Mycoplasmas and Bovine Respiratory Disease: Studies Related to Pathogenicity and the Immune Response—A Selective Review

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Three species of mycoplasma have been established as being of importance as causes of pneumonia in housed calves, based on pathogenicity studies and frequency of association with the disease. These three species are Mycoplasma bovis, M. dispar, and Ureaplasma diversum. M. bovis is the most pathogenic of these species but the disease outbreaks with which it is associated are sporadic. M. dispar is regularly isolated from pneumatic calves but is also found causing mild superficial and asymptomatic infections of the respiratory mucosa. The bovine ureaplasmas are serologically complex. They are distinct from ureaplasmas isolated from other non-ruminants by PAGE analysis, G + C content of DNA, and serology. A second species within the genus ureaplasma has been proposed to accommodate the bovine ureaplasmas, U. diversum. Control of mycoplasma respiratory infections of cattle based on immunization might be possible. Calves have been immunized against M. bovis and immunity has been related to antibody in the lung. M. dispar appears less immunogenic in calves than M. bovis and this may contribute to its pathogenicity.

THE COMPLEX NATURE OF BOVINE RESPIRATORY DISEASE

A variety of viruses, typical bacteria, and mycoplasmas are associated with pneumonia of housed calves, and these agents probably interact to produce more severe lesions than would be produced by any single entity. Several of the common respiratory viruses of calves (e.g., parainfluenza type 3, infectious bovine rhinotracheitis, and respiratory syncytial virus) usually cause an acute transient infection, although a carrier state sometimes follows. Decreased resistance of the lung to bacterial infection is often a consequence of viral infection; the best studied example of this is Sendai virus in mice [1]. A similar effect is apparent in calves [2,3]. Viral infections can also predispose to subsequent mycoplasma infections. However, several mycoplasmas are primary pathogens and their possible chronic existence may be exacerbated by a viral infection. Thus the timing of viral-mycoplasmal interactions is likely to be less critical than that of viral-bacterial interactions in the production of respiratory disease [4].

Brennen et al. [5] reported increased severity of disease following the inoculation of bacteria and mycoplasmas together into the respiratory tract, compared to the disease observed when either was inoculated alone. This could simply be an additive effect of introducing two agents at the same time. Alternatively, Schewen and
Wilkie [6] reported *Pasteurella haemolytica* had a toxic effect for bovine alveolar macrophages and this type of effect could aid mycoplasma multiplication. Mycoplasmas do not appear to predispose to secondary bacterial infection in the same way as described for viruses such as Sendai and parainfluenza type 3 [4,7]. The authors presume this is because they do not affect the same host cells.

**THE RELATIVE IMPORTANCE OF THE MYCOPLASMA SPECIES ISOLATED FROM THE BOVINE RESPIRATORY TRACT**

A large number of species of mycoplasma have been isolated from the respiratory tract of calves. These isolations have been from both non-diseased and diseased tissues [8]. Not all of these species are proven pathogens for cattle. Furthermore, only a few of the proven pathogens are frequently isolated from housed calves with pneumonia. The three most important species, based on studies of occurrence and pathogenicity, are *Mycoplasma bovis*, *M. dispar*, and *Ureaplasma* sp. The occurrence and pathogenicity of each of these three species is different and each needs to be studied individually.

**ISOLATION AND PATHOGENICITY OF M. DISPAR**

Isolation of this glucose-fermenting species from calves is not possible on conventional mycoplasma media and a modified medium similar to that developed for *M. hyopneumoniae* was originally used to isolate *M. dispar*. Failure to grow on conventional media has probably led to a considerable underestimate of the prevalence of *M. dispar* in the calf population [8,9,10]. The membrane of *M. dispar* is surrounded by a layer of material that stains with ruthenium red. This layer is more extensive than that seen in several bovine mycoplasma species, apart from *M. mycoides* subsp. *mycoides* [11]. It has been proposed that this "capsular" material plays a role in the virulence of the organism. Attachment to bovine erythrocytes and to the epithelium of bovine tracheal organ cultures is via a trypsin-sensitive attachment site. Thomas and Howard [12] reported ciliastasis following the inoculation of *M. dispar* into bovine tracheal organ cultures, and this fact is likely to play an important role in the pathogenicity of this species.

Gourlay et al. [13] reported isolations from the lungs of 30 percent of calves that died with severe pneumonia or were killed in extremis and 60 percent of calves with subclinical pneumonia. Subsequent to these findings in England, isolations have been made in other parts of the world [8,9]. *M. dispar* was isolated from nasopharyngeal swabs taken from 93 percent of apparently healthy Ayrshire calves obtained from farms in southern England [14]. Later studies on animals from the same source have indicated that many of them have subclinical pneumonia, usually involving 2–10 percent of the lung surface, and *M. dispar* was isolated from lungs of 97 percent of 33 animals examined. Higher levels of colonization were apparent in pneumatic, compared to non-pneumatic, animals. Thus of 32 lungs examined, 14 had lesions involving ≤ 2 percent of the surface and the number of *M. dispar* isolated from these was ≤ 10^4 ccu in 12/14 cases. Of the remaining 18 lungs, which had lesions involving ≥ 3 percent of the surface, ≥ 10^4 ccu *M. dispar* were isolated from 17/18. The principal histopathological lesion in these animals was an alveolitis with pronounced lymphoid accumulations rarely being present. *M. dispar* has also been isolated from animals free from macroscopic pneumonia, more frequently from the bronchus than from the lung, with peak colonization at three months [15].

Pneumonia was apparent in gnotobiotic calves following the introduction of *M. dispers* into the respiratory tract and the lesions extended over 0 to 17 percent of the
lung surface. The predominant histopathological lesion in these young gnotobiotic calves involved a cellular infiltration, and thickening, of the alveolar walls [16,17].

*M. dispar* has also been shown to cause mastitis in cattle following its introduction via the teat canal into the mammary gland. Infection seems to be limited to the inoculated quarter and can continue for several months. Associated with the infection is a persistent and markedly raised cell count in the milk. Both virulent and avirulent strains, as judged by ability to induce mastitis, are present in the respiratory tract of cattle [18]. In conclusion, this pathogenic species is clearly a very common inhabitant of the bovine respiratory tract, being typically found in the bronchi and lungs of calves.

**SEROLOGICAL RESPONSE TO INFECTION WITH M. DISPAR**

Attempts have been made to measure antibody to *M. dispar* in the sera and lung washings from several groups of naturally infected cattle in order to determine whether infection or disease associated with *M. dispar* could be diagnosed by serological tests.

The first group of sera examined were from five groups of eight calves selected from each of five groups of approximately one hundred animals reared on a beef unit in southern England [19]. Sera taken from these animals at about monthly intervals over a period of up to 200 days were examined for IgG antibody to *M. dispar* by enzyme-linked immunosorbent assay (ELISA) [20]. A small increase in the mean antibody titer of two of the five groups was observed, but the mean titer was always $<10^3$ units of antibody. It has been proposed that this represents the serological response in colonized animals in the absence of severe disease involving *M. dispar*.

A second group of sera examined consisted of paired samples, the first taken at the onset of outbreaks of respiratory disease and the second about thirty days later [21]. Fourfold, or greater, rises in IgG antibody to *M. dispar* were noted in association with disease in several of these animals. Furthermore, sera from several animals contained $>10^3$ units of antibody. Rises in antibody titers were also detected by single radial hemolysis (SRH) [20,21]. Thus, antibody rises to *M. dispar* can be demonstrated in association with some outbreaks of respiratory disease.

A third group of sera examined were from Ayrshire calves. Most of these animals had pneumatic lesions and were colonized with *M. dispar*, as noted above. Serum antibody was not detected. However, antibody to *M. dispar* was found in lung washings by ELISA. Of nine lung washings examined, two had IgG1 antibody to *M. dispar*, one had IgG2 antibody, and seven had IgA antibody demonstrable. Six gnotobiotic calves inoculated previously with respiratory syncytial virus were used as controls. None had antibody to *M. dispar* detectable in lung washings. In this case *M. dispar* appears to have generated only a local immune response and infection was presumably limited to the surface of the lung.

Other evidence for *M. dispar* producing infections that do not progress beyond the mucosal barrier come from an examination of experimental mastitis. When sera from infected animals is examined by SRH, which detects bovine IgM and IgG1, serum antibody can be demonstrated in some cows. Others appear not to have produced a detectable response, even though they had developed severe mastitis.

In conclusion the progression of *M. dispar* infection in cattle might be as follows. Calves become colonized in the nasopharynx and then in the lung. Possibly a subclinical pneumonia with a local IgA response is produced, but no serum antibody response. In association with other agents, *M. dispar* may be involved in more severe clinical pneumonia. In this case a serum antibody response is produced. Conversely,
it can be argued that the presence of serum antibody to *M. dispar* indicates more than colonization of the respiratory tract, probably lung lesions and possibly spread beyond the mucosal surface to which it is usually limited.

**M. BOVIS, ISOLATION, PATHOGENICITY, AND SEROLOGICAL RESPONSE**

This mycoplasma has been isolated from pneumonic calves as well as other diseased cattle, notably those with mastitis, arthritis, and urogenital infections [9]. Although not as ubiquitous as *M. dispar*, isolation rates within a given herd, in association with pneumonia, can be very high. A serum antibody response detectable by a variety of techniques, including indirect hemagglutination, single radial hemolysis, complement fixation, and ELISA, almost invariably follows infection. This is in contrast to *M. dispar* and probably reflects the more invasive nature of *M. bovis*.

Pneumonia develops following inoculation of the respiratory tract of gnotobiotic calves. Severe clinical mastitis, sometimes with spread to other quarters, follows intramammary inoculation. Arthritis can be produced by injecting calves intravenously. Thus, this species is more pathogenic than *M. dispar* but less prevalent.

**ISOLATION AND PATHOGENICITY OF BOVINE UREAPLASMAS**

Ureaplasmas were first isolated from cattle in 1967. Originally, isolations were from the urogenital tract [22]. Subsequently, ureaplasmas were isolated from pneumonic calf lungs [13]. The diseases from which ureaplasmas have been isolated include: vulvitis, infertility, pneumonia, and keratoconjunctivitis. Ureaplasmas are also commonly isolated from apparently healthy urogenital tracts [9,23,24].

These mycoplasmas have been shown to produce mastitis in cattle, pneumonia in gnotobiotic calves, vulvitis, and ocular infections [16,25,26,27]. Both virulent and avirulent strains exist. Since ureaplasmas have been shown to be pathogenic, a possible role in any disease from which they are isolated should be considered.

**ANTIGENIC STRUCTURE OF BOVINE UREAPLASMAS**

The genus *Ureaplasma* was proposed in 1974 by Shepard et al. [28], some 20 years after the original description of the organisms. A single species, *U. urealyticum*, accommodates the human isolates. Originally eight serotypes were proposed, but this serotyping scheme has subsequently been expanded [29].

The bovine ureaplasmas are serologically complex. Initial studies using antisera prepared in rabbits indicated there were three distinct groups, two of which were clusters of serologically similar, but not identical, strains [30]. Similar conclusions were reached by Ogata et al. [31]. Eight strains were selected from these studies as representing the range of antigens synthesized by the bovine isolates. Antisera against these eight strains were used to serotype fresh isolates from various countries. Most (72/77) isolates reacted with at least one of the sera, but three more strains had to be added to the original eight to complete the range of antigens expressed [32].

Subsequently one strain from each of the three antigenic clusters was selected and antiserum against it prepared in calves. All of the representative strains previously selected from our studies, as well as those of the Japanese workers [31], were found to react with at least one of the bovine antisera [33]. Thus, bovine antisera were less discriminating than rabbit antisera. It should be possible to identify all bovine ureaplasmas by using just three antisera; 96/96 strains examined were identified as
being of cluster A (represented by strain A417), B (represented by strain D48), or C (represented by strain T44).

The serological relationship of antigens possessed by bovine ureaplasmas is represented in Fig. 1. The smaller clefts represent differences distinguished by rabbit antisera and the larger ones by bovine antisera and polyacrylamide gel electrophoresis (PAGE). This illustrates the relationship of the 11 selected strains and possibly not the entire spread of antigens present in nature. Thus, within the groups A, B, or C there may be a continuous spectrum of antigens with no "clefts," but with strains at distant parts of the spectrum being distinct. The clefts between clusters A, B, and C may be genuine, and strains may not exist to fill them. However, some strains have been isolated that react with antigens of clusters A and C and, if sufficient fresh isolates are examined, there may be a continuous spectrum of antigens evident for the whole of the bovine group.

For the future, it should be possible to identify bovine isolates using just three antisera and to use three antigen preparations for serodiagnosis. Finally, immunity between strains of the same serological cluster might occur. However, strains from different clusters (A and B) do not cross-protect [34].

**RELATIONSHIP OF BOVINE UREAPLASMAS TO U. UREALYTICUM**

Bovine ureaplasmas are distinct from *U. urealyticum* strains when compared by growth inhibition, metabolism inhibition, and immunofluorescence using antisera prepared in rabbits and cattle [31,32,33]. However, it appears that there is at least one cytoplasmic antigen common to both species [35,36].

The range of guanine plus cytosine (G + C) contents of DNA from *U. urealyticum* strains (27.0–27.8) and bovine isolates (29.0–30.2) was found not to overlap. This indicates the organisms comprise two distinct populations [37,38,39]. Analysis of the polypeptides by PAGE, in both one and two dimensions [40,41,42], indicated the bovine strains were distinct from the human. Furthermore there were clusters of strains by PAGE that were coincident with the serological clusters (see Fig. 1).

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**FIG. 1.** Diagrammatic representation of relation between selected typical bovine ureaplasma strains based on studies of serology and polypeptides.
Based on these findings, it has been proposed that a second species should be recognized within the genus *Ureaplasma* with the bovine isolate A417 named the type strain of this species—*U. diversum*. Within this species there are three clusters of strains, Groups A, B, and C, represented by three strains A417, D48, and T44.

**COMPARISON OF BOVINE UREAPLASMAS WITH THOSE FROM OTHER ANIMAL SPECIES**

In Fig. 2 a diagram has been constructed representing the relationship between ureaplasmas from various animal species. The strains have been arranged according to G + C values with serological and PAGE studies used to further position groups of organisms.

Within the species *U. diversum* (cattle isolates) are the three clusters A, B, and C which are recognized by serology and PAGE. The sheep and goat isolates have similar but slightly higher G + C values. Two major clusters of these strains exist based on studies with rabbit or bovine antisera and PAGE of polypeptides [43,44]. Whether the two groups of sheep and goat strains are further groups of *U. diversum*, or another species, will be apparent from future studies.

*U. urealyticum* is distinct from the ruminant animal group. The several serotypes of this species comprise two clusters based on PAGE, and DNA homology [45]. Mouches et al. [42] showed that chimpanzee and Talapoin monkey strains were similar to *U. urealyticum* strains by two-dimensional PAGE. The dog and cat strains have similar G + C values to *U. urealyticum*, but the canine isolates are serologically distinct and comprise four clusters [46]. Kotani et al. [47] found that the feline strains contain two more serological clusters. The strains from a squirrel monkey and marmoset are distinct from *U. urealyticum* and *U. diversum* by PAGE and G + C and should not be regarded as being of either species.

**FIG. 2.** Diagram illustrating possible relation to each other of members of genus *Ureaplasma* isolated from various animal species (based on G + C content of DNA, PAGE of polypeptides, and serology).
This diagram is certainly not complete; uncharacterized ureaplasmas from other animal species are not included and it reflects only the information available now. It would seem certain that a number of further species will be proposed in the future as more tests and comparisons are made.

IMMUNIZATION STUDIES WITH \textit{M. BOVIS} AND \textit{M. DISPAR}

A series of experiments have been done to determine whether it was possible to immunize against respiratory infections with \textit{M. bovis} and \textit{M. dispar} by injecting killed organisms [48,49,50]. The first series of experiments was stimulated by the work of Pierce and Gowans [51] who found that systemic (intraperitoneal) priming, followed by local boosting, was an effective way of producing a local immune response.

Animals were injected with the killed \textit{M. bovis} intramuscularly (im) twice, intratracheally (it) twice, im + it or subcutaneously (sc) three times. In summary, only animals injected im + it or sc × 3 showed increased resistance to \textit{M. bovis} respiratory infection. Immunity was related to the antibody, IgG, in lung washings but not in the serum. This is consistent with the general consensus that IgG is a major component of bovine secretions [52] and of the lung rather than the upper respiratory tract in general [53].

With \textit{M. dispar} im + it injections of antigen did not produce detectable protection from colonization. Calves injected 3 × sc with antigen and oil adjuvant had less mycoplasmas in their lungs \((p < 0.05)\) than control calves. However, this difference was not as great as observed with \textit{M. bovis} and a relatively low immunogenicity appeared to be a feature of \textit{M. dispar}.

Further studies with \textit{M. dispar} and \textit{M. bovis} [54] have indicated that young calves respond poorly to injections of \textit{M. dispar} antigen compared to older animals. With \textit{M. bovis}, the age effect is less marked. The poor immunogenicity of \textit{M. dispar} in young calves might have affected the histopathological lesion observed following its inoculation into the respiratory tract of gnotobiotic calves. For \textit{M. dispar} this was found to be primarily an alveolitis, but with \textit{M. bovis} lymphoid accumulations were observed. Thus, failure of young calves to produce a proliferative response is related to absence of, or poor, immune response following infection compared to \textit{M. bovis}.

No evidence for destruction of \textit{M. dispar} antigens by formalin or increased response with increased dose was found. One possible explanation for the poor response of young calves is that the lymphocyte clones that recognize and react with the surface antigens of \textit{M. dispar} develop late in the ontogeny of the calves' immune system. This has been described for certain other antigens [55]. Further immunological studies will provide an insight into the mechanisms of immunity to mycoplasmas in the calf respiratory tract that should enable effective vaccination procedures to be employed.

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