

# *Mycoplasma* Mastitis

## Causes, Transmission, and Control

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### KEYWORDS

• *Mycoplasma* • Mastitis • Epidemiology • Control

### KEY POINTS

- *Mycoplasma* sp are categorized as contagious mastitis pathogens, and it appears that *Mycoplasma* mastitis is a growing problem in the United States.
- The herd prevalence of mycoplasma mastitis pathogens has been estimated through culture and analysis of bulk tank milk samples.
- *Mycoplasma* sp that have been associated with mastitis have been considered contagious in nature, transmitted at milking time from a reservoir, the infected udder; via fomites, hands of a milker, milking unit liners, or udder wash cloths; to an uninfected cow. Additionally, evidence is presented that would suggest that *Mycoplasma* sp are spread on dairy herds by aerosols, nose to nose contact, and are spread hematogenously to the mammary gland to cause mastitis and arthritis.

### INTRODUCTION

The first reported case of *Mycoplasma* mastitis was that of Hale and coworkers.<sup>1</sup> This Connecticut research group described the difficulties in isolating the pathogen that infected approximately 30% of a dairy herd. They had success when they allowed incubation of milk cultures to proceed for 5 days under 10% CO<sub>2</sub>. They named the isolated organism *Mycoplasma agalactiae* var *bovis*, currently known as *M bovis*. This first described outbreak was remarkable in that it affected a large proportion of the herd, spread to multiple quarters of the same cow, and the agent was difficult to culture. Shortly after this report, Carmichael and coworkers of New York,<sup>2</sup> as reported by Jasper<sup>3</sup> and Stuart and coworkers of Great Britain,<sup>4</sup> reported *Mycoplasma* mastitis cases. One can imagine that following the report by Hale and coworkers,<sup>1</sup> researchers<sup>1,4</sup> and others applied the culture techniques described and were able to isolate *Mycoplasma* sp from cases of mastitis that might have previously been considered

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**Table 1**  
Percentage of cases of *Mycoplasma mastitis* by species

Report	<i>M bovis</i>	<i>M californicum</i>	<i>M bovigenitalium</i>	Other
Jasper (1980) <sup>15</sup>	51	16	5	28
Kirk et al (1997) <sup>16</sup>	48	11	25	16
Boonyayatra et al (2011) <sup>17</sup>	85	5	1	9

idiopathic. Thus, 50 years ago it was apparent that *Mycoplasma mastitis* was a problem, perhaps an emerging problem.

Today it is recognized that *Mycoplasma mastitis* affects cattle around the world.<sup>5,6</sup> *Mycoplasma* sp are categorized as contagious mastitis pathogens<sup>7</sup> and it appears that *Mycoplasma mastitis* is a growing problem in the United States.<sup>3,8–10</sup> Moreover, given the difficulty in culturing the pathogen that was first noted 50 years ago, there is reason to suspect that cases of *Mycoplasma mastitis* are underreported.<sup>11</sup> In this review the epidemiology of *Mycoplasma mastitis* will be discussed, followed by a discussion of the host–pathogen interaction and elements associated with control of the disease. A focus of this article will be the presentation of recent findings that would explain why *Mycoplasma* may be an emerging mastitis pathogen.

## EPIDEMIOLOGY

*Mycoplasma* sp are pathogens associated with several cattle diseases, primarily otitis media, inflammation of the urogenital tract, arthritis, pneumonia, and mastitis.<sup>12,13</sup> The most prevalent species causing these diseases is *M bovis*.<sup>5,14</sup> With respect to *Mycoplasma mastitis*, *M bovis* is the predominant causative agent and *M californicum* and *M bovigenitalium* appear to the next most common (Table 1).

Jasper<sup>15</sup> summarized the agents associated with cases of clinical *Mycoplasma mastitis* during a 14-year period and found that *M bovis* and *californicum* were the most common. The third most common was *M alkalescens*, which comprised approximately 12% of intramammary infections, followed by *M bovigenitalium* at 5% (see Table 1). Kirk and coworkers<sup>16</sup> surveyed bulk tank milk from a cooperative of 267 dairies in CA monthly for 6 years. The annual prevalence of tanks with *Mycoplasma* sp known to be mastitis agents ranged from 1.2% to 3.1% of tank samples. They reported that *M bovis*, *californicum*, and *bovigenitalium* were the most consistently the *Mycoplasma mastitis* agents isolated. Boonyayatra and colleagues<sup>17</sup> examined milk samples from 248 cases of clinical mastitis from a variety of sources over several years and reported 85% were *M bovis*, 5% were *M californicum*, and only 1% were *M bovigenitalium*. In the surveys reported in Table 1, it is clear that *M bovis* and *M californicum* appear to be the 2 most prevalent *Mycoplasma mastitis* pathogens. Other species that have been noted as causes of *Mycoplasma mastitis* include *M arginini*, *bovirhinis*, *canadense*, *dispar*, *bovine group 7*, and *F-38*.<sup>18</sup>

## Prevalence

Prevalence of contagious mastitis pathogens estimates have been made through culture and analysis of bulk tank milk samples.<sup>9,19</sup> The major contagious mastitis pathogens identified this way in the United States are *Staphylococcus aureus*, *Streptococcus agalactiae*, and *Mycoplasma* sp, with herd level prevalence of 43.0%, 2.6%, and 3.2%.<sup>9</sup> In this survey,<sup>9</sup> the herd size affected the prevalence of only *Mycoplasma mastitis*, with the prevalence of other contagious mastitis pathogens

unaltered by the number of cows per herd. In large herds (>500 cows), the prevalence of *Mycoplasma* mastitis was 14.4%. Results from a previous study were similar as it was reported that the percentages of *Mycoplasma* positive bulk tanks from herds with less than 100, 100 to 499, and more than 500 cows was 2.1%, 3.9%, and 21.7%.<sup>8</sup> In the later survey, regional differences were noted with 9.4% of the operations in the West having one positive *Mycoplasma* bulk tank culture, with operations in the Northeast and Midwest with less than 3% and the Southeast having 6.6%. Presumably, the regional differences are a function of herd size as herds in the West tend to have the most cows and herds in the Northeast and Midwest tend to have the fewest number of cows.<sup>20</sup>

Based on bulk tank surveys, the prevalence of *Mycoplasma* mastitis varies across the globe. In the European Union countries of Belgium, France, and Greece, the range in prevalence was less than 1% to 5.4% of herds.<sup>21–23</sup> Yet surveys done in Mexico,<sup>24</sup> Iran,<sup>25</sup> and Australia<sup>26</sup> indicate prevalence estimates as high as 55% to 100% of herds. In New Zealand, McDonald and coworkers<sup>27</sup> surveyed 244 herds and could not detect *Mycoplasma* sp in any bulk tank samples, suggesting a very low prevalence. The wide variation in global prevalence may be a function of exposure to these agents. Importation and mixing of cattle have been reported to lead to outbreaks of *Mycoplasma* diseases. For example, the first reported case of *Mycoplasma* cattle disease in Ireland occurred in 1993 and was attributed to the relaxation of import controls within the European Union.<sup>28</sup> Exposure of naïve cattle to this agent led to the appearance and then a significant increase in bovine *Mycoplasma* diseases.<sup>28</sup> Herd replacement cattle exposed to cattle outside the herd, either imported or reared off-site, increased with increasing herd size, a biosecurity risk factor.<sup>29</sup> It was found that herd size<sup>10,30</sup> and culling<sup>30</sup> were risk factors for increased herd prevalence of *Mycoplasma* mastitis. Presumably this is a result of herd expansion, the entrance of new cattle with symptomatic, or asymptomatic carriage of new strains of *Mycoplasma* sp into the herd. Thus, the elevated prevalence of *Mycoplasma* mastitis in herds, and herds of some countries, where cattle movement into and out of a herd is common, could explain the increased prevalence of this disease.

Cow-level prevalence is more difficult to estimate. It has been reported that in Great Britain, less than 1% of cows are affected by *Mycoplasma* mastitis.<sup>31</sup> *Mycoplasma* mastitis has most often been reported as a clinical disease. A survey of clinical mastitis in New York indicates that *Mycoplasma* sp are the cause of 1.5% of cases.<sup>32</sup>

### **Transmission**

*Mycoplasma* sp that have been associated with mastitis have been considered contagious in nature, transmitted mostly at milking time from a reservoir, the infected udder; via fomites, hands of a milker, milking unit liners, or udder wash cloths; to an uninfected cow.<sup>7</sup> Strict milking time hygiene practices of disinfectant of udders before milking using single service towels, use of gloves by milkers, post-milking unit disinfection, and disinfection of teats post-milking were very effective in controlling the traditional contagious mastitis pathogens of *S aureus* and *S agalactiae*.<sup>33</sup> It has been assumed, but not tested, that such practices would be effective in the control of *Mycoplasma* mastitis.

*Mycoplasma* sp can spread from one bovine body site to another presumably via lymph or peripheral blood systems. *Mycoplasma* sp associated with mastitis have been isolated from the blood of cattle.<sup>34–36</sup> In outbreaks with *Mycoplasma* mastitis, it is not unusual to find cases of *Mycoplasma* arthritis.<sup>37–41</sup> Similarly, a field outbreak of *Mycoplasma*-associated bovine respiratory disease was associated with outbreaks of

arthritis.<sup>41</sup> The link between arthritic *Mycoplasma* disease events and mastitis or pneumonia is indicative that internal somatic spread of this agent is not uncommon. Often multiple organ sites of cattle can be colonized and it is clear that the strain causing the disease is most often the same strain that is widely disseminated throughout the body.<sup>35</sup> This is also been shown by Jain and colleagues,<sup>42</sup> who experimentally induced intramammary infections with *Mycoplasma* sp in lactating cows and found that the apparent strain inoculated was shed at the mucosal surfaces of the eyes, nose, vagina, and rectum, within hours to days after inoculation. With this experiment, they also demonstrated vertical transfer of the agent as a calf, born during the trial from one experimentally infected cow, became colonized by the agent.<sup>42</sup> Moreover, in an outbreak of *Mycoplasma* mastitis, the agent was found colonizing the nares of cattle, both cows and/or calves.<sup>43,44</sup> The strain causing mastitis was found from nasal swab samples collected from cows and calves.<sup>40</sup> Thus, transmission of *Mycoplasma* sp associated with bovine mastitis may occur within the cow internally, from one infected organ site to the udder or reverse; and between cows from indirect udder to udder contact at milking time; or perhaps by shedding of the pathogen through external mucosal surfaces of an infected or colonized animal to a naïve animal.

Transmission of *Mycoplasma* sp from environmental sources to the udder has been discussed.<sup>18</sup> In this review, the authors report on 2 studies, 1 in Italy and 1 in Germany, where it was found that *M bovis* survived in and on multiple surfaces at various temperatures for up to 8 months. Materials studied were those that could be typically found on dairies including sponges, stainless steel, wood, rubber, glass, and water. Justice-Allen and coworkers<sup>45</sup> in Utah discovered that *Mycoplasma* could live for up to 8 months in a sand pile. The sand originated from a herd with an outbreak of *Mycoplasma* mastitis. *Mycoplasma* was also isolated in sand from 2 other dairies. The authors<sup>45</sup> suggested that sand could be a reservoir for *Mycoplasma* mastitis. However, in a separate investigation where there appeared to be a link between sand bedding and a clinical mastitis outbreak, it was found that the strains of *Mycoplasma* sp in the bedding had a completely different DNA fingerprint than those causing mastitis (Fox and Corbett, unpublished data, 2008). Utah researchers<sup>46</sup> investigated the possible transmission of *M bovis* from sand to naïve dairy calves during a 105-day trial. Although calves housed on sand bedding with *M bovis* carried this agent for periods of time during the trial, there was no evidence of carriage beyond transient colonization and no specific antibody titers formed against the agent. The authors concluded that there was no evidence that the contaminated bedding would serve as a source of *M bovis* disease transmission to naïve dairy calves. Thus, although it is clear that environmental sources could serve as a reservoir for *Mycoplasma* mastitis, there is no evidence to support that *M bovis* transmission from the environment to a cow is a likely mechanism involved in *Mycoplasma* mastitis.

### Carriage

Most cases of mastitis are subclinical and the greatest loss to a dairy is a result of the subclinical nature of the disease.<sup>47</sup> Jasper<sup>48</sup> indicates that a significant number of cows might be shedding *Mycoplasma* pathogens in their milk without symptoms. Perhaps given the difficulty, expense, and the historically low prevalence of the *Mycoplasma* mastitis, a good estimate of the prevalence of subclinical *Mycoplasma* mastitis infections has not been reported.

It is well established that *Mycoplasma* sp can be isolated from mucosal surfaces of clinically normal calves and cows.<sup>40,49</sup> The prevalence of calves shedding *M bovis* at the nares was 34% in herds with noted *Mycoplasma* mastitis and only 6% in herds

apparently free of disease.<sup>49</sup> The prevalence of mucosal surface shedding by asymptomatic carriers with the same clone of *M bovis* causing a *Mycoplasma*-associated disease outbreak may be as high as 21% to 47% of cattle in a dairy herd.<sup>40</sup> These findings indicate that *Mycoplasma* shedding by ostensibly healthy cattle is not uncommon but may be far more likely in herds experiencing a current outbreak.

The role of the asymptomatic *Mycoplasma* carrier animal in an outbreak of mastitis is not clear. It is known that *M bovis* carriage in the lungs of beef cattle calves is approximately that of dairy calves in situations without apparent *Mycoplasma* disease. Carriage increases when cattle are stressed, such as when they are moved from their place of rearing and then comingled in different locations as in feedlots.<sup>12</sup> Climatic stresses and *Mycoplasma* disease outbreaks have also been documented. Episodes of *Mycoplasma pneumoniae* were observed in a closed beef herd where a number of calves became diseased after a spring storm.<sup>50</sup> Only 1 strain of *M bovis* was identified from pulmonary samples. Given the herd<sup>50</sup> was closed, it could be suspected that the strain identified was asymptotically carried by cattle of this herd, and with climatic stresses and potentially associated compromised hosts, the *M bovis* strain was able to transform cattle from symptomless to diseased. Thus, a change in the environment of the calf, a move away from their accustomed setting, a change in climate, and/or the exposure to potential new strains of *Mycoplasma* sp can increase the prevalence of carriage of these agents and such carriage might be associated with subclinical or clinical disease.

A dairy herd will generally increase the exposure of its herd, to outside animals, through the purchase of replacements and via off-site rearing of calves. The University of Idaho dairy with approximately 90 to 100 lactating cows was historically free of *Mycoplasma* mastitis, and ostensibly other *Mycoplasma* diseases were rare or nonexistent. An outbreak of *Mycoplasma*-associated diseases at the University of Idaho dairy began shortly after a state institutional herd contracted with the dairy to raise their calves. The institutional herd also leased their primiparae to the university dairy.<sup>40</sup> Within 2 months of initiating the contract, several cases of *Mycoplasma* diseases in calves and mastitis in cows developed. Diseased animals were culled from the herd, and during the third month of the initial outbreak, samples of mucosal surfaces of all animals were collected. Nearly 25% of all animals were shedding the same clone of *M bovis* from the mucosal surfaces as that causing disease. Yet, within 6 months only 1 cow and 1 calf were shedding the clone. During the course of the next year, the outbreak strain was infrequently detected. However, the outbreak clone was the only cause of *Mycoplasma* mastitis, with 4 cases occurring in total. One case spontaneously cured, and the other 3 cases were removed from the herd. New strains of *Mycoplasma* sp were detected, and these strains appeared to be very similar to the outbreak clone. None of these similar strains caused disease. These findings suggest an outbreak strain may be widely disseminated within a herd initially, with a few cases of disease, but concomitant with the dissolution of the outbreak is the reduction of shedding of the agent from mucosal surfaces. Additionally, the authors<sup>40</sup> concluded that the outbreak strain originated with the animals exposed to the institutional dairy herd and thus was imported into the herd. Punyapornwithaya and colleagues<sup>41</sup> also reported on an outbreak of *Mycoplasma* mastitis that appeared to originate with an imported heifer. The *M bovis* clone that caused mastitis in the original heifer at parturition also caused mastitis, pneumonia, and arthritis in the home herd of lactating cows. The strain then “ran its course” and disappeared after 4 months. A similar outbreak of *M bovis* disease was reported to start with mastitis.<sup>44</sup> Here an imported heifer developed mastitis at parturition, and within a few weeks several of the

homebred cows developed *M bovis* mastitis, 1 cow developed arthritis, and several calves developed pneumonia. These reports<sup>40,41,44</sup> demonstrate that in an outbreak, there is the potential for multiple animals to become infected with several forms of *Mycoplasma* disease. In aggregate, these studies<sup>40,41</sup> indicate that a single clone of *M bovis* can readily transmit through the herd, but only a small proportion of cows become infected, and both asymptomatic carrier(s) or diseased animals can be the nidus of the outbreak. The nature of transmission might have been during milking time in one herd<sup>41</sup> but in the other<sup>40</sup> it was concluded by the authors that nose-to-nose contact was the most likely means of transmission. Pulmonary transmission would account for the rapid spread, the involvement of both lactating and nonlactating animals, and the involvement of both respiratory and joint diseases. Both Bicknell and colleagues<sup>51</sup> and Jasper<sup>49</sup> discuss the role of the asymptomatic carrier in *Mycoplasma* mastitis disease outbreaks. Both warn that asymptomatic carriers may be reservoirs of disease, although neither author presents evidence that such an outcome is likely or unlikely.

Jasper<sup>49</sup> indicates that some dairy managers will cull asymptomatic *Mycoplasma* carriers and some will isolate carriers until shedding subsides; successful control can be achieved with either method. The odds of an asymptomatic carrier causing an outbreak is unknown. Additionally, preferential culling or isolation of carrier animals was not apparently necessary to control *Mycoplasma* mastitis, and no animal appeared to be an asymptomatic carrier prior to the appearance of *Mycoplasma* mastitis.<sup>40</sup> Additionally, a cow or cows with *Mycoplasma* mastitis may not pose a risk to the development of an outbreak and may not need to be preferentially culled to control transmission.<sup>52</sup> It appears that asymptomatic carriage of *Mycoplasma* sp is involved in a *Mycoplasma* mastitis outbreaks. However, the definitive role carrier animals play in the outbreak and how they should be controlled are unclear. If culling asymptomatic carriers is chosen as a *Mycoplasma* mastitis control strategy, then it should be used judiciously while considering the number of potential culls and their proximity to susceptible animals. Isolation of affected animals and monitoring new carrier and infected animals might be effective tools of control of *Mycoplasma* mastitis.

#### CHARACTERISTICS OF PATHOGENIC *MYCOPLASMA* SP

Razin and Hayflick<sup>52</sup> have recently reviewed the research on *Mycoplasma* sp. They report that the *Mycoplasma* sp evolved from gram-positive bacteria in a degenerative evolution where these simple organisms lost the ability to produce a cell wall, one manifestation of the diminution of the genome. Razin and Hayflick<sup>53</sup> wrote that *Mycoplasma* cells have essentially 3 organelles: cell membrane, ribosomes, and densely packed circular DNA. The *Mycoplasma* cell is spherical about 0.3 to 0.8  $\mu\text{m}$  in diameter. The species have a significant requirement for fatty acids and sterols and intermediate metabolic pathways are often truncated. *Mycoplasma* sp are perhaps the smallest and most simple self-replicating bacteria.<sup>54</sup> Given their simple nature and fastidious growth requirements, they find ecological niches within their host. In cases of intramammary infections, *Mycoplasma* sp do not appear to often cause a significant, if any, febrile response,<sup>48,55,56</sup> which may be consistent with their nature to colonize cows asymptotically.

*Mycoplasma* sp lack a cell wall and thus are inherently resistant to beta-lactam antibiotics. The study of the pathogenicity of *Mycoplasma* organisms is diverse given that there are more than 100 *Mycoplasma* sp, with most of these pathogens specific for one or a few host species. Yet it appears that the pathogenic characteristics of *Mycoplasma* sp in general are (1) adherence to host cells, (2) internalization into host

cells, (3) immunomodulatory characteristics, and (4) ability to colonize host tissue without causing fulminant disease.

Several *Mycoplasma* sp including *M bovis* possess adhesion molecules as part of their cell membranes, which allow them to bind to host tissue cells.<sup>57</sup> *M pneumoniae*, for example, possesses a protein complex (P1, P30, P116, HMW1-3, A, B, and C) that provides for structural and functional adherence to cells and enables gliding mobility.<sup>58</sup> *M bovis* possesses variable surface lipoproteins (Vsps) that are involved in adherence to host cells.<sup>59–61</sup> These Vsps are part of a complex bacterial system that is notably most antigenically diverse and associated with much variation in gene expression.<sup>60,62,63</sup> Browning and colleagues<sup>64</sup> describe the high-frequency phase variation of the multigene families that encode surface proteins that are part of the *Mycoplasma* sp genome. They indicate that it has been generally accepted that the antigenic variation that results from the genetic phase variation is an immune evasion characteristic, although this hypothesis has not been tested.

Adherence to mammary epithelial surfaces is a characteristic of contagious mastitis pathogens, and this adherence characteristic appears to differentiate the contagious from the noncontagious mastitis pathogens.<sup>65,66</sup> It would be logical to assume that since *Mycoplasma* sp are considered contagious mastitis pathogens and as *Mycoplasma* mastitis pathogens are likely to produce cytoadhesins, they would also have the ability to adhere to mammary epithelial cells, although this has been untested.

There may be other benefits to these adhesion proteins. The ability to adhere to host cell mucosal surfaces may enable the *Mycoplasma* sp to access nutrients including amino acids, nucleic acids, fatty acids, and sterols.<sup>67</sup> *Mycoplasma* sp tend to have truncated intermediate metabolic pathways<sup>53</sup> and thus have significant nutrient requirements, especially for sterols and fatty acids.

Pathogens that have the ability to invade and survive within the host cell have the advantage of the protection that the host cell affords against the host's own immune response and antimicrobial therapy. The mastitis pathogen *Staphylococcus aureus* has been described to possess this factor.<sup>68</sup> The ability to invade mammary epithelial cells may be a function of the virulence of the *S aureus* mastitis pathogen.<sup>69</sup> *Mycoplasma* sp have the ability to invade eukaryotic host cells.<sup>70–72</sup> There is evidence to indicate that *M bovis* can invade peripheral blood mononuclear cells and erythrocytes in vitro<sup>73</sup> and in both renal tubular epithelial cells and hepatocytes in clinically diseased bull calves determined at necropsy.<sup>74</sup> van de Merwe and colleagues<sup>73</sup> acknowledge that what might have seemed to be *M bovis*-induced invasion might have been a phagocytic response by specific immunocytes. However, *M bovis* appeared to be internalized by lymphocytes and erythrocytes. Not only would such internalization afford the pathogen protection from the immune response and antibiotic treatment, but this characteristic would enable it to reach multiple organ systems, consistent with the ability of *M bovis* to spread to multiple body sites of diseased cattle.<sup>35,55</sup>

*M bovis* has the ability to modulate the immune system. Findings by van der Merwe and colleagues<sup>73</sup> and Vanden Bush and Rosenbusch<sup>75</sup> indicate the pathogen secretes a peptide, a factor that can inhibit lymphocyte proliferation. This factor appears in the culture supernatant.<sup>73</sup> In addition, *M bovis* can cause immunomodulation of both the humoral and cell-mediated responses. Antibody titers may be reduced in *M bovis*-affected cattle,<sup>76</sup> and the ratio of IgG1 to IgG2 was reversed in some pneumonic calves.<sup>77,78</sup> An alteration in the T-helper cell response to *M bovis* lung infections was noted,<sup>79</sup> and there was evidence indicating that anti-inflammatory cytokine production was altered by an *M bovis* infection.

## CONTROL

Historically, it has been thought that *Mycoplasma* mastitis might be best controlled by a test and slaughter program. Cows with *Mycoplasma* mastitis need to be identified and culled from the herd.<sup>6,18,80</sup> A critical component of this *Mycoplasma* mastitis control program is a monitoring system. First, a potential problem with *Mycoplasma* mastitis must be known and cows suspected of *Mycoplasma* mastitis must be identified and verified as diseased. Culture of bulk tank milk on a regular basis is a method to monitor a herd's *Mycoplasma* mastitis status,<sup>10,16,81</sup> and such regular sampling and culture of bulk tank milk as a monitor of *Mycoplasma* mastitis in a herd have been advocated.<sup>52</sup> It is generally believed that the culture of *Mycoplasma* sp from bulk tank milk is indicative of at least one herd cow having *Mycoplasma* mastitis, although a negative culture does not necessarily indicate that the herd is free of this disease.<sup>82</sup> If a herd has zero tolerance of *Mycoplasma* mastitis, then a positive bulk tank culture must be followed by the identification of cows with mastitis. Generally, cows with recent or chronic cases of clinical mastitis would be identified and milk from infected mammary quarters cultured and tested for *Mycoplasma* sp. Additionally, cows with elevated milk somatic cell counts would be identified and milk cultured. Cows once identified with *Mycoplasma* mastitis would be culled from the herd. However, the process of collection of a sample, transport to the laboratory, and culture and identification of the agent can take at least 4 to 7 days, an interim period. Cows may be penned with other infirm cows without *Mycoplasma* mastitis during this interim period. The transmission of *Mycoplasma* mastitis within these hospital pens might be as much as 100-fold more than in the cow's home pen.<sup>41</sup> Thus, hospital pen cows must be managed carefully to control this disease such that *Mycoplasma* mastitis is not transmitted to the home pens, when cows falsely believed to be free of this disease are returned.

The test and slaughter method of control might not be required. Some<sup>53,83,84</sup> reported that control could be achieved without culling, although another report indicated success with specific removal of cows with *Mycoplasma* mastitis.<sup>85</sup> *Mycoplasma* mastitis as a contagious mastitis pathogen should be controlled by full milking time hygiene practices that include disinfectant in the udder premilking wash, single service towels used to clean and dry udders premilking, use of clean gloved hands by milkers, milking unit backflush, and postmilking teat disinfection.<sup>52</sup> Biosecurity practices of isolation of all cattle before entry into a new herd, the testing of those cattle for carriage of *Mycoplasma* sp and elimination of those testing positive prior to entry into the herd, would in theory be an effective control strategy. Yet such a strategy does not appear to be a most common practice.<sup>52</sup> Quarantine of incoming animals requires considerable management as a practice. Quarantine as a control of a disease like *Mycoplasma* mastitis, that is emerging but affects a minority of cattle and herds, may not be cost effective. Yet *M. bovis* was believed to be asymptotically carried from imported cows into a herd believed to have been free of *Mycoplasma* mastitis.<sup>40</sup> Such carriage resulted into an outbreak of *Mycoplasma* diseases, mastitis, arthritis, and pneumonia, in cows and replacements in this herd.

Control of *Mycoplasma* mastitis via treatment is generally not viewed as a primary strategy. It is clear from previous discussion that the immune system will respond to *Mycoplasma* sp as a foreign agent. Yet it is also clear that *Mycoplasma* sp have the ability to evade the immune system by altering their surface proteins and inducing immunomodulatory effects. Perhaps the latter 2 characteristics would explain in part the heretofore lack of a successful development of mastitis vaccines against this agent.<sup>6,18</sup> An excellent review of *Mycoplasma* mastitis therapy can be found in

Jasper.<sup>3</sup> In that review, it is clear that although in vitro sensitivity of *Mycoplasma* mastitis agents exists for a broad range of non-beta-lactam antibiotics, success with antibiotic therapy in vivo has been unrewarding. Bushnell<sup>80</sup> indicated that based on his field experience, antibiotic therapy of *Mycoplasma* mastitis was not an economically viable control strategy.

## SUMMARY

*Mycoplasma* mastitis is an emerging mastitis pathogen. Herd prevalence has increased over the past decade, and this increase parallels the increase in average dairy herd size. It has been documented that the importation of cattle into a herd can result in new cases of *Mycoplasma* disease in general and *Mycoplasma* mastitis specifically. Thus, expanding herds are likely to have a greater incidence of this disease. Transmission of the agent can result from either contact with diseased animals or with colonized or asymptotically infected cattle. Initial transmission might occur via nose-to-nose contact and result in an outbreak of *Mycoplasma* mastitis, or it might occur during the milking time. This would suggest that new, incoming animals should be quarantined before being commingled with original herd animals. Quarantining does not seem to be a biosecurity strategy often practiced in control of *Mycoplasma* mastitis and may not be warranted in herds with excellent milking time hygiene practices. The ability to monitor for the incipient stages of an outbreak, often done through bulk tank milk culturing, is recommended.

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