the only known natural route of infection. Abortion is the only clinical sign in *Neospora* infected cows, and multiple abortions can occur over a few months. Most abortions are at 4-6 months of gestation but can occur any time between 3-8 months of gestation. Calves infected congenitally can be underweight, weak,

unable to rise, uncoordinated, or normal. Most cases of neosporosis have been associated with dairy cattle, but congenital infections and abortions also have been documented in beef cattle.

Diagnosis of neosporosis is difficult in live cattle but can be accomplished in aborted fetuses by a combination of immunohistochemistry on the tissues and serology from the dam. Aborted fetuses and placentas should be handled with caution, because there is a real possibility that this parasite can infect humans, although it has not been proven yet.

Prevention programs are difficult to design because of the lack of knowledge of the life cycle. No vaccines or drugs are available for treatment. Aborted fetuses and placentas should be burned or otherwise properly disposed of to prevent the potential intermediate host or other cows from being infected by eating these tissues. Feed and water should be protected from fecal contamination by domestic or wild animals. Infection of the cow before or during pregnancy can cause abortion, but not all infections result in abortion. Seropositive cows are two times more likely to abort from neosporosis, and cows that abort once from neosporosis can abort again from the disease.

If abortions from neosporosis are high in number, control measures may need to be taken. Many control programs exist. One possible first step is to screen 35-50 cows, depending on herd size, with serology (ELISA or IFA) to find the prevalence of *Neospora* in the herd. If the prevalence is low, the entire herd can be screened, and the seropositive animals culled. If the prevalence is too high to cull out the seropositives, one possibility is to test all dams and daughters of cows that have aborted and cull the seropositives. Also, testing replacement animals and permitting only seronegative replacements to enter the herd will reduce congenital infections. If embryo transfer is done, use only seronegative recipients, and don't purchase a heifer whose recipient was seropositive.

Neospora is a major cause of abortions in cattle. Prevention and control are complicated by the unknown life cycle of the parasite. Diagnosis is difficult in live cows, but antibodies can be detected to help point out cattle at higher risk of aborting. Testing all new animals before they enter the herd and protecting feed and water from contamination by other animals are keys to prevention.

Cryptosporidiosis

Cryptosporidium parvum, also known as "Crypto", is a protozoan parasite closely related to coccidia. It is present in virtually all calf-raising environments and can cause calf diarrhea. It can infect all mammals, including humans, in which it causes severe headaches, vomiting, nausea, and weakness in addition to severe diarrhea. In elderly and otherwise immunosuppressed people, it can be life threatening. The organism commonly infects calves 1 to 4 weeks of age, causing a nonbloody, yellow, watery malabsorptive diarrhea that is usually self-limiting, and mortality is low. Diagnosis can be made by fecal flotation or fecal smear stained with acid-fast stain.

Infected calves shed billions of infective oocysts in their feces, and adult cattle can shed some as well, but far fewer than calves. Transmission from calf to calf or calf to human is primarily by the fecal-oral route, although it can be transmitted by the aerosol route as well. The most common place for calves to become infected is in the calf-rearing area. Control of "Crypto" focuses mainly on management. The organism is hardy and has been shown to survive from 2 to 6 months at 4°C (40°). The organism is resistant to most disinfectants, but bleach at half strength is effective, as is formalin (formaldehyde). Formalin is very toxic and should be used only under strict supervision. Putting calves in hutches or on a clean pasture with low stocking density will reduce the level of exposure to *C. parvum*, and thereby reduce losses from "Crypto" diarrhea.

If calves become ill with “Crypto” diarrhea, the only treatment is supportive care, especially electrolyte and fluid therapy. The use of anticoccidial drugs has been suggested for treatment and prevention but has not proven to be effective.

Salmonella Infections

Many species of the bacterium *Salmonella* affect cattle. They can cause sporadic abortions and, more commonly, neonatal diarrhea, especially in dairy calves. They are not common causes of diarrhea in beef calves. Humans can be infected with *Salmonella* by drinking unpasteurized milk or handling infected placentas and fetuses. Most cattle become infected by ingestion of contaminated feed, water, or milk. *Salmonella* can be shed by asymptomatic carrier cows and calves, and it can survive in a damp environment for months. Rodents can also be a source of the bacterium.

Good hygiene is essential to halt the continued spread of *Salmonella*. The calving area should be clean, and hutches should be used. The hutches should be cleaned and disinfected after each group of calves, and the feeding utensils should be cleaned between feedings. A rodent control program should be instituted. All replacements should be tested to be sure they are not carriers, and aborting animals should be isolated. Vaccinating cows with two doses of a killed bacterin may help control *Salmonella* in calves less than 3 weeks of age, but vaccination of the calves is usually not protective. Good hygiene, not vaccination, should be the main focus for controlling *Salmonella* diarrhea.

Johne’s Disease

Johne’s disease, also known as paratuberculosis, is caused by *Mycobacterium paratuberculosis*, a slow growing bacterium that can survive in the environment for approximately 1 year. It is best known in the dairy industry, where it costs U.S. dairy producers an estimated \$1.5 billion annually, but cases in beef herds, especially seedstock operations, can be devastating as well. In addition to death loss, premature culling, and decreased weight at slaughter, losses

from decreased milk production and increased susceptibility to other diseases such as mastitis can be major. Johne’s disease is a reportable disease in Kansas. Recently, *M. paratuberculosis* has been associated with Crohn’s disease in people, but scientific evidence is not available to prove or disprove its involvement in the disease at this point.

Most cattle with Johne’s disease were infected as young calves, which are most susceptible. Calves have no clinical signs, and, therefore, this stage of the disease has been called the “silent” stage. After an incubation period of 2-10 years, infected adult cows can be more prone to mastitis or infertility. These animals can be shedding the organism in their feces at undetectable levels, which can contaminate the environment. Within a few weeks, clinical signs such as gradual weight loss with a normal appetite, diarrhea, and decreased milk production can appear. In advanced cases, animals are very weak, have profuse, “pipestream” diarrhea, and can have intermandibular edema, or “bottle jaw”, and death follows shortly. For every such case of advanced Johne’s disease on a farm, 15 or 25 other animals likely are infected.

The major route of infection of calves is ingestion of colostrum and/or milk contaminated with fecal material. Calves also can be infected *in utero*, especially if their dam is clinically ill. Such infection is unlikely in early, subclinically infected dams. Infected cows can shed the organism directly in colostrum or milk as well, which is another potential source of infection for calves. Adults can be infected from contaminated feed, but they are less susceptible than calves, and because of the long incubation time, likely will be culled before they shed the organism. The organism also can be transmitted by semen, in uterine fluids, by rectal examinations, and by wildlife, but these are not likely sources of infection. Embryo transfer and artificial insemination are not likely sources of infection because of frequent testing. However, all embryo transfer recipient cows should be tested, because fetal infection can occur transplacentally.

Prevention

Because of the nature of the disease, prevention is much more economical than control once it has entered the herd. Herds are infected primarily by purchasing infected animals. These animals may show no clinical signs for many years and may even test negative on serologic and fecal culture tests. The sensitivity of tests for Johne's disease is only about 50%, which means 50% of animals with the disease will not test positive. Therefore, it is best to maintain a closed herd or purchase replacements from herds that are certified to test negative. If this is not possible, prepurchase testing of the seller's entire herd should be done. If none or very few test positive, chances are very good the animals purchased are not infected. At the minimum, replacements should be purchased from reputable herds with no clinical history of Johne's. All new animals should be isolated and tested before they enter the herd. The risk of bringing in paratuberculosis in an animal from a sale barn has been estimated at 10% per animal. Another preventative measure that should be practiced on all farms is proper cleaning of calving areas and calf hutches.

Many tests are available to test individual animals and screen herds for Johne's disease. The sensitivity of these tests for early detection is low because of the slow progression of the disease. Fecal culture is best for detection of infected animals in a herd. It is 100% specific, which means every positive test truly indicates an infected animal, and 50% sensitive. The major drawback to this test is the 12- to 16-wk incubation period before results are available. A new culture method is available that has only a 4- to 7-wk incubation period, but it is more expensive. Three serum tests that detect antibodies to paratuberculosis are used commonly. They are the complement fixation test (CF), the agar gel immunodiffusion test (AGID), and the enzyme-linked immunosorbent assay (ELISA). Results from these tests are available in 2 to 4 days, and they are nearly 100% specific and quite sensitive in detecting infected animals, especially those with clinical signs. ELISA has a sensitivity of 99% and a specificity of 15 to 87%, depending on the stage of the disease, but overall specificity of 45%. It is the most sensi-

tive and specific of the serum tests. A DNA probe test, which is fast but expensive and less sensitive than fecal culture, also is available. Rectal scrapings or histopathology of tissues are both sensitive in detecting clinically ill animals. The newest test method, which tests for cellular immunity, is not proven yet, but has a promising future. The Johnin Test, which has been used in the past, is no longer recommended.

Control

Control and/or eradication of Johne's disease on a farm that has had confirmed cases of the disease is a long, difficult process and should be undertaken only if management changes can be instituted. No "cookbook" method of control works for every farm, but a summary of key points follows. Control programs have two fundamental objectives. The first is to prevent highly susceptible newborn calves and young animals from ingesting manure, colostrum, and milk from infected cows. To accomplish this, remove the calves early; put them in hutches; and feed them only uninfected colostrum, milk, or milk replacer. Improving hygiene to reduce exposure of calves to *M. paratuberculosis* also reduces exposure to *Salmonella*, *E. coli*, *Crypto*, and coccidia because of decreased fecal contact. The second objective is to reduce total farm environmental contamination by culling infected animals. To accomplish this objective, screen all animals over 20 months of age with the ELISA test or fecal culture, and cull all animals that test positive. A more aggressive strategy is to cull all offspring from cows that test positive, because of the possibility they were infected *in utero*. The ELISA test is recommended for the first screening, because it is the least expensive test with similar sensitivity and specificity to fecal culture. Within 1 year after the test-positive cattle have been culled, all animals over 20 months of age should be tested again, with either ELISA or fecal culture, and the test-positive animals culled. Herd screening should continue, but the time between screenings varies depending on multiple factors. Elimination of Johne's disease takes many years, and biosecurity measures as outlined above should be practiced along with the control program.

A killed vaccine is available for use by accredited veterinarians, usually under the supervision of the state veterinarian, and its usage varies from state to state. It does not prevent infection, but it does delay the onset of clinical signs. However, it interferes with diagnostic tests for Johne's disease and is not recommended.

Mastitis

Mastitis is inflammation of the mammary gland. The majority of mastitis causes are caused either by contagious mastitis pathogens such as *Streptococcus agalactiae* and *Staphylococcus aureus* or environmental pathogens such as *Escherichia coli* and *Klebsiella pneumoniae*. Mastitis also can be caused by other organisms such as *Nocardia* sp. and yeast.

Environmental mastitis is caused by pathogens present in feces, bedding, or other places in the cow's environment. They are impossible to eliminate, so steps to prevent environmental mastitis include decreasing exposure of teat ends to pathogens and increasing the resistance of cows to intramammary infections. Conditions that increase exposure to environmental pathogens include overcrowding; elevated temperature and humidity in barns; poor ventilation; accumulation of manure, urine, and water; poor stall design; access to ponds or muddy lots; and dirty maternity stalls or calving areas. To increase the resistance of cows to intramammary infections with environmental pathogens, they should be fed a well-balanced diet that is sufficient in vitamin E and selenium. The J-5 bacterin-toxoid has proven effective in decreasing incidence and severity of these infections as well.

Contagious mastitis is transmitted from cow to cow via infected milk at milking time through the milker's hands, milking units, common sponges and towels, and other items used during milking. Most *Strep. agalactiae* and some *S. aureus* infections respond to most commercial intramammary antibiotic products in both the lactating and dry periods. Chronic *S. aureus* and *Strep. agalactiae* infections may not respond to antibiotic therapy, and chronically infected cows should be identified, segregated, and milked last at every milking. They should be culled when

daily feed costs exceed income from milk production. Prevention and control or eradication of *S. aureus* and *Strep. agalactiae* infections can be accomplished by good milking practices, especially proper udder preparation using single-use towels and post-milking teat dips. Also, purchase animals from herds with low somatic cell counts and test individual animals before they enter the milking herd. New animals should be milked last until negative test results for contagious mastitis are returned.

Cases of *Nocardia* sp. and yeast mastitis often are due to contaminated mastitis treatment preparations and infusion needles. Yeast mastitis also can result from multiple antibiotic treatments, because yeast are resistant to antibiotics.

Hairy Heel Warts

Papillomatous digital dermatitis, also known as hairy heel warts, is a contagious infection of the skin, usually on the back of the foot between the bulbs of the heel, most often on the rear feet. It is primarily a disease of housed dairy cattle and rarely occurs in pasture cattle. Unlike true bovine warts, which are caused by a virus, heel warts are most likely caused by spirochetes, which are spiral-shaped bacteria. Early heel warts can be flat and circular and later become raised masses and develop hair-like projections. They bleed easily if traumatized and can cause severe pain and lameness, resulting in economic losses from decreased milk production, decreased reproductive efficiency, and cost of treatment. Only about 50% of cows with heel warts are actually lame, which makes control difficult.

Introduction of the bacteria into a clean herd may be difficult to prevent in anything but a closed herd, but isolation of replacement animals for at least 1 month and checking all their feet very closely before introducing them into the herd are good practices. Also, hoof trimmers, veterinarians, and any other visitors should clean and disinfect their boots and equipment before working on the herd.

Once introduced, the warts spread quickly through the herd, and eradication is difficult or impossible because of the subclinical cases and lack of immune clearance. Lactating heifers and

young cows may show more lameness than older cows, but this pattern does not always hold true.

Treatment of heel warts is by extra-label drug use, so your veterinarian must be consulted for a treatment protocol. Whole-herd treatment is necessary to control heel warts, because of the high incidence of subclinical cases that can perpetuate the problem in the herd and the propensity of the warts to recur. The most cost-

effective method of whole-herd treatment is by topical spray of antibiotics, such as tetracycline, after washing the feet. Foot baths are effective only if properly prepared and maintained, which can be very expensive. Whole-herd treatment is not a cure and will have to be repeated on a regular basis to limit the incidence of this disease in the herd.