Contagious caprine pleuropneumonia and other pulmonary mycoplasmoses of sheep and goats

F. THIAUCOURT* and G. BÖLSKE**

Summary: Contagious caprine pleuropneumonia (CCPP) is now a well-defined disease that is caused by Mycoplasma capricolum subsp. capripneumoniae. CCPP is infectious, contagious and fulfils the classic Koch postulates that characterise such types of disease. The distribution of the disease is not exactly known, but reports of mycoplasma isolation and official declarations to the Office International des Epizooties (OIE) enable a probable distribution map to be obtained. There are many other mycoplasmas that can infect goat and sheep lungs and induce pleuropneumonia. However, pleuropneumonia is often restricted to young animals and the prominent symptom is mastitis in lactating does. Other symptoms may also occur, contributing to a syndrome that has been tentatively described in this paper as ‘MAKePS syndrome’ for mastitis, arthritis, keratitis, pneumonia and septicaemia.


INTRODUCTION

The first clinical description of contagious caprine pleuropneumonia (CCPP) was reported in 1873 in Algeria (66). Soon after, in 1881, the disease was proved to be caused by a contagious agent (27, 28). In spite of this early description, the causative agent was only isolated and characterised a century later, in 1976 (46, 48). Recently, this causative agent, a mycoplasma, has been given a definitive name (38).

Many factors could explain the long period between the description of the disease and that of the causative agent. First, Mycoplasma capricolum subsp. capripneumoniae (MccF38) is very difficult to grow in vitro, which explains why it was isolated only in 1976, whereas the causative agent of contagious bovine pleuropneumonia (CBPP), a similar mycoplasma disease of cattle, had been isolated in 1898 (51). Moreover, MccF38 belongs to the so-called mycoides ‘cluster’, a group
of six mycoplasma species or subspecies which share many antigenic and genotypic characteristics (11). The common traits exhibited by these mycoplasmas have often confused the diagnosticians or taxonomists.

A good description of the pulmonary mycoplasmoses of small ruminants was given in 1987 (40). Since that time, tools developed in molecular biology have allowed significant progress in knowledge of the disease.

Owing to these technical advances, CCPP can be clearly distinguished from the other pulmonary mycoplasmoses which affect goats. CCPP is a very precise entity, whereas the others belong to what could be described as the 'MAKePS syndrome', for mastitis, arthritis, keratitis, pneumonia and septicaemia. The lack of precise definition of this syndrome reflects the diversity that exists among the mycoplasma strains that can cause such a syndrome (1, 3, 8). Finally, sheep do not seem to be very susceptible to pulmonary mycoplasmoses, which is why little attention has been devoted to these animals.

CONTAGIOUS CAPRINE PLEUROPNEUMONIA

Definition

CCPP is an infectious disease which affects only goats. This disease is caused by a mycoplasma, Mycoplasma capricolum subsp. capripneumoniae (MccF38).

Aetiology

The causative agent of CCPP has recently been named Mycoplasma capricolum subsp. capripneumoniae (38). Previously, this agent was known as Mycoplasma sp. type F38, as the taxonomic position had not been clearly determined. Each isolate was compared with one of the strains that had been isolated in Kenya: the F38 strain. The authors will use the abbreviation 'MccF38' for these strains, in an attempt to link the new name with the former denomination.

It is important to remember that MccF38 was characterised only in 1976, meaning that all previous publications must be analysed with care, since Mycoplasma mycoides subsp. capri (Mmc) was considered responsible for CCPP before this finding (16, 24, 30, 42, 44, 47, 50). The aetiological role of MccF38 cannot be questioned now. Numerous workers have successfully reproduced the disease with that agent (35, 49, 55). Since diagnostic tools have been improved, MccF38 is regularly demonstrated in clinical cases in which there is a strong suspicion of CCPP (7). Therefore, CCPP and MccF38 fulfil all Koch postulates, as these have been revisited (17):

- the parasite occurs in every case of the disease in question and under circumstances which account for the pathological changes and clinical course of the disease
- the parasite occurs in no other disease as a fortuitous and non-pathogenic parasite
- after being fully isolated from the body and repeatedly grown in pure culture, the parasite can induce the disease anew.

Mycoplasma capricolum subsp. capripneumoniae belongs to the mycoides 'cluster' (11), which consists of six species or subspecies of mycoplasmas which infect ruminants and share multiple phenotypic or genomic properties (Table 1). This group can be subdivided into two subgroups: mycoides (sensu stricto), and capricolum.
### TABLE I

**Relationships within the mycoides ‘cluster’**

(32)

<table>
<thead>
<tr>
<th>Mycoides subgroup</th>
<th>Capricolum subgroup</th>
</tr>
</thead>
<tbody>
<tr>
<td>M. mycoides subsp. mycoides biotype SC</td>
<td>M. capricolum subsp. capricolum</td>
</tr>
<tr>
<td>M. mycoides subsp. mycoides biotype LC</td>
<td>M. capricolum subsp. capripneumoniae</td>
</tr>
<tr>
<td>M. mycoides subsp. capri</td>
<td>M. sp. group 7 of Leach</td>
</tr>
</tbody>
</table>

### Symptoms and lesions

All goats can be affected, whatever their age or sex. Acute cases can be observed in regions where CCPP is introduced for the first time to naive populations. The incubation period generally lasts 10 days but may vary between two and 28 days. The first symptom to appear is a reluctance to walk. Fever is extreme (41°C) but the animals continue to feed and ruminate. Abortions are frequent in pregnant goats. Gradually, the respiratory symptoms become prominent. Respiration is accelerated and painful, and accompanied by violent coughing. In the terminal stages, the animals are unable to move. They stand with their legs wide apart, the neck is stiff and extended, saliva continuously drips from their mouth and their nose is obstructed by a mucopurulent discharge.

The duration of the disease varies according to the environmental conditions. This duration may vary from a few days for animals that are subjected to harsh conditions, such as underfeeding, polyparasitism, the necessity to walk long distances for watering, bad climatic conditions, etc. However, animals can survive more than one month or even recover if they are placed in good conditions.

In acute cases, lesions are typical, occurring only in the thoracic cavity. Very often lesions occur in one lung only. Affected lungs can be totally hepatised, of a port wine colour. A lung section shows a fine granular texture and a colour that varies from purple to greyish. There is often an abundant pleural exudate and conspicuous pleuritis. The pleural exudates can solidify and form a gelatinous covering on the whole lung.

Subacute or chronic forms can be observed in regions where CCPP is enzootic. The symptoms are similar to those of the acute cases, but are not as strong. Coughing is irregular and usually follows a physical effort, and nasal discharge may be absent.

A complete ‘matity’ or ‘flatness’ of one of the lungs can be observed easily when examining a live animal. Affected lungs can sometimes degenerate into a voluminous abscess, as a consequence of bacterial secondary infections. Adhesions between the lung and the pleura are very common and often very thick (49). In the absence of antibiotic treatment, mortality varies between 60% and 100%.

### Epidemiology

**Affected species**

Only goats are affected.
Geographical distribution

It is difficult to give an exact picture of the distribution of the disease. When considering MccF38 isolation records, affected countries are as follows: Kenya (48), Sudan (26), Tunisia (56), Chad (39), Oman (34), Ethiopia (63), the United Arab Emirates, and certainly Turkey (67) and Yemen. When considering some clinical descriptions which have been published, Algeria and India can certainly be considered to be infected as well.

Recent findings have extended this list. CCPP has been confirmed by isolation of MccF38 in Uganda (6) and in Niger (Y. Maïkano and F. Thiaucourt, personal communication). Considering the contagiousness of the disease and the movements of nomadic goat herds, the whole region delineated by Tunisia, Niger, Uganda, Turkey and Yemen can be considered to be infected. Some uncertainty remains concerning the extension of the disease to the west or south of the African continent or to the east, in Asia (Fig. 1). Numerous publications have documented the presence of goat pleuropneumonia in Nigeria (53) and in India (58) without the isolation of MccF38. Usually the isolated mycoplasmas belong to M. mycoides subsp. capri. The only description of MccF38 isolation in India has been recorded from cattle milk (36), but doubts have been raised on the exactness of this identification (62).

Compared to the few descriptions of isolation, there are numerous countries (approximately 30) which have officially declared that they are infected by CCPP (Table II).

Evolution of the disease

It seems that CCPP may become enzootic in some regions and occur in epizootic waves in others. These differences may be due to differences in livestock management. In Oman, the disease is enzootic and many herds are affected each year. In Chad, the disease seemed to have disappeared after its first description in 1987, reappearing only recently.

Transmission

CCPP always appears after the introduction of an infected animal into a susceptible herd. The transmission is direct, by the aerogenic route, through droplets released during coughing. Very short periods of contact are sufficient to transmit the disease, but intimate contact is needed. Indirect transmission does not seem to occur, as mycoplasmas are not very resistant and are rapidly inactivated.

Some animals may become latent chronic carriers and thus play an important role in the transmission of the disease (69). For example, the introduction of CCPP into the Cape Province in 1881 may have been due to such a carrier in a group of animals from Turkey. These animals had been on the journey for more than seven weeks, i.e. longer than the known incubation period. The stress of transportation might have reactivated a latent MccF38 infection.

The exact location of the mycoplasmas in latent carriers is not known. Unlike in the case of contagious bovine pleuropneumonia, no sequestra have been described for CCPP. In two reports, sheep have been shown to harbour MccF38 in the nares (41) or in the lungs (6); however, their possible role as carriers has not yet been demonstrated. There is growing evidence that mycoplasmas do not have strict host specificity, as
Probable distribution of contagious caprine pleuropneumonia

Countries in which there is a confirmed description of contagious caprine pleuropneumonia, with isolation of *Mycoplasma capricolum* subsp. *capripneumoniae* have been isolated. The probable extension of the disease is marked in pale grey (this relies solely upon clinical descriptions of the disease).
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Africa</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Angola</td>
<td>+*</td>
<td>+T*</td>
<td>+*</td>
<td>+..</td>
<td>+..</td>
<td>...</td>
</tr>
<tr>
<td>Central African Republic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cameroon</td>
<td>-T*</td>
<td>-T*</td>
<td>-T*</td>
<td>-T*</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Côte d’Ivoire</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>+TV</td>
<td>1993</td>
<td></td>
</tr>
<tr>
<td>Djibouti</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Equatorial Guinea</td>
<td>+..</td>
<td>+..</td>
<td>?</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eritrea</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ethiopia</td>
<td>+TV</td>
<td>+TV</td>
<td>+..TV</td>
<td>+TV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Guinea</td>
<td>(+)</td>
<td>(+)</td>
<td>(+)</td>
<td>(+)</td>
<td>(+)</td>
<td>(+)</td>
</tr>
<tr>
<td>Guinea Bissau</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td>?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kenya</td>
<td>+TV</td>
<td>+TV</td>
<td>+TV</td>
<td>+TV</td>
<td>+TV</td>
<td>+</td>
</tr>
<tr>
<td>Libya</td>
<td>++T</td>
<td>++T</td>
<td>+T</td>
<td>+T</td>
<td>+T</td>
<td>+</td>
</tr>
<tr>
<td>Mauritania</td>
<td>-</td>
<td>(+)*</td>
<td>(+)*</td>
<td>(+)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Niger</td>
<td>?V</td>
<td>+SV</td>
<td>+</td>
<td>++QiT*</td>
<td>++QiT*</td>
<td>++</td>
</tr>
<tr>
<td>Nigeria</td>
<td>+</td>
<td>+T</td>
<td>+</td>
<td>+</td>
<td>+T</td>
<td>+</td>
</tr>
<tr>
<td>Rwanda</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Somalia</td>
<td>+PaQiV</td>
<td>+PaQiV</td>
<td>+PaQiV</td>
<td>+PaQiV</td>
<td>+PaQiV</td>
<td>+PaQiV</td>
</tr>
<tr>
<td>Sudan</td>
<td>+</td>
<td>-1990T</td>
<td>+++</td>
<td>++</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Tunisia</td>
<td>-</td>
<td>-</td>
<td>-1988</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Uganda</td>
<td>+OQiT*</td>
<td>+OQiT*</td>
<td>+OQiT*</td>
<td>...QiT*</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td><strong>Asia</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Afghanistan</td>
<td>++</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bahrain</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>India</td>
<td>+V</td>
<td>+V</td>
<td>+V</td>
<td>+T</td>
<td>+T</td>
<td>+</td>
</tr>
<tr>
<td>Iran</td>
<td>++(PaQfTV)</td>
<td>++(PaQfTV)</td>
<td>++PnTV</td>
<td>++PnTV</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>Kuwait</td>
<td>++T</td>
<td>++T</td>
<td>?T</td>
<td>+</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Laos</td>
<td>+T</td>
<td>+T</td>
<td>++T</td>
<td>++T</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Lebanon</td>
<td>++T</td>
<td>++T</td>
<td>++T</td>
<td>++T</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Lebanon</td>
<td>+</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oman</td>
<td>+TT</td>
<td>+T</td>
<td>+T</td>
<td>++T</td>
<td>++T</td>
<td>++</td>
</tr>
<tr>
<td>Pakistan</td>
<td>+V</td>
<td>(0)</td>
<td>(0)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Qatar</td>
<td>+V</td>
<td>+TV</td>
<td>+V</td>
<td>+V</td>
<td>+</td>
<td>+</td>
</tr>
<tr>
<td>Saudi Arabia</td>
<td>+</td>
<td>+QT</td>
<td>+QT</td>
<td>1992</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
thought previously. Thus, the occurrence of \textit{MccF38} in hosts other than goats, especially sheep (but also cattle) should be investigated.

\textbf{Factors affecting receptivity}

In regions where CCPP already occurs, the severity of the disease may depend on the following factors:

- the proportion of immune animals, as an animal which has survived a previous infection is thought to be protected
- the presence of co-existing viral infections, orf (contagious ecthyma) or peste des petits ruminants, for example, which may favour the development of CCPP
- poor climatic conditions, such as a large temperature difference between day and night or an abrupt change of climate, especially during the period between the dry and rainy seasons
- stress due to movement over long distances.

\begin{table}[h]
\centering
\begin{tabular}{|l|c|c|c|c|c|c|}
\hline
\hline
\textbf{Asia (cont.)} & & & & & & \\
Turkey & +V & +V & +V & +V & + & \\
United Arab Emirates & +T & +TV & +T & & & \\
Yemen & +TV & +TV & ? & ++ & & \\
\hline
\textbf{Europe} & & & & & & \\
Greece & + & +( & +( & +( & ++ & ++( \\
\hline
\end{tabular}
\end{table}

-: not reported

year: year of last occurrence

?: suspected but not confirmed

(+): exceptional occurrence

+: low sporadic occurrence

++: enzootic

+++: high occurrence

+.: disease exists; distribution and occurrence unknown

(): confined to certain regions

): ubiquitious

...: no information available

Pa: control programme only for some areas of the country or for certain types of breeding

Pn: control programme for the whole country

Q: quarantine, movement control and other precautions at frontier and inside the country

Qf: quarantine and other precautions at frontier

Qi: quarantine measures and movement control inside the country

S: stamping-out policy

T: treatment

V: vaccination

*: notifiable disease
Pathogenesis

There is very little information on the pathogenic mechanisms of \textit{MccF38}, although some hypotheses can be drawn from comparison with other mycoplasmoses and especially with CBPP. A striking feature of CCPP is the strict host and tissue specificity of the causative agent, as lesions are produced only in goat lungs. Mycoplasmas usually grow on tissues which provide them with essential metabolites. Some mycoplasmas have adhesins, but no such component has yet been described for \textit{MccF38}. The expression of pathogenicity may depend on other factors that favour penetration and multiplication, which may explain why it is sometimes difficult to reproduce the disease experimentally. Although \textit{MccF38} is present in high quantities in affected lungs, there is no dissemination to other organs. This may be due to a specific reaction of the lung tissue towards a mycoplasmal component which leads to an exacerbated inflammatory response. More studies are needed to understand the intimate mechanisms that explain this strict host and organ specificity. Such comprehension might help in defining better treatments and/or prophylactic measures.

The polysaccharide capsule of \textit{MccF38} (57) may have a similar role to that described for \textit{Mycoplasma mycoides} subsp. \textit{mycoides} small colony type (\textit{MmmSC}) in CBPP.

\textbf{THE MASTITIS, ARTHRITIS, KERATITIS, PNEUMONIA AND SEPTICAEMIA SYNDROME IN GOATS}

\textbf{Definition}

The MAKePS syndrome is caused by mycoplasmas and occurs in goats. It is characterised by an association of mastitis, arthritis, keratitis, pneumonia and septicaemia, alone or together, in individual animals or in many animals within a herd.

\textbf{Aetiology}

Many different mycoplasmas can cause this syndrome, namely (Table III):

- \textit{Mycoplasma mycoides} subsp. \textit{mycoides} LC (\textit{MmmLC}) (2)
- \textit{Mycoplasma mycoides} subsp. \textit{capri} (\textit{Mmc}) (31, 54)
- \textit{Mycoplasma capricolum} subsp. \textit{capricolum} (\textit{Mcc}) (5)
- \textit{Mycoplasma putrefaciens} (\textit{Mp}) (12)
- \textit{Mycoplasma agalactiae} (\textit{Ma}).

The infection due to \textit{M. agalactiae} in sheep and goats is also known as contagious agalactia, and is dealt with by Bergonier and Poumarat in another paper in this issue (4). However, this disease cannot be differentiated from those due to other mycoplasmas of the \textit{mycoides} 'cluster' (\textit{MmmLC}, \textit{Mmc} and \textit{Mcc}), or to \textit{Mp}, and is therefore integrated into the 'MAKePS syndrome'.

These five species share a common characteristic. They are very heterogeneous from a genetic as well as an antigenic point of view (37). This contrasts markedly with the great homogeneity of species which cause very well-defined diseases, such as CCPP and CBPP (\textit{MccF38} and \textit{MmmSC}, respectively). This heterogeneity can be
<table>
<thead>
<tr>
<th>Species</th>
<th>Taxonomic position</th>
<th>Taxonomic position (ARN 16S)</th>
<th>Usual hosts</th>
<th>Pathogenic power</th>
<th>Growth characteristics</th>
</tr>
</thead>
<tbody>
<tr>
<td><em>Mycoplasma capricolum</em> subsp. capripneumoniae</td>
<td><em>Mycopotes</em> ‘cluster’</td>
<td>Spiroplasma group</td>
<td>Goats</td>
<td>P</td>
<td>Very difficult</td>
</tr>
<tr>
<td><em>M. capricolum</em> subsp. capricolum</td>
<td><em>Mycopotes</em> ‘cluster’</td>
<td>Spiroplasma group</td>
<td>Goats, sheep</td>
<td>MAKePS</td>
<td>Very easy</td>
</tr>
<tr>
<td><em>Mycoplasma</em> sp. group 7 (Leach)</td>
<td><em>Mycopotes</em> ‘cluster’</td>
<td>Spiroplasma group</td>
<td>Cattle</td>
<td>MA</td>
<td>Very easy</td>
</tr>
<tr>
<td><em>M. mycoides</em> subsp. mycoides LC</td>
<td><em>Mycopotes</em> ‘cluster’</td>
<td>Spiroplasma group</td>
<td>Goats, sheep</td>
<td>MAKePS</td>
<td>Very easy</td>
</tr>
<tr>
<td><em>M. mycoides</em> subsp. capri</td>
<td><em>Mycopotes</em> ‘cluster’</td>
<td>Spiroplasma group</td>
<td>Goats, sheep</td>
<td>MAKePS</td>
<td>Very easy</td>
</tr>
<tr>
<td><em>M. putrefaciens</em></td>
<td>Near <em>mycoides</em> ‘cluster’</td>
<td>Spiroplasma group</td>
<td>Goats, sheep</td>
<td>MAKePS</td>
<td>Very easy</td>
</tr>
<tr>
<td><em>M. agalactiae</em></td>
<td>Near <em>M. bovis</em></td>
<td>Hominis group</td>
<td>Goats, sheep</td>
<td>Dubious</td>
<td>Easy</td>
</tr>
<tr>
<td><em>M. ovipneumoniae</em></td>
<td>ND</td>
<td>ND</td>
<td>Goats, sheep</td>
<td>None</td>
<td>Difficult</td>
</tr>
<tr>
<td><em>M. arginini</em></td>
<td>ND</td>
<td>Hominis group</td>
<td>Goats, sheep</td>
<td>C</td>
<td>Easy</td>
</tr>
<tr>
<td><em>M. conjonctivae</em></td>
<td>ND</td>
<td>ND</td>
<td>Goats, sheep</td>
<td>ND</td>
<td>Easy</td>
</tr>
<tr>
<td><em>M. auris</em></td>
<td>ND</td>
<td>ND</td>
<td>Goats</td>
<td>ND</td>
<td>Easy</td>
</tr>
<tr>
<td><em>M. cottewi</em></td>
<td>ND</td>
<td>ND</td>
<td>Goats</td>
<td>ND</td>
<td>Easy</td>
</tr>
<tr>
<td><em>M. yeatsii</em></td>
<td>ND</td>
<td>ND</td>
<td>Goats</td>
<td>ND</td>
<td>Easy</td>
</tr>
</tbody>
</table>

ND: not determined

P: pleuropneumonia

MA: mastitis, arthritis

MAKesPS: mastitis, arthritis, keratitis, pleuropneumonia, septicaemia

C: conjunctivitis
easily detected by whole cell protein electrophoresis in sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) (3, 8, 36, 64), for instance.

**Symptoms and lesions**

All animals can be affected. Mastitis/agalactia is usually the prominent symptom in lactating does. Adult animals frequently also suffer arthritis and keratitis while kids usually suffer severe arthritis, pleuropneumonia and septicaemia. Some particular mycoplasma strains may exhibit a marked tropism for the lung, and sometimes environmental factors may favour a spread to the respiratory tract. In this case, the symptoms may be very similar to those seen in CCPP. A pulmonary tropism is often seen with strains belonging to the MmmLC or Mmc subspecies but has also been recorded with *M. agalactiae* strains.

In the case of pleuropneumonia, macroscopic lesions differ slightly from those of CCPP, as there is frequently enlargement of the interlobular septa. This thickening is less pronounced than the thickening seen in CBPP lesions. Thickening may not be present consistently and confusion with CCPP lesions is very likely.

Histological lesions are usually characterised by interstitial pneumonia, but this is not pathognomonic (32). In natural cases, the presence of other pathogens, such as bacteria or viruses, may alter the picture. For example, the presence of *Pasteurella haemolytica* may give a picture of fibrinous pneumonia.

**Epidemiology**

**Affected species**

Sheep and goats can both be affected by the MAKePS syndrome but usually goats are more susceptible. Kids are more frequently affected by pleuropneumonia than adults.

**Geographical distribution**

This syndrome has a world-wide distribution. All mycoplasma species which can cause this syndrome have been isolated from regions where goat-raising is developed.

**Transmission**

Contaminated milk may be the main vector for the dissemination of the disease, by mechanical transmission from one animal to another through milking and to the kids by suckling or by inhalation of infected droplets. In kids, this contamination is often followed by septicaemia and dissemination to other organs.

**Carrier state**

An affected herd may recover, remain clinically healthy for many years and then suffer from the same disease again. This can be due to new contamination but many authors have raised the possibility of a long-term carrier state in some animals, which may lead to new outbreaks. Mycoplasmas may survive in the organs that were originally affected, such as the mammary gland or lung, in which case re-excretion may follow some physiological modification, such as the start of new lactation.
Mycoplasmas can also survive in a distant organ such as the ear canal; an original finding which was clearly demonstrated by Cottew and Yeats and DaMassa and Brooks (9, 10, 13). Since the original description, numerous mycoplasma species have been isolated from the ear canal of healthy goats. The pathogenicity of some of these species is unknown, as in the case of *M. auris*, *M. cottewi* or *M. yeatsii*, but all species which can cause MAKePS have already been isolated from the ear canal. Furthermore, it has been shown that mites which colonise goat ears can harbour these mycoplasmas and play an active role in transmission. This type of carrier state and mode of transmission may well jeopardise many efforts to eradicate the disease.

**Factors affecting receptivity**

All the factors that are well known to favour pulmonary infections may play a role in the occurrence of pulmonary mycoplasmoses in goats: bad climatic conditions, overpopulation or other infectious predisposing factors. Respiratory disease usually occurs in kids during the immunological gap that exists when passive immunity, provided by maternal colostrum, has waned and the immune system is not yet mature.

**Clinical diagnosis**

Clinical diagnosis can be performed at the herd level but not from individual cases. It is quite unusual to observe all the symptoms in a single animal. Usually, agalactia is the prominent symptom in the herd but cases of arthritis, keratitis and pneumonia can be observed in some animals. When there are only pulmonary symptoms, clinical diagnosis is difficult and confirmation should come from the laboratory.

**Laboratory diagnosis**

**Isolation**

All classical techniques which are used to grow and identify mycoplasmas (23, 40) can be used to confirm MAKePS. All causative mycoplasmas grow easily *in vitro*. It is noteworthy that they grow much more rapidly than *MccF38*. This must be kept in mind when *MmmLC* or *Mmc* are isolated from pulmonary lesions that look like CCPP lesions. Multiple mycoplasmas may well co-exist in goat lungs and, in that case, only the strains which grow well will be recovered. This may well explain the origin of the confusion on the aetiology of CCPP which existed until recently.

**Polymerase chain reaction**

Until now there has been no polymerase chain reaction (PCR) protocol that permits the specific amplification of DNA fragments of each of the mycoplasmas that may cause MAKePS. Nevertheless, it is possible to use protocols that specifically amplify a region of the *M. agalactiae* genome (15), or that amplify a fragment which is common to all mycoplasmas belonging to the *mycoides* ‘cluster’. In the latter case, a more precise identification can be achieved by digestion of the amplified product with an endonuclease and analysis of the fragments obtained (15, 61). Considering the easy growth of the mycoplasmas that can cause the MAKePS syndrome, PCR is not yet used routinely for direct diagnosis. Such a technique might also give dubious results in the case of samples that contain multiple mycoplasma species.
The complement fixation test (55) may be an efficient method if certain precautions are taken. This test must be conducted with a representative number of sera. It must be performed with different antigens (Ma, Mcc, Mmc, MmmLC, Mp) in parallel and, finally, it must be performed shortly after the outbreak, when complement-fixing antibodies are still present. The short-term presence of complement-fixing antibodies does not permit the use of this technique to screen a large number of sera in wide serological surveys when there is no record of disease.

Indirect enzyme-linked immunosorbent assay (ELISA) has been proposed for the detection of antibodies directed towards *M. agalactiae* or towards mycoplasmas of the *mycoides* ‘cluster’. With this type of method, antibodies may be detected over a longer period and the distinction will be easily made between *agalactiae* and *mycoides* ‘cluster’, as there is no antigenic homology between the two. Distinction from CCPP can be achieved through the results of a competitive ELISA that is strictly specific for that disease (65).

In the case of MAKePS, it must be remembered that infection might not always be followed by a rise in antibody, especially when this syndrome affects kids which are immunologically immature.

**Treatment**

As a general rule, tetracyclines or antibiotics belonging to the macrolide group, such as tylosin and spiramycin, are recommended. The recommendations of the manufacturer must be followed carefully, especially the dosage and the duration of the treatment, even though symptoms might disappear rapidly.

**Prevention**

Owing to the wide diversity of agents that can cause MAKePS, there is no universal vaccine that can protect a herd completely. Some vaccines have been designed against *Ma* or *Mmc* (58, 59). Their efficacy has not been clearly demonstrated. The wide variation that exists among mycoplasma strains might be an insurmountable obstacle to the design of efficient vaccines.

Sanitary prophylaxis may be the sole measure that can prevent a herd from being infected by MAKePS. The most important measure is to prevent the introduction of animals from herds that have already suffered from MAKePS syndrome in the past. This is very difficult to implement as a negative serological response does not guarantee that the herd is not harbouring latent carriers. Implementation is even more difficult for nomadic herds which have frequent contacts with other animals.

**OTHER MYCOPLASMAS OF THE RESPIRATORY TRACT**

*M. ovipneumoniae* has often been isolated from goats with pneumonia or pleuropneumonia (43), but the significance of this is unclear since *M. ovipneumoniae* frequently also occurs in healthy young goats. However, in the experimental infection
of six goats with *M. ovipneumoniae*, pleuropneumonia was produced in one goat, pleuritis in a second and focal pneumonia in a third (25).

*M. arginini*, another agent sometimes isolated from the respiratory tract of goats, is not known to be pathogenic.

*M. bovis*, a pathogen for cattle, has, in a few cases, been isolated from goats with pneumonia. Whether this mycoplasma is pathogenic and of any importance for goats is not known.

**RESPIRATORY MYCOPLASMOSES IN SHEEP**

Sheep are less susceptible to pulmonary mycoplasmoses than goats. In this species, agalactia is the prominent symptom where mycoplasma infections are concerned. *Mycoplasma agalactiae* is the most frequently isolated mycoplasma, but isolations of *M. mycoides* subsp. *mycoides* LC or *M. capricolum* subsp. *capricolum* have also been reported. The latter was also found in the respiratory tract in one study (60).

Respiratory disease in sheep is often of multiple origins, with an association of viral infection with pasteurellic infection. Mycoplasmas may also be involved and *M. ovipneumoniae* has been incriminated as an aetiological agent for atypical pneumonia in sheep (33).

As in the case of goats, climatic conditions seem to play an important role in the development of respiratory infections. In temperate climates, there is a significative correlation between the value of an index of ‘rain and windchill’ and the occurrence of pneumonia in sheep after two months (45).

Sheep seem to harbour mycoplasmas in the external ear canal (13) but this may be less frequent than in goats.

**CONCLUSION**

Although the aetiological agent of CCPP, *Mycoplasma capricolum* subsp. *capripneumoniae*, was identified more than 20 years ago, some authors still continue to attribute the origin of CCPP to other mycoplasmas. However, it is necessary to distinguish CCPP from the other respiratory mycoplasmoses because their distribution is very different, as is, obviously, their mode of spread. Hence, the prophylactic measures that can be implemented will vary accordingly. The introduction of CCPP into CCPP-free zones would result in great losses to the goat industry and international animal health policies should aim at combating this disease. The other respiratory mycoplasmoses of small ruminants form part of what has been called in this paper the ‘MAKePS syndrome’. This new definition has been created to match the diversity of symptoms that can be observed with the different pathogenic mycoplasmas that affect small ruminants. This grouping is considered logical, as all these mycoplasmas share similar properties. The MAKePS syndrome may then include the classic ‘contagious agalactia’ that would be restricted to diseases caused by *M. agalactiae*.

* *
PLEUROPNEUMONIE CONTAGIEUSE CAPRINE ET AUTRES MYCOPLASMOSES PULMONAIRES DES OVINS ET CAPRINS. – F. Thiaucourt et G. Bölske.

Résumé : La pleuropneumonie contagieuse caprine (PPCC) est une maladie désormais bien définie, due à Mycoplasma capricolum subsp. capripneumoniae. La PPCC est une maladie infectieuse, contagieuse, qui satisfait au postulat de Koch applicable à ce type de maladie. On n’en connaît pas encore avec précision la répartition géographique, mais des rapports sur l’isolement de mycoplasmes et des déclarations officielles adressées à l’Office international des épizooties permettent de dresser une carte de distribution probable. Nombre d’autres mycoplasmes peuvent infecter les poumons de caprins et d’ovins et entraîner une pleuropneumonie. Néanmoins, la pleuropneumonie est souvent limitée aux jeunes animaux et la mammite en est le symptôme majeur chez les femelles allaitantes. D’autres symptômes peuvent également apparaître, constituant un syndrome auquel les auteurs ont donné le nom de « syndrome MAKePS » (pour mammite, arthrite, kératite, pneumonie et septicémie).


* * *


Resumen: La pleuroneumonía contagiosa caprina (PPCC) es hoy una enfermedad bien definida, causada por Mycoplasma capricolum subsp. capripneumoniae. La PPCC es infecciosa y contagiosa, y obeede al postulado clásico de Koch que caracteriza las enfermedades de esta índole. No se conoce con exactitud la distribución de la enfermedad, aunque es posible trazar un mapa de distribución probable a partir de los informes sobre casos de aislamiento de Mycoplasma y de las declaraciones oficiales a la Oficina Internacional de Epizootias. Existen muchos otros micoplasmas capaces de infectar los pulmones de cabras y ovejas y de provocar con ello una pleuroneumonía. No obstante, la pleuroneumonía afecta generalmente de forma exclusiva a los individuos jóvenes. El síntoma dominante es la presencia de mastitis en las hembras lactantes. También pueden manifestarse otros síntomas, que contribuyen a formar lo que los autores proponen definir como «síndrome MAKePS» (por las denominaciones inglesas de mastitis, artritis, queratitis, neumonía y septicemia).


* * *
REFERENCES


