www.canadianveterinarians.net/larounds

Large Animal VETERINARY Rounds™

AUGUST/SEPTEMBER 2003
Volume 3, Issue 7

AS PRESENTED IN THE ROUNDS OF THE DEPARTMENT OF LARGE ANIMAL CLINICAL SCIENCES OF THE WESTERN COLLEGE OF VETERINARY MEDICINE, UNIVERSITY OF SASKATCHEWAN

Mycoplasma bovis-associated disease: new syndromes and emerging problems

By Farshid Shahriar, DVM, MVSc; Edward G. Clark, DVM, MVSc

***Mycoplasma bovis* is traditionally known as a major contributing etiological agent in enzootic pneumonia of calves,[1] mastitis of cows,[2] and arthritis of young and adult bovine animals. Recently, a chronic pneumonia-polyarthritis syndrome (CPPS) with distinct pulmonary and arthritic lesions caused important economic losses in the beef industries of Canada and the United States.[3-5] This condition often fails to respond to antimicrobial treatment. Once established in a herd, the infection has a chronic course and is difficult to control. Pathogenic mycoplasmas have a strong affinity for mucosal surfaces and a specific preference for the respiratory and urogenital tracts, mammary glands, and serosal membranes. *Mycoplasma bovis* contains a series of prominent membrane lipoproteins on the cell surface that are highly variable in size and expression. It is thought that they may play a critical role in allowing the organism to escape the host immune system.**

This issue of *Large Animal Veterinary Rounds* reviews the major diseases associated with *Mycoplasma bovis* in cattle, and discusses the diagnosis, clinical findings, the recent advances in research on basic molecular mechanisms of its pathogenicity, and developments in laboratory diagnosis of this infectious agent.

Bovine mycoplasmas

Mycoplasmas are wall-less prokaryotes (class Mollicutes) that contain a diverse group of organisms; they are considered the smallest self-replicating organisms. Among the approximately 200 species of Mollicutes from animals, only a small number, mainly *Mycoplasma* species, are described as pathogens. *Mycoplasma mycoides* subsp *mycoides* small colony (SC) type, is the etiological agent of contagious bovine pleuropneumonia (CBPP) and was the first *Mycoplasma* species described and cultured at the end of the 1800s.[6] It is still one of the most important threats to the cattle industry in parts of the world and is a reportable disease in North America. The disease was eradicated from North America in 1892.

Mycoplasma bovis was first isolated in the USA in 1962 and it is believed that the infection is now worldwide[7] with a growing prevalence.[8-10] The prevalence of *M. bovis* is most likely underestimated, since most laboratories do not routinely monitor for mycoplasmas. *M. bovis* is considered the second most pathogenic bovine mycoplasma after *Mycoplasma mycoides* subsp *mycoides* SC.

Mycoplasmas are generally host-specific, and most diseases associated with animal mycoplasmas are chronic with high morbidity and low mortality.[11] Table 1 describes the major diseases associated with mycoplasmas in cattle.

Diseases associated with *Mycoplasma bovis*

Pneumonia

Bovine respiratory disease (BRD) complex is the most important cause of mortality and culling of weaned calves and young animals after arrival at a feedlot.[12] The cost of *M. bovis* infections in the USA is estimated to be $32 million per year.[13] In spite of advances in immunization, treatment, and control

WCVM
WESTERN COLLEGE OF VETERINARY MEDICINE

Department of Large Animal Clinical Sciences
Western College of Veterinary Medicine

Western College of Veterinary Medicine
Department of Large Animal Clinical Sciences
52 Campus Drive
University of Saskatchewan
Saskatoon, Saskatchewan S7N 5B4

The editorial content of *Large Animal Veterinary Rounds* is determined solely by the Department of Large Animal Clinical Sciences, Western College of Veterinary Medicine

The Canadian Veterinary Medical Association recognizes the educational value of this publication and provides support to the WCVM for its distribution.

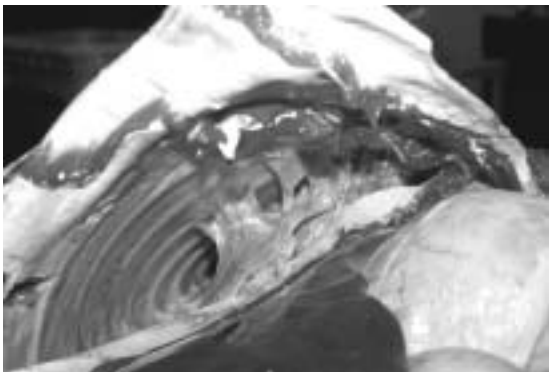
Table 1: The most important pathogenic Mollicutes in cattle[11]

Mollicutes species	Disease
M. mycoides subsp *mycoides* SC	Contagious pleuropneumonia
Mycoplasma sp. Bovine group 7	Pneumonia and arthritis
M. bovis	Pneumonia, polyarthritis, mastitis, abortion, sterility
M. dispar	Pneumonia
M. californicum	Mastitis
M. canadense	Mastitis
M. bovigenitalium	Mastitis and genital disease
M. bovocculi	Conjunctivitis
Ureaplasma diversum	Sterility, abortion
Eperythrozoon wenyoni	Anemia

of specific pathogens, *M. bovis* pneumonia infections are increasingly observed and isolated in feedlot calves and adult bovine animals worldwide.[5,9] *Mycoplasma bovis* pneumonia usually occurs in calves shortly after their arrival in the feedlots. Affected animals are clinically characterized by depression, increased respiratory rate, coughing, nasal discharge, and fever. Arthritis in one or multiple joints may be associated with the pneumonia. A number of these animals become chronically ill and fail to thrive. This disease condition has been named chronic pneumonia-polyarthritis syndrome (CPPS) in feedlot cattle (Dr. Colleen Pollock, personal communication). At necropsy, affected animals have variable degrees of bronchopneumonia with fibrinous to fibrous pleuritis (Figure 1).

M. bovis lesions of the lung vary considerably from case to case. Most show variable degrees of cranioventral chronic bronchopneumonia with nodular, firm, yellow lesions within the lung lobes that often project above the pleural surface. On the cut surface, these lesions vary from miliary (1-2 mm) to large (often 1-2 cm or larger) areas of caseous-like necrosis (Figure 2). Unlike a true abscess, the masses are not encapsulated and the exudate is seldom creamy. These nodular mycoplasma lesions may be few or large in number and are randomly scattered throughout all of the lung lobes. The nodules are usually accompanied by dark red, firm, collapsed lung. The necrotic lung nodules are usually delineated by thin fibrous tissue similar to chronic necrotic lesions of *Mannheimia haemolytica*. Sometimes the lung lobes may be diffusely involved, accompanied by pleural and interlobular fibrosis. Linear yellow necrotic lesions are often seen in the interlobular septa where *M. bovis* has gained access to the interlobular lymphatics. The organism is consistently isolated from the bronchial lymph nodes in these cases. It is the

Figure 1: Chronic pleuritis with costal adhesions in *M. bovis* infected lung in a feedlot calf.



authors' impression that the organism in lung tissue begins to proliferate within the lumen of airways. It continues to expand outward in a centrifugal-like fashion, coalescing with adjacent necrotic areas, and eventually invading and disseminating further via the perivascular and interlobular lymphatics. In cases where arthritis is the principal clinical presentation, the lung lesions may be confined to the right middle lobe or perhaps only the accessory lobe of the right lung. Cases have been observed with a single mycoplasma lung lesion, yet with very extensive and seemingly more chronic arthritic lesions. Histopathologically, the mycoplasma lesions are zones of diffuse hypereosinophilic, amorphous, necrotic lung tissue. The outer edges of the necrotic zones usually show lymphoid cells and some macrophages rather than neutrophils. However, in small lesions, neutrophils are often seen and probably make up much of the necrotic cells in zones of necrosis. The Warthin Faulkner silver stain is useful because it will stain the extremely tiny *Mycoplasma* organisms black. More importantly, this stain will also stain any other types of bacteria, helping to confirm whether the mycoplasma alone is responsible. The organisms are most numerous in the outer zones of the necrotic lesions and in the immediately adjacent viable lung tissue.

In cases with severe pleuritis and lung necrosis, the lesions are very similar to contagious bovine pleuropneumonia caused by *Mycoplasma mycoides* subsp *mycoides* SC and this similarity is of great concern in countries free of CBPP.[14] In two studies, experimental *M. bovis* infection produced similar clinical and pathological findings to that seen in naturally occurring *M. bovis* cases.[15-17]

M. bovis infection is usually introduced to *M. bovis*-free herds by clinically healthy carrier calves or young cattle shedding mycoplasmas. Once *Mycoplasma* infection is established, it is difficult to eradicate. In one study, *M. bovis* was detected in nasal swabs of 48.6% of a group of calves at at age 5 days and from 91.4% of the same calves at age 26 days, demonstrating the gradual spread of *M. bovis* infection.[18] Cattle infected with *M. bovis* shed the mycoplasma from the respi-

Figure 2: Multiple foci of caseous necrosis (arrow) and a large area of coagulative necrosis (star) in a lung section of *M. bovis* pneumonia in a feedlot calf.

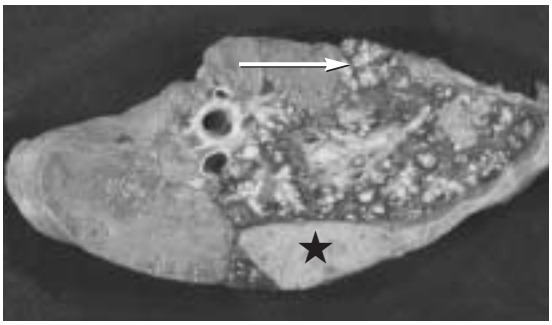
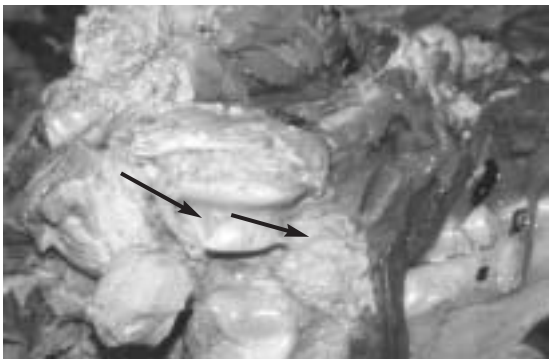


Figure 3: Stifle joint in a feedlot calf with severe *M. bovis* fibrinous arthritis. Note the fibrin masses (arrows) in the joint cavity.



ratory tract for many months and even years, acting as a reservoir of infection.[19] Co-infection with viral and bacterial organisms including *Mannheimia haemolytica, Pasteurella multocida, Haemophilus somnus,* and bovine virus diarrhea virus (BVDV), is commonly reported with *M. bovis* pneumonia[4,5] and a synergistic effect of these organisms with *M. bovis* has been reported.[20] Other concurrent viral, bacterial, and environmental factors play a role in BRD; however, some studies suggest that *M. bovis* is the predisposing factor leading to invasion from other bacterial pathogens by compromising the host immune system.[21,22] In feedlot cattle, primary BVDV infection and the associated immunosuppression may be potentially important in *M. bovis*-induced disease.

Other *Mycoplasma* species may be isolated from *M. bovis*-infected lungs (eg, *Mycoplasma bovirhinis, M. arginini, M. bovigenitalium, M. californicum, M. canis*). Although these mycoplasmas are not considered primary bovine respiratory pathogens, it is reported that under certain conditions they may become pathogenic.[23]

Low responsiveness to antimicrobial treatment of pneumonia and/or arthritis is typical of *M. bovis* infection in feedlot cattle.[4,24] Those antibiotics that are effective *in vitro* are often ineffective *in vivo.* Currently, despite many efforts, there are no vaccines available that induce complete protection against pneumonic strains of *M. bovis* in North America. However, there are reports of successful trials and immunogenic vaccines for *M. bovis* outside of North America.[8,25]

Arthritis

Mycoplasma bovis arthritis commonly occurs as a sequel to or concurrent with pneumonia or mastitis in cattle, and its occurrence depends on the level of infection challenge.[19] Polyarthritis caused by *M. bovis* in feedlot calves is a major cause of weight loss and culling of calves. Affected calves are reluctant to move or they walk with a stiff gait. As the disease progresses, the animals become recumbent. Arthritis usually affects multiple joints, mainly the femorotibial, radiocarpal, and tibiotarsal, but involvement of a single stifle joint is also very common. Affected joints are warm and swollen, and contain increased, opaque synovial fluid mixed with fibrinopurulant exudate (Figure 3). The infection commonly extends into tendon sheaths, bursae, and joint capsules, and is characterized by fibrinonecrotizing tenosynovitis.[3] Adjacent soft tissues, including skeletal muscle, are often involved as well. In affected animals, all these tissues should be examined for lesions and abnormalities.

Arthritis may occur without any obvious clinical signs of pulmonary disease.[26] The co-occurrence of respiratory disease and arthritis may suggest that *M. bovis* is able to enter the blood circulation from the lung and induce arthritis in cattle. Like pneumonia, other mycoplasmas may be isolated from infected joints and their significance is not clear. Submission of tissue samples for histology and bacteriology is helpful in diagnosing the disease. Pieces of affected synovial membrane and tendon sheaths are recommended for histopathology. Cultures of joint fluid and affected synovial membrane are used to isolate the causative organism. Treatment usually is ineffective, especially in chronic cases of mycoplasmal arthritis. Attempts to produce a protective vaccine against *M. bovis* arthritis have not been successful.

Mastitis

M. bovis was first isolated in 1962 from cases of severe mastitis in cattle in the USA.[2] *M. bovis* mastitis is a highly contagious and costly disease in the dairy industry.[27] Initially, affected mammary glands have a light brown secretion containing a flaky sediment. After a few days, the secretion becomes purulent and the gland edematous and tender. Infection can spread to other quarters.[27,28] The affected quarters appear enlarged with a firm texture. The cut surface is nodular and has a moderate gray-yellow discoloration. Two to 3 weeks after infection, the same quarters are clearly reduced in size, but still in firm consistency. Histologically, there is degeneration of the alveolar epithelium and an accumulation of highly homogenous, eosinophilic, purulent exudate within the mammary

ducts and intra-alveola 4 to 9 days postinfection. Between 2 to 3 weeks from infection, the mammary tissue has alveolar atrophy and interstitial infiltration of plasma cells, lymphocytes, and macrophages.[29] Formation of multiple abscesses has been reported in the chronic stages of the disease, suggesting the potential for intermittent shedding of the organism.[30] Contaminated milk from cases of subclinical mastitis is of great concern for the spread of mastitis and as a source of infection for calves. In one report, the feeding of waste milk containing *M. bovis* from mastitic cows to calves was suggested as the possible source of infection causing otitis media in dairy calves.[31] There is no treatment for mycoplasmal mastitis. Control of the disease by identifying the infected cows, segregating contaminated milk, and imposing routine hygienic procedures in herds are helpful measures.[30]

Other M. bovis-related diseases

Mycoplasma bovis infection can cause reproductive disorders in cows (metritis, salpangitis, reduced conception rates, and abortion) and bulls (seminal vesiculitis, epididymitis, and orchitis).[32,33] The authors detected mycoplasma antigen by immunohistochemistry from the placenta of an aborted bison.

Disease entities with features other than pneumonia, arthritis, and mastitis have been reported with *M. bovis* infection. Polyarthritis accompanied by meningitis, probably originating from intrauterine infection, was reported in 2 herds of 1- to 3-week-old dairy calves.[34] Decubital abscesses were reported in a herd of Holstein cattle. Affected calves did not have any joint lesions and the route of entry was not determined; however, rubbing the skin against a hard wooden floor contaminated with *M. bovis* or transmission of the organism from nasal discharge by licking the region was suggested.[35] During the last several years, the authors have detected *M. bovis* antigens in a variety of lesions including necrotizing myocarditis, necrotizing laryngitis and tracheitis, nephritis, panniculitis, vegetative endocarditis, and peritonitis. These findings may indicate the ability of this pathogen to circulate through the body via the bloodstream and localize in different tissues.

Laboratory analysis and detection of *M. bovis*

Isolation or detection of *M. bovis* in affected tissues or submitted samples is of critical importance in diagnosis, control, and possible treatment of disease. Several methods for detection of *M. bovis* are being used in diagnostic laboratories. Each method has advantages and disadvantages and each can be used for different purposes.

• Bacterial culture is conventionally used to detect mycoplasmas. Cultures of lung samples are considered the most effective method for isolating pulmonary pathogens, but are limited to fatal cases. Cultures are time-consuming and can be compromised by bacterial contamination of the sample; however, they have high specificity and sensitivity and they can be applied to a wide variety of clinical specimens.[36] Furthermore, other *Mycoplasma* species present in the same sample can also be isolated.

• Nasal swabs (NS) or bronchioalveolar lavage (BAL) are used in living animals and present interesting alternative techniques for defining pulmonary pathogens. In a recent study, the efficacy of these two sampling procedures in detecting pulmonary mycoplasmas in cattle was compared.[37] It was concluded that BAL is the best method for isolating *M. bovis* in cattle with respiratory disease, while NS was not representative of the mycoplasmas present in the lower respiratory airways. In another study, there was moderate agreement between NS and BAL at the group level, but not in the individual calf.[38]

• The polymerase chain reaction (PCR) technique is becoming a routine application in diagnostic laboratories. PCR is used to detect *M. bovis* in milk samples and the supernatant of cell cultures. This method is superior to other methods of detection in terms of sensitivity, specificity, and speed.[36] While it is undoubtedly a highly sensitive method, there is a detection limit for this method[36] and unlike immunohistochemistry, it is not possible to associate the organism with the lesion.

• In the Western College of Veterinary Medicine (WCVM)/Prairie Diagnostic Services diagnostic laboratory, immunohistochemistry is widely used for *M. bovis* detection in tissues processed for histology. It is a useful tool to detect and locate the antigen associated with lesions in affected tissues and has been used in several studies.[4,5] However, it is mainly a postmortem test and requires a few days for results to be available. Moreover, possible confounding effects of tissue fixation on antigens and possible cross-reactions with other antigens should be considered.

Pathogenesis of *M. bovis*

Mycoplasmas can have a close interaction with mammalian host cells for long periods without causing cytopathic effects. It has been suggested that the virulence of *M. bovis* is induced by its production of tumour necrosis factor-alpha (TNF-α),[39] specific cytoadhesive structures,[40-42] and a capability to evade the host's immune system by surface antigenic variation. Major lipoprotein antigen expression on the surface of *M. bovis* was shown to spontaneously undergo noncoordinated phase variation between "ON" and "OFF" expression states at a high frequency.[43,44] Differences in adhesion between pathogenic and nonpathogenic strains of *M. bovis* have been demonstrated.[45] *M. bovis* has been shown to be

immunosuppressive, affecting both cell-mediated and humoral immunity.[22,46] *M. bovis* can also induce apoptosis of bovine lymphocytes.[47]

Summary

Recent evidence indicates that *M. bovis* may be an emerging pathogen in cattle around the world. In feedlot cattle, a history of respiratory disease unresponsive to treatment and followed by polyarthritis is suggestive of *M. bovis* infection. The exact cause of the increased incidence and pathogenicity of *M. bovis* is not known; however, factors such as the elimination of other bacterial pathogens through use of potent antibiotics and vaccines, increased virulence of *M. bovis* strains, interactions with other pathogens (eg, BVDV), and management factors may be important. To evade the immune system, *M. bovis* is able to alter its surface antigens by variable surface protein gene rearrangement.

Acknowledgment

The authors wish to thank Drs. Elemir Simko and Dorothy M. Middleton for their advice on this manuscript.

Dr. Farshid Shahriar *obtained a Master of Veterinary Science (MVSci) in pathology from the Department of Veterinary Pathology at WCVM in 2000 and is currently enrolled in a Ph.D. program at the WCVM, University of Saskatchewan.*

Dr. Edward G. (Ted) Clark *obtained his DVM from the University of Guelph (OVC) in 1966. After 9 years in large animal practice, he obtained his MVSci in pathology from the Department of Veterinary Pathology at WCVM in 1977. He has been employed as a diagnostic pathologist at WCVM since that time. He is currently an employee of Prairie Diagnostic Services, Inc.*

References

1. Bryson DG. Calf pneumonia. *Vet Clin North Am Food Anim Pract* 1985;1:237-257.
2. Hale H, Helmbold CF, Plastridhe WN, Stula EF. Bovine mastitis caused by a *Mycoplasma* species. *Cornel Vet* 1962:582-591.
3. Adegboye DS, Halbur PG, Nutsch RG, Kadlec RG, Rosenbusch RF. *Mycoplasma bovis*-associated pneumonia and arthritis complicated with pyogranulomatous tenosynovitis in calves. *J Am Vet Med Assoc* 1996;209:647-649.
4. Haines DM, Martin KM, Clark EG, Jim GK, Janzen ED. The immunohistochemical detection of *Mycoplasma bovis* and bovine viral diarrhea virus in tissues of feedlot cattle with chronic, unresponsive respiratory disease and/or arthritis. *Can Vet J* 2001; 42:857-60.
5. Shahriar FM, Clark EG, Janzen E, West K, Wobeser G. Coinfection with bovine viral diarrhea virus and *Mycoplasma bovis* in feedlot cattle with chronic pneumonia. *Can Vet J* 2002; 43:863-868.
6. Taylor-Robinson D, Tully JG. *Mycoplasmas*, *Ureaplasmas*, *Spiroplasmas* and related organisms. In: Balows A, Duerden BI, eds. *Microbiology and Microbial Infections.* 9th ed. Vol. 2. London: Arnold; 1998:797-827.
7. ter Laak EA, Wentink GH, Zimmer GM. Increased prevalence of *Mycoplasma bovis* in the Netherlands. *Vet Q* 1992;14:100-104.
8. Nicholas RA, Ayling RD, Stipkovits LP. An experimental vaccine for calf pneumonia caused by *Mycoplasma bovis*: clinical, cultural, serological and pathological findings. *Vaccine* 2002;20:3569-75.
9. Kusiluka LJ, Ojeniyi B, Friis NF. Increasing prevalence of *Mycoplasma bovis* in Danish cattle. *Acta Vet Scand* 2000;41:139-146.
10. Brice N, Finlay D, Bryson DG, Henderson J, McConnell W, Ball HJ. Isolation of *Mycoplasma bovis* from cattle in Northern Ireland, 1993 to 1998. *Vet Rec* 2000;146:643-644.
11. Frey J. Mycoplasmas of animals. In: Razin S, Herrman R, eds. *Molecular biology and pathogenicity of mycoplasmas.* New York: Kluwer Academic/Plenum; 2002:73-90.
12. Kelly AP, Janzen ED. A review of morbidity and mortality rates and disease occurrence in North American feedlot cattle. *Can Vet J* 1986;27:496-500.
13. Rosengarten R, Citti C. The role of ruminant mycoplasmas in systemic infection. In: Stipkovits L, Rosengarten R, Frey J, eds. *Mycoplamas of ruminants: pathogenicity, diagnosis, epidemiology, and molecular genetics*. Vol. 3. Brussels: European Commission; 1999: 71-74.
14. Bashiruddin JB, De Santis P, Varga E, Stipkovits L. Confirmation of the presence of *Mycoplasma bovis* in Hungarian cattle with pneumonia resembling pleuropneumonia. *Vet Rec* 2001;148:743-746.
15. Rodriguez F, Bryson DG, Ball HJ, Forster F. Pathological and immunohistochemical studies of natural and experimental *Mycoplasma bovis* pneumonia in calves. *J Comp Pathol* 1996;115:151-162.
16. Linker H, Poumarat F, Le Grand D, et al. Pathological and immunohistochemical findings in calf lungs after experimental infection with a clonal variant derived from *Mycoplasma bovis* strain PG 45. In: Bergoneir D, Berthelot X, Frey J, eds. *Mycoplamas of ruminants: pathogenicity, diagnosis, epidemiology, and molecular genetics.* Vol. 4. Brussels: European Commission; 2000: 67-70.
17. Stipkovits L, Glavits R, Ripley P, Molnar T, Tenk M, Szeredi L. Pathological and immunohistochemical studies of pneumonia in calves experimentally induced by *Mycoplasma bovis*. In: Bergoneir D, Berthelot X, Frey J, eds. *Mycoplamas of ruminants: pathogenicity, diagnosis, epidemiology and molecular genetics.* Vol. 4. Brussels: European Commission; 2000:27-30.
18. Stipkovits L, Ripley P, Varga J, Palfi V. Clinical study of the disease of calves associated with *Mycoplasma bovis* infection. *Acta Vet Hung* 2000;48:387-395.
19. Pfutzner H, Sachse K. *Mycoplasma bovis* as an agent of mastitis, pneumonia, arthritis and genital disorders in cattle. *Rev Sci Tech* 1996;15:1477-1494.
20. Doherty ML, McElroy MC, Markey BK, Carter ME, Ball HJ. Isolation of *Mycoplasma bovis* from a calf imported into the Republic of Ireland. *Vet Rec* 1994;135:259-60.
21. Poumarat F, Le Grand D, Philippe S, et al. Efficacy of spectinomycin against *Mycoplasma bovis* induced pneumonia in conventionally reared calves. *Vet Microbiol* 2001;80:23-35.
22. Bennett RH, Jasper DE. Immunosuppression of humoral and cell-mediated responses in calves associated with inoculation of *Mycoplasma bovis*. *Am J Vet Res* 1977;38:1731-1738.
23. Hewicker-Trautwein M, Feldmann M, Kehler W, et al. Outbreak of pneumonia and arthritis in beef calves associated with *Mycoplasma bovis* and *Mycoplasma californicum*. *Vet Rec* 2002;151: 699-703.
24. Ayling RD, Baker SE, Peek ML, Simon AJ, Nicholas RA. Comparison of *in vitro* activity of danofloxacin, florfenicol, oxytetracycline, spectinomycin and tilmicosin against recent field isolates of *Mycoplasma bovis*. *Vet Rec* 2000;146:745-7.
25. Urbaneck D, Liebig F, Forbrig T, Stache B. Experiences with the use of herd-specific vaccines against respiratory infections with *Mycoplasma bovis* in a large cattle herd. *Praktische Tierarzt* 2000;81: 756-763.
26. Henderson JP, Ball HJ. Polyarthritis due to *Mycoplasma bovis* infection in adult dairy cattle in Northern Ireland. *Vet Rec* 1999; 145:374-376.
27. Cullor JS. The control, treatment, and prevention of the various types of bovine mastitis. *Vet Med* 1993;88:571-579.
28. Jasper DE. *Mycoplasma* and *mycoplasma* mastitis. *J Am Vet Med Assoc* 1977;170:1167-1172.

29. Seffner W, Pfutzner H. Untersuchungen zur Mykoplasmenmastitis des Rindes. 8. Mitteilung: Pathologische Anatomie und Histologie der experimentellen *Mycoplasma bovis* mastitis. *Arch Exp Veterinarmed* 1980; 34:817-26.
30. Gonzalez RN, Wilson DJ. Mycoplasmal mastitis in dairy herds. *Vet Clin North Am Food Anim Pract* 2003;19:199-221.
31. Walz PH, Mullaney TP, Render JA, Walker RD, Mosser T, Baker JC. Otitis media in preweaned Holstein dairy calves in Michigan due to *Mycoplasma bovis*. *J Vet Diagn Invest* 1997;9:250-254.
32. Byrne WJ, Brennan P, McCormack R, Ball HJ. Isolation of *Mycoplasma bovis* from the abomasal contents of an aborted bovine fetus. *Vet Rec* 1999;144:211-212.
33. Hirth RS, Nielsen SW, Plastridge WN. Bovine salpingo-oophoritis produced with semen containing a *Mycoplasma*. *Path Vet* 1966;3:616-32.
34. Stipkovits L, Rady M, Glavits R. Mycoplasmal arthritis and meningitis in calves. *Acta Vet Hung* 1993;41(1-2):73-88.
35. Kinde H, Daft BM, Walker RL, Charlton BR, Petty CR. *Mycoplasma bovis* associated with decubital abscesses in Holstein calves. *J Vet Diagn Invest* 1993;5:194-197.
36. Sachse K, Pfutzner H, Hotzel H, Demuth B, Heller M, Berthold E. Comparison of various diagnostic methods for the detection of *Mycoplasma bovis*. *Rev Sci Tech* 1993;12:571-580.
37. Thomas A, Dizier I, Trolin A, Mainil J, Linden A. Comparison of sampling procedures for isolating pulmonary mycoplasmas in cattle. *Vet Res Commun* 2002;26:333-9.
38. Allen JW, Viel L, Bateman KG, Rosendal S, Shewen PE, Physick-Sheard P. The microbial flora of the respiratory tract in feedlot calves: associations between nasopharyngeal and bronchoalveolar lavage cultures. *Can J Vet Res* 1991;55:341-346.
39. Jungi TW, Krampe M, Sileghem M, Groit C, Nicolet J. Differential and strain-specific triggering of bovine alveolar macrophage effector functions by mycoplasmas. *Microb Pathog* 1996;21:487-498.
40. Sachse K, Helbig JH, Lysnyansky I, et al. Epitope mapping of immunogenic and adhesive structures in repetitive domains of *Mycoplasma bovis* variable surface lipoproteins. *Infect Immun* 2000;68:680-7.
41. Sachse K, Pfutzner H, Heller M, Hanel I. Inhibition of *Mycoplasma bovis* cytadherence by a monoclonal antibody and various carbohydrate substances. *Vet Microbiol* 1993;36(3-4):307-16.
42. Sachse K, Grajetzki C, Rosengarten R, Hanel I, Heller M, Pfutzner H. Mechanisms and factors involved in *Mycoplasma bovis* adhesion to host cells. *Zentralbl Bakteriol* 1996;284:80-92.
43. Behrens A, Heller M, Kirchhoff H, Yogev D, Rosengarten R. A family of phase- and size-variant membrane surface lipoprotein antigens (Vsps) of *Mycoplasma bovis*. *Infect Immun* 1994;62:5075-5084.
44. Lysnyansky I, Rosengarten R, Yogev D. Phenotypic switching of variable surface lipoproteins in *Mycoplasma bovis* involves high-frequency chromosomal rearrangements. *J Bacteriol* 1996;178:5395-5401.
45. Thomas A, Sachse K, Dizier I, et al. Adherence to various host cell lines of *Mycoplasma bovis* strains differing in pathogenic and cultural features. *Vet Microbiol* 2003;91(2-3):101-113.
46. Boothby JT, Jasper DE, Zinkl JG, Thomas CB, Dellinger JD. Prevalence of mycoplasmas and immune responses to *Mycoplasma bovis* in feedlot calves. *Am J Vet Res* 1983;44:831-838.
47. Vanden Bush TJ, Rosenbusch RF. *Mycoplasma bovis* induces apoptosis of bovine lymphocytes. *FEMS Immunol Med Microbiol* 2002;32:97-103.

Do you have a review article suitable for large animal practitioners? Please contact the editor by E-mail, Jon.Naylor@usask.ca for publication and remuneration details.

Abstract of Interest

Mycoplasma bovis: disease, diagnosis, and control.

NICHOLAS RA, AYLING RD.

Mycoplasma bovis is a major, but often overlooked, pathogen causing respiratory disease, mastitis and arthritis in cattle. It is found worldwide and has spread into new areas, including Ireland and parts of South America, in the last decade. In Europe, it is responsible for at least a quarter to a third of all calf pneumonia although this may be an underestimate as few laboratories regularly monitor for mycoplasmas. Like all Mollicutes, *M. bovis* is inherently refractory to certain groups of antibiotics because it does not possess a cell wall; furthermore, evidence is accumulating that strains of *M. bovis* are becoming resistant to antibiotics, including tetracycline, tilmicosin and spectinomycin, traditionally used for their control. No vaccines are presently available for the control of *M. bovis* infections. *Res Vet Sci* 2003;74(2):105-112.

Upcoming Meetings

1-2 November 2003 (11-12 CE Hrs.)
2003 Atlantic Veterinary College Fall Conference
Charlottetown, Prince Edward Island, Canada.
Animal Welfare, Companion Animal, Food Animal and Equine topics.
CONTACT: Susan Stensch, Atlantic Veterinary College
Tel.: (902)566-0780
Email: sstensch@upei.ca
Website: www.upei.ca/-avc/conted.html

2-4 November 2003
From the Farm Gate to the Dinner Plate – Conference on Food Safety Issues and Scientific info about advances in agricultural technologies and products
Westin Hotel, Edmonton, Alberta
CONTACT: Agricultural Institute of Canada Foundation
Tel.: 1-800-868-8776 or (613)732-7068
Fax: (613)732-3386
Email: profedge@renc.igs.net

21-25 November 2003
49th Annual Convention
American Association of Equine Practitioners
New Orleans, LA
CONTACT: AAEP
Tel.: (859)233-0147 or toll-free 800-443-0177
Website: www.aaep.org

9-12 July 2004
23rd World Buiatrics Congress
Montreal, Quebec
CONTACT: www.wbc2004.ca

Change of address notices and requests for subscriptions to *Large Animal Veterinary Rounds* are to be sent by mail to P.O. Box 310, Station H, Montreal, Quebec H3G 2K8 or by fax to (514) 932-5114 or by e-mail to info@snellmedical.com. Please reference *Large Animal Veterinary Rounds* in your correspondence. Undeliverable copies are to be sent to the address above.

This publication is made possible by an educational grant from

Schering-Plough Animal Health

117-024R

SNELL