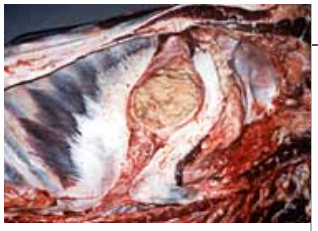

SANTÉ ANIMALE FICHES MALADIES

Contagious Caprine Pleuropneumonia

Names

Pathogen(s)

Preferred Name: Mycoplasma capricolum subsp. capripneumoniae.

Disease/Parasitosis:
Other Names Used: Mycoplasma strain F38

Disease/Parasitosis Names
Preferred Name : Contagious caprine pleuropneumonia

Acronyms (English): CCPP

Overview

The causative agent of contagious caprine pleuropneumonia (CCPP) is Mycoplasma capricolum subsp. capripneumoniae (Mccp), which was previously known by the strain name of its type species, F38. It is a member of the Mycoplasma mycoides cluster which includes M. mycoides subsp. mycoides SC (MmmSC), M. mycoides subsp. mycoides LC (MmmLC), M. mycoides subsp. capri (Mmc), M. capricolum subsp. capricolum (Mcca) and Mycoplasma species bovine group 7 (Bg7), an uncharacterized bovine isolate, which causes other diseases of ruminants. CCPP is a significant disease of goats in Africa, the Middle East and Western Asia, and is characterized primarily by its contagious nature. The disease causes interstitial, fibrinous pleuropneumonia, interlobular oedema and hepatization of the lung causing high mortality rates of up to 80%.

Definite diagnosis is made by culture of the causative agent from lung samples or pleuritic fluid taken at postmortem. Liquid and solid mycoplasma media are inoculated and filtered subcultures from liquid medium may be required if there is evidence of bacterial contamination. Isolates may be identified by biochemical, immunological and molecular tests. Serological tests for the detection of specific antibodies have relied on the complement fixation test which is the test prescribed by the Office International des Epizooties (OIE) for international trade. A latex agglutination test is also available, and is used routinely in some parts of Africa.

History
CCPP is an infectious disease which affects only goats, and was first described in the late 19th century (Hutcheon, 1889; McMartin et al., 1980). Before the isolation and identification of Mycoplasma strain F38 by MacOwan (1976), and the subsequent demonstration of its causal relationship with CCPP (MacOwan and Minette, 1976), M. mycoides subsp. capri was considered to be the aetiological agent of CCPP (Edward, 1953; Jonas and Barber, 1969). So far M. capricolum subsp. capripneumoniae is the only mycoplasma that fulfils KochÇs postulates for CCPP and is believed to be the sole cause of CCPP (MacOwan, 1984). Mycoplasma strain F38 has recently been reclassified and now all F38-like mycoplasmas are known as Mycoplasma capricolum subsp. capripneumoniae (Leach et al., 1993).

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Animals Affected

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Medline

CCPP has been reported to affect only goats (Thiaucourt and Bàlske, 1996) and it does not cause disease in sheep, neither spontaneously nor experimentally (McMartin et al., 1980). However, there are some reports describing the isolation of M. capricolum subsp. capripneumoniae from healthy sheep in Kenya that have been in contact with goat herds affected by CCPP (Litamoi et al., 1990), and from sick sheep mixed with goats in Uganda suffering from the disease (Bàlske et al., 1995). The isolation of M. capricolum subsp. capripneumoniae from cattle with mastitis has also been reported (Kumar and Garg, 1991), and these reports confound the perceived host specificity of M. capricolum subsp. capripneumoniae.

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Epidemiology

The most important distinguishing features of CCPP, with respect to the other goat respiratory mycoplasmoses, were defined by Hutcheon and are quoted as follows: (1) the disease is readily contagious to susceptible goats; (2) sheep and cattle are not affected; (3) local oedematous reactions do not occur in goats when infective inoculum is given subcutaneously (Hutcheon, 1889). In natural infections, the organisms are acquired by susceptible goats by inhalation of contaminated droplets from infected goats (MacOwan, 1984). The environment as a whole plays an important role in the appearance, evolution and severity of CCPP. Due to the high sensitivity of mycoplasmas to the external environment, close contact is essential between infected and naive animals for transmission to take place, and, overcrowding and confinement favours close contact and circulation of mycoplasmas. Stress factors due to malnutrition and movement over long distances can predispose the animal to disease.

In Africa where extensive and traditional husbandry is practised, pathogens spread when animals meet at watering points and grazing areas. Breed and sex appear not to affect the epidemiology of CCPP, but age is an important factor. Though all age groups are susceptible, mortality is higher among young animals than adults. Infective M. capricolum subsp. capripneumoniae may persist in chronic, latent carriers, such as goats or sheep which have recovered from infection without becoming bacteriologically sterile, and are considered to be responsible for the perpetuation of the disease in a herd (Thiaucourt and Bàlske, 1996; Wesonga et al., 1998). This aspect of the epidemiology was described as early as 1881 by Hutcheon (McMartin et al., 1980) in the case of CCPP in South Africa (LefÂvre et al., 1987b). Viruses are important factors that predispose lung tissue to invasion by mycoplasmas. In Africa the virus, peste des petits ruminants (PPR) and capripox viruses are important (Lefåvre et al., 1987b).

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Distribution Table

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Distribution

Although a precise description of the distribution of CCPP is not available, the clinical disease has been reported in 30 countries mainly in Africa and Asia (Thiaucourt and Bàlske, 1996). The only African countries where M. capricolum subsp. capripneumoniae has been isolated are Chad (Lefåvere et al., 1987a), Eritrea, Ethiopia (Thiaucourt et al., 1992), Kenya (MacOwan and Minette, 1976), Niger, Sudan (Harbi and El-Tahir, 1981), Tanzania (Kusiluka et al., 2000), Tunisia (Perreau et al., 1984), and Uganda (Bàlske et al., 1995). In Asia and the Mediterranean, isolations have been reported in Oman (Jones and Wood, 1988), Turkey (Jones and Wood, 1988), the United Arab Emirates, and Yemen (Rurangirwa et al., 1987b). In Mali, goats have been suspected of infection based on serological evidence (Rurangirwa et al., 1990).

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Economic Importance and Impact

Goats are important commodities to a large segment of the worldÇs population as a source of meat, milk, and skin. CCPP is a disease of major economic importance in Africa and Asia, posing a major constraint to goat production. The direct losses of the disease result from its high mortality, reduced milk and meat yield, cost of treatment, control, disease diagnosis and surveillance. In addition to this, there are indirect losses due to the imposition of trade restrictions.

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Disease Systems

Respiratory Small Ruminants

Disease Course

Acute cases can be observed in regions where CCPP is introduced for the first time into naive populations. Particularly in animals with primary infection, the illness lasts for about two days and death ensues, while in other cases it may last several days. The primary clinical signs are cough with animals tending to lie down or lag behind the flock. Affected animals continue to graze for some time but eventually become anorexic, breathing becomes laboured with painful grunting and a rise in temperature up to 41ŠC. Gradually, the respiratory symptoms become prominent, respiration is accelerated and painful, and is followed by violent coughing (McMartin et al., 1980; Thiaucourt and Bàlske, 1996).

In the terminal stages, the animals are unable to move. They stand with their legs abducted, the neck is stiff and extended downward, saliva continuously drips from their mouth and their nose is obstructed by mucopurulent discharge. The tongue protrudes and the animals bleat distressingly. In fully susceptible flocks that encounter an outbreak, morbidity is usually 100% and mortality is up to 70% (McMartin et al., 1980). The organism is not reported to affect organ systems other than the respiratory tract of goats. In endemic areas subacute and chronic cases are common and the symptoms are milder, dominated by intermittent coughing.

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Pathology

The gross pathological lesions are localized exclusively to lung and pleura and are often unilateral. Affected lungs can be totally hepatized, and have a port wine colour (Thiaucourt and Bàlske, 1996). A lung section shows a fine granular texture with various colours, but usually without any thickening of the interlobular septa. There is often an abundant pleural exudate and conspicuous pleuritis. The pleural exudates can solidify and form a gelatinous covering sometimes over the whole lung. In acute cases, the pleural cavity contains an excess of straw-coloured fluid with fibrin flocculations (Kaliner and MacOwan, 1976; Wesonga et al., 1993). In chronic cases there is a black discolouration of the lung tissue and sequestration of the necrotic lung areas. Adhesions between the lung and the pleura are very common and often very thick (MacOwan and Minette, 1977).

Histological examination of the lung tissues may show acute serofibrinous to chronic fibrino-necrotic pleuropneumonia with infiltrates of serofibrinous fluid and inflammatory cells, mainly neutrophils, in the alveoli, bronchioles, interstitial septae and subpleural connective tissue. Intralobular oedema is more prominent but interlobular oedema has also been reported. Peribronchial and perbronchiolar lymphoid hyperplasia with mononuclear cell infiltration is also present (MacOwan and Minette, 1976; Kibor, 1990; Wesonga et al., 1998; Msami et al., 1998).

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Symptoms Table

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Diagnosis

In the field, diagnosis of mycoplasma pneumonia can not be established on clinical signs or on postmortem examinations alone. In outbreaks of classical acute CCPP, the high mortality and typical early thoracic lesions in goats are highly indicative of M. capricolum subsp. capripneumoniae infection, but all cases of caprine mycoplasmosis need additional laboratory tests to establish a presumptive diagnosis. It may be difficult to distinguish CCPP from an infection by M. mycoides subsp. mycoides LC or M. mycoides subsp. mycoides SC, which have a pulmonary location. In the case of M. mycoides subsp. mycoides LC infection, thickening of the interlobular septa may be evident. These lesions are similar to those observed in the case of CBPP. Sometimes the thickening is absent or inconspicuous and laboratory confirmation is needed. Recently, sequestra in the lungs of goats infected with M. mycoides subsp. mycoides SC have been described (Kusiluka et al., 2000).

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Growth, Isolation and Transport Media

Definite diagnosis is made by the isolation of M. capricolum subsp. capripneumoniae from clinical samples, usually lung tissue and may be a long and difficult process. The success of isolation depends primarily on the attention that is given to sample collection. The main difficulties in isolating M. capricolum subsp. capripneumoniae is that it grows very poorly in vitro and samples are often contaminated by other mycoplasmas (Freundt, 1983a; Thiaucourt et al., 1996) which are generally faster growing and overgrow M. capricolum subsp. capripneumoniae. In addition to this the frequent use of antibiotic therapy has impaired the cultivation of these mycoplasmas from clinical material. An immunobinding assay that detects M. capricolum subsp. capripneumoniae in pleural fluids that overcomes some of these problems has been described (Guerin et al., 1993). Liquid medium and a solid agar medium which allows the presumptive identification of M. capricolum subsp. capripneumoniae by the production of coloured colonies is available commercially (Bashiruddin and Windsor, 1998). An antigen detection system using latex coated antibodies has also been described (March et al., 2000).

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Biochemical Tests

Only a limited number of biochemical tests perform a useful function as a preliminary screening system and are based on specific enzyme activities or nutritional capabilities. For instance, digitonin sensitivity distinguishes mycoplasmas from acholeplasmas, and serum digestion differentiates members of the M. mycoides cluster from all other small ruminant mycoplasmas (Freundt, 1983b). Also, phosphatase production separates M.capricolum subsp. capricolum from other members of the cluster (Bradbury, 1983). Substantial metabolic differences between M.capricolum subsp. capricolum and M. capricolum subsp. capripneumoniae exist, but differences in glucose metabolism were described between strains of M. capricolum subsp. capripneumoniae (Abu-Groun et al., 1994). These tests can not differentiate M. capricolum subsp. capripneumoniae from all members of M. mycoides cluster (Bàlske, 1995). The interspecies variation in some biochemical reactions is often considerable, rendering their application valueless (Jones, 1989; Rurangirwa, 1996).

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Growth Inhibition Test

The growth inhibition (GI) test is the simplest and most specific, but the least sensitive of the tests available. It depends on the direct inhibition of mycoplasma growth on solid media by specific hyperimmune serum, and detects primary surface antigens (Dighero et al., 1970). The GI test is particularly useful in identifying M. capricolum subsp. capripneumoniae because they appear to be serologically homogeneous, and antiserum to the type strain produces wide inhibition zones free of çbreakthroughÇ colonies against field isolates from diverse sources. M. capricolum subsp. capripneumoniae cross-reacts with LeachÇs Bg7, M. equigenitalium and M. primatum in the GI test, but since these cross-reactive species do not occur in goats, they present no difficulties when identifying field isolates. However, a small proportion of M. capricolum subsp. capripneumoniae isolates also cross-react in the GI test with antiserum to M.capricolum subsp. capricolum which may confuse the identification of field isolates. A monoclonal antibody has been produced which specifically inhibits the growth of M. capricolum subsp. capripneumoniae but not of other members of the M. mycoides cluster (Rurangirwa et al., 1987c). It was later demonstrated that this monoclonal antibody was not specific. Cross-reactions with some strains of Bg7 were observed with the GI test and with a strain of M.capricolum subsp. capricolum in the immunofluorescence test (Belton et al., 1994).

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Fluorescent Antibody Test
The direct and indirect fluorescent antibody tests are one of the most effective, simple and rapid serological methods of identification for most mycoplasma (Rosendal and Black, 1972). Several forms have been described, the most commonly used one is the indirect fluorescent antibody (IFA) test which is applied to unfixed colonies on agar.

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Serological Tests
The complement fixation test (CFT) was used for the detection of CCPP infection (MacOwan, 1976; MacOwan and Minette, 1977), and it was found to be more specific, though less sensitive, than the indirect haemagglutination test (Muthomi and Rurangirwa, 1983). The latex agglutination test uses latex beads sensitized with the polysaccharide produced by M. capricolum subsp. capripneumoniae in culture supernatant in a slide agglutination test (Rurangirwa et al., 1987a). The use of the more defined antigen such as the polysaccharide provides greater sensitivity without cross-reactivity with sera against the other three principal caprine mycoplasmas. The indirect haemagglutination test (IHA) (Cho et al., 1976) has been used for the diagnosis of CCPP (Muthomi and Rurangirwa, 1983). The specificity of IHA test for the M. mycoides cluster has been evaluated and results were found to show cross-reactivity between these organisms (Jones and Wood, 1988). An indirect immunosorbent assay (ELISA) was developed to screen goat sera at a single dilution of antibody to M. capricolum subsp. capripneumoniae (Wamwayi et al., 1989). Some problems due to cross-reactions from other members of the M. mycoides cluster were encountered, but in spite of these, ELISA was more sensitive than CFT in detecting antibodies in serum. More recently, a competition ELISA (c-ELISA) was developed which permitted the specific detection of antibodies in sera from animals affected by CCPP (Thiaucourt et al., 1994). Analysis of field sera showed that seroconversion did not occur in all animals, whatever test was used. The percentage positive animals in affected herds varied between 30 and 60% with this test. The test was therefore unsuitable as an individual screening test (Thiaucourt et al., 1996).

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Molecular Diagnosis
Until recently, isolation was the only way to confirm the presence of CCPP. A DNA probe which differentiates M. capricolum subsp. capripneumoniae from other members of the M. mycoides cluster was developed (Taylor et al., 1992). Diagnostic systems based on PCR have been developed for the rapid detection, identification and differentiation of members of the M. mycoides cluster and the specific identification of M. capricolum subsp. capripneumoniae (Bashiruddin et al., 1994; Hotzel et al., 1996). The sequence of the gene for 16S ribosomal RNA has also been used to develop a PCR-based test where the final identification of M. capricolum subsp. capripneumoniae is made dependant on the pattern of the products after digestion of the PCR product with the restriction enzyme Pst1 (Bascu“ana et al., 1994; Bàlske et al., 1996).

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Disease Treatment Table

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Disease Treatment

The macrolides (erythromycin, spiramysin, and tylosin), tetracyclines, quinolones and chloramphenicol are active against M. capricolum subsp. capripneumoniae. Tylosin, tetracyclin, tiamulin or streptomycin are recommended (Hassan et al., 1984; Onovarian, 1974) but their success depends on early intervention and treatment.

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Disease Prevention and Control

An experimental vaccine inactivated with saponin that protects goats for approximately a year has been produced in Kenya (Rurangirwa et al., 1984).

Movement restrictions and slaughtering infected animals is recommended for countries that are newly infected.

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Vaccines

No data

Zoonoses and Food Safety

CCPP is not a zoonotic disease.

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References

Abu-Groun EAM, Taylor RR, Varsani H, Wadher BJ, Leach RH, Miles RJ, 1994. Biochemical diversity within the 'Mycoplasma mycoides cluster'. Microbiology (Reading), 140(8):2033-2042; 30 ref.

Bascu“ana CR, Mattsson JG, Bàlske G, Johansson K-E, 1994. Characterisation of the 16S rRNA genes from Mycoplasma sp. strain F38 and development of an identificaion system based on PCR. Journal of Bacteriology, 176:2577-2586.

Bashiruddin JB, Taylor TK, Gould AR, 1994. A PCR-based test for the specific identification of Mycoplasma mycoides subspecies mycoides SC. Journal of Veterinary Diagnostic Investigation, 6 (4):428-434; 14 ref.

Bashiruddin JB, Windsor GD, 1998. Coloured colonies of Mycoplasma mycoides subsp. mycoides SC and Mycoplasma capricolum subsp. capripneumoniae on solid agar media for the presumptive diagnosis of CBPP and CCPP. In:Proceedings of the ARC-Onderstepoort OIE International congress with WHO-cosponsorship on Anthrax, Brucellosis, CBPP, Clostridial and Mycobacterial diseases. 9-15, August 1998, Kruger National Park, South Africa, 226-229.

Belton D, Leach RH, Mitchelmore DL, Rurangirwa FR, 1994. Serological specificity of a monoclonal antibody to Mycoplasma capricolum strain F38, the agent of contagious caprine pleuropneumonia. Veterinary Record, 134(25):643-646; 21 ref.

Bloch N, Diallo I, 1991. Serological survey on sheep and goats in four departments of Niger. Revue d'ålevage et de Mådecine Våtårinaire des Pays Tropicaux, 44(4):397-404; 9 ref.

Bàlske G, 1995. Respiratory mycoplasmoses in goats: especially with regard to contagious caprine pleuropneumonia. PhD. Thesis, Uppsala, Sweden.

Bàlske G, Johansson KE, Heinonen R, Panvuga PA, Twinamasiko E, 1995. Contagious caprine pleuropneumonia in Uganda and isolation of Mycoplasma capricolum subspecies capripneumoniae from goats and sheep. Veterinary Record, 137(23):594; 7 ref.

Bàlske G, Mattsson JG, Bascun^macron~ana CR, Bergstràm K, Wesonga H, Johansson KE, 1996. Diagnosis of contagious caprine pleuropneumonia by detection and identification of Mycoplasma capricolum subsp. capripneumoniae by PCR and restriction enzyme analysis. Journal of Clinical Microbiology, 34(4):785-791; 41 ref.

Bradbury JM, 1983. Phosphatase activity. In: Razin S, Tully JG, eds. Methods in Mycoplasmology Vol. 1. Mycoplasma characterization. New York, USA: Academic Press, 363-366.

Cho HJ, Ruhnke HL, Langford EV, 1976. The indirect haemagglutination test for detection of antibodies in cattle narurally infected with mycoplasmas. Canadian Journal of Comparative Medicine, 40:20-29.

Cottew GS, Brerard A, DaMassa AJ et al., 1987. Taxonomy of the Mycoplasma mycoides cluster. Israel Journal of Medical Sciences, 23:632-635.

Dighero MW, Bradstreet PCM, Andrews BE, 1970. Dried paper discs for serological identification of human mycoplasmas. Journal of Applied Bacteriology, 33:750-757.

Edward DGF, 1953. Organisms of the pleuropneumonia group causing disease in goats. Veterinary Record, 65:873-875.

Freundt EA, 1983. Culture media for classic mycoplasmas. In: Razin S, Tully JG, eds. Methods in Mycoplasmology Vol. 1. Mycoplasma characterization. New York, USA: Academic Press, 127-135.

Freundt EA, 1983. Preteolytic activity. In: Razin S, Tully JG, eds. Methods in Mycoplasmology, Vol. 1. Mycoplasma characterization. New York, USA: Academic Press, 367-371.

Guerin C, Thiaucourt F, Mady V, Breard A, Lefevre PC, 1993. Rapid diagnosis of contagious caprine pleuropneumonia in pleural fluids by immunobinding assay. Small Ruminant Research, 12 (2):193-200; 33 ref.

Harbi MSMA, El-Tahir MS, 1981. Mycoplasma strain F38 and contagious caprine pleuropneumonia in the Sudan. Veterinary Record, 108:261.

Hassan SMel, Harbi MSMA, Bakr MIA, 1984. Treatment of contagious caprine pleuropneumonia. Veterinary Research Communications, 8(1):65-67; 7 ref.

Heldtander M, Wesonga H, Bàlske G et al., 2001. Genetic diversity and evolution of Mycoplasma capricolum subsp. capripneumoniae strains from eastern Africa assessed by 16S rDNA sequence analysis. Veterinary Microbiology, 78:13-28.

Hotzel H, Sachse K, Pf•tzner H, 1996. A PCR scheme for differentiation of organisms belonging to the Mycoplasma mycoides cluster. Veterinary Microbiology, 49(1/2):31-43; 21 ref.

Hutcheon D, 1889. Contagious pleuro-pneumonia in goats at Cape Colony, South Africa. Veterinary Journal, 29:399-404.

Jonas AM, Barber TI, 1969. Mycoplasma mycoides var. capri isolated from a goat in Connecticut. Journal of Infectious Diseases, 119:126-131.

Jones GE, 1983. Mycoplasmas of sheep and goats: a synopsis. Veterinary Record, 113(26/27):619-620; 11 ref.

Jones GE, 1989. Contagious caprine pleuropneumonia. Technical Series - Office International des Epizooties, No. 9:63 pp.; 132 ref.

Jones GE, Wood AR, 1988. Microbiological and serological studies on caprine pneumonias in Oman. Research in Veterinary Science, 44(1):125-131; 45 ref.

Kaliner G, MacOwan KJ, 1976. The pathology of experimental and natural contagious caprine pleuropneumonia in Kenya. Zentrablat Veterinary Medicine B, 23:652-661.

Kibor AC, 1990. Methods for the laboratory diagnosis of cantagious carprine pleuropneumonia (CCPP). In: Alton GG, Carter GR, Kibor AC, Pesti L, eds. Veterinary Diagnostic Microbiology. A manual of laboratory procedures for selected diseases of livestock. Rome, Italy: Food and Agricultural Organisation of the United Nations, 169-200.

Kokotovic B, Bàlske G, Ahrens P, Johansson K-E, 2000. Genomic variations of Mycoplasma capricolum subsp. capripneumoniae detected by amplified fragment length polymorphism (AFLP) analysis. FEMS Microbiology Letters, 184:63-68.

Kumar A, Garg DN, 1991. Isolation of mycoplasma F-38 from the milk of mastitic cows. Veterinary Record, 128(18):429; 12 ref.

Kusiluka LJM, Semuguruka WD, Kazwala RR et al., 2000. Demonstration of Mycoplasma capricolum subsp. capripneumoniae and Mycoplasma mycoides subsp. mycoides, small colony type in outbreaks of caprine pleuropneumonia in eastern Tanzania. Acta Veterinaria Scandinavica, 41:331-319.

Leach RH, Ernñ H, MacOwan KJ, 1993. Proposal for designation of F38-type caprine mycoplasmas as Mycoplasma capricolum subsp. capripneumoniae subsp. nov. and consequent obligatory relegation of strains currently classified as M. capricolum (Tully, Braile, Edward, Theodore and Ernñ 1974) to an additional new subspecies M. capricolum subsp. capricolum subsp. nov. International Journal of Systematic Bacteriology, 43:603-605.

Lefevre PC, Breard A, Farouk Ial, Buron S, 1987. Mycoplasma species F38 isolated in Chad. Veterinary Record, 121(24):575-576; 2 ref.

LefÀvre PC, Jones GE, Ojo MO, 1987. Pulmonary mycoplasmoses of small ruminants. Revue Scientifique et Technique, Office International des åpizooties, 6(3):713-757, 759-799; 117 ref.

Litamoi JK, Wanyangu SW, Siman PK, 1990. Isolation of Mycoplasma biotype F38 from sheep in Kenya. Tropical Animal Health and Production, 22(4):260-262; 11 ref.

MacOwan KJ, 1976. A mycoplasma from chronic caprine pleuropneumonia in Kenya. Tropical Animal Health Production, 8:28-36.

MacOwan KJ, 1984. Role of mycoplasma strain F38 in contagious caprine pleuropneumonia. Israel Journal of Medical Sciences, 20 (10):979-981; [Contribution to the 5th International Mycoplasmology Conference]; 22 ref.

MacOwan KJ, Minette JE, 1976. A mycoplasma from acute contagious caprine pleuropneumonia in Kenya. Tropical Animal Health Production, 8:91-95.

MacOwan KJ, Minette JE, 1977. The role of Mycoplasma strain F38 in contagious caprine pleuropneumonia (CCPP) in Kenya. Veterinary Record, 101:380-381.

March JB, Gammack C, Nicholas R, 2000. Rapid detection of contagious caprine pleuropneumonia using a Mycoplasma capricolum subsp. capripneumoniae capsular polysaccharide-specific antigen detection latex agglutination test. Journal of Clinical Microbiology, 38:4152-4159.

McMartin DA, MacOwan KJ, Swift LL, 1980. A century of classical contagious pleuropneumonia:from original description to aetiology. British Veterinary Journal, 136:507-515.

Msami HM, Kapaga AM, Bàlske G et al., 1998. Occurance of contagious caprine pleuropneumonia in Tanzania. Tanzania Veterinary Journal, 18:285- 297.

Muthomi EK, Rurangirwa FR, 1983. Passive haemagglutination and complement-fixation as diagnostic tests for contagious caprine pleuropneumonia caused by F-38 strain of mycoplasma. Research in Veterinary Science, 35:1-4.

Onovarian O, 1974. The comparative efficacy of some antibiotics used to treat experimentally induced mycoplasma infection in goats. Veterinary Record, 94:418-420.

Perreau P, Breard A, Goff C le, 1984. Experimental infection of goats with type F.38 mycoplasma strains (CCPP). Annales de Microbiologie, 135A(1):119-124.

Pettersson B, Leitner T, Ronaghi M et al., 1996. Phylogeny of the Mycoplasma mycoides cluster as determined by sequence analysis of 16S rRNA genes from the two rRNA operons. Journal of Bacteriology, 178:4131-4142.

Pettersson B, Uhlån M, Johansson KE, 1996. Phylogeny of some mycoplasmas from ruminants based on 16S rRNA sequences and definition of a new cluster within the hominis Group. International Journal of Systematic Bacteriology, 46(4):1093-1098; 24 ref.

Pettersson B, Bàlske G, Thiaucourt F et al., 1998. Molecular evolution based on Mycoplasma capricolum subsp. capripneumoniae strains, based on polymorphisms in the 16 rRNA genes. Journal of Bacteriology, 180:2350-2358.

Rodwell AW, 1982. The protein fingerprints of mycoplasmas. Review of Infectious Diseases, Supplement 4:8-17.

Rosenbusch RF, Minion FC, 1992. Cell envelope:Morphology and Biochemistry. In: Maniloff J, McElhaney RN, Finch LR Baseman JB, eds. Molecular Biology and Pathogenesis. Washington DC, USA: American Society for Microbiology, 73-77.

Rosendal S, Black FT, 1972. Direct and indirect immunofluorescence of unfixed and fixed mycoplasma colonies. Acta Pathologica et Microbiologica Scandinavica, 80:615-622.

Rurangirwa FR, 1996. Contagious caprine pleuropneumonia. In: Manual of Standards for Diagnostic Tests and Vaccines. Office International des Epizooties, 374-383.

Rurangirwa FR, Masiga WN, Muthomi EK, 1984. Immunisation of goats against contagious caprine pleuropneumonia using

sonicated antigens of F-38 strain of mycoplasma. Research in Veterinary Science, 36(2):174-176; 9 ref.

Rurangirwa FR, McGuire TC, Magnuson NS, Kibor A, Chema S, 1987. Composition of a polysaccharide from mycoplasma (F-38) recognised by antibodies from goats with contagious pleuropneumonia. Research in Veterinary Science, 42(2):175-178; 16 ref.

Rurangirwa FR, McGuire TC, Kibor A, Chema S, 1987. A latex agglutination test for field diagnosis of contagious caprine pleuropneumonia. Veterinary Record, 121(9):191-193; 11 ref.

Rurangirwa FR, McGuire TC, Musoke AJ, Kobore A, 1987. Differentiation of F38 Mycoplasmas causing contagious caprine pleuropneumonia with a growth-inhibiting monoclonal antibody. Infection and Immunity, 55:3219-3220.

Rurangirwa FR, Kouyate B, Niang M, McGuire TC, 1990. CCPP: antibodies to F38 polysaccharide in Mali goats. Veterinary Record, 127(14):353; 11 ref.

Salih MM, Ernñ H, Simonsen V, 1983. Electrophoretic analysis of isoenzymes of Mycoplasma species. Acta Veterinaria Scandanavica, 24:14-33.

Taylor TK, Bashiruddin JB, Gould AR, 1992. Relationships between members of the Mycoplasma mycoides cluster as shown by DNA probes and sequence analysis. International Journal of Systematic Bacteriology, 42(4):593-601; 19 ref.

Thiaucourt F, Bàlske G, 1996. Contagious caprine pleuropneumonia and other pulmonary mycoplasmoses of sheep and goats. Revue Scientifique et Technique - Office International des åpizooties, 15(4):1397-1414; 69 ref.

Thiaucourt F, Breard A, LefÀvre PC, Mebratu GY, 1992. Contagious caprine pleuropneumonia in Ethiopia. Veterinary Record, 131(25/26):585; 3 ref.

Thiaucourt F, Bàlske G, Libeau G, Goff Cle, LefÀvre PC, 1994. The use of monoclonal antibodies in the diagnosis of contagious caprine pleuropneumonia (CCPP). Veterinary Microbiology, 41 (3):191-203; 36 ref.

Thiaucourt F, Bàlske G, Leneguersh B, Smith D, Wesonga H, 1996. Diagnosis and control of contagious caprine pleuropneumonia. Revue Scientifique et Technique - Office International des åpizooties, 15(4):1415-1429; 35 ref.

Thiaucourt F, Lorenzon S, David A, Breard A, 2000. Phylogeny of the Mycoplasma mycoides cluster as shown by sequencing of a putative membrane protein gene. Veterinary Microbiology, 72:251-268.

Tully JG, Bovå JM, Laigret F, Whitcomb RF, 1993. Revised taxonomy of the class Mollicutes: proposed elevation of a monophyletic cluster of arthropod-associated mollicutes to ordinal rank (Entomoplasmatales ord. nov.), with provision for familial rank to separate species with nonhelical morphology (Entomoplasmataceae fam. nov.) from helical species (Spiroplasmataceae), and emended descriptions of the order Mycoplasmatales, family Mycoplasmataceae. International Journal of Systematic Bacteriology, 43(2):378-385; [see erratum in IJSB, 43: 630 (1993)]; 54 ref.

Wamwayi HM, Wafula JS, Litamoi JK, Nandokha EN, 1989. Detection of antibody to mycoplasma F38 in goat sera by an enzyme-linked immunosorbent assay. Tropical Animal Health and Production, 21(1):43-49; 14 ref.

Weisburg WG, Tully JG, Rose DL, Petzel JP, Oyaizu H, Yang D, Mandelco L, Sechrest J, Lawrence TG, Etten Jvan, Maniloff J, Woese CR, 1989. A phylogenetic analysis of the mycoplasmas: basis for their classification. Journal of Bacteriology, 171 (12):6455-6467; 50 ref.

Wesonga HO, Litamoi JK, Kagumba M, Wakhusama E, 1993. Relationship between clinical signs and early lesions of contagious caprine pleuropneumonia caused by Mycoplasma strain F38. Small Ruminant Research, 10(1):45-54; 15 ref.

Wesonga HO, Lindberg R, Litamoi JK, Bàlske G, 1998. Late lesions of experimental contagious caprine pleuropneumonia caused by Mycoplasma capricolum ssp. capripneumoniae. Journal of Veterinary Medicine. Series B, 45(2):105-114; 22 ref.

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