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Review

Contagious agalactia due to *Mycoplasma* spp. in small dairy ruminants: Epidemiology and prospects for diagnosis and control

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ABSTRACT

Contagious agalactia (CA) is a serious disease of small dairy ruminants that has a substantial economic impact on the goat and sheep milk industries. The main aetiological agent of the disease is *Mycoplasma agalactiae*, although other species, such as *Mycoplasma mycoides* subsp. *capri*, *Mycoplasma capricolum* subsp. *capricolum* and *Mycoplasma putrefaciens*, are pathogenic in goats. There are two clinical–epidemiological states of CA in sheep and goats; herds and flocks may exhibit outbreaks of CA or may be chronically infected, the latter with a high incidence of subclinical mastitis and only occasional clinical cases. The complex epidemiology of CA is related to the genetic characteristics and mechanisms of molecular variation of the *Mycoplasma* spp. involved, along with presence of CA-mycoplasmas in wild ruminant species. In goats, the situation is particularly complex and asymptomatic carriers have been detected in chronically infected herds. The coexistence of other non-pathogenic mycoplasmas in the herd further complicates the diagnosis of CA and the design of efficient strategies to control the disease. Routes of infection, such as the venereal route, may be involved in the establishment of chronic infection in herds. Current challenges include the need for improved diagnostic methods for detection of chronic and subclinical infections and for the design of more efficient vaccines.

Introduction

Contagious agalactia (CA) is a multi-aetiological syndrome that has a serious economic impact in countries with goat and/or sheep dairy industries, mainly in the Mediterranean. Clinical disease typically involves the mammary glands, joints and eyes, although respiratory signs and reproductive disorders, including abortions, have been also reported. Clinical CA outbreaks in herds and flocks are often attributable to the entry of infected animals or to a decrease in herd immunity (Bergonier et al., 1997). In areas where CA is endemic, the most common clinical–epidemiological situation is one of chronically infected herds, with no apparent signs of disease. This situation is characterised by the presence of animals with subclinical mastitis caused by *Mycoplasma* spp., with progression to clinical mastitis in some animals. In infected goat herds, large numbers of asymptomatic carriers exist and these are often serologically negative for CA-causing *Mycoplasma* spp.

Developments in the molecular diagnosis of CA and the genetic typing of isolates are starting to clarify epidemiological aspects of the disease. These advances have improved our prospects for the design of effective strategies to prevent, diagnose and control CA. In this paper, we review the epidemiology of CA in dairy goats and sheep, and discuss future prospects for its control.

Taxonomy of *Mycoplasma* spp. causing contagious agalactia

Contagious agalactia is caused by four different species of *Mycoplasma*: *Mycoplasma agalactiae* (Ma), the classic agent of the disease in sheep and goats, *Mycoplasma mycoides* subsp. *capri* (Mmc), *Mycoplasma capricolum* subsp. *capricolum* (Mcc) and *Mycoplasma putrefaciens* (Mp). In addition to Ma, Mmc has also been identified in sheep and, in decreasing order, Mmc, Mcc and Mp have been implicated in disease in goats (Bergonier et al., 1997; Chazel et al., 2010).

Among the four *Mycoplasma* spp. incriminated in CA, Mmc and Mcc phylogenetically belong to the *M. mycoides* cluster within the group *Spiroplasma*. The causal agent of CA, previously defined as *M. mycoides* subsp. *mycoides* large colony (LC), has been reclassified as Mmc on the basis of phylogenetic data (Fig. 1) (Manso-Silván et al., 2007 and 2009; Shahram et al., 2010). Mp also occurs in the *Spiroplasma* group and is phylogenetically close to the *M. mycoides* cluster. In contrast, Ma is phylogenetically distant from the remaining *Mycoplasma* spp. causing CA and has been ascribed to the group *Hominis* (Fig. 2). Horizontal gene transfer takes place

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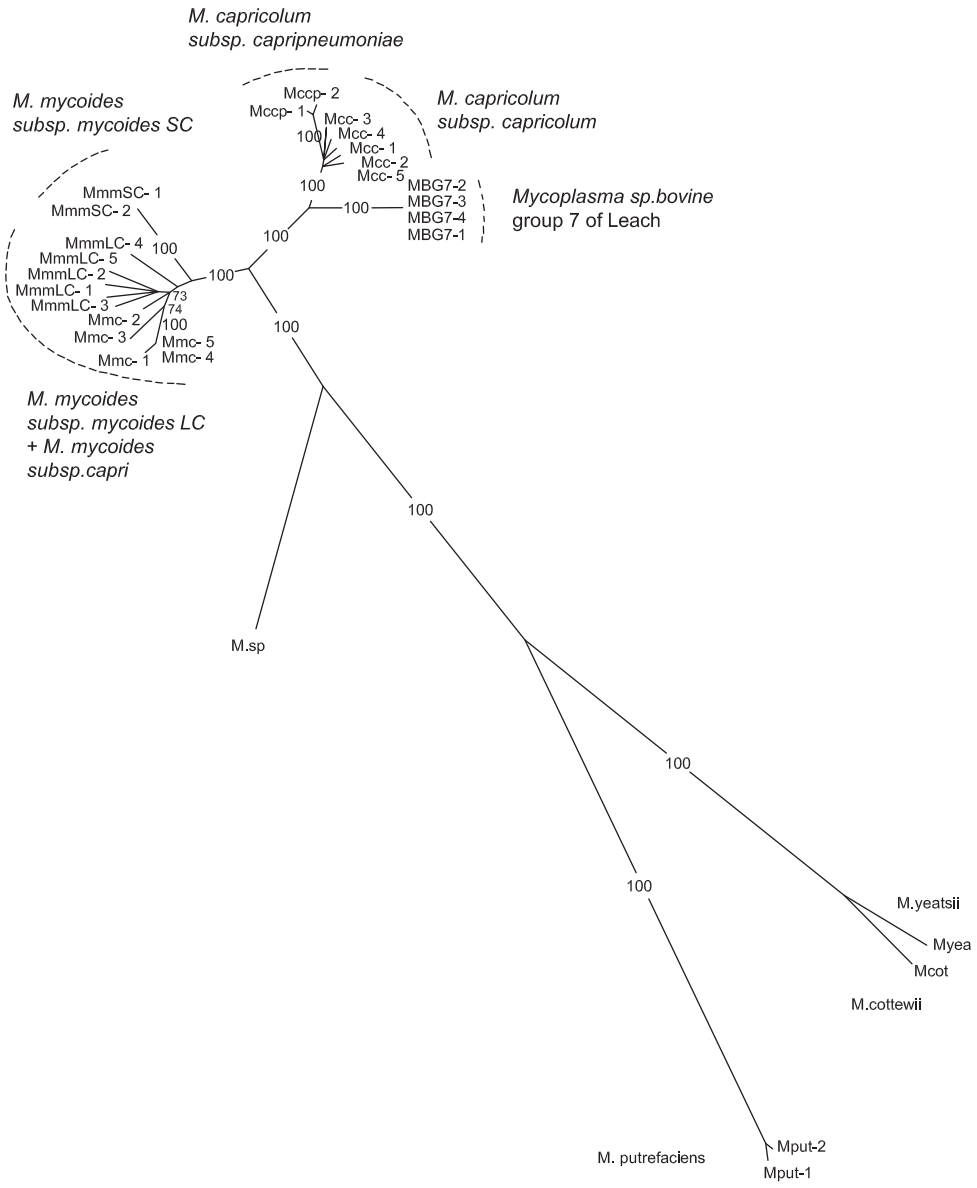

Fig. 1. Phylogenetic tree of *Mycoides* cluster, *Mycoplasma putrefaciens*, *Mycoplasma yeatsii* and *Mycoplasma cotewii* derived from distance analysis of five concatenated protein coding sequences, *fusA*, *glpQ*, *gyrB*, *lepA* and rpoB (Manso-Silván et al., 2007).

among the four *Mycoplasma* spp. and it is estimated that Ma shares ~18% of its genome with the *M. mycoides* cluster (Sirand-Pugnet et al., 2007).

Economic impact of contagious agalactia

In herds and flocks of domestic small ruminants, economic losses due to CA are mainly due to loss of milk production in affected animals, mortality, abortions, reduced growth rates, early culling and the costs generated by control measures. The impact of mycoplasmas on milk quality is probably underestimated (Contreras et al., 2008). The risk of exceeding the legal limits for somatic cell counts (SCC) in bulk tank milk (BTM) samples is high during an outbreak of CA and affected dairy goat and sheep herds may suffer serious mastitis problems. However, SCC cannot be used for the indirect detection of chronically infected herds of goats (Corrales et al., 2004) or flocks of sheep (Gonzalo et al., 2005), particularly in areas where the disease is endemic.

Mycoplasma spp. may also affect fertility, since Ma, Mmc and Mp exhibit tropism for the reproductive tract in sheep and goats (Gil et al., 2003; Szeredi et al., 2003; Kidanemariam et al., 2005a). In goats, there is a negative correlation between mycoplasmal infection, gestation rates and the number of kids weaned (Gil et al., 2003), and it is likely that the effects of mycoplasmas on reproductive performance have been underestimated.

Antimicrobial therapy is one of the main tools used to control CA (Bergonier et al., 1997), but requires a withholding period, resulting in milk production losses. In addition, the presence of antimicrobial residues in milk poses a risk to the consumer (Allison, 1985; Dewdney et al., 1991) and could impair bacterial fermentation required for cheese production (Mourot and Loussouarn, 1981). None of four screening tests for detection of residues of 20 antimicrobial agents (including members of the aminoglycoside, macrolide, tetracycline, sulphonamide and quinolone groups) in goat's milk were able to detect European Union maximum permissible levels of tetracyclines or quinolones currently used against CA (Sierra et al., 2009), indicating a need to improve

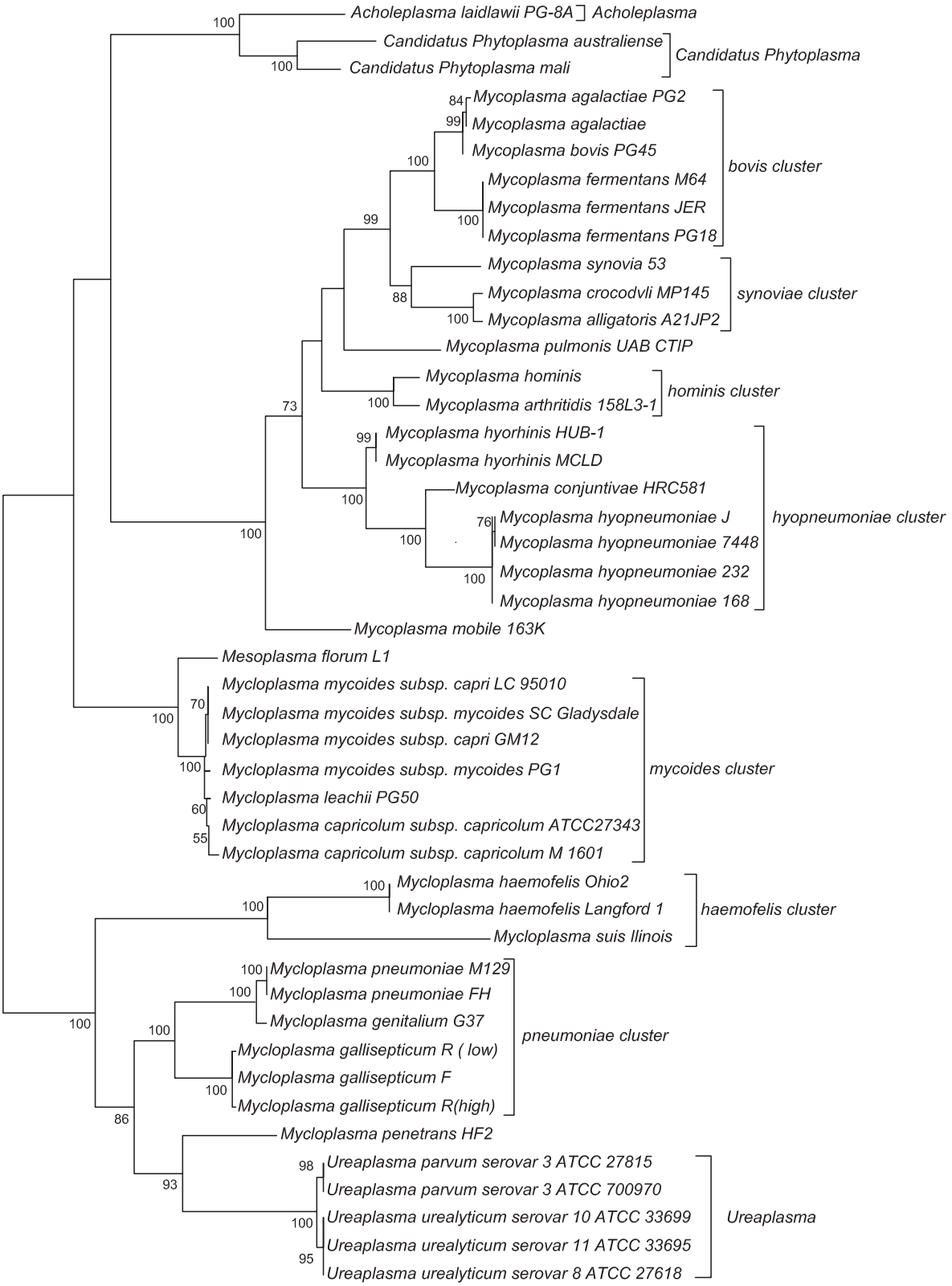

Fig. 2. *Mycoplasma* phylogenetic tree of the 16S RNA (Thompson et al., 2011).

the sensitivity of the methods routinely used in antimicrobial residue programmes.

Molecular epidemiology of *Mycoplasma* spp.

Multilocus sequence (MLST), variable number tandem repeats (VNTR) and pulsed field gel electrophoresis (PFGE) have been used to identify several strains of Ma circulating in Europe (McAuliffe et al., 2008, 2011). In goats, molecular studies have revealed a high genetic diversity of Ma isolates in an area endemic for Spanish CA (De la Fe et al., 2012), in contrast with a relatively low genetic diversity in Ma from sheep in France (Nouvel et al., 2012), Italy (Tola et al., 1999) and Spain (Ariza-Miguel et al., 2013). This genetic diversity of Ma in goats needs to be considered in the design of vaccines.

A single pulsotype of Mmc was detected during a CA outbreak in goats, while different pulsotypes were observed in asymptomatic auricular carriers and these in turn differed from isolates obtained from clinical cases, suggesting the existence of non-pathogenic strains of Mmc (Tardy et al., 2007). However, clinical signs developed in goats after experimental inoculation using from the clinical outbreak, as well as isolates from asymptomatic animals, indicating a lack of correlation between genomic diversity and pathogenicity (Tardy et al., 2011).

Genetic systems that allow mycoplasmas to undergo high frequency surface antigenic variations include *Vpma* in Ma and *Vmc* in Mcc (Glew et al., 2000; Wise et al., 2006). Molecular mechanisms generating high antigenic variability has been recently reviewed (Citti et al., 2010). In Ma, *Vpma* phase variation is not necessary for establishing infection, but may influence the survival and persistence of the organism (Chopra-Dewasthaly et al., 2012). In a comparison of two Ma genomes, most differences were attributable to mobile genetic elements, including integrating conjugative elements (ICE) and insertion sequences (IS), and elements related to the variable expression of surface proteins (Nouvel et al., 2010).

Genomic sequencing has revealed a Ma prophage resembling a prophage identified in *M. conjunctivae*, which has been linked to outbreaks of keratoconjunctivitis in the Alpine ibex (*Capra ibex*) (Tardy et al., 2012). These findings point to the highly dynamic nature of Ma. Several coding sequences identified in Ma are conserved in other ruminant *Mycoplasma* spp., and some of these genome fragments have been implicated in a substantial horizontal gene transfer event between Ma and other ruminant mycoplasmas, such as *M. bovis* and members of the *Mycoides* cluster (Skapski et al., 2011).

Analytical epidemiology of contagious agalactia

CA is distributed worldwide and has been reported in Africa, Asia and America (OIE, 2012). However, the Mediterranean region traditionally has been the focus of attention because of its large dairy goat industry and the endemic status of CA in the region (Bergonier et al., 1997).

Although Ma is the main agent responsible for CA in sheep, Mmc has also been identified sporadically in this species and is able to produce lesions (Trichard et al., 1993; Kidanemariam et al., 2005a; Chazel et al., 2010). In goat herds, the aetiology of CA seems to be more complex, since up to four species of *Mycoplasma* spp. have been defined as causative agents (Ma, Mcc, Mmc and Mp; OIE, 2012) and mixed infections are common at individual or herd level. This, along with the presence of other non-pathogenic mycoplasmas, makes the diagnosis and control of CA particularly difficult (Gil et al., 2003; Gómez-Martín et al., 2012a). Contact between different *Mycoplasma* spp. may also promote the transfer of genetic material (Nouvel et al., 2010).

Outbreaks of disease caused by Ma and Mmc have been detected in several wild ruminants, including the Spanish ibex (*Capra pyrenaica*) (Verbisck et al., 2008, 2010), Alpine ibex (Chazel et al., 2010; Giangaspero et al., 2010) and chamois (Tardy et al., 2012). Pneumonia associated with Mcc has been reported in the markhor (*Capra falconeri*) (Ostrowski et al., 2011). The occurrence of these outbreaks in wild ruminants has been linked to interactions with domestic species sharing grazing lands and water troughs, movement of livestock, high population density of wild ruminants, unfavourable climate conditions and the availability of forage sources (Verbisck et al., 2008; Ostrowski et al., 2011). Further work is needed to clarify the role of wild ruminants in the epidemiology of CA in domestic sheep and goats.

The epidemiological role of asymptomatic carriers, especially auricular carriers, has been investigated extensively. All four *Mycoplasma* spp. involved in CA can be isolated from the external ear canal of goats, both in animals with clinical disease and asymptomatic animals (Bergonier et al., 1997). Carriers of Ma and Mmc have been also detected in wild goat populations (Chazel et al., 2010). Several hypotheses have been evoked to explain the localisation of *Mycoplasma* spp. in the ear: (1) spread by vectors, such as blood-feeding mites (Cottew and Yeats, 1982; DaMassa and Brooks, 1991); the presence of Mp is correlated with the presence of mites in the external ear canal (Otero et al., 2009); (2) damage to the tympanic membrane; simultaneous infection of the middle ear and external auditory canal has been reported in goats (Cottew and Yeats, 1982) and this mode of localisation appears to occur in calves infected with *M. bovis* (Walz et al., 1997); (3) via the auditory tube, which favours spread of Mmc from the respiratory system to the middle ear (Gómez-Martín et al., 2012a); Mmc is able to colonise the ear via the oronasal route following experimental infection (DaMassa and Brooks, 1991); and (4) via the bloodstream; after experimental infection in the absence of mites, the external ear canal is colonised by Ma following mycoplasmaemia (De la Fe et al., 2011). In a study of bucks that were auricular carriers of Mmc, the organism was detected in chronic lesions in the respiratory, genitourinary and nervous systems (Gómez-Martín et al., 2012a).

The host humoral immune response does not seem to be able to prevent colonisation of the ears by *Mycoplasma* spp. (Castro-Alonso et al., 2009; De la Fe et al., 2011) and a high proportion of goats with auricular colonisation by Ma and Mmc are seronegative (De la Fe et al., 2010; Gómez-Martín et al., 2012b). In endemic areas, in which CA in most herds is chronic, the presence of many asymptomatic carriers indicates that the infection is constantly perpetuated, compromising disease control and eradication measures (Thiaucourt and Bölske, 1996; Mercier et al., 2007). In these chronically infected herds, the presence of mycoplasmas may lead to outbreaks under conditions of stress and/or a diminished immune response (Thiaucourt and Bölske, 1996; De la Fe et al., 2007a). Mmc isolates associated with septicaemia cannot be distinguished on the basis of genetic or pathogenic characteristics from isolates carried in the external ear canal (Tardy et al., 2011). Further work is needed to clarify the pathogenesis and epidemiological role of CA carriers.

The possibility of venereal transmission of CA in goats has been suggested by: (1) detection of Ma and Mmc in the semen of naturally infected bucks (De la Fe et al., 2009a; Gómez-Martín et al., 2012b); (2) isolation of Mp and Mmc from the female reproductive tract (Gil et al., 2003; Szeredi et al., 2003); and (3) the detection of Ma, Mmc and Mcc in bucks in artificial insemination centres (Amores et al., 2011a). Viable Ma, Mmc and Mp have also been detected at several sites in the male reproductive tract, including the testes, bulbourethral glands and prepuce of naturally infected bucks (Gil et al., 2003; De la Fe et al., 2010; Gómez-Martín et al., 2012a, 2012b).

The potential risk of transmitting mycoplasmal infection from males to females has prompted the design of specific control measures linked to genetic improvement programmes for dairy goat breeds: (1) preventing the entry of asymptomatic auricular carriers

Fig. 3. Contagious agalactia is characterised by the classical triad of (a) joint, (b) mammary and (c) eye disease. (d) Disease is sometimes severe and can lead to emaciation and death. (e) Respiratory signs range from coughing to dyspnoea, leading to death in severe cases, especially in young animals. (f) Abortion can occur sporadically or as outbreaks.

into genetic selection centres based on the results of ear swab cultures and PCR; and (2) detecting infected animals at artificial breeding centres by routine testing of semen ejaculates by culture and PCR (Gómez-Martín et al., 2012b). Ma is shed intermittently in goat buck semen (De la Fe et al., 2009), indicating a need to continuously monitor the semen produced at these centres (Amores et al., 2011a; Gómez-Martín et al., 2012a).

Clinical disease associated with contagious agalactia

In addition to the classic triad of diseases associated with CA (mastitis, arthritis and conjunctivitis) (Fig. 3), respiratory and reproductive disorders have been observed in goats infected with CA-associated mycoplasmas (Bergonier et al., 1997). In chronically infected dairy goat herds, subclinical mastitis is mostly frequently observed, among which episodes of clinical mastitis occur sporadically. However, in these flocks, mycoplasmal infection does not have substantial effects on milk quality (Corrales et al., 2004; De la Fe et al., 2009b).

Mmc and Mp have been identified in the brain and meninges of goats with neurological disease, as well as in animals without clinical signs or lesions (DaMassa et al., 1987; Gómez-Martín et al., 2012a). Neurological signs associated with CA include opisthotonus, circling and coma (DaMassa et al., 1987; Kinde et al., 1994; Bajmocy et al., 2000).

The presence of mycoplasmas in the reproductive tract of females may be associated with reduced fertility (Gil et al., 2003; Di Provvido et al., 2009). Ulcerative balanoposthitis and vulvitis in sheep have also been associated with reduced fertility (Trichard et al., 1993; Kidanemariam et al., 2005a). However, Mmc has been detected in the genital tract of apparently healthy sheep (Kidanemariam et al., 2005a). Therefore, it remains to be determined whether mycoplasmas in the genital tract of small ruminants represent a reservoir of infection within a herd or flock, as suggested for mycoplasmas in horses (Spergser et al., 2002).

Diagnosis of contagious agalactia

The diagnosis of CA, especially in endemic zones, is complex and depends on laboratory testing (Bergonier et al., 1997). Recent studies indicate that milk samples for isolating mycoplasmas should not be frozen, since freezing reduces the viability of the organisms (Amores et al., 2010a). Therefore, it is preferable to use fresh samples or to treat samples with a bacteriostatic preservative, such as azidiol (Amores et al., 2010a). The advent of diagnostic molecular tests, especially real-time PCR has provided rapid, sensitive and robust tests for identifying CA-causing mycoplasmas (Fitzmaurice et al., 2008; Oravcová et al., 2009; Becker et al., 2012). These tests have proved valid for the systematic anal-

Table 1
Characteristics of *Mycoplasma* spp. infecting small ruminants.

Species	Growth in vitro	Typical colony size	Biochemical features[a]						Host	Disease	Samples
			S	A	U	T	F	C			
M. agalactiae	Good	Small	–	–	–	+	+	+	Sheep/goat	Contagious agalactia	Milk, joint fluid, ocular swabs, nasal swabs, ear swabs, semen, udder, lymph nodes, lung lesions, brain
Mycoplasma mycoides subsp. *capri*	Good	Large	+	–	–	+	–	–	Goat/sheep	Contagious agalactia	Milk, joint fluid, ocular swabs, nasal swabs, ear swabs, semen, udder, lymph nodes, lung lesions, brain
Mycoplasma capricolum subsp. *capricolum*	Good	Large	+	–	–	+	+	–	Goat	Contagious agalactia	Milk, joint fluid, ocular swabs, nasal swabs, ear swabs, udder, lymph nodes, lung lesions, brain
M. putrefaciens	Good	Small	+	–	–	+	+	+	Goat	Contagious agalactia	Milk, joint fluid, udder, lymph nodes, lung lesions
Mycoplasma capricolum subsp. *capripneumoniae*	Fastidious	Large	+	–	–	+	+	–	Goat	Caprine pleuropneumonia	Nasal, ear swabs, lung lesions, pleural fluid, mediastinal lymph nodes
M. ovipneumoniae	Fastidious	Centreless	+	–	–	+	–	–	Sheep/goat	Non-progressive pneumonia	Nasal swabs, bronchoalveolar lavage, lung lesions, pleural fluid, bronchopulmonary lymph nodes
M. conjunctivae	Fastidious	Small	+	–	–	+	–	–	Sheep	Infectious keratoconjunctivitis	Eye swabs
M. arginini	Good	Small	–	+	–	–	–	–	Sheep/goat	Opportunistic pathogen	Nasal swabs, bronchoalveolar lavage, lung lesions, pleural fluid, bronchopulmonary lymph nodes
M. cotewii	Good	Variable	+	–	–	+	–	–	Goat/sheep	Non-pathogenic	Ear swabs
M. yeatsii	Good	Variable	+	–	–	+	–	–	Goat/sheep	Non-pathogenic	Ear swabs, milk
M. auris	Good	Variable	–	+	–	+	+	–	Goat	Non-pathogenic	Ear swabs

[a] S, fermentation of sugars; A, hydrolysis of arginine; U, hydrolysis of urea; T, reduction of tetrazolium; F, phosphatase activity; C, formation of films and spots in solid medium.

ysis of BTM (Chazel et al., 2010), even in the presence of preservatives, such as bronopol or sodium azide (Amores et al., 2011b).

In endemic areas, BTM testing is recommended to determine the health status of herds (Contreras et al., 2008), noting that detection of mycoplasmas in milk is affected by the number of shedding animals and the final tank volume (Amores et al., 2012). Intermittent Ma shedding in milk has also been reported in sheep BTM samples in CA endemic areas; 26.3% of farms with a positive test result for the disease returned a negative result at the next sampling (Ariza-Miguel et al., 2013). It has been proposed that samples from clinical cases of mastitis should be tested at the same time as BTM to increase diagnostic sensitivity at the herd level, since herds have been identified in which five consecutive BTM samples were negative for Ma and Mmc, whereas CA infection was confirmed on testing of samples from cases of clinical mastitis (Amores et al., 2012).

Culture combined with PCR is the best method of detecting CA in individual cases (Amores et al., 2010b; Gómez-Martín et al., 2012a) and in BTM for diagnosis at the herd level (Ariza-Miguel et al., 2012). However, non-pathogenic *Mycoplasma* spp., such as *Mycoplasma yeatsii*, *Mycoplasma cotewii* and *Mycoplasma auris*, have also been isolated from small ruminants, especially goats (Chazel et al., 2010), indicating the need to correctly identify the species of *Mycoplasma* involved (Table 1).

Serological tests are available for detection of antibodies against CA (Bergonier et al., 1997; Nicholas et al., 2008). However, some commercial ELISA kits have a poor ability to detect antibodies against some Ma strains and test performance varies according to host species and geographical origin of samples (Poumarat et al., 2012). The low sensitivity of some serological tests may explain the lack of a detectable antibody response in some infected animals (Amores et al., 2011a). Conversely, cross-reactions among *Mycoplasma* spp. will yield false positive results, sometimes corresponding to the presence of non-pathogenic mycoplasmas (Di Provvido et al., 2009; Poumarat et al., 2012). These shortcomings have limited the use of serology to assess the health status of herds or flocks (Bergonier et al., 1997; Schubert et al., 2011; Gómez-Martín et al., 2012b; Agnone et al., 2013a). The sensitivity of serological testing may be improved by combining ELISA with Western blot analysis (Schubert et al., 2011; Agnone et al., 2013a).

Control of contagious agalactia

Current measures to control CA in affected areas have mainly been through the use of antibiotics and vaccines, with few changes over the last 15 years (Bergonier et al., 1997). These treatments are an alternative to culling infected animals of high genetic value, as well as rare domestic breeds and wild species in danger of extinction. Several studies have determined antibiotic resistance profiles of CA-causing *Mycoplasma* spp. (Loria et al., 2003; Al-Momani et al., 2006; Antunes et al., 2007a, 2007b, 2008); resistance may lead to failure of some antibiotic treatments (Gómez-Martín et al., 2013).

The antibiotics of choice against Mmc and Ma are fluoroquinolones, tetracyclines and macrolides. While erythromycin is effective against Mmc, Mcc and Mp, it is inefficient against the strains of Ma tested so far (Loria et al., 2003; Al-Momani et al., 2006; Antunes et al., 2007a, 2007b, 2008). Enrofloxacin, florfenicol, oxytetracycline and spiramycin have been used to treat ulcerative balanitis and vulvitis in sheep caused by Mmc (Kidanemariam et al., 2005b); Mmc exhibits resistance to nalidixic acid, gentamicin, streptomycin and spectinomycin. Similarly, Ma is resistant to streptomycin and nalidixic acid (Antunes et al., 2007a, 2008). The *Hominis* group shows an intrinsic resistance to erythromycin and streptomycin (Furneri et al., 2001; Königsson et al., 2002). A low sensitivity to tetracyclines has also been observed in some strains of Mp (Antunes et al., 2007b).

In a recent study, systemic marbofloxacin treatment was assessed in goat bucks that were asymptomatic auricular carriers of CA-causing mycoplasmas at an artificial breeding centre (Gómez-Martín et al., 2013). Despite the known high susceptibility of these microorganisms to fluoroquinolones and their good tissue penetration, these agents were unable to eliminate Ma and Mmc from the external ear canal of affected animals and also had detrimental effects on sperm motility. Thus, current antimicrobial strat-

egies have limited effects on carriers in chronically infected herds, with implications for controlling CA in artificial breeding centres.

The strategies employed for vaccination against CA have also varied little over the years and conventional monovalent or polyvalent killed vaccines are still used (Nicholas et al., 2008). Under field conditions, vaccines may prevent the appearance of new clinical signs and reduce mycoplasmal excretion, but are unable to prevent transmission of infection (De la Fe et al., 2007b; Agnone et al., 2013a, 2013b). The low efficacy of currently available vaccines may be due to the multi-aetiological nature of CA, the molecular characteristics of the causative species and the ability of CA-causing mycoplasmas to avoid the host immune response.

The best clinical results with inactivated vaccines have been observed when saponin has been used as an adjuvant or inactivating agent (De la Fe et al., 2007a, 2007b; Agnone et al., 2013a, 2013b). The beneficial effect of saponin in vaccines is linked to the timing of T lymphocyte memory cell activation (Agnone et al., 2013a). In an experimental study in sheep, a live attenuated vaccine against Ma also induced good clinical protection (Agnone et al., 2013b). It is likely that a new generation of recombinant vaccines in the next 10 years will provide better protection against mycoplasmal infection, especially against Ma.

Another tool proposed to eliminate a source of CA infection for offspring is pasteurisation of colostrum. Treatment of experimentally contaminated colostrum samples for 60 min at 60 °C successfully eliminates viable Mmc colonies (Paterna et al., 2012). Although treatment of colostrum for 30 min at 56 °C significantly reduces Ma counts, this *Mycoplasma* spp. is able to survive after treatment of colostrum for 120 min at 60 °C (Paterna et al., 2012).

Conclusions

A better understanding of the genetic characteristics of the mycoplasma species involved in CA and the molecular mechanisms driving their variability and pathogenicity is essential to explain the complex epidemiology of CA, especially in goat herds. There is a need to clarify the consequences of the coexistence of several mycoplasmas in the same animal or herd, the epidemiological role of asymptomatic carriers and the potential risk of transmitting mycoplasmal infection from wild animals to domestic small ruminants. More sensitive diagnostic methods should be targeted at detecting asymptomatic carriers in chronically infected herds. Finally, to achieve real control of CA, new efficient control tools are needed to prevent the infection entering a herd.

Conflict of interest statement

None of the authors of this paper has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

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