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Cross-Infection Between Cats and Cows: Origin and Control of *Streptococcus canis* Mastitis in a Dairy Herd

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ABSTRACT

Group G streptococci in animals usually belong to the species *Streptococcus canis* and are most commonly found in dogs and cats. Occasionally, *Strep. canis* is detected in milk from dairy cows. An outbreak of *Strep. canis* mastitis in a dairy herd is described. Based on results from bacterial culture and ribotyping, a cat with chronic sinusitis was the most likely source of the outbreak. Subsequent cow-to-cow transmission of *Strep. canis* was facilitated by poor udder health management, including use of a common udder cloth and failure to use postmilking teat disinfection. Infected cows had macroscopically normal udders and milk, but significantly higher somatic cell counts than *Strep. canis*-negative herd mates. The outbreak was controlled through antibiotic treatment of lactating cows, early dry-off with dry cow therapy, culling of infected animals, and implementation of standard mastitis prevention measures. Cure was significantly more likely in dry-treated cows (87.5%) and cows treated during lactation (67%) than in untreated cows (9%). Whereas mastitis due to group G streptococci or *Strep. canis* in dairy cows is usually limited to sporadic cases of environmental (canine or feline) origin, this case study shows that crossing of the host species barrier by *Strep. canis* may result in an outbreak of mastitis if management conditions are conducive to contagious transmission. In such a situation, measures that are successful in control of *Strep. agalactiae* can also be used to control *Strep. canis* mastitis. (**Key words:** *Streptococcus canis*, mastitis, host species barrier, group G streptococcus)

Abbreviation key: BMSCC = bulk milk somatic cell count, DCT = dry cow treatment, GGS = group G streptococcus, LCT = lactating cow treatment, MRSA = methicillin-resistant *Staphylococcus aureus*, QMPS = Quality Milk Production Services.

INTRODUCTION

Streptococci are a common cause of mastitis in dairy cows. In many areas, contagious mastitis caused by *Streptococcus agalactiae* has largely been controlled (Loeffler et al., 1995; Andersen et al., 2003), but other streptococci, specifically *Streptococcus dysgalactiae* and *Streptococcus uberis*, continue to be highly prevalent throughout the world (Wang et al., 1999; Zadoks et al., 2004). Identification of streptococcal species in mastitis diagnostics is usually based on hemolytic patterns, esculin splitting, and the CAMP reaction (National Mastitis Council, 1999). Serological grouping in accordance with the Lancefield system can also be used for typing of some streptococcal species from milk, most importantly for group B streptococci or *Strep. agalactiae* (Facklam, 2002). In addition, group G streptococci (GGS) are occasionally found in bovine milk samples.

Mastitis caused by GGS in dairy cows is relatively rare. In herd surveys from Iowa and New York State, the prevalence was 0.7% of 455 streptococcal cultures from 72 herds (McDonald and McDonald, 1976), 4 of 250 dairy herds (1.6%) (Hamilton and Stark, 1970), and 125 of 105,083 surveyed cows (0.1%) (Wilson et al., 1997). However, herd outbreaks due to GGS have been reported from many places, including Washington, DC (Miller and Heishman, 1940); Ontario, Canada (Barnum and Fuller, 1953); Denmark (Romer, 1948); New York (Hamilton and Stark, 1970); Pennsylvania (Eberhart and Guss, 1970); Israel (Bergner-Rabinowitz et al., 1981); Louisiana (Watts et al., 1984); The Netherlands (O. C. Sampimon, personal communication, 2003); and Italy (P. Moroni, personal communication, 2003). In 1986, the name *Streptococcus canis* was coined (Devriese et al., 1986) to describe GGS found in dogs and cattle. Animal GGS or *Strep. canis* differed in physiological, biochemical, and DNA hybridization characteristics from human GGS isolates which belong to the species *Strep. dysgalactiae* spp. *equisimilis* (Devriese et al., 1986). In fact, *Strep. canis* is more closely related to *Streptococcus pyogenes* or group A streptococcus than to GGS of humans (Facklam, 2002). In dogs and cats, *Strep. canis* is found on skin and mucosa of asymptomatic carriers and in many pathological conditions, in-

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cluding infections of the skin, urogenital, and respiratory tract, polyarthritis, abortion, septicemia, canine streptococcal toxic shock syndrome, and necrotizing fasciitis (Devriese et al., 1986; DeWinter et al., 1999; Hassan et al., 2003).

In this paper, we describe an outbreak of bovine mastitis caused by *Strep. canis* in a New York State dairy herd. The source of infection and routes of pathogen transmission are identified through bacteriology, molecular typing of GGS isolates, and analysis of herd management. The impact on affected cattle and the outcome of antibiotic treatments and management changes that were instituted to curb the outbreak are presented. This case study serves both as a suggestion on how to deal with *Strep. canis* in dairy cattle, and as an example of the combination of traditional herd-health approaches with modern DNA-based methods for problem solving in a situation where crossing of the host-species boundary by a pathogen resulted in an unusual disease outbreak.

MATERIALS AND METHODS

Case History

In April 1999, Quality Milk Production Services (QMPS) personnel were requested to visit a dairy herd in central New York State for the first time to perform a whole-herd mastitis screening survey. The herd, consisting of 90 lactating head of Holstein-Friesian cattle with mean 305-d milk production of 6700 kg/cow, was in danger of losing its milk market because 2 of the last 4 official bulk milk somatic cell counts (BMSCC) were greater than 750,000 cells/mL. Bulk milk SCC had been 173,000/mL in December 1998, but counts had risen steadily since that time. The most recent BMSCC was 1,800,000/mL. Standard plate count was 41,000 cfu/mL.

The herd was housed in a tie-stall barn with concrete floors. Stalls were covered with rubber mats and minimal amounts of old hay. The milking system included a 5.08-cm (2-inch) pipeline around the barn with 8 milking units. Cows were milked twice daily by the producer and his wife. Cows' teats were forestripped and then washed with water and a common towel. Teats were not dried before attachment of the milking unit. Post-milking teat dip was not applied, and gloves were not worn by the producer or his wife. Cows were milked once a day for 3 d before dry off and then treated in each quarter with a long-acting penicillin-dihydrostreptomycin treatment. The herd had been closed for 40 yr and had always been housed at the same location. Several cats had access to the barn.

A second visit followed in May 1999. Bulk milk SCC on the latest test was 560,000/mL. Quarter samples for bacteriologic culture were collected from those lactating

cows that were diagnosed with GGS at the whole herd survey in April. From the remaining 50 cows, composite cow milk samples were collected. In addition, swabs or samples were collected from milking unit inflations, nasal secretions, and hand surfaces of the producer's wife, udder wash towels, dip cups, and feline nasal and anal secretions. Personnel from QMPS returned to the herd in July and October 1999, for whole herd surveys. Bulk milk SCC were 560,000/mL and 470,000/mL, respectively, at those surveys. Because BMSCC was consistently below the legal limit and the producer planned to sell the herd in 2000, no further treatments or surveys were undertaken.

Milk Samples, Bacteriology, and SCC

Composite milk samples from each lactating cow were collected aseptically into sterile vials, in accordance with National Mastitis Council guidelines, at the morning milking. Samples were cooled rapidly and transported to the laboratory for immediate bacteriologic culture. Aliquots (0.01 mL) of each sample were plated on trypticase soy agar plates containing 5% sheep blood and 0.1% esculin (Becton Dickinson, Sparks, MD). Plates were incubated aerobically at 37°C and examined for growth at 24 and 48 h. Colonies were presumptively identified as streptococci by colony morphology, hemolytic patterns, and esculin reaction, and were confirmed by Gram stain and catalase-negative reaction. Representative colonies were tested for the CAMP reaction. Biochemical tests were performed on representative isolates with the API 20 Strep system (BioMérieux, Hazelwood, MO), and serologic grouping was accomplished on all streptococcal isolates with the PathoDx latex agglutination system following the manufacturer's recommendations (Diagnostic Products Corporation, Los Angeles, CA). Based on this method, isolates could be identified as GGS, without differentiation between *Strep. canis* and *Strep. dysgalactiae* spp. *equisimilis*. Swab samples were inoculated in Todd-Hewitt broth upon collection and taken back to the laboratory for processing within a few hours. In the laboratory, samples were incubated for 3 to 4 h in a water bath at 37°C. Swabs were subsequently streaked onto trypticase soy agar plates containing 5% sheep blood and 0.1% esculin. Plates were processed and evaluated as described for milk samples.

Additional composite milk samples that were collected during the second herd survey (May 1999) were used to measure SCC (Fossomatic FC; Foss, Eden Prairie, MN). Antibiotic sensitivity of a limited number of isolates (n = 5) was determined using the Kirby-Bauer agar disk diffusion method in accordance with standards from the National Committee for Clinical Labora-

tory Standards (NCCLS, 1997). Antibiotics tested were amoxicillin, cephalotin, novobiocin, oxacillin, penicillin/novobiocin, penicillin, pirlimycin, and tetracycline. Based on the diameters of the inhibition zones, bacteria were classified as resistant, intermediately resistant, or susceptible.

Interventions

Because of the impending loss of the milk market, a decision was made to treat or cull all cows that were infected with GGS. Seven cows were culled immediately because of poor production or infertility. Cows due to dry off ($n = 7$) were treated in all 4 quarters for 3 consecutive milkings with 62.5 mg of amoxicillin and then abruptly dried off with 1,000,000 IU of penicillin and 1 g of dihydrostreptomycin (dry cow treatment; **DCT**). Lactating cows ($n = 18$) were treated with 62.5 mg of amoxicillin in all 4 quarters for 3 consecutive milkings (lactating cow treatment; **LCT**). Postmilking teat dipping with 0.5% iodine was also instituted. After the second survey (July 1999), 2 additional cows were treated with amoxicillin based on these culture results, and 1 additional cow with GGS was culled from the herd. Eleven cows infected with GGS remained in the herd but did not receive treatment. Allocation of treatment was decided by the farmer and was not based on formal randomization. After the second survey, use of the common towels was discontinued, and individual paper towels were used for udder preparation.

Molecular Typing

During routine bacteriology, isolates were identified as GGS based on serology. To determine whether GGS isolates belonged to the species *Strep. canis*, PCR using species-specific 16S rDNA as target (Hassan et al., 2003) was used to test 1 feline isolate and 12 bovine isolates. Strain typing of confirmed *Strep. canis* isolates was performed by means of automated ribotyping using the RiboPrinter Microbial Characterization system (Qualicon, Wilmington, DE) and restriction enzymes *EcoRI* or *PvuII*. Based on results for this selection of all GGS isolates in combination with the herd situation, typing of additional isolates was not deemed necessary.

Statistical Analyses

For analysis of SCC data, cows were grouped based on culture results, and SCC were log-transformed. Cows were classified as 1) infected with GGS, 2) infected with other major pathogens, 3) infected with *Corynebacterium bovis*, 4) infected with other minor pathogens (*Staphylococcus* spp.), or 5) culture-negative. Group as-

signment was hierarchical, i.e., any sample with GGS was assigned to group 1, irrespective of presence of other bacterial species; any sample without GGS but with other *Streptococcus* species or *Staphylococcus aureus* was considered group 2, irrespective of presence of minor pathogens, etc. Comparisons of SCC between groups were done using box and whisker plots and 1-way ANOVA. Cure rates for LCT and DCT were analyzed by logistic regression and χ^2 analysis. Because several cells in the χ^2 analysis had values less than 5, Fisher's Exact tests were also performed. Significance was declared at $P < 0.05$. All analyses were done in Statistix version 8.0 (Analytical Software, Tallahassee, FL).

RESULTS

Bacteriology

At the first survey (April 1999), 46 of 90 cows (51%) cultured positive for GGS. Sixteen cows (18%) were infected with *Staph. aureus*. Six cows had concurrent infections with both organisms and are included in both counts. Results of the second survey (May 1999) indicated that 17 of 77 (22%) lactating cows were infected with GGS. Cultures positive for GGS were identified from 1 milking unit liner and from nasal secretions from a cat with chronic sinusitis. The samples from the producer's wife, collected because she suffered from chronic pharyngitis and sinusitis, tested negative for GGS in tests performed by QMPS (skin swabs, nasal secretion) or by the family physician (pharyngeal swab). At the third and fourth surveys (July and October 1999) 19 and 11 cows, respectively, were culture-positive for GGS. Moreover, GGS was consistently isolated from nasal secretions from the cat with chronic sinusitis that had tested positive in May. For analysis of cure data, quarters that were positive for GGS before treatment and had 2 negative cultures at 2 subsequent surveys after treatment were considered cured.

SCC

Udders of cows infected with GGS were normal on palpation and none of these cows showed evidence of clinical mastitis. Average logSCC was 6.7 (equivalent to 4.8 million cells/mL) for GGS-positive milk samples ($n = 16$), 6.2 (equivalent to 1.5 million cells/mL) for milk samples containing other major pathogens ($n = 8$), 6.0 (equivalent to 1 million cells/mL) for milk samples that only contained *C. bovis* ($n = 27$), 5.8 (equivalent to 631,000 cells/mL) for milk samples with other minor pathogens ($n = 7$), and 5.5 (equivalent to 353,000 cells/mL) for culture-negative milk samples ($n = 20$). LogSCC for GGS-positive samples was significantly higher than

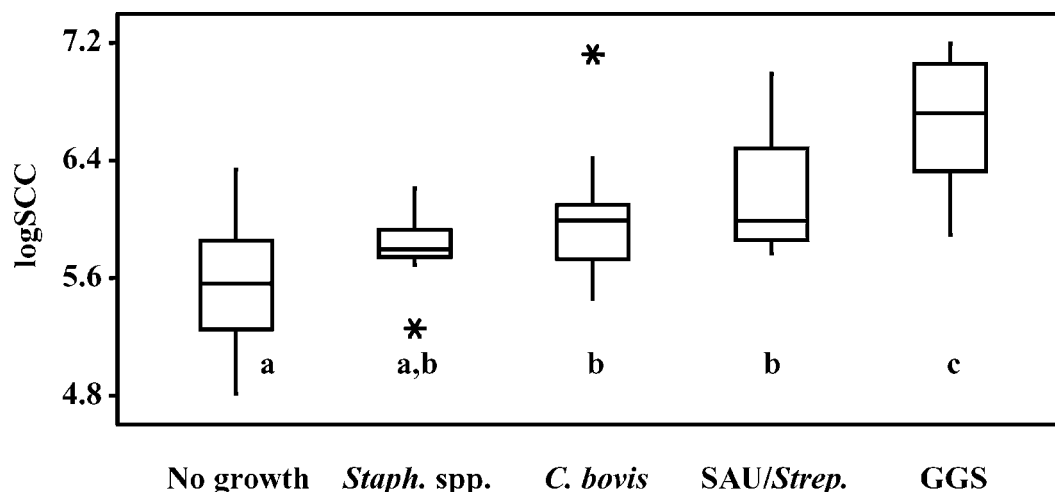


Figure 1. Boxplot of logSCC by culture status of composite cow milk samples. Different letters indicate that average logSCC is significantly different between groups ($P < 0.05$). Culture status: No growth = culture-negative; *Staph. spp.* = positive for other minor pathogens (*Staphylococcus* spp.); *C. bovis* = positive for *Corynebacterium bovis* (irrespective of presence of other minor pathogens); SAU/*Strep.* = positive for *Staphylococcus aureus* or *Streptococcus* species other than group G streptococci (GGS) (irrespective of presence of other minor pathogens); GGS = positive for GGS (irrespective of presence of other pathogens). Bottom of each box represents first quartile; central line indicates median; top of box represents third quartile; and whiskers indicate range. Possible outliers, defined as values that are outside the box boundaries by more than 1.5 times the size of the box, are indicated by an asterisk.

for any other type of sample, and logSCC for culture-negative samples was significantly lower than for samples harboring GGS, other major pathogens, or *C. bovis* (Figure 1).

Molecular Typing

One feline isolate and 12 bovine isolates of GGS were tested by PCR and confirmed to be *Strep. canis*. Among the bovine *Strep. canis* isolates, which originated from quarter milk samples of 12 different cows, 1 ribotype was identified by means of *EcoRI* ribotyping. The same ribotype was obtained for *Strep. canis* isolated from nasal secretion of the cat with chronic sinusitis. Ribotyping of a feline and bovine isolate with *PvuII* yielded indistinguishable patterns too (Figure 2). Ribotype images for all isolates used in this study are publicly available in the searchable PathogenTracker database (www.pathogentracker.net). Ribotypes are identified by a code (e.g., 116-783-3) consisting of instrument ID (i.e., 116) and pattern ID (e.g., 783-3).

Treatment and Cure

Antibiotic sensitivity was determined for the feline isolate and 4 bovine isolates. Resistance was not detected. Because the outbreak appeared to result from contagious transmission of one strain, no additional sensitivity testing was done. Thirteen of 20 cows receiving LCT with amoxicillin were cured (65%), as were 6

of 7 (87.5%) cows receiving DCT with amoxicillin and penicillin-dihydrostreptomycin. Eleven infected cows remained in the herd but were not treated. One of those cows (9.0%) showed a spontaneous cure whereas the rest cultured positive on subsequent samples. Lactating cow treatment and DCT did not differ significantly from each other in probability of cure ($P = 0.6$), but both were significantly more likely to result in cure than no treatment ($P < 0.001$).

DISCUSSION

In this case study, we describe an outbreak of *Strep. canis* mastitis in a dairy herd that was most likely initiated by cross-host-species transmission of *Strep. canis* from a cat with chronic sinusitis to a cow, and subsequently spread from cow to cow in a contagious manner as a result of poor milking hygiene. The outbreak was controlled through implementation of routine mastitis prevention measures, i.e., use of single-use udder towels and postmilking teat disinfection (aimed at a reduction in the incidence of new infections), in combination with culling, dry-off, and lactational treatment of infected cows (aimed at reduced prevalence of infection). Reduction in prevalence was needed to decrease exposure to GGS and incidence of new cases, and to bring BMSCC back down to a level that allowed for shipping of milk.

Streptococcus canis mastitis in dairy cows is relatively rare but its occurrence has been reported from

Host species	Source	Enzyme	RiboPrint pattern	Number of isolates typed
Cat	Nasal secretion	<i>EcoRI</i>		1
Cow	Milk	<i>EcoRI</i>		12
Cat	Nasal secretion	<i>PvuII</i>		1
Cow	Milk	<i>PvuII</i>		1

Figure 2. Ribotyping results for *Streptococcus* from a cat with chronic sinusitis and cows with mastitis on a dairy farm.

several countries and continents around the world. In some cases, anecdotal information specifically implies a role for a canine source (barn dog licking cows' teats; personal communication to RNZ from a Dutch veterinarian, 2003). Based on strain typing, we showed that a cat may have been the source of the outbreak reported here. The cat had chronic sinusitis that predated the mastitis outbreak, had access to the cows, and shed *Strep. canis*. It is also possible that the cat became infected through consumption of raw cows' milk, but because the cat had sinusitis before the outbreak, we think it more likely that the cat infected the cows than vice versa. Although many bacterial species or strains are more or less host-specific, crossing of host species boundaries has been described for several pathogens of animals and humans. For example, *Staph. aureus*, in particular methicillin-resistant *Staph. aureus* (MRSA), has been transmitted between humans and animals in both directions. Dogs (Cefai et al., 1994) and cats (Scott et al., 1988) can act as a source of MRSA carriage in humans, and humans can be a source of MRSA for animals. An outbreak of mastitis in dairy cows caused by MRSA has been attributed to introduction by a milker (Devriese and Hommez, 1975). Host-adapted strains have also been described for *Strep. agalactiae*, another udder pathogen of dairy cows (Bisharat et al., 2004; Sukhnanand et al., 2005). Some clones of *Strep. agalactiae* are predominantly found in humans, whereas different clones are found in milk from dairy cows (Sukhnanand et al., 2005). However, human strains of *Strep. agalactiae* have been found in other animals, i.e., dogs and cats, suggesting an epidemiological connection and possible transfer of bacteria across host species (Yildirim et al., 2002). Thus, although a host species barrier exists, pathogens may infect species that are not their natural host.

Theoretically, the fact that ribotyping results were identical for all cows could be the result of infection of each cow individually by the cat. However, it seems far

more likely that the host species barrier was crossed once, and that subsequent cow-to-cow transmission of *Strep. canis* occurred. Two obvious shortcomings in milking time hygiene that may have contributed to pathogen spread are the use of a communal udder cloth and the failure to use postmilking teat disinfection. An outbreak of streptococcal mastitis in the absence of postmilking teat disinfection due to another streptococcal species that is not contagious under good management conditions, *Strep. uberis*, has been described before (Zadoks et al., 2001). In that outbreak, as in the current case study, mastitis-causing streptococci were isolated from inflations, implying that the milking machine, like the communal udder cloth, may act as a fomite for transmission (Zadoks et al., 2003). It has been known for decades that postmilking teat disinfection can curb the transmission of many *Streptococcus* species (Neave et al., 1969; Eberhart et al., 1983). Because speciation and strain typing results were the same for all samples that were characterized with molecular methods, confirmation of all GGS isolates as *Strep. canis* and ribotyping of all isolates was not deemed necessary.

The incidence of GGS infections in humans is increasing in many parts of the world (Lewthwaite et al., 2002; Sylvetsky et al., 2002; Ikebe et al., 2004). Our interest in this outbreak of GGS mastitis in dairy cows was partially ignited by the possibility of milk acting as a source of GGS in people. Human and animal GGS can each cause chronic pharyngitis and sinusitis in their respective hosts (Ikebe et al., 2004). On the farm, chronic sinusitis/pharyngitis was present in a human and a cat; hence, samples from both hosts were analyzed. Only the cat was shown to carry GGS and more specifically, *Strep. canis*. In general, GGS from dairy cows belong to the species *Strep. canis* (Devriese et al., 1986; unpublished results from our laboratory) whereas GGS in humans rarely does (Zaoutis et al., 1999; Woo et al., 2003; Ikebe et al., 2004). A few exceptions to

this rule are cases of *Strep. canis* septicemia in elderly people that were bitten by a dog (Takeda et al., 2001) or that had a condition predisposing them to secondary infection (Bert et al., 1997; Whatmore et al., 2001).

There is no known public health impact of *Strep. canis* in cattle but infections with the pathogen have a serious impact on udder health and milk quality. Clinical mastitis and damage to udder secretory tissue because of GGS infection is variable and ranges from no gross alterations of milk to severely clotted samples (Hamilton and Stark, 1970). Leukocytosis appears to be a common finding (Miller and Heishman, 1940; Barnum and Fuller, 1953; Eberhart and Guss, 1970; Watts et al., 1984). In the outbreak reported in this case study, gross abnormalities of the udder were not noted but SCC of GGS-infected cows was significantly higher than that of cows that did not have GGS, including cows that had infections with other major pathogen species such as *Staph. aureus* or nonagalactiae streptococci other than GGS. In previous studies, response of GGS infections to intramammary antibiotic therapy has been variable (Miller and Heishman, 1940; Barnum and Fuller, 1953; Eberhart and Guss, 1970; Watts et al., 1984) despite demonstration of good susceptibility of the bacteria to penicillin in vitro (Devriese et al., 1986; Libertin et al., 1988). Hamilton and Stark (1970) state that "the infected quarters were readily cured with commercially prepared mastitis infusions containing procaine penicillin G and dihydrostreptomycin" without specifying dose or length of treatment or number of treated cases. Good cure with procaine penicillin G was also reported in a Pennsylvania herd (Eberhart and Guss, 1970). In contrast, Watts et al. (1984) reported only 24% cure for LCT with a penicillin-based product, whereas 55 and 70% cure was achieved with cephalosporin as LCT or DCT, respectively. In the herd described in the current paper, treatment with amoxicillin and (in the case of DCT) penicillin-dihydrostreptomycin was successful in the majority of cases. Treatment was warranted because of the impending loss of the milk market; and resulted in reduction of BMSCC and return of the herd to an economically viable status.

CONCLUSIONS

To summarize, we showed how strain typing was used to demonstrate contagious transmission of a pathogen, and that a cat was the most likely source of an unusual outbreak of mastitis in a dairy herd. We also demonstrated how cow and herd level data were used to identify contributing causes to the problem, such as poor milking time hygiene, as well as opportunities for control of the disease outbreak. Through culling of cows with poor prognosis for cure and treatment of

cows during lactation or at dry off, infection prevalence and BMSCC were reduced. The decrease in prevalence, combined with management measures aimed at a decrease in infection incidence through diminished pathogen transmission, resulted in return of the herd to an udder health status and milk quality level that met the producer's needs and industry standards. Thus, this case study shows how routine bacteriology and analysis of milking management, combined with use of molecular methods, can help resolve disease outbreaks, and specifically how an outbreak of *Strep. canis* mastitis in a dairy herd may be handled.

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